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Original Articles

PSEUDO-HYPERTROPHIC MUSCULAR ATROPHY.

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I. INTRODUCTION.

Cases of muscular atrophy may be of at least three distinct types, viz.: (1) those due to lesions in the central nervous system; (2) those due to lesions in the peripheral nerves; and (3) those due to a primary lesion of the muscles themselves. The last of these forms is also spoken of as a muscular dystrophy; and as the case here reported is of this form, it may be of interest to see how this type is defined. I quote here Dr. Osler's[1] definition. He says that it is a "muscular wasting, with or without an initial hypertrophy, beginning in various groups of muscles, usually progressive in character, and dependent on primary changes in the muscles themselves." Pseudo-hypertrophic Muscular Atrophy is that form of muscular dystrophy which affects infants, which is preceded by a pseudo-hypertrophy and, which attacks first the muscles of the calves of the legs.

II. HISTORICAL STATEMENT.

The first cases of muscular atrophy mentioned in the literature upon this subject were studied with no reference to the microscopical condition of the spinal cord, the peripheral nerves, or of the muscles themselves. The diagnosis was based entirely on the history and on the clinical findings in the case; nor was any serious attempt made to find the real cause of the various symptoms that were observed. Thus Bell (1830),[2] though he made no microscopical examination of the tissues, was the first investi-

gator to describe such cases of muscular atrophy as have of late been called Pseudo-hypertrophic Muscular Atrophy. In 1836 he made the first clinical description of a case of Progressive Spinal Paralysis. Aran (1850)[3] was the first investigator to demonstrate changes in the spinal cord of cases of Progressive Spinal Paralysis, while Meryon (1852)[4] was the first to demonstrate that in cases of Pseudo-hypertrophic Muscular Atrophy no microscopical changes are present in the spinal cord, but that the muscles had undergone a "fatty and granular degeneration."

Duchenne (1868)[5] also recognized that this disease is independent of all changes in the central nervous system and gave it the name "Pseudo-hypertrophic Muscular Atrophy."

Charcot (1872)[6] discovered Amyotrophic Lateral Sclerosis, and pointed out the differences between it and Progressive Spinal Mucular Atrophy. Friedreich (1873)[7] discussed fully the reasons for separating the muscular dystrophies from the forms of spinal muscular atrophy. Dejerine (1882) and others, showed that numerous cases formerly supposed to be due to spinal lesion were really due to multiple neuritis. Erb (1882)[8] described the form of muscular dystrophy which he called "juvenile," and later (1884)[9] gave a full account of the same. Landouzy and Dejerine (1884) described the facio-scapulo-humeral form of muscular dystrophy, and later (1885)[10] gave a more extended discussion of the same. Gowers (1879)[11] collected and studied 160 cases of Pseudo-hypertrophic Muscular Dystrophy, and more than 140 cases have been reported, by various writers, since the publishing of his paper. Concerning the condition of the spinal cord in this disease, Gowers (1899)[12] wrote: "The motor nerves, when examined, have been found normal. The spinal cord has been found perfectly normal in most cases in which it has been examined . . ., the anterior gray matter was unaffected. Hemorrhages have been occasionally found. The neuroglia cells have been found increased in number."

III. CLASSIFICATION.

It is therefore evident that the forms of muscular atrophy may well be arranged into three groups:

(a) Those due to changes in the spinal cord: Progressive Spinal Muscular Atrophy, etc.

(b) Those due to changes in the peripheral nerves: Multiple Neuritis, Progressive Neural Muscular Atrophy, etc.

(c) Those due to primary changes in the muscles: The muscular dystrophies.

Erb (1891)[13] has classified the forms of muscular dystrophies as follows:

I. Dystrophia Muscularis Progressiva Infantum.
   1. Hypertrophic form,
      a. With pseudo-hypertrophy or lipomatosis, Duchenne's Pseudo-hypertrophic Muscular Atrophy.
      b. With real hypertrophy.
   2. Atrophic form,
      a. With primary involvement of the face (Duchenne's infantile form or the Landouzy-Dejerine type).
      b. Without involvement of the face.

II. Dystrophia Muscularis Progressiva Juvenum vel Adulterum (Erb's juvenile form).

According to another classification the types of the disease and the muscle-groups affected are as follows:

1. Duchenne's pseudo-hypertrophy,—muscles of calves of legs.
2. Erb's juvenile dystrophy,—muscles of arms and shoulders.
3. Sachs'-Hoffmann type,—peroneal muscles of legs.
4. Landouzy-Dejerine type,—muscles of face and arms.
5. Charcot-Marie-Tooth type,—muscles of forearms, legs, and back.

The above classifications are given in order that the reader may be better able to place the case here reported.

IV. CLINICAL HISTORY OF THE CASE REPORTED.

His mother, as well as his paternal grandfather died of cancer. One of his sisters was still-born, and a brother shows neurotic tendencies. An aunt on his father's side has been confined in an insane asylum. A cousin is said to have had cerebro-spinal meningitis leaving one leg crippled. There is no history of any other case of Pseudo-hypertrophic Muscular Atrophy among his ancestors or relatives.

The following communication from the boy's father seems to be of sufficient importance to warrant its insertion. He says:

"I do not know that he was ever sick with any disease before the onset of the trouble with his feet."—"I don't remember the two days'sleeping spell to which you refer. The child did have a seeming desire to be held and acted sort of droopy and whined when not asleep as if in pain, and this lasted the greater part of three months, I think. This was before anything wrong was noticed about his ankles, and it was in his ankles that the trouble made its first appearance. As I think I stated in the other letter, for quite a long while the trouble was so slight that we thought little of it. All the physicians consulted, and they were not a few, made light of the trouble. They said it was nothing worthy of notice. To me it seemed many times that the calves of the boy's legs were large, and a great deal of the time were devoid of feeling. For instance, one night when we were living in Tacoma, Wash., a lot of children went out coasting on the hill and took this boy with them. As he could not walk, they hauled him up the hill on the sled, and when they came in, the calves of his legs were nearly frozen without his knowing it. He was then nine years old."

The first abnormality noticed was his going on all fours, using the sides of his feet, when moving about on the floor. He began to walk before he was two years old, but never learned to walk well. He stumbled and fell frequently, as he was unable to flex dorsally either his toes or his feet. When about six years old he wore a brace for a short time. At ten, he found it necessary to use a cane while walking; and at twelve, crutches. He was always frail and this became more apparent as he grew older. He was, however, intelligent and very fond of reading. His arms seemed normal as regards strength, but the movements of his hands were both slow and clumsy. When about twelve years of age he experienced, occasionally, a difficulty in retaining the contents of his rectum, though he was able to perceive the inclination to evacuate his bowels. He seemed normally developed sexually, and never had any trouble in retaining the contents of the bladder. Especially since twelve years of age, have the calves of his legs been abnormally thin. He had always considerable difficulty in going up stairs, and frequently lost his balance. Except when very young, only the balls of his feet touched the ground while walking. He was able to remain in a sitting posture with no difficulty; but was unable to arise from this position without assisting himself with his hands. In arising from the prone position he would first roll over on his face, get up on his hands and knees, then on all fours, and finally raise his trunk by putting his hands on his knees. He showed a lessened sensibility to cold in his feet and in the calves of his legs, though the other sensations were apparently normal. In the later years of his life his shoulder-blades were rather prominent: but this was, no doubt, partly due to the emaciated condi-

tion in which he then was. He had attacks of both measles and whooping-cough, but not until after the onset of his trouble.

The immediate cause of his death was an attack of typhoid fever, which lasted six or seven weeks. During this attack he was in the St. Luke's Hospital, Chicago, where he died February 24, 1903, at the age of twenty years.

(The above history has been obtained from friends and relatives since the death of the patient.)

V. POST MORTEM EXAMINATION.

This was made by Dr. H. G. Wells (1903),[14] Chicago, and showed the following conditions present:

"Anatomical Diagnosis.—Extreme emaciation; typhoid ulceration of the colon; hypertrophic pulmonary emphysema; purulent bronchitis; bronchopneumonia; hypostatic pneumonia of left lung; edema of the right lower lobe of lung; acute lymphadenitis, peribronchial and mesenteric; hyperplasia of the aorta; hypertrophy of the left ventricle of the heart; parenchymatous degeneration of the liver and the kidneys; fibrous pericholecystitis; atrophy of appendix vermiformis; hyperplasia of the spleen; omentum almost devoid of fat; calcification of the peripancreatic and mesenteric lymph nodes; diffuse, chronic nephritis; decubitous ulcerations; callosities over base of the left metatarsal bones; hyperplasia of the testicles; fibrous increase in the thyroid gland; atrophy of the lower extremities with replacement of fatty fibrous tissues; contractures of the feet, both in a spiral with big toe to plantar surface (pes varus et pes equinus); slight scoliosis."

The heart muscle showed a slight increase in the interstitial tissue. The recti muscles were nearly normal except that the cross-striations, in some places, were scarcely visible, and some fibers showed a lessened staining capacity. In the gastrocnemii muscles there were only a few normal fibers, as here atrophy was well marked, and the muscular tissue had the appearance of broken bundles of narrow fibers separated by large amounts of fibrous and adipose tissue. In places the muscle fibers were entirely replaced by connective tissue. In other places the muscle bundles had undergone degeneration to such an extent that only the nuclei of the fibers were present. Some fibers that were apparently nearly normal as regards degenerative changes, seemed swollen. An attempt to regenerate the muscle fibers was also observed in a few places. The small arteries in the muscles showed in many instances a diffuse thickening. Organized thrombi were also observed in a few small blood vessels.

The following study of the nervous tissues of this case was commenced in the Neurological Laboratory of the University of Chicago, and completed in the Pathological Laboratory of the State Hospital for Insane, at Independence, Iowa.

VI. HISTO-PATHOLOGICAL EXAMINATION OF THE SPINAL CORD AND THE ROOTS OF THE SPINAL NERVES.

The material here investigated was kindly furnished me by Dr. H. G. Wells, of the University of Chicago.

Sections from different levels of the spinal cord were stained according to the following methods, and a careful histological examination made.

(*a.*) The Marchi Method.—The nerve tissue prepared according to this method had been preserved in formalin and was subsequently treated with Müller's fluid for 20 days, and with an osmic acid mixture for 7 days. After dehydrating in alcohol, the tissue was imbedded in paraffin. Sections were made 20 micra thick, cleared in xylol, and mounted in balsam.

Sections from the following segments of the spinal cord were studied by this method: cervical VIII., lumbar III., IV., V., sacral I. The result of this examination was negative. Only occasionally were nerve fibers found having a black color. We must consequently conclude that in these segments of the cord there was practically no degeneration of nerve fibers.

(*b*) The Iron Hematoxylon Method.—The material studied by this method was preserved in formalin, dehydrated in alcohol, imbedded in celloidin, and sectioned 30 micra thick. After being treated with an ammonio-ferric sulphate mordant, the sections were washed in water and stained with hematoxylon.

Sections prepared according to this method were made from the following segments of the spinal cord: thoracic VI., lumbar III., IV., V. All these sections showed a fairly well marked proliferation of the neuroglia beneath the ependyma of the central canal of the spinal cord. The ependyma was consequently in many places pushed into the central canal in well marked folds. There was also a slight increase in the neuroglia of the gray matter of the spinal cord. Some of the nerve cells have shrunk so that they are surrounded by open spaces and stain rather faintly indicating that chromatolysis has taken place. The blood vessels in the meninges of the spinal cord are seen to be much dilated in several places.

(*c.*) The Nissl Method.—This method is especially servicable in demonstrating the Nissl-bodies, or the tigroid masses in the nerve cell. When the cell body undergoes degeneration, the Nissl bodies usually disintegrate and the cell content becomes more homogeneous than it is normally—in other words, they undergo chromatolysis. The nerve tissue studied by this method was preserved in formalin. It was afterwards fixed still further and dehydrated by alcohol, cleared in cedar oil-xylol and imbedded in paraffin. The sections were made 10 micra thick, stained with Nissl's methylen blue, cleared in oil of cajeput and mounted in colophonium.

The sections studied by this method were from the following segments of the spinal cord: cervical VIII., thoracic VIII., lumbar III., IV., V., and sacral, I., I., III. These show a proliferation of the neuroglia cells around the central canal, as well as the congestion of the blood vessels of the meninges of the spinal cord already mentioned. Chromatolysis of many nerve cells is also evident. This is especially marked in the segments below and including lumbar IV. In these segments there are fewer cells present, especially in the lateral horn, and those present more frequently show chromatolysis. In several instances the nucleus is displaced so as to lie nearer to the periphery of the cell body, and the side of the nuclear membrane nearest to the center of the cell is, in several instances, thickened and wrinkled. The dendrites of the affected cells also shows chromatolysis and have frequently a shrunken appearance.

In concluding the study of the spinal cord of this case of Pseudo-hypertrophic Muscular Atrophy, it is well to keep in mind that this disease is considered one involving primarily the muscles, that lesions in the spinal cord are rarely present, and that when these lesions are present, they are considered secondary or subsequent to the disease of the muscles. The investigation here made substantiates this view except in one respect, viz., the lessened number of lateral horn cells in the segments of the cord below and including lumbar IV., and the frequent chromatolysis of the remaining cells in these segments.

Sano (1898)[15] has demonstrated that most of the muscles of the leg below the knee are innervated by columns of nerve cells in the lateral horn of the segments of the spinal cord below and including lumbar IV. It is therefore but natural to conclude that the chromatolysis observed, and the possible diminution in the number of cells of the lateral horn of lumbar V, is secondary to the atrophy of the muscles.

Since this patient died from Typhoid Fever it is doubtful if much importance can be given to the other findings,—the proliferation of the neuroglia beneath the ependyma of the central canal of the spinal cord, the slight increase in the neuroglia of the gray matter, and the congested condition of the blood vessels of both the cord and its meninges.

The fact that several of the ventral and lateral horn cells in the lumbar region of the spinal cord had undergone chromatolysis led to the investigation of the roots of the spinal nerves, especially the motor or ventral roots, to determine whether or not these nerve roots contain a smaller number of medullated nerve fibers than do the normal nerve roots. As the author three years ago measured the areas and counted the medullated nerve fibers of both the motor and the sensory nerve roots from the spinal cord of a normal subject, such a comparison becomes possible. According to

the author's results, published June, 1903[16], the dorsal or sensory spinal roots of the left side have together an area in cross-section of 54.93 mm². and contain 653,627 medullated nerve fibers; and according to the results published in 1904[17], the ventral or motor spinal roots of the left side have together an area in cross-section of 26.50 mm². and contain 203,700 medullated nerve fibers. In other words the ratio of the areas of the cross sections of the ventral and dorsal roots is as 1 :2.07, while the ratio of the numbers of medullated nerve fibers is as 1 :3.2. From the author's results similar comparisons can also be made for each pair of nerve roots. The nerve roots investigated were studied both by Marchi's and Pal-Weigert's methods. The Marchi method gave only negative results. The few black droplets present were not sufficiently numerous to indicate much degeneration. But as this was a case of long standing, it is most probable that the disintegrated myelin of the degenerated nerve fibers had disappeared by absorption.

In using the Pal-Weigert method the material was first fixed in formalin, and later in Müller's fluid. It was then washed in water, dehydrated in alcohol and ether, imbedded in celloidin, and sectioned 30 micra thick. The sections were stained in Weigert's hematoxylon, and differentiated with potassium permanganate, oxalic acid, and acid potassium sulphate. After washing in water, dehydrating in alcohol, clearing in creosote, they were mounted in balsam.

In determining the areas of the cross sections of the nerve roots, camera lucida projections were made on millimeter paper, the square millimeters counted, and from these results were calculated the true areas by dividing the results thus obtained by the square of the magnification of the projections. In counting the medullated nerve fibers a Zeiss microscope fitted with a Zeiss objective, 4 mm., aperture 0.95, and oculus No. 6 was used. In the oculus was placed an ocular micrometer ruled into square millimeters. The counting was done by means of an automatic register.

The following table shows a comparison of the areas of the cross sections of the left nerve roots of the spinal cord of a large, normal man, with similar areas from the nerve roots of this small man here reported as suffering from Pseudo-hypertrophic Muscular Atrophy.

	Areas of cross-sections of nerve roots from normal cord.			Areas of cross-sections of nerve roots from abnormal cord.		
	Motor Roots.	Sensory Roots.	Ratio.	Motor Roots.	Sensory Roots.	Ratio.
Lumbar IV.	1.27mm²	2.93mm²	1 :2.3	0.47mm²	1.66mm²	1 :3.4
V.	2.17 "	3.20 "	1 :1.5	1.28 "	1.99 "	1 :1.6
Sacral I.	1.98 "	3.44 "	1 :1.7	1.49 "	2.34 "	1 :1.6
II.	0.61 "	1.92 "	1 :3.2	0.45 "	1.39 "	1 :3.1

The nerve roots of the lower lumbar and the upper sacral nerves only were investigated as the muscles of the calves of the

legs—those most atrophied in this case—are mainly innervated by these nerves. The areas of the cross sections of the nerve roots from the spinal cord of the case here reported are found to be considerably smaller than those from the nerve roots of the normal cord. This is probably partly due to the difference in the sizes of the two subjects.

From the fore-going table it is evident that the ratio between the areas of the cross sections of the motor and sensory roots of the 4th lumbar segment differs in the two cases. The ratio of the normal roots being 1 : 2.3; and that from the roots of the case here reported 1 : 3.4. This is without doubt due to the fact that the motor root (lumbar IV) is smaller that it should be.

To determine whether there is also a corresponding diminution in the number of nerve fibers in these roots a count was made of the nerve fibres in the motor root of the left spinal nerve of the 5th lumbar segment. The result of this count was 5171 nerve fibers as compared with 10,366 in the corresponding normal root. That the different sizes of the two bodies from which the material was obtained partly accounts for this seems evident. This is, however, not the only reason for this difference.

In counting the fibers in the nerve root from the case here reported it was noticed that there were practically no small nerve fibers present. From the author's former investigation (1904) it was determined that the corresponding normal nerve root contains 15% of small fibers (less than 7 micra in diameter). This difference in the number of small nerve fibers in the two roots compared suggest the idea that possibly there was present a diminution in the number of fibers without a corresponding proportionate diminution in the area of the cross section of the nerve root from the case here reported. That this is the case is seen from the fact that the normal root contains, on the average, 4800 nerve fibers per mm^2, while the other root contains on the average 4040. It is therefore evident that the root here investigated not only has a smaller area than the normal root, but contains a smaller number of fibres, as well as, a smaller number of fibers per mm^2.

This diminution in the area of the cross section and in the number of fibers in the root under discussion is without doubt due to a degeneration of some of its nerve fibers and agrees well with the fact already referred to that some of the motor cells of the lateral and ventral horns of the lumbo-sacral segments of the cord were undergoing chromatolysis—some having even disappeared.

Boughton (1906)[18] demonstrated that in the oculomotor nerve of the white rat and of the cat the "small" fibers appear after the period of most rapid growth. This fact suggested the idea that the diminution in the number of small fibers in the roots here studied might be due to the fact that the onset of the muscular

atrophy occurred at such an early period as to prevent the normal growth of the "small" nerve fibers.

The fact that the ratios of the cross sections of the motor and sensory roots from the other segments of the cords compared are so nearly equal would seem at first thought to indicate that there was no degenerative process here present. This conclusion is, however, not necessarily correct. As was pointed out above there is evidence of a greater diminution in the number of nerve fibers than in the area of the cross sections of the nerve root investigated. This makes it probable that in the other motor nerve roots of the lumbo-sacral segments there is also present a diminution in the number of nerve fibers.

VII. DIAGNOSIS.

The diagnosis of Pseudo-hypertrophic Muscular Atrophy in this case is based on the following facts:

1. The early onset.—The first symptoms were observed before the child was two years of age. It was apparent to the father that something was wrong with the child's feet and ankles; but this was not sufficiently marked at first to satisfy those who saw the child that the trouble was of a serious nature.

2. The slow progress of the trouble.—The trouble was for sometime unnoticed, and, when sufficiently advanced to be apparent, was observed to grow worse only very gradually. He gradually became emaciated and weak, and died at the age of twenty years from an attack of Typhoid-Fever. During the last days of his illness, a hypostatic pulmonary congestion was present.

3. The calves of the legs first affected.—The first muscles affected were those of the calves of both legs. These at first seemed large to the father, but after a time became thinner than in normal children.

4. The difficulty in walking.—He never learned to walk well, and fell frequently when attempting to walk. This difficulty was due to contractures, described under another heading.

5. The difficulty in arising from a sitting posture.—He invariably found it necessary to use his hands in arising.

6. The characteristic way of arising from the recumbent position.—He would roll over on his stomach, get up on his knees and elbows, then on all fours, and finally by placing his hands on his knees, raise the trunk to the erect position.

7. Contractures and deformity of his feet.—This resulted

first in *pes varus*—the walking on the outer sides of his feet; and later in *pes equinus*—the walking on the balls of the feet and the heel not touching the ground. He was unable to flex his feet dorsally and as the disease advanced, was compelled to use canes and crutches in walking.

8. The scoliosis.—The spinal column showed a fairly well marked lateral curvature.

9. The vaso-motor disturbances in the calves of his legs.—His feet and the calves of his legs were, after the disease had progressed a few years, generally cold and he seemed to have, as might be expected, a lessened sensibility for cold sensations in them.

10. The microscopical examination of the gastrocnemius muscles revealed a condition characteristic of pseudo-hypertrophic atrophy.

11. An apparent disturbance of the function of the rectum.—He experienced an occasional inability to hold the contents of the rectum, especially when he felt the need of evacuating his bowels. Since he felt the need of evacuating his bowels, there may be some doubt that this disturbance is of nervous origin. It is highly probable that his extreme emaciation, and that the local ulceration of the rectum revealed by scars at the post-mortem examination, were responsible for this disturbance of function. The fact that there was no disturbance of the function of the bladder also supports this view.

12. The prominent shoulder-blades.—It is not certain whether this was due to the general emaciation or to an atrophy of the muscles concerned.

13. The impaired function of the hands.—This, in all probability, was due to an atrophy of the muscles of the hand—a condition not rare in this disease.

14. The unimpaired intellect.

It is to be regretted that we have no data as to the presence or absence of fibrillary contractions in the affected muscles, as to the condition of the deep reflexes, nor as to the electric reaction of the muscles.

In favor of the diagnosis here made of Pseudo-hypertrophic Muscular Atrophy, and against a possible diagnosis of Acute Poliomyelitis Anterior, is the absence of such characteristic symptoms of the latter disease as:—(1) the fever attack, (2) the

acute onset,—sudden paralysis, (3) the partial recovery of the affected muscles, etc.

VIII. CONCLUSION.

1. The most important pathological changes observed in the muscle fibers of the muscles most affected in this case were: cross-striations less marked, lessened staining capacity, pseudo-pertrophy, fatty degeneration, etc. Some of the small blood vessels in the muscles show the diffuse thickening and organized thrombi.

2. The proliferation of the neuroglia beneath the ependyma of the central canal, the slight increase in the neuroglia of the gray matter, and the congested condition of the blood vessels of the spinal cord were probably due to the Typhoid Fever from which the patient died.

3. The chromatolysis of the nerve cells of the gray matter of the cord, especially of the lateral horn, in the segments below and including lumbar IV, is best explained by considering it secondary to the degenerative changes in the muscles.

4. The column of nerve cells in the lateral horn of the lumbo-sacral cord designated No. 7 by Sano[15] seemed to show a diminution in the number of its cell bodies. This was most marked in the 4th and 5th lumbar segments of the cord.

5. The roots of the spinal nerves of the 4th and 5th lumbar and 1st and 2nd sacral segments, both motor and sensory, were found to have a much smaller area of cross-section than those from a normal subject. Thus the area of the cross-section of the left motor root of lumbar IV is only 0.47 mm^2 as compared with 1.27 mm^2 for the normal root. This makes the ratio of the areas of the cross-sections of the motor and sensory roots here studied 1 : 3.4 as compared with 1 : 2.3 for that of the normal roots. This difference in the ratios is due to the abnormal size of the motor root mentioned. The small size of the roots, both motor and sensory, is probably due to several causes,—the material being from a small man, and some nerve fibers having undergone degeneration.

6. The left motor root of the 5th lumbar segment contained by actual count 5,171 medullated nerve fibers as compared with 10,366 for the normal root. In addition to the reasons given for the small areas of the cross-sections of the spinal roots here stud-

ied, it has been suggested that possibly the early onset of this disease prevented the "small" fibers from developing.

7. The muscles most markedly atrophied in this case were those of the calves of the legs—muscles innervated by the cell-bodies of the lateral horns of the lumbo-sacral segments of the spinal cord. This atrophy probably accounts for the chromatolysis of the motor cells referred to, as well as, for the diminution in the number of medullated nerve fibers in the motor roots counted (lumbar V).

8. The histo-pathological findings here reported add support to the conclusion of other investigators that Pseudo-hypertrophic Muscular Atrophy is primarily a disease of the muscles, and that the changes in the spinal cord and in the spinal nerve roots are secondary.

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MYOPATHY OF THE DISTAL TYPE AND ITS RELATION TO THE NEURAL FORM OF MUSCULAR ATROPHY (CHARCOT-MARIE, TOOTH TYPE).*

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Various forms of muscular atrophy have been distinguished and sharply separated from one another. While it is possible to make these classifications for typical cases, the borderline cases cause great confusion, and make clinical diagnosis at times extremely difficult.

The primary neurotic muscular atrophy of Charcot-Marie and Tooth was supposed to be so sharply defined that confusion with other types of muscular atrophy could not occur. The characteristics of the type as given by Charcot and Marie[1] are:

Progressive muscular atrophy implicating first the feet and legs, and not appearing in the upper limbs (hands and later forearms) until several years later; the progression of the atrophy being slow.

Relative integrity of the muscles of the limbs near the trunk, or at least, much longer preservation of these than of the muscles of the distal ends of the limbs. Integrity of the muscles of the trunk, shoulders and face.

Fibrillary contractions in the atrophying muscles.

Vasomotor disturbances in the portions of the limbs atrophied.

No pronounced contractions of tendons in the atrophied limbs.

Sensation usually intact, but sometimes affected.

Cramps frequent.

Reaction of degeneration in the atrophying muscles.

Commencement of the affection usually in childhood, the disease often found in several brothers and sisters, and sometimes in the previous generations.

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*Read before the American Neurological Association, June 4 and 5, 1906.

[1]Charcot and Marie. Revue de médecine, 1886, p. 97.

Charcot and Marie based their conclusions on five cases of their own, and on a few cases in the literature which they believed belong to this type.

I think it well to emphasize the fact that Charcot and Marie in their original paper acknowledged the possibility of implication of the muscles of the limbs near the trunk, at least to some extent. They said that the muscles of the thighs seem to preserve their power and volume during a certain period, but that this integrity often is not real. The vastus internus is first involved. In their summary at the end of their paper they speak of relative integrity of the muscles of the limbs near the trunk, or at least, much longer preservation of these muscles than of those of the distal ends of the limbs. Unquestionably, however, they emphasized the earlier and greater involvement of the muscles at the distal ends of the limbs as the most characteristic feature of the disease.

If we insist on one of the features emphasized by Charcot and Marie; viz., the almost constant absence of all contraction of tendons, many cases classed as progressive neurotic muscular atrophy must be regarded as doubtful.

Cases conforming strictly to the type as described by Charcot and Marie occur, and these probably have a distinct pathology. Such a case is the following, which has been in the Philadelphia General Hospital many years, several times under my care, and so far as I know has not been reported:

N. Hutt, 59 years of age, hostler, was admitted Feb. 15, 1901, at which time the following notes were made:

Chief complaint: Weakness in the arms and legs.

Family history: Father died at about the age of 60 years from some affection of throat, with which he had been ill for one month. Mother died at about the age of 60 years from a second "stroke" of apoplexy. One brother died in infancy and four brothers and three sisters are healthy. The patient is not aware of any family diseases.

He has had only the milder diseases of childhood, and was healthy as a boy and a man, except for the present complaint, and typhoid fever at 17 years. He says he was a moderate user of alcohol, positively denies venereal disease, and has never been married.

History of present illness: He states that at the age of 15 years he began to have pains in the legs and feet, which would last for a week or two, and incapacitate him for work.

These attacks seem to have come on usually in the spring of the year, and he would then be free until the next spring. He had no weakness of the limbs, and was able to work as a hostler, though at about the age of 35 he began to have pain in his hands and arms, and he noticed that he was becoming weak in

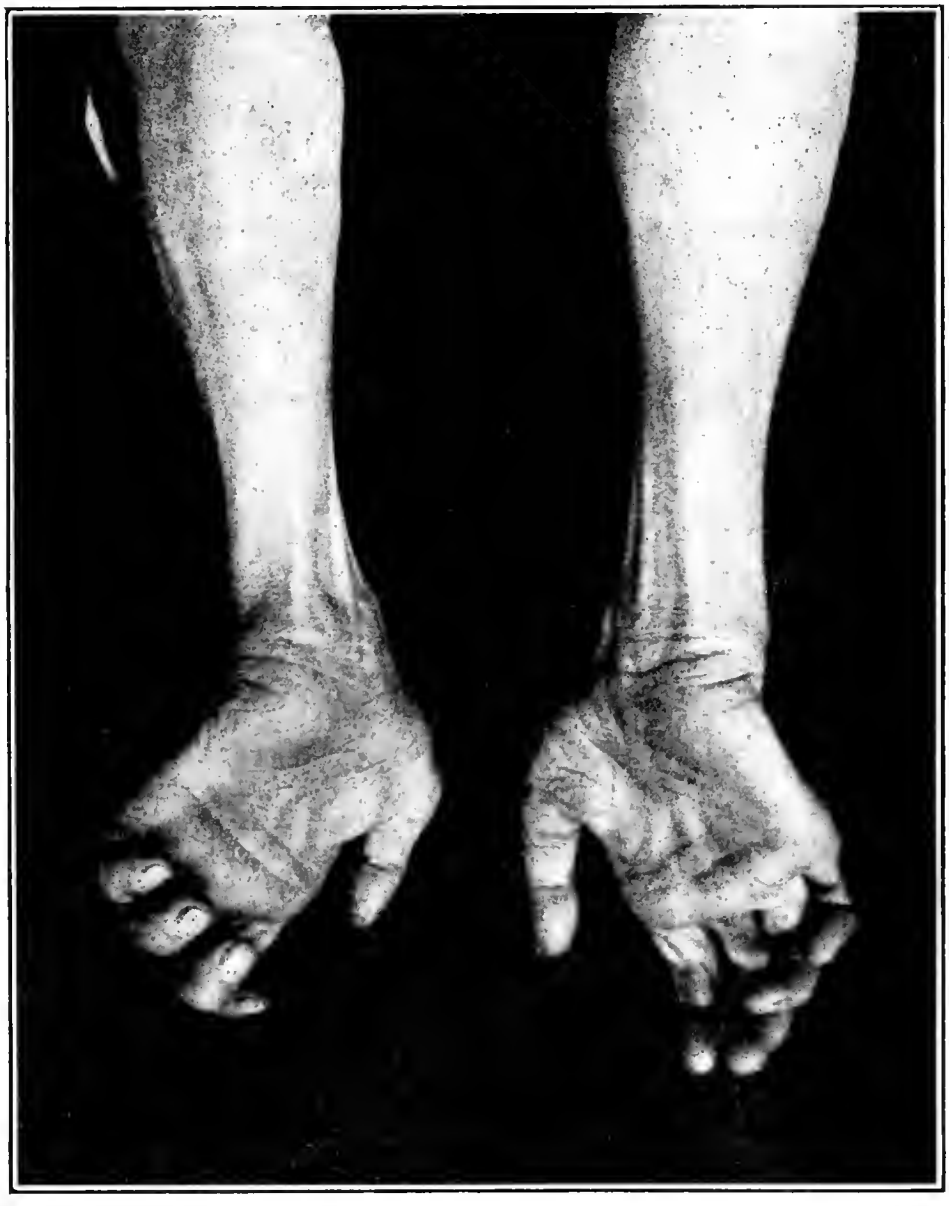

Fig. 1. The neurotic form of muscular atrophy, hands and forearms involved. (Photographed by Dr. Ralph Pemberton.)

the legs. These grew thinner and became somewhat deformed. From that time until the present the progress of the disease has been constant, though he has now much less pain than formerly, and only in his feet. During the past five years he has been unable to do any work. The atrophy of the muscles of the

legs preceded that of the upper limbs ten years, according to the patient's statement.

He is well nourished, pupils are equal and react to light. Tongue is clean and shows no tremor. Chest and abdomen are well formed, the expansion of the chest is fair, lungs are normal, heart sounds are muffled and distant.

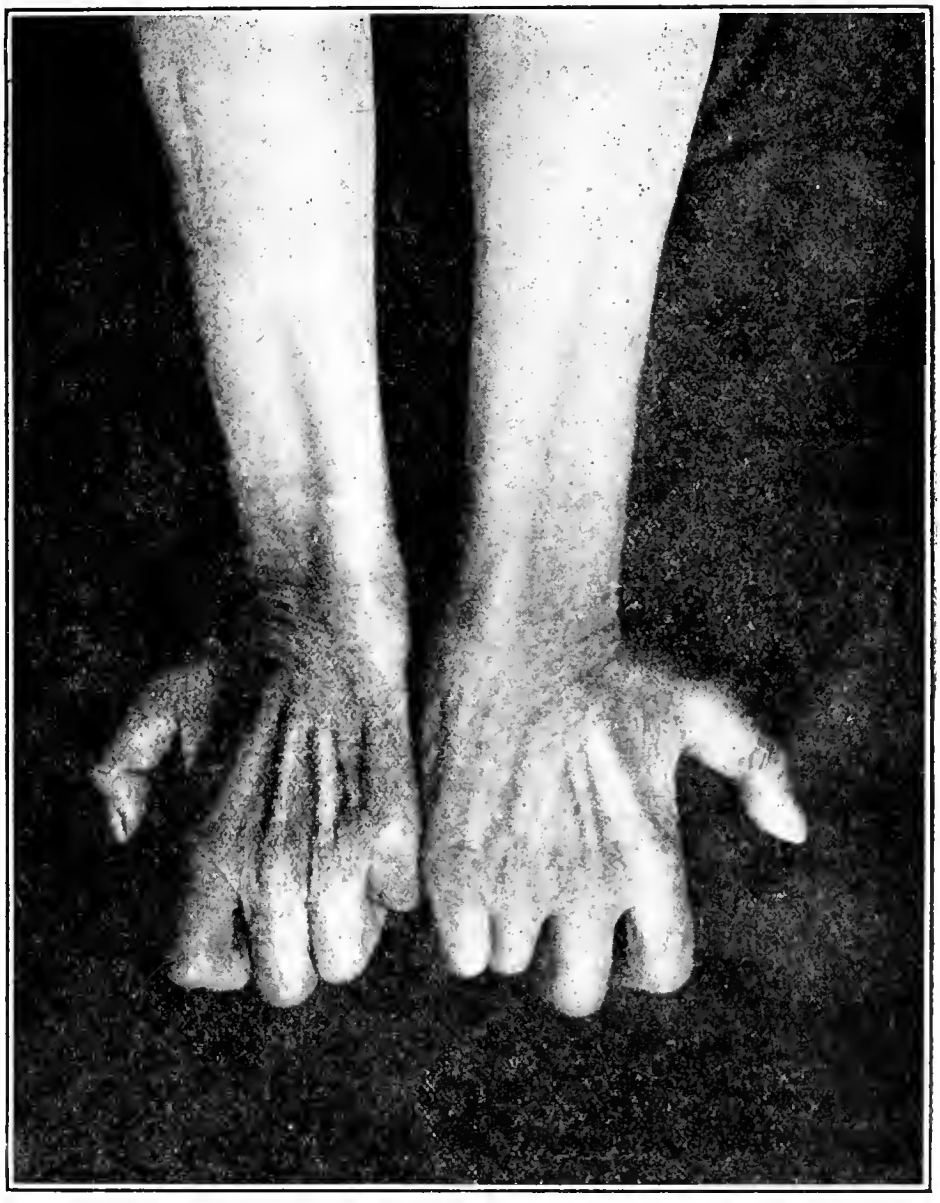

Fig. 2. The neurotic form of muscular atrophy, hands and forearms involved. (Photographed by Dr. Ralph Pemberton.)

Examination of eyes by Dr. Shumway, Oct. 20, 1904: The pupils are equal, respond promptly; eye movements are good.

Ophthalmoscopic examination: Haziness of lens, retinal veins full and show pressure signs, result of arteriosclerosis,

otherwise fundus is normal. Vision, O. D. 5-8; O. S., 5-10; fields are normal.

Condition of the patient at my examination March 7, 1906: The atrophy is intense in the hands (figs. 1 and 2), distinct in the forearms, but the arms above the elbows are well developed. Sensations for touch, pain and temperature are normal in the

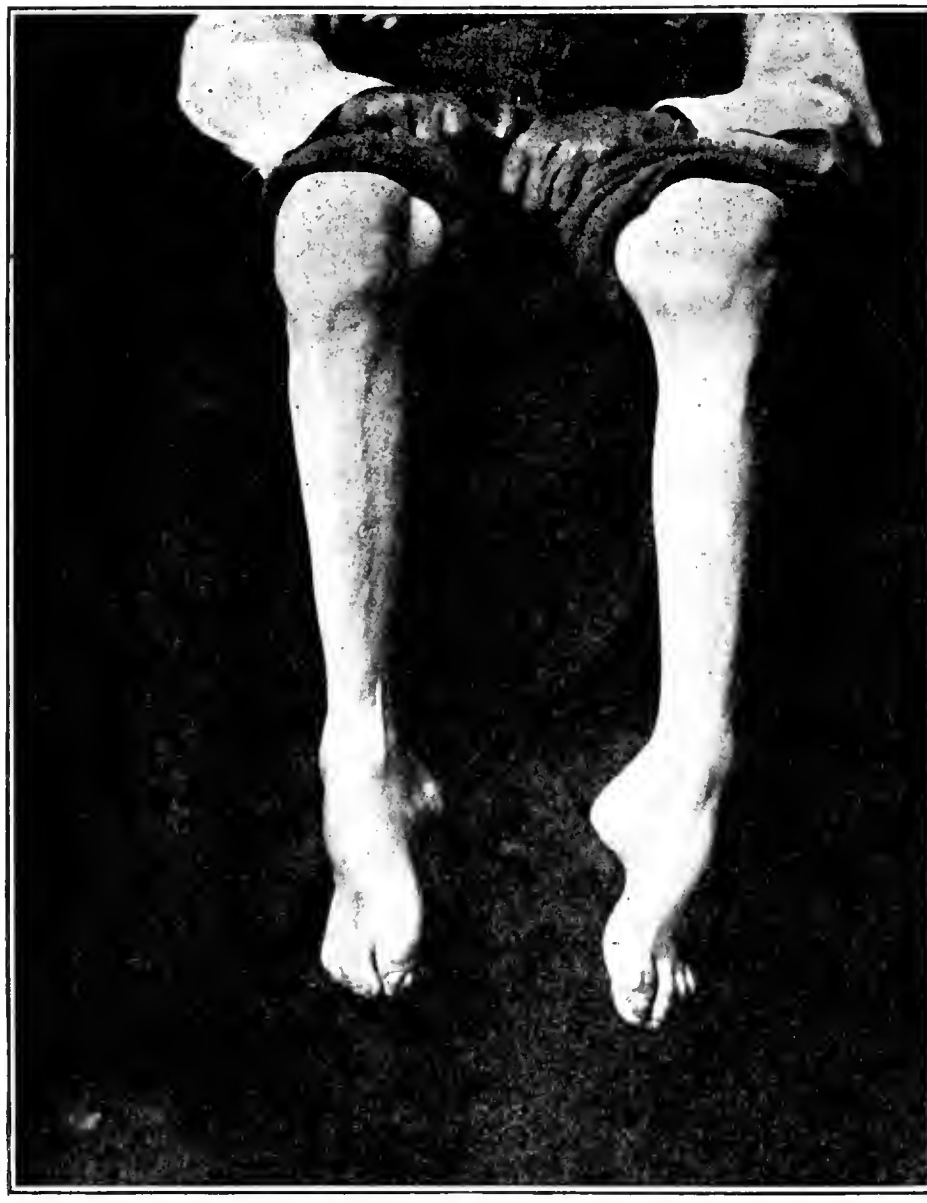

Fig. 3. The neurotic form of muscular atrophy, feet and legs (not thighs) involved. (Photographed by Dr. Ralph Pemberton.)

upper limbs. Sense of position is normal in the fingers. The grasp of each hand is very feeble. The muscles of the trunk and head are well developed. He raises the upper limbs above the head with much power, and is able to dress himself and handle his clothing with considerable skill, notwithstanding

the intense atrophy of the hands. Biceps and triceps reflexes are weak.

The lower limbs, above the knees, are well developed, and if there is any atrophy here it is very slight. Below the knees the atrophy is marked. The feet are inverted and talipes equino-varus (fig. 3) is very pronounced on each side. The patellar reflex and Achilles tendon reflex are lost on each side. The toes are somewhat cyanotic. Sensations for pain, touch and temperature are normal in the lower limbs. He is able to walk without assistance and without crutch or cane, but his gait is rather slow, with legs far apart and his feet turned inward. His gait is somewhat steppage in character.

If we hold to the type of muscular atrophy as presented by this patient; viz., atrophy confined strictly to the distal parts of the limbs, we shall be able to recognize the neurotic muscular atrophy as a distinct type, with a distinct pathology. The cases of this kind with necropsy are very rare, we are obliged to depend on the findings of Marinesco, Sainton, and Dejerine and Armand-Delille. Oppenheim includes the cases of Dubreuilh and Siemerling, but Sainton regards the former as far from typical, and does not refer to the latter, which should, I think, be excluded.

In Siemerling's[2] case there was great atrophy of all the extremities in the upper parts as well as in the lower, and of the trunk; flaccid complete paralysis of the lower limbs, so that all voluntary movements of these limbs were lost. Siemerling found degeneration of the posterior and lateral columns, most intense in the lower thoracic and upper lumbar regions, degeneration of peripheral nerves and muscles; atrophy of the cells of the anterior horns, of the columns of Clarke, of the anterior roots, and of the spinal ganglia.

He thinks there can be no doubt that the case belongs to the spinal neurotic atrophy, but if we accept this opinion we cannot limit the type to those cases in which the atrophy affects the distal portions of the limbs, and the proximal portions and trunk escape.

In the case of the neurotic muscular atrophy studied by Paul Sainton[3] the lesions were:

Sclerosis of the posterior columns, especially of the columns of Burdach. Slight degeneration of both pyramidal tracts. Al-

[2]Siemerling Archiv. für Psychiatrie, Vol. 31, 1898, p. 105.
[3]Paul Sainton Nouvelle Iconographie de la Salpêtrière, Vol. 12, 1899.

teration of the columns of Clarke. Atrophy of the cells of the anterior horns. Slight degeneration of the intramuscular nerves. Slight sclerosis of the nerves of the forearms and legs, very distinct only in the peroneal nerves. Atrophy of the muscle fibers even causing complete disappearance of some fibers with proliferation of connective tissue.

In this case the atrophy began in the upper limbs. The lesions resembled those observed by Marinesco in his case, except that Marinesco found the anterolateral columns intact. Judging from these two cases Sainton regards as the lesions of this form of muscular atrophy: sclerosis of the columns of Burdach and Goll, atrophy of the cells of the anterior horns, and alterations of the peripheral nerves of varying intensity, sometimes slight.

The lesions found by Dejerine and Armand-Delille[4] in a case of neurotic muscular atrophy were degeneration of the posterior columns, degeneration of some of the nerve cells of the anterior horns of the cervical and lumbar regions without diminution in the number, chronic meningitis, degeneration of the muscles of the hands and feet, alteration of the intramuscular nerve fibers in the hands and feet (*i.e.*, many nerve fibers of small size, many empty nerve sheaths and a few nerve fibers in the process of degeneration). The nerve trunks, the cutaneous sensory nerves, and the anterior and posterior nerve roots with slight exception were normal.

The literature contains many cases reported as belonging to neurotic muscular atrophy in which some atypical features were present. Sachs'[5] two cases were probably the first reported in America. In his first case the patient had pronounced kyphosis of the lumbo-sacral region, slight wasting of the infraspinatus muscle, and decided diminution in the power of thighs as well as of legs.

The younger brother of the first patient had general emaciation of all parts of the upper extremities, very distinct atrophy of the infraspinatus and marked general atrophy of the legs, which seems to imply involvement of the thighs.

In discussing the cases together Sachs says the atrophy caused a weakness of the thigh muscles as well as of the muscles

[4]Dejerine and Armand-Delille. Revue Neurologique, 1903, p. 1198.
[5]Sachs, Brain, 1890, Vol. 12, p. 447.

of the legs, and he speaks of atrophy involving all the muscles of the lower extremities as common in the neurotic muscular atrophy.

In one of Hoffmann's[6] cases the lower limbs were wasted in all parts, and chiefly below the knees, but the strength of the thigh muscles was good. Fibrillary tremors were not observed. In another case, Hoffmann found wasting of the upper part of the left trapezius muscle and flattening of the supraspinous and infraspinous fossæ, without any loss of function. Fibrillary tremors were not seen. In another case he says the thigh muscles were distinctly weak and much wasted, especially in the lower part of the thigh. The thoracic muscles were poorly developed and the ribs were prominent. In still another case all the thigh muscles were much atrophied, as well as the muscles of the buttocks and the lower part of the extensors of the back. The patient had a distinct lumbosacral kyphosis, and experienced difficulty in rising from the ground, such as is seen in progressive muscular dystrophy. The legs were kept far apart and the patient climbed upon himself as in muscular dystrophy.

Hoffmann accepts involvement of the facial muscles as a part of the neurotic atrophy.

In the case reported by George W. Jacoby,[7] and recorded as one of progressive muscular atrophy of the peroneal type, severe pain was felt in the lower extremities when the child was two years old. When she was four years old one leg was found to be weaker and thinner than the other. Later atrophy of the thigh on the side opposite to that of the affected leg was noticed. There was marked lordosis and slight lateral curvature. The muscles of both thighs were in a continual state of unrest, almost like fibrillary twitchings. The right foot was in equinovarus position. There were no sensory disturbances, but there was partial reaction of degeneration in the affected muscles. The arms were not affected.

Jacoby acknowledged that the case was not typical, inasmuch as the atrophy, although bilateral, was asymmetrical, one leg being affected and the opposite thigh.

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[6]Hoffmann. Deutsche Zeitschrift für Nervenheilkunde, Vol. 1, p. 95.

[7]G. W. Jacoby, JOURNAL OF NERVOUS AND MENTAL DISEASE, 1894, p. 259.

Sachs, however, concurred in the diagnosis of muscular atrophy of the peroneal type.

In at least one of Diller's[8] two cases (brother and sister), the thighs also were wasted, and the patient climbed upon his lower limbs as is seen in muscular dystrophy. He had left talipes equinus. Response to electricity in the paralyzed legs was very sluggish and reaction of degeneration was obtained.

In the first of the two cases reported by Given Campbell[9] the disease progressed in the lower limbs until these limbs became useless and the muscles of the thighs also were much wasted. The deltoid muscles, especially the left were weak. The weakness and wasting developed hand in hand, and began in the peroneal group of muscles. The small muscles of the hands were not affected. The muscles of the trunk were not wasted, unless possibly the abdominal muscles to a slight extent.

I could add to these cases others to show that atypical features have not prevented the diagnosis of muscular atrophy of the Charcot-Marie-Tooth type, and therefore it is not surprising that cases of muscular dystrophy have been diagnosed clinically as belonging to the type. In this connection it is interesting to read Sachs' words published in 1889 (Brain) when the peroneal type of muscular atrophy was little known. He said: "There is a consensus of opinion, however, on this one point, that the cases in question do not belong to the category of primary muscular dystrophies." He was speaking of the peroneal type of atrophy. Oppenheim and Cassirer[10] showed that this consensus of opinion no longer exists.

They pointed out that the clinical picture of the neurotic muscular atrophy is not so sharp as Hoffmann believed, and that the pathology is still uncertain. In their case the muscular atrophy had existed about two years. The symptoms began with severe pain in the lower and upper limbs. The distal portions of the lower limbs, especially those innervated by the peroneal nerves, were affected, and in the upper limbs only a part of the small hand muscles, and later the triceps and supinator longus, were implicated. In some muscles incomplete reaction of degeneration was obtained, in others all electrical response

[8]Diller. Philadelphia Medical Journal, March 17, 1900, p. 642.
[9]Campbell. JOURNAL OF NERVOUS AND MENTAL DISEASE, 1900, p. 274.
[10]Oppenheim and Cassirer. Deutsche Zeitschrift für Nervenheilkunde. Vol. 11, p. 143.

failed. The tendon reflexes were diminished. The orbicularis palpebrarum on each side was involved. Later the calf muscles became atrophied. The diagnosis seemed to lie between chronic multiple neuritis and neurotic muscular atrophy. The central and peripheral nervous systems were normal, but the muscles were much degenerated.

As regards the involvement of the orbicularis palpebrarum, Oppenheim and Cassirer refer to the fact that in Sachs' cases the infraspinati were atrophied, in a case of Hoffmann's the upper portion of the left trapezius and the supraspinati and infraspinati were atrophied, in a case of Donkin's reaction of degeneration was obtained in the left trapezius, in a case of Hülemann's the pictoral, romboid, latissimus dorsi muscles were somewhat atrophied, in a case of Eisenlohr's the atrophy of the upper limbs was like that of myopathy, but of the lower limbs like that of the neurotic musclar atrophy. All these cases were regarded as belonging to neurotic muscular atrophy, and yet they showed some features in common with muscular dystrophy. Especially interesting are two cases in sisters observed by Dähnhardt, in one, the wasting resembled the neurotic muscular atrophy, in the other the progressive muscular dystrophy.

Oppenheim and Cassirer concluded that the clinical picture of progressive neurotic muscular atrophy may be caused by muscular dystrophy, and that the pathology of the former is not always the same and not as yet clearly defined.

Gowers[11], in reporting a clinical case of distal myopathy in 1902, said he had not seen a similar case nor did he know that one had been recorded. The patient, a boy, was eighteen years old, and was the only member of the family affected. The symptoms first attracted notice when he was ten or twelve years old; then it was noticed that he often caught his toes against the ground in walking. At a later date his hands were found to be weak. The feebleness of hands and feet steadily increased until it became pronounced. He was unable to flex the ankles, although he could just extend the toes and could move each foot slightly in and out by the tibialis anticus and the peronei. He could extend the ankle joint by the calf muscles with some force. The movements of the knee and hip were performed with good power, the knee jerks were present, but the left was less than

[11]Gowers. British Medical Journal. July 12, 1902.

the right. The thigh muscles were of normal size; the anterior tibial muscles were distinctly smaller on the left side; the calves were rather large and firm, and were sufficiently large to suggest a resemblance to pseudo-hypertrophic paralysis. The grasp was extremely feeble, with the right hand he could not move the dynamometer, and with the left he could only move the index to 2 k° instead of 50 or 60. Extension was also feeble; he could get the fingers with the wrist into line with the forearm, but could not fully extend them when the wrist was overextended. The muscles of the forearms and hands were small, but presented no wasting comparable to that which is seen in progressive spinal atrophy. Above the forearm the muscles had fair power and presented no wasting; only a trifling atrophy of the middle part of the trapezii could be observed. Electrical excitability was lowered in the affected muscles in proportion to their feebleness, and equally to faradism and voltaism.

The neck muscles were normal except the sterno-mastoids. He could not raise the eyebrows at all, and closure of the eyes by the orbicularis muscles were weaker than normal.

Gowers said this case differed from all recognized forms in the purely distal distribution of the affection in the limbs and the normal state of the muscles near the trunk. The case resembled therefore the neurotic muscular atrophy, but the implication of the sterno-mastoid, frontalis and orbicularis palpebrarum muscles probably prevented Gowers from classing the case under this head.

In the case of myopathy of the Aran Duchenne type reported by Dejerine and Thomas,[12] the muscular atrophy was confined to the upper limbs and the hands were much wasted. The muscles of the head, neck, face, trunk and lower limbs were not at all affected. Fibrillary contractions were seen in the upper limbs. The symptoms began when the patient was 49 years old, and lasted thirty-one years. It is not surprising therefore that the case was regarded as one of myelopathy during the lifetime of the patient. The spinal cord and nerves, even the intramuscular nerves, were normal. The muscles were much altered, many muscular fibers had disappeared, and those that remained were much atrophied, many had lost the transverse

[12]Dejerine and Thomas. Revue Neurologique, No. 24, Dec. 30, 1904.

and longitudinal striations, the sarcolemma nuclei were multiplied and some were enlarged.

The case of muscular dystrophy affecting the hands and feet reported by C. Macfie Campbell[13] was without necropsy. Other members of the family were similarly affected. Fibrillary twitching in the interossei and short flexor of the thumb, and reaction of degeneration in two of the atrophied muscles of the lower extremities raises the suspicion that the case possibly may belong to the type of neurotic muscular atrophy.

These are the only instances in literature I know of where muscular dystrophy gave distinctly the clinical signs of neurotic muscular atrophy, but a case of this character with necropsy has come under my observation. I hesitated during the lifetime of the patient to make the diagnois of neurotic atrophy, although the case was regarded by some as belonging to this type. I hesitated because the thighs and upper arms and trunk were also much atrophied, and I felt that the diagnosis should be left open. The commencement of the atrophy in the lower part of the lower limbs, the slight reaction of degeneration in these parts, the talipes equino-varus on each side, the symmetry and the later involvement of the upper limbs suggested the neurotic form. The microscopical examination has shown a normal central and peripheral nervous system with much atrophy and disappearance of muscle fibers.

G. R., age 28 years, was admitted to the Philadelphia General Hospital Oct. 11, 1898, complaining of pain about the knee and difficulty in walking.

Family history: Father died of heart disease at about 25 to 30 years of age, mother living and well, at 50 years of age.

The patient was married at 23 years or age. He has one child, three years old, in good health. He uses neither tobacco nor alcoholic drinks, and has been employed as a watch case engraver since his fifteenth year.

Past medical history: He has had childhood diseases. When ten years old he fell and injured his back and could not walk for two months (the condition was diagnosed as spinal meningitis), but he recovered the use of his limbs satisfactorily in about one month after getting up. Three years ago he had an attack of cystitis.

History of present illness: About two years ago he fell from a street car, striking on his hip and back of his head,

[13]Campbell. Review of Neurology and Psychiatry, March, 1906, p. 192.

badly bruising his right hip and thigh. He suffered no particular inconvenience from this at the time, except a slight but constant pain in the knee joint. About six months later, upon arising one morning, he fell to the floor and found himself unable to walk, but this was probably not the first manifestation of weakness. Two or three days later he was able to walk with a cane, and after a few days could walk without it. He walked without a cane about two weeks. During this time, while walking along the street, he fell and had to be assisted to a car, and could not walk during the remainder of the day. About a week after this he was obliged to use the cane again, and has continued doing so since. For the past year and a half the inability to walk has steadily progressed, and coincident with this has been the muscular wasting. No involvement of bladder has occurred, except a cystitis three years ago, previous to his accident. He had noticed weakness first in the left foot, later in the right foot, and three months still later his upper limbs became affected. He stated that the wasting began in the feet, extended to the legs and thighs, and that the upper limbs were affected first in the distal parts.

Condition upon admission: He is a somewhat poorly nourished adult male, of above average height; pupils are dilated, the left more so than the right, both react to light, accommodation and convergence. Speech is normal. Tongue protrudes straight and shows no tremor. Pulse is small, quite irregular; arteries are soft.

Chest and lungs: Chest is somewhat emaciated; expansion is good. Lungs are normal.

Heart: Apex beat is visible and palpable in the 6th interspace, 1½ inches within the nipple line. First sound is loud and strong; second sounds are both accentuated. Liver and spleen are normal.

Arms: Grip, right hand, is 21; left is 17. The arms are emaciated, but not atrophied. Ataxia is not detected.

Legs: Marked general wasting of the muscles, apparently equal in both legs. Foot-drop is marked. Toes are slightly flexed. Spasticity of legs is not present.

Reflexes: Knee jerks are absent; no ankle or patellar clonus; superficial reflexes are present. Tactile sensation is preserved and normal. Pain and temperature sensations are normal.

Nov. 7, 1898: Patient's physical condition is improved since admission. No change in walking, or in rising from chair. He is gaining slightly in weight, but complains of some pain in the legs and ankles.

An examination of the eyes April 14, 1899, by Dr. Charles A. Oliver showed healthy eyegrounds, paresis of left external rectus and left superior oblique, with diplopia, which had been present a few days. This was not persistent.

At this time he was unable to walk, and could not stand alone,

although he could move his legs somewhat. Foot drop was marked on each side. The legs could not be extended. Muscular atrophy had been progressive.

On June 25, 1899, a note was made that the patient could walk, but had inversion of the foot on attempting to walk.

Sept. 10, 1899, the patient was granted a two-days pass on which he went out Friday. He was brought back by a patrol wagon last night, having been picked up at Market St. ferry, where he had suddenly lost all power of his legs. When admitted he was complaining of abdominal and lumbar pain, loss of power in legs as well as a feeling of complete exhaustion. This evening he is much better, and already can move his legs fairly well, has lost much of the lumbar and abdominal pain, and feeling of exhaustion.

Sept. 26, 1899. He has improved rapidly since his return; he can walk, but is still kept in bed.

April 13, 1901. Examination by Dr. Pearce. Both upper extremities, both above and below the elbow, respond quickly and well to faradism. There is no reaction of degeneration. Elbow jerks and biceps jerks are present, but much diminished. There is no localized wasting in upper extremities. In the lower extremities there is marked quantitative change below the knees, and very slight qualitative change in the anterior and posterior groups of muscles. There is no sensory change anywhere. Knee jerks are absent. Plantar reflex is present. No arthropathies; pupillary reactions are normal.

July 19, 1903. He has not walked for two and a half years. Sensations of touch and pain are normal.

Aug. 20, 1903. Dictated by Dr. Weisenburg. Patient is unable to walk; he can flex the right thigh on the hip slightly; left thigh movement is very poor. No movements possible in ankles; he can flex or extend the toes of both feet very slightly. Both feet are in a position of talipes equino-varus, the left more than the right. Both feet are cold. Movements of both upper limbs are free in all directions. Grip in the hands is almost nil. No power in elbows at all. Power in shoulders fairly good. Both upper extremities, shoulders, back and chest are wasted; abdomen not so much. Wasting is about equal on both sides. Thenar and hypothenar eminences on both hands are almost absent. Atrophy of interossei is marked. In the forearm the flexor group is more atrophied than the extensor; the biceps is atrophied on each side. Deltoid is slightly atrophied. Pectoral muscles wasted, also the interossei on both sides. Supraclavicular and infraclavicular fossae well marked, also interscapular region. All muscles of the shoulder girdle are atrophied, including the latissimus dorsi on each side. No fibrillary twitching is noticed in the muscles. All reflexes in the upper limbs are lost.

He has never had any urinary or rectal disturbances. The pupils are equal, respond to light, accommodation and convergence. No apparent weakness of left internal rectus or left superior oblique. No involvement of 5th or 7th nerves. Tongue protruded straight; no tremor or atrophy. No atrophy of the muscles of the face or neck.

At present the wasting in both lower extremities is about equal. The femoral group on each side is very much wasted;

Fig. 4. Piece of muscle from the sole of the foot in a case of myopathy of the distal type. (Photographed by Dr. Alfred Reginald Allen.)

the calf muscles are wasted, as are the thigh muscles of each side. All the reflexes in the lower extremities are lost. Plantar irritation produces flexion of all the toes. Abdominal and cremasteric reflexes are present and active.

He says he has dull pains in the legs and arms, but has never had any sharp shooting pains, nor any sensory disturbances like pins and needles. Sensation is apparently normal.

Oct. 12, 1904. Dr. Shumway: Right vision, 5-5; left vision, 5-6. External ocular movements are normal. Pupil reaction is prompt. No diplopia in any part of the field. Fields full; perfectly normal eyegrounds.

July 21, 1905. Examination by Dr. Pickett: Ankles are almost fixed in position of foot drop. The right knee cannot be extended completely. Strongest movements of the body are extension of the feet (plantar flexion), supination of the fore-

Fig. 5. A piece of muscle from the sole of the foot in a case of myopathy of the distal type. (Photographed by Dr. Alfred Reginald Allen.)

arm, then after this pronation and extension of wrist and fingers. Preserved supinators and extensors make the forearm bulge as if it and the bones were bent. His movements in dressing are at times so irregular by the unequal strength of the different muscles as to constitute a kind of ataxia.

Dec. 6, 1905. Patient's nervous condition is the same as described above. He has a cough and expectorates moderately;

has night sweats, and the sputum shows the presence of tubercle bacilli.

Urine analysis: Dec. 6, 1905. Yellowish; slight sediment; acid reaction; Sp. Gr. 1010. No sugar or albumin. Microscopic: red blood cells, a few; few calcium oxylates, epithelial cells and debris.

He died Jan. 11, 1906.

This man was examined frequently and lectured upon by me several times. He was repeatedly in my charge at the hospital. His condition at the time of death was as follows:

The lower and upper limbs were greatly atrophied and equally so at all parts. Contracture at the right knee was present, so that the leg could not be fully extended upon the thigh. The left leg could be fully extended on the thigh. There seemed to be contracture at the right hip. Talipes equino-varus was present on each side. No contractures were observed in the upper limbs. Emaciation was shown also in the muscles of the trunk and face. Slight lordosis was present in the lumbo-sacral region, and there was some protrusion of abdomen, probably secondary to the lordosis. There was no evidence at the necropsy of old spinal injury, and excepting the lordosis, there was no deformity of the vertebræ. The wasting of the face may have been caused by tuberculosis, as it was not present in 1903.

Sections from the cervical and lumbar regions of the spinal cord stained by the hematoxylin method of Weigert or the method of Marchi, by acid fuchsine, hemalum or thionin are normal. A piece of muscle from the foot (fig. 4 and 5) shows no recent degeneration by the Marchi method, but the long-standing degeneration is pronounced, and is better shown by other methods of staining. The fibrous and fatty connective tissues are much increased, the muscle fibers are greatly atrophied. The longitudinal and transverse striations are well preserved, and the sarcolemma nuclei are increased in number in some of the muscle fibers. Nerve bundles between the muscle fibers stain well by the Weigert hematoxylin stain, as do also sections of one of the plantar nerves. No degeneration is seen by the Marchi method in the latter, but the blood vessels of the nerve are thickened. A piece of muscle from the back of the trunk appears normal.

CONSCIOUSNESS IN THE BRUTES.[1]

By George V. N. Dearborn, M.D., Ph.D.,

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The brutes cannot tells us humans directly whether they are conscious or not, because they are "speechless" if for no other reason. Opinions on the matter range from that of the zoophilist, (who is apt to believe them all as richly endowed with thought and fancy as himself) to the occasional physiologist, now rarer and rarer, in whose vocabulary the term consciousness, save as the name of an epiphenomenon in man, a "secretion" of the brain, does not ever appear. Trite as the subject is, new biological view-points give the matter even a somewhat novel appearace and so lend new material to its philosophical discussion.

The problem may be approached from any one of at least three sides, or from all three. The first of these is that of untechnical common-sense, which means the pure reason beneath all our notions, some times at fault. By "the man in the street" the brutes are considered conscious as a matter of course, because he knows them to be built substantially like himself and because he sees them act practically as he would act under like conditions, if the conditions were not too complex or too abstract—and the average man knows himself conscious if he knows nothing else, this being the essence of humanity. The usual man then takes it for granted that the brutes, especially the "higher" brutes are conscious by the innate criterion of analogy, the perception of identity, by the doctrine of probability, by the inherent chances of the facts as he sees them. It is likely that no man could do better in principle than this, nor will he, until the protozoa learn to talk! The second side from which the problem may be approached is that of epistemology and metaphysics, and the more natural trend of this way of looking is toward pan-psychism, the climax of rational idealism, at no variance with science. A third way the matter may be discussed is in the light of biology, physiology, chemistry, and phy-

[1]Read before the American Philosophical Association in Philadelphia, Dec. 29, 1904.

sics concurrently with psychology, the former gradually describing the details of the somatic process while the latter science analyzes even more gradually by the aid of experiment further and further into the description of consciousness —two "parallel" lines of details which persistently refuse, and more and more persistently, to "correspond." This third method of approach rests also on analogy for any satisfaction it may result in, but on an analogy so minute and sooner or later so complete that to deny its consequences as to the consciousness of brutes is mere obstinacy, the child's principle of self-supporting opposition. In theory, this method carried out fully would amount to a demonstration to everyone and be the best which we could hope ever to do. In practice, the details of the similarity in function and structure between man and brute are not so complete but that some still say, "We are conscious, but horses and crabs and butterflies are not," forgetful that this form of solipsism is natural although illogical, and that probably could they think and speak the horses and the crabs and butterflies would each so express itself of all outside the limits of his own communication. The evidence lacks only this mere affirmation of being universally conclusive. The present paper attempts to approach the question as to the consciousness of animals from what is practically this last direction. It is the viewpoint of the physiologist, the vivisector, the medical man, interested in the chemistry and the physics as well as in the gross and minute anatomy of animal bodies from ameba's to his own. It is also the viewpoint of the amateur psychologist *speculating* where experiment is vain.

It seems to be more and more obvious that to trace out any sort of a one-to-one correspondence between the terms of the mind-series and the terms of a brain-series is impossible. Whether these two be considered as causally or only as concomitantly related, whether the viewer be materialist or pan-psychist, the members of these two empirical series cannot be made to correspond and the likelihood of tracing out any exact back-and-forth relation is more remote now than formerly, as detailed knowledge of the two orders of events increases. I say this probably too dogmatically for I say it somewhat feelingly, since the conviction comes to me I confess with a tinge of disappointment. I started out on this quest with a most unphilosophical but not unnatural bias, a firm presupposition as a matter of course that psycho-physical

parallelism between nerve and mind was very probable and that only the details remained to be worked out. I have little idea how it is with my hearers, but in myself the more I racked my ingenuity in comparing the two series of processes back and forth, the better, too, the details of the two series here and there became known, the more hopeless the comparison seemed. Sometimes I tried to make myself believe that it was only because the details were so dim as yet, especially in the mental series, that the correspondence did not appear and the two halves fit together like the sunset sky and the skyline beneath it of hills and forest. This sort of harmony one would like to experience and add to his understanding of "things as they are"; it arouses one's scientific curiosity as few things beside and the sense of being baffled is correspondingly unpleasant. The pseudo-satisfaction of reducing consciousness to a process of cortical "associative memory," as is now the fashion among a certain school of physiologists, is a "satisfaction" as remote as any which can be imagined, for after all the brain is something different from a "phonograph," and mental process unlike vibrations.

Like many others then in the same dilemma, students of both mind and body, it seems to me inevitable that one should take refuge in a purely logical syllogism and conclusion that somehow or other there are not two series there at all, but only one, and that the other inheres in this, that the body is a phenomenal aspect of the mind or of mind. Idealism truly offers all men rational satisfaction (but least when we try to orient in it our own bodies), although at the same time we must sympathize with and try to satisfy the natural realist, and even the out-and-out dualist when he refuses to repress his human curiosity as to the magic means by which the reason so readily fools, or let us say outreasons, its hand-maid, sense. What the scientists need, indeed, to be taught by students of philosophy in return for facts, is the truth, the reality, and the importance of the idealistic view. Epistemology should not exist for students of philosophy alone, and should not for lack of cöoperation, be considered by the average biologist and physician and man of science a system of notions merely, pleasant to discuss sometimes, but useless after all. If idealism is worth its historic name, it is fit substratum for every science in the schools, and its principles simple in themselves lend simplicity to many a complicated scientific snarl. On the idealistic basis

science gets a richness as well as a truthfulness which students of science should not be allowed by their instructors to miss out of their knowledge, a richness which develops into one's whole life.

The pan-psychistic aspect of the relations of mind and body as processes, fails to show to common sense (and it is common sense which most of us are trying to impart) a working hypothesis on which the scientific student may rest when, as usual, he is not in a metaphysical mood. And of course the scientific problem is quite independent of the philosophic problem though not of the philosophic student, for his puzzles are also in part those of the psychologist. So far as most of us know for a certainty, mind is inherent only in living tissues and it is inevitable that every active inquiring imagination should insist on some sort of parallelism if there is any sort of basis for any such conception. The fact that disembodied consciousness is to say the least doubtful and a body therefore always accompanying mind to say the least probable, makes this demand reasonable, and renders necessary some sort of parallelistic theory if mind itself is to be understood. The physiologist as a scientist is somewhat better off, more independent of mind than the psychologist is of body, as one sees evidenced in comparing the text-books of physiology with those of psychology and in observing therein that whereas the physiologies, the older ones, scarcely refer to mind, the psychologies, the newer ones, refer everywhere to the bodily substratum.

Notwithstanding this dependence of an adequate empirical psychology on the somatic process for guiding description in at least some directions, no one has ventured to attempt to define a plan for the psychophysical correspondence in general, and now the problem seems, as has been said above, further from solution than ever before although the light at present is greater than ever. Never before was so much really known about the nervous system with which it is customary to attempt to correlate mind, while systematized introspection continually shows us more of the molecules, so to say, of mind. Psychophysical parallelism must evidently take a new standpoint if it is to serve a useful explanatory or even descriptive purpose.

The reason for this failure of correspondence more and more lies partly in the change which our notion of the nervous system has undergone very recently. It has lost some of the former im-

portance accorded to it in the hierachy of the tissues and organs. There are conditions inherent in the neurone theory of the structure of the nervous system which supported the belief that the neurones were in a way the physical basis of mind; for example, they allowed of a belief in the existence of perfectly definite centers in the brain and cord with which it was comparatively easy to imagine that various psychical functions, for example volitions, were connected in some way directly. Again, the nerve cells might be similarly used, while that fugitive retraction-theory of the neurones, although never really accepted as certain by anyone, doubtless helped to make more tangible a vague correspondence between a period of consciousness and the same period of the functions of the nervous system. Now the tendency is more or less away from the neurone theory. Many physiologists begin to see in the nervous system an immensely complex reticulum of very minute fibrils, combined mostly into axis-cylinders and here and there surrounded and enmeshed by masses of different yet neural protoplasm, the nerve-cells. These nerve cells are no longer considered by many as wholly the functional centers of the fibrils as a battery-element is the center of a telephone system, but appear rather chiefly as organs for controling the nutrition of the fibrillar network, having also important uses in cöordinating and changing more or less the impulses coming into them over the fibrils. In short, the nerve-cells no longer have in many minds the importance they seemed to have, while the fibrils, the conducting paths, have acquired new meaning. It is the white matter composed of fibrils which most largely serves the functions of the nervous system, the chief of these functions being to connect the separated parts of the body and the body at large with the sense organs by which it is related to its environment. These separated "organs" are not only the massive viscera, the muscles, the brain, etc., but they are also single cells and groups of cells in muscles, glands, and supporting tissues. From this point of view the important use of the nervous system is to connect, to cöordinate, and to adjust by its own control over impulses which are originated not within but outside of its substance, by forces either mechanical or chemical or electrical. It is, by the newer view, somewhat, but not much, more than a vastly complicated system of protoplasmic bridges connecting more or less active and extensive structures and the individual with its environment.

Some of these structures which the fibrils of the nervous system connect exhibit their lack of dependence. In other ways the predominance of muscular tissue in psychophysical processes is equally obvious. In the perception of space and of time every ultimate criterion is muscular in all probability, since time-perception depends on bodily rhythms and space-perception, largely at least, on movements (two-dimensional space getting its appreciation from eye movements and three-dimensional from the contractions of other muscles especially those of the arms and legs). Even monocular and instantaneous perception of depth may contain within it the universal influence of afferent and efferent impulses connected with the tonal or more powerful contractile movements of muscles. The category of causality seems to have a quite similar origin in the individual consciousness, the child obtaining this notion clearly only from his actual experience that he can cause changes and things himself by the actuation of his muscles. Thus the muscles, more than half the weight of the body, concern themselves most intimately with the most basal categories of the mind, which develop as the muscles' use and control develop in the child. The unstriated and involuntary muscles differ in no wise from this in principle, since they appear to represent only another form adapted by force of the law of habit to work even more automatically on its services very readily, for example the heart and the glands. We need not review the large amount of work on the contraction of muscle lately performed; here it is sufficient to summarily say that it is now known that practically all the unstriated muscle of the body, and the heart (although partaking of the nature of striated muscle) can carry on their rhythmic action indefinitely when supplied with nutriment, heat, etc., without nervous control; of this the heart's apex, containing no known neural tissue, is the most striking example. If we transfer our attention to the protoplasm of simple animals instead of to the tissues of the "higher" forms, we find quite analogous results. Thus Yerkes studying the movements of the medusoid Gonionema (a jellyfish-like animal of common occurrence), finds that the reactions of special organs or parts are not dependent for their execution upon the functional activity of the central nervous system, nor spontaneity of movement, nor cöordination, but that this last depends rather on the rapid transmission of what is probably a muscular impulse, the

others on stimuli inherent in the tissue itself (that is on nutriment, ions, or what-not). In short the tissues and smooth muscle in particular are automatic and depend on the nerves probably only for cöordination in cases where the impulses would not be carried fast enough through the protoplasm itself, undifferentiated into a conducting tissue. So far as the process of secretion by epithelium is concerned it is nothing at all new that the varied chemical reactions go on quite independently of nervous influence save that which regulates their supply of nutriment and that which controls the health of the protoplasm perhaps. This secretory process is next in importance to the contraction of muscle.

Thus the nervous system is gradually being forced into its proper place as a series of protoplasmic connections between parts of tissues other than itself. If a blow on the forehead causes the so-called loss of consciousness while an equal blow on the thigh would cause only pain, it is perhaps because the blow in the former place by its concussion disturbs the nutrition of and partly disorganizes a multitude of connecting fibers each bearing an important message, while the blow on the leg disturbs relatively few connections and so causes "loss of consciousness" only in the foot perhaps or up and down the leg. The nervous system connects, cöordinates, and adjusts the influences given out by the body's varied bioplasm. What it conducts it does not create (save in the sense discussed below)—it is at least as passive as it is active. Its business is largely conduction, and in doing this it gives the semblance of representing mind, partly because the association of ideas does perhaps depend at once upon it, and partly because the totality of a continuum, such as consciousness empirically is, demands a unification in the physical basis which unification it is certainly the function of the conducting nervous system to supply.

In the hierarchy of the tissues of a mammal or even of any vertebrate there are properly grades of value if we make active service the criterion of usefulness. From such a viewpoint the muscles are the master tissues, as indeed Foster calls them, for it is they which most serve the purposes of the individual. Besides, epithelium supplies the chemical products used in the organic machine, the nervous system cöordinates and adjusts "internal relations to external relations," the connective tissue binds the parts together structurally as does the nervous system

functionally, the bones support the body, etc. Formerly, not too long ago, the nervous system was supposed to dominate and control the whole, not only cöordinating but directing, the one tissue in command, nearest to the soul, immediate agent of the individual will.

Sometimes, as a mere matter of feeling, if you please, it seems absurd to try to define any sort of correspondence between the quite indescribable sentient experience and the body at all. Such an attempt seems a contradiction. The sort of localization which would call what we know as consciousness a product of the material process seems almost an indignity, an insult, and the impression is not premeditated but comes from an underlying awareness of the utter contrast of the two kinds of events when looked at casually, phenomenally and compared. Who can imagine even that the manifold of joy and sorrow, heaven and earth, past and present and to come of that we know and feel as *life* is the outcome, the accompaniment even, save as cause, of a small complex of physical currents or of vibrations in a brain? In some moments such a comparison seems sheer nonsense and absurdity, and in proportion to the smallness of the supposed accompanying mass, to the simplicity of the supposed accompanying process do the absurdity and the nonsense of such attempts impress the mind. Only on empirical evidence which cannot be denied is a psychophysical correspondence believable or even supposable at all.

The notion of Descartes that the soul had its seat in the pineal "gland" we hear now-a-days most often with a tendency to smile and yet this mode of thinking is not wholly outgrown as is evidenced by the present habit of locating consciousness in the nervous system, or even in the cortex of the brain. The absurdity of Descartes's supposition lies largely in the small size of the pineal gland—there is an inevitable feeling that the more definitely localized we consider consciousness the closer are we to the old outworn conception of a substantial soul. Yet the customary mode of viewing the matter is not much different, only that now the location has been extended to a brain cortex whose gray matter, even in man, weighs only about one-fortieth of one per cent. of what the body weighs and whose energy is in little if in any larger proportion. The action of the white matter is apparently as passive as that of any tissue in the body, so that

although quite unmeasured it is insignificant compared with that of the easily fatigued and injured gray matter in the nerve cells. But I for one practically am quite as unable to relate consciousness to the seventeen grams of gray matter in the human cortex as to the two or three grams of the pineal gland although theoretically the former sort of tissue has claims which the unnervous tissue of the conarium does not possess. Consciousness has the earmarks (they may be specious) of experiences we know as forceful or energetic, and it is not easy to see how so little energy in a supposed physical concomitant could stand for experiences at times so overwhelmingly significant and powerful. But if we realize that the nervous system is the more or less passive distributor of impulses and influences which originate without its area in other sorts of protoplasm, we shall grade its status more rightly. It is obvious that such is the case with those two large thirds of consciousness called feeling and will, for much research has shown conclusively that when feeling or willing is at all predominant in a period of consciousness, that practically every portion of the organism is concerned either actively or passively, in either case causing mechanical changes and the stimulation of a myriad sense organs in all the tissues. How greatly indeed feeling and will as aspects of mind predominate over cognition we all realize, so that one is bound to admit, if he realizes how perfect a unit the body is, that much of its protoplasm is concerned in nearly all of consciousness. Two theoretical aspects of mind, however, namely, "pure thought" and " pure sensation" are apparently relatively confined to the nervous system, without involving protoplasm outside it. The former of these, thought, we shall consider later on; the latter, pure sensation unaccompanied by elements of will or of emotion, is an abstraction as you all realize for descriptive purposes only, rarely or never experienced, for on the one hand the personal element is always present in greater or less degree, and on the other hand it is only practically under artificial conditions that one sense organ or even one sort of sense organ only is stimulated. But suppose a single pain-spot on the skin to be incited to action and that affective elements in the consequent consciousness are absent,—even then the stimulation would only serve to direct the attention, the focus of consciousness, without involving the great sensation-mass, for

the instant submerged but by no means abolished, as experiment readily shows.

Another problem germain to the question of how far the nervous system is adequate to represent consciousness in all sorts of animals depends on that rather intangible inquiry as to the extensity of consciousness, or rather perhaps, more precisely, as to its having or not having as its basis a sensation-mass, although the two problems are not theoretically quite the same. Is empirical consciousness like the electric currents darting singly here and there over an intricate system of telegraph wires, bearing with them meaning of various sorts and one always more conspicuous than the rest, or is it more like the mass of water making up a brook? In other words, has consciousness only time-relations and intensity or has it, is it, also something comparable in a figurative sense to mass or substance as well as time relation, intensity, and relative degrees of meaning? Is the varying focus of attention all, or is there behind and beneath the attention focus of the moment a sort of sensational substance like to a process? Is it phenomena or noumena? Only a meaningful direction of experience or a meaningful reality? I for one confess my entire inability to see it other than in the latter light, something real and essential because complex, index of all reality besides, and in a strictly figurative sense massive and substantial, made up mostly of sensations.

The simile of the brook has much of suggestiveness in it — the physical basis beneath it, the mass of on-flowing subconsciousness here and there disturbed by bodily conditions and determining largely the waves of attention on its surface, although these are susceptible readily also to stimuli from without —all this is a simile which is perhaps not only suggestive but to a degree descriptive. Consciousness has a measure of extensity. Even when the narrow ear drums receive the stimuli we notice it, for we distinguish a difference between the violin solo and the music of thirty playing in unison aside from that of intensity, while the difference between a burn on the finger-tip and the sensation coming from sinking into a bath of hot water is one of extensity almost wholly. No explanation of this difference is so close at hand as that based on a limited atomism in consciousness, but an atomism which the nervous system is quite inadequate to repre-

sent. Witness the astonishing phenomena of the sub-conscious mind; hypnosis, split-off consciousness or personality, automatism, and even of normal sleep and its varied puzzles in psychology. These have begun to reveal to us that consciousness has a basis and that in itself it is complex and multifold. Somehow the consciousness so exhibited has too much of something akin to extensity to be represented by the disparate currents along isolated neural fibers or in nerve cells, particles of neural protoplasm scarcely less minute and themselves made up mostly of fibrils. This complexity and this extensity argue for some sort of representation in the animal body outside of a system of only trivial mass and metabolism. Considered so, parallelism takes on a new sense, the details of which because of their minuteness and complexity may never be laid bare, no microscopes corresponding to those which explore the structure of the tissue cells being at hand to apply to conscious processes. In this way of seeing the theory of psychical atomism, even, so much derided formerly by some, acquires some sort of real significance, for beneath the discrete elements of consciousness is the cellular atomism, so to say, of the animal body.—both body and consciousness being empirical continua with more or less corresponding relations, each composed of parts not obvious to the unanalyzing view yet each an individual series of empirical events.

(To be Continued.)

PERIPHERAL OBLITERATING ARTERITIS AS A CAUSE OF TRIPLEGIA FOLLOWING HEMIPLEGIA.*

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We published in the *American Journal of the Medical Sciences* for June, 1905, a paper entitled "Peripheral Obliterating Arteritis as a cause of Triplegia following Hemiplegia, and of Paraplegia." In it we described a not very infrequent but much neglected form of palsy brought about by obliterating arteritis in the extremities affected. In this condition the leg on the non-paralyzed side becomes as powerless as the other. Both thighs become flexed on the abdomen and the calves on the thighs. There is marked rigidity and some muscular wasting, not a local atrophy picking out individual muscles but a general decrease in size. The skin is mottled, bluish-red and looks and feels thin. The final condition presents a clinical picture superficially resembling that seen when diffuse myelitis has occurred in hemiplegia, but there is not true palsy of the bladder and rectum. The condition must not be mistaken for the triplegia which sometimes occurs in syphilis as the result of multiple cerebral and spinal lesions, nor for double hemiplegia or hemiplegia plus monoplegia resulting from bilateral cerebral lesions. It slowly follows a single cerebral apoplexy, and is caused not by secondary involvement of the spinal cord but by disease of the arteries of the legs themselves. It occurs only in old people whose arteries are diseased throughout the entire body. There is no reason why it should not affect the arms as well as the legs but we have never seen such a case.

During the past winter another instance of this condition has come under our care and we now report it. The history of the case is as follows:

W. E., a white man, 80 years old, was admitted to Dr. Burr's service in the Philadelphia Hospital in March, 1904, and died in

*Read at the meeting of the American Neurological Association, June 4 and 5, 1906.

January, 1906. On admission to the hospital he was suffering from a partial right hemiplegia and aphasia. All that we could discover concerning the history of his illness was that he had had a stroke a few days previously. There was slight right-sided facial palsy. He could move the tongue well in all directions. There was paralysis of the right arm. Both legs were somewhat weak but weakness was greater on the right side. There was no disturbance of movement in the left arm or leg. The knee jerk on the right side was somewhat decreased, on the left it was normal. Stroking either sole caused slight flexion of all the toes followed on the right side by extension of the great toe. Sensibility was normal except in the right hand and forearm where sense of touch and pain was lost. The skin over the hands and feet was distinctly atrophic. The pupils were equal, reacted promptly to light, slowly to accommodation. All movements of the eyeballs were normal. The patient understood all that was said to him but could speak very little using only single words. He could recognize objects by sight and indicated that he knew the use of them but could rarely call their name. He could recognize objects by touch in the left hand but not in the right. He could understand short written or printed words. After some days his speech had improved a great deal and the anesthesia passed away. At the first examination there were no contractures of either leg or arm. By January, 1906, he was completely bedridden. Both thighs were flexed upon the abdomen, the knees closely pressed together, the calves flexed upon the thighs, and one foot crossed over the other. This condition slowly increased for months. It was not due to active spasm but apparently to shortening of the muscles. The right arm was also markedly contractured, the left not at all. The femoral pulse was palpable on either side, but the popliteal pulses could not be felt. Both legs were cold, the feet bluish and the toe-nails almost black. The right knee jerk could not be obtained. (This may have been either on account of the great rigidity or because of the distortion of the knee from old disease in it.) The left knee jerk was quite marked. Stroking the right sole caused drawing up of the whole leg without any movement of the toes. Stroking the left side caused no movement of any kind. There was slight Achilles jerk on the right side, none on the left. The biceps jerk was present on both sides. He had lost all power of articulation except to say "yes" and "no." He understood all simple verbal commands. Several days later he became stuporous and died shortly after.

At autopsy the brain appeared superficially normal. There was slight but not great atheroma of the arteries. There was slight enlargement of the ventricles. The popliteal arteries on both sides contained thrombi. The iliac arteries were much thickened. On cross section of the brain a small area of softening was

found in the left internal capsule and another in the island of Reil. The other lesions found at necropsy were chronic interstitial nephritis, and chronic myocarditis.

Histological examination of the paracentral lobules showed much congestion within the sections. The blood vessel walls were only moderately thickened and there was slight fibrous thickening of the pia. The Betz cells, though exceedingly pigmented, appeared normal by the thionin stain. The Mallory neuroglia stain showed moderate subpial glial hypertrophy.

A series of sections of the right internal capsule showed it to be normal by the hemalum-acid-fuchsin and Weigert hematoxylin stains. On the left side a series of sections showed sclerosis in the island of Reil and an area of degeneration of fibers in the posterior limb of the internal capsule when stained by the Weigert hematoxylin method. A series of sections of the cerebral peduncles showed the left to be distinctly smaller than the right and there was an area of degeneration occupying the middle third of the left crusta, but otherwise the sections appeared normal. In the pons serial sections showed degeneration of some of the bundles of fibers of the pyramidal tract on the left side when stained by the Weigert hematoxylin method, but no degeneration on the right side. There was complete degeneration of the left pyramidal tract in the medulla, but the right pyramidal tract stained very well by the Weigert method. The small intramedullary blood vessels were very prominent, caused rather by congestion than by thickening of their walls. There was a fairly well marked subpial gliosis but no round cell infiltration about the blood vessels nor in the pia. The cells of the nuclei of the cranial nerves stained by the thionin method appeared to be in fairly good condition.

Sections from the cervical region of the spinal cord stained by the Weigert method showed an area of degeneration in the right crossed pyramidal tract, and very slightly in the left direct pyramidal tract. The Marchi method revealed recent degeneration in the same location. There was no degeneration in other parts of the section. Sections stained by the hemalum-acid-fuchsin method showed a well marked thickening of the walls of the finer blood vessels making them appear unduly prominent. There was an overgrowth of glia about the blood vessels and also a moderate subpial gliosis. The nerve cells in the anterior horns stained by the thionin method while intensely pigmented were otherwise normal. Sections from the thoracic and lumbar regions of the spinal cord do not require separate description, as the same conditions were there present as in the cervical region, except that there was no degeneration in the left direct pyramidal tract below the level of the fourth thoracic segment.

To summarize: the findings in the central nervous system were

only those found in an ordinary case of right-sided hemiplegia, an area of sclerosis in the left cerebral hemisphere and the usual secondary degeneration of the pyramidal tract on that side. There was no evidence of a lesion on the opposite side. Other findings in the brain and spinal cord cannot be regarded as pathological in a man of the patient's age, as they are found in numerous persons in whom during life there are no nervous symptoms.

Sections of the popliteal nerves stained by the hemalum-acid-fuchsin method showed a marked increase of connective tissue between the nerve bundles, and to a less extent within them. There was no evidence of any inflammatory change, and by the Weigert hematoxylin method the nerve fibers stained well. There was no notable difference between the nerves from the two legs. When stained by the Marchi method there was seen, in longitudinal sections, black staining masses within the myelin not having, however, a typically degenerated appearance.

Sections of the left external iliac artery showed marked irregular thickening of the intima (endarteritis nodosa) and in one portion a hemorrhage into the intima. There was marked fibrosis of the media with calcification. Sections of the right external iliac artery showed even more thickening of the walls and constriction of the lumen than the left. The left popliteal artery was filled with a recent thrombus and its walls were greatly thickened, especially the intima. A small blood vessel seen in the same section, a branch of the popliteal, was almost completely occluded by intimal thickening. The right popliteal resembled the left.

Sections of the right tibialis posticus muscle stained by hematoxylin eosin showed a moderate increase in the connective tissue and of the connective tissue nuclei. The small intramuscular nerve fibers stained well by the Weigert hematoxylin method, as did also a nerve fiber seen within a muscle spindle. Sections of the left tibialis posticus stained by the hematoxylin-eosin appeared nearly normal. There was a slight increase in the amount of connective tissue between the muscle fasiculi without any increase of connective tissue nuclei. The muscles presented no apparent increase in sarcolemma nuclei. Stained by the Marchi method they showed distinct recent degeneration, a finely granular fatty change, present in numerous fibers throughout the sections.

In the nerves there was but a slight fibrosis not abnormal when the age of the patient is considered. The blood vessels showed intense arterio-sclerosis more marked in the smaller arteries where it produced obliteration in many. The muscles showed a distinct degenerative change by the Marchi method of staining.

It is of course well known that peripheral arterial disease may cause a palsy of the affected extremities. Many years ago Charcot showed that disease of the larger arterial trunks is the cause of intermittent claudication. It is well established that in many

instances the motor disability of old age (often in senile paraplegia for example) is frequently brought about not by cerebral or spinal cord disease but by lack of muscular nutrition on account of poor blood supply through the much thickened and diseased arteries and arterioles. This was the condition found in our patient. When there has been no preceding hemiplegia the picture is simple enough but when the two are combined the result resembles superficially a severe spastic myelitis.

Society Proceedings

AMERICAN NEUROLOGICAL ASSOCIATION.

Thirty-second Annual Meeting, Held at Boston, June 4 and 5, 1906.

The President, Dr. Henry R. Stedman, in the Chair.

(*Continued from page 779, vol. 33.*)

Myopathy of the Distal Type and Its Relation to the Neural Form of Muscular Atrophy (Charcot-Marie Tooth Type.)—By Dr. William G. Spiller. (See this journal, p. 14.)

Dr. B. Sachs said it was some time since this question had been brought up before this association, and naturally one's views undergo a change, yet he thought the change in views is not very great, and what changes there are can be very readily explained. First of all it is a question of adhering strictly to a type. He could never believe that all cases must present no variations from that type, however, they must in the main adhere to the form as laid down.

Now as far as the case Dr. Spiller presented is concerned, there was involvement of face and shoulder muscles, and of course that is a case Charcot himself would not have included under that special type, but it seemed to Dr. Sachs a case of that type. This gradual atrophy of the entire limb is so characteristic that it cannot be mistaken for another disease. Dr. Sachs thought it was a form of the Charcot-Marie-Tooth type, but an unusual form of the disease. With our present-day views of the neurone it seemed to him that the way to look at all these progressive muscular atrophies is to regard them as due to disease of various parts of the secondary neurone. It matters not whether in some cases it is purely spinal, or whether in some cases it is purely peripheral, clinically it is important to maintain the well-established types. There is no doubt that there are some causes at work producing the affection of the secondary motor neurone with the muscular attachments.

Dr. Gordon said Dr. Spiller's paper shows how difficult it is to classify these cases. We see them very frequently in hospital work.

As far as he could remember, in looking up the literature on the subject the earlier writers mentioned that in addition to the partial atrophy there was almost always involvement of the muscles of the hands, and that it was very difficult to divide sharply the classical types of the disease. Take for example the question of reaction of degeneration. Quite frequently we find R. D. in cases which, according to the general symptomatology, should be placed among the myopathies. Moreover, not infrequently we see cases which present some symptoms of one group of amyotrophy and some symptoms of another group.

Recently Raymond and Guillain published a paper in the *Presse Médicale* in which they called attention to an entirely new type of muscular atrophy, but close reading of the record shows elements of all types of muscular atrophy, so it is very difficult to define sometimes the cases of muscular atrophy.

Dr. Spiller said he believed we can recognize this distal type when the atrophy is confined to the distal parts, but as soon as it extends into the upper part of the limbs it becomes much more difficult of recognition. We may then say that it is *probably* a case of the Charcot-Marie type, but with the reservation that it may not be. Dr. Spiller was always doubtful

regarding the diagnosis in his case, because of the involvement of the upper portion of the limbs.

Peripheral Obliterating Arteritis as a Cause of Triplegia, Following Hemiplegia.—By Dr. C. W. Burr and Dr. C. D. Camp. (See this journal, p. 42.)

Dr. McCarthy thought this paper was something like Dr. Collins' paper on arteriosclerosis. He called attention to this peculiar gait in his paper. Erb recently has been calling attention to exhaustion of the upper extremities, not limiting it to the lower extremities. Many of these cases of exhaustion of the lower extremities are senile types of paraplegia.

The matter of arteriosclerosis of the extremities is one which should be carefully looked into. In his own autopsy work he made it a point to examine the nerves. It is surprising to find arteriosclerosis in the peripheral muscles. A thing which must appeal to everyone is the fact that arteriosclerosis is not a disease which affects the nervous system alone.

Brain Tumor Symptom, Complex with Termination in Recovery.—By Dr. Herman H. Hoppe. (To be published in this journal.)

Symptoms Following the Occlusion of the Posterior Inferior Cerebellar Artery.—By Dr. H. M. Thomas.

Case I. Man, aged fifty-eight. In hospital for pleurisy with effusion, and cardiac disturbance. Sudden vertigo, pain in right side of face, tendency to fall towards right. Vomited. Attack followed by slight ptosis of right eye, right pupil smaller than left, no sweating on right side of face, transient lateral nystagmus. Some difficulty in swallowing. Pain and temperature perception disturbed on right side of face, left trunk and limbs. Ataxia of right arm. Fidgety movements of both legs. Some improvement in focal symptoms. Sudden death twelve days after onset. Autopsy. Thrombosis of right vertebral and posterior inferior cerebellar artery. General arterial sclerosis, etc. Absence of marked microscopical lesions. Case II. Man, aged forty-eight. Recurrent attacks, during a month, of numbness in left side of face, vertigo, difficulty in speech, tendency to fall to left. Never recovered from last attack. Examination six days after the last attack. Vertigo from right to left, tendency to fall to left. Pupils practically equal. Crossed dissociated sensory disturbance. Left face, right arm, body and leg. Paralysis of left vocal cords. Sweating on right side of face, not on left. Conjunctival, nasal and pharyngeal reflexes diminished on left side. Slight ataxia in left arm, more marked in left leg. Condition improved to a certain point and remained stationary nearly two years.

The similarity of these cases is evident and the symptoms point squarely to a lesion of one side of the medulla. The author has collected about twenty-five cases in the literature in which the symptoms indicated a similar lesion, and in seven of these the autopsy showed a unilateral lesion in the dorsal aspect of the medulla. These are the cases of Dumenil, Leyden, Senator, van Oordt, Hun, Wallenberg and Babinski and Negeotte. The positions of the lesions in these cases corresponded for the most part accurately and involved more or less extensively the lateral part of the medulla dorsal to the olive and implicated the following important structures:

(1) The lateral part of the reticular formation.

(2) The descending root of the V with its nucleus.

(3) Gowers' ventro-lateral ascending tract.

(4) The direct cerebellar tract and other fibres which pass into the inferior cerebellar peduncle.

The lesion in each case was a necrotic process and Wernicke (1881), who made the anatomical examination of Senator's cases, suggested that it was due to an occlusion of the posterior-inferior cerebellar artery. Wernicke directed special attention to this artery in 1895, when he reported his cases clinically, and again in 1901 when he was able at the autopsy to demonstrate the thrombosis of the artery as well as the area of necrosis in the medulla almost exactly as he had predicted it. Other writers usually

assume an occlusion of this artery, but none has demonstrated it. In the author's first case, there was a thrombosis of the artery, but no softening was found in the medulla, due, probably, to the rich collateral circulation. The posterior-inferior cerebellar artery has been studied by Duret and Wallenberg.

Mr. M. T. Burrows, of the Johns Hopkins Medical School, at Dr. Thomas' suggestion, examined the museum preparations and made a careful injection of the artery, a drawing of which was shown. The anastomosis between it and the neighboring arteries is very free, but varies in different specimens. However, it appears that it very generally gives off to the lateral aspect of the medulla dorsal to the olive a number of little arteries which do not anastomose and are end arteries. This freedom of anastomosis explains why the area of necrosis is so limited after the occlusion of an artery with such a comparatively wide distribution, and indeed why it may not occur.

The clinical picture in these cases is remarkably constant and sharp-cut. The onset is sudden without loss of consciousness, and is ushered in by intense vertigo, with nausea and vomiting and a tendency to fall to one side. There is usually pain or some other sensory phenomenon on the side of the face toward which the patient falls; that is, the side of the lesion in the medulla. There may be a difficulty of speech and an inability to swallow, troublesome singultus and profuse sweating. Transient double vision and weakness of the face on the side of the lesion are frequently recorded. Upon physical examination, the most distinctive changes are in relation to sensation. There is very generally a crossed or alternate sensory paresis involving the face on the side of the lesion and the contralateral body and limbs. The sensory loss in the great majority of cases is to pain and temperature alone, although in four cases all the sense qualities were involved.

There is a marked tendency to fall toward the side of the lesion, and in most cases there has been a disturbance of co-ordination in the arm and leg on that side. The tongue is not generally affected, but the muscles of the soft palate and those used in the act of swallowing have been paralyzed in a number of cases. The muscles of mastication are normal and the facial muscles usually act well, although some slight asymmetry of the face has been noted.

The condition of the eyes in some of the cases has been most interesting, and it was to this point that Dr. Thomas wished to call particular attention. The pupil on the side of the lesion is smaller than its fellow, although it reacts equally well. There is a slight ptosis causing the narrowing of the visual aperture on the same side, and the retraction of the eyeball has also been observed; that is, the eye shows the condition characteristic of a paresis of the cervical sympathetic, as was pointed out by Hoffman, in his cases there was no abnormality in the secretion of sweat, and Hun's case is the only one in which such an abnormality was noted, where there was an increase of the secretion on the side opposite to the lesion. In both of the author's cases, there was a loss or a marked decrease in the secretion of sweat on the side of the lesion, and so completing the picture of paralysis of the cervical sympathetic. In his second, no abnormality of the eye was noted, but he thinks this may have been overlooked. In explanation of the symptoms, the crossed disassociated sensory disturbance is believed to be due to an involvement of the descending branch of the V and the tract of Gowers', or tracts which ascend with it. The vertigo and tendency to fall to the side of the lesion is explained by an involvement of fibres which enter the cerebellum by its anterior peduncle, or perhaps by a more direct disturbance of the vestibular nerve. The ataxy of the homolateral arm and leg should be brought into relation to an interruption of tracts which enter the restiform body; the direct cerebellar tract and fibres from the nuclei of the dorsal column, and perhaps others, although one must think of the tracts which descend from the cerebellum to the cord on the same side.

This paper in full will be published later.

The After-Care Movement, with Special Reference to Organization to Help the Hospital Physicians.—By Dr. Adolf Meyer. (To be published in this journal.)

Dr. Channing said he would like to endorse this work, and he hoped the public understands that it is important that it should stand behind it because it means so much to the people in every community.

Dr. Tomlinson said it may be of interest to know that the State Conference of Charities in Minnesota last year began an organized effort to establish after-care of the insane. This is at present a purely philanthropic undertaking, and will necessarily have to be for some time. They were trying to get physicians and philanthropic persons interested in the welfare of patients discharged from the hospitals, and are organizing and perfecting the machinery for carrying out this undertaking. The scheme, as outlined at the last meeting of the State Conference, is substantially the same as that described by Dr. Meyer, but too short a time has elapsed for us to report results.

Dr. Riggs said it is an experience common to all of us that after the patient's return home he finds great difficulty, as a rule, in adopting himself to the new environment. The unstable brain responds only too readily to every disturbing influence of his surroundings, and it is a matter of months before it receives its final poise.

The necessity for such aid as Dr. Meyer suggests for those convalescing from the psychoses has long been recognized by Dr. Riggs, and some six years ago he read a paper on this subject at the annual meeting of the Minnesota Association of Corrections and Charities at Faribault, advising essentially the same course. Nothing could be more unfortunate than that these persons should be thrown on the charity of the community, which is hostile toward them.

Dr. Meyer said that the Manhattan Hospital has one paid agent, and also some voluntary helpers, who visit the environment of patients who have been discharged. As a rule the relations have been remarkably satisfactory. The agent and helpers have been welcomed by the families they have visited, and they have helped many unfortunates to be started properly by the community.

Miss V. M. Clarke, Assistant Secretary of the State Charities Association, has forwarded some circulars. Unfortunately they did not get here for distribution at this session, but they will be distributed later.

The Insane Commission of the St. Louis City Jail, an Experiment in Civic Medicine.—By Dr. Sidney I. Schwab. (To be published in this journal.)

Dr. Channing thought this was a very admirable effort on the part of the gentlemen concerned. He would like to ask who employs them, and how many cases they have examined. He would also like to know how they can afford the time it must take to give such an examination as they are obliged to make.

Dr. Schwab, in answer to Dr. Channing's question, said they were not employed. Any judge has a right to say, "Will you come and help me with the mental question of this case?" He has the right to ask three physicians to examine an insane case and report. In the course of a year they had examined thirty or thirty-five cases. In some cases a long examination was not needed. They were not obliged to examine them all at a certain time. The cases waited in jail until they could be examined. The court takes time to get around, so they had time enough. In St. Louis the city jail physician is also the city physician, and he has a right to ask them to examine a case, or the prosecuting attorney's office would notify them.

A Case of Double Consciousness, Amnesic Type.—By Dr. Edward B. Angell. The subject of the sketch, a frank, open-hearted Englishman, was married on Christmas last, and within a few days had disappeared from

home. Some ten days later a somewhat incoherent letter from him to his wife located him, and he was brought home in a dazed and somewhat confused state of mind. This mental condition so closely resembled hypnotic state that it suggested a means of treatment. Under hypnosis, easily induced, the suggestion was made that on awakening his mind would be clear. Such was the result, and gradually he became alert, clear-minded and able to discriminate between the unreal, dreamy states of consciousness and the real facts of normal existence. For a dreamer of dreams his altered personality disclosed him to be. The tale he told first differed materially from a later one, while both became radically changed when normal consciousness had become established. He had assumed a name under which he was married, different from his own, insisted upon a genealogy, which was fictitious, claimed a college education and a service in South Africa, which he had not experienced; in fact, much of his memory registration was absolutely wrong. Careful investigation disproved most of his experiences. His tales were but creatures of an unstable imagination. His consciousness, when in the abnormal state, so akin to hysteria, registered fact and fiction alike; no discrimination being made between objective fact and subjective image. Such is the condition of the hypnotic. There is a subjective, unconscious falsification of memory, a species of amnesia, for the real events of an uneventful existence and the gap is filled with visions, with real unrealities, with plausible impossibilities. If the facts of such dual existence could be proven much that has been accepted as actual occurrences during the dispossession of the ego would be found illusions. They are but shadows of reality, misty radiographs which rapidly fade from the mind when Richard is himself again. In the present instance the honesty of purpose and frankness of mind are unquestionable. Whatever be the nature of this disturbance of mind it is real not fictitious. Memory is unstable not character.

A Case of Alteration of Personality.—By Dr. Richard Dewey. An alteration of consciousness of sixteen days' duration in a girl of twenty-three, not amounting to double personality, being incomplete and of rudimentary form. The symptom-complex embracing a history of migraine, hysteria and an eroto-mania of homo-sexual character. The altered consciousness being preceded by an evolution of systematized delusions (or pseudo-systematized delusions invented by the patient). The altered personality consisting in an assumption by the patient of the name and character of a person known to her and in authority over her. There being also a total change of handwriting during the sixteen days, the same being vertically upside down and horizontally reversed; *i. e.*, running from right to left.

A Case of Double Consciousness.—By Dr. Edward B. Angell.

Dr. Gordon said in the April number of the *American Journal of the Medical Sciences* will be found an article by him on double ego that deals with a case much like the cases described to-day. It was a case of a young man above the average intelligence. It happened several times that the manager of the place where he worked would give him an order to do a certain thing and he would not obey, while in other circumstances he would do it at once. Sometimes he would raise his hand to strike his wife, while at other times he was known as a most loving husband. When reminded of it, he would be surprised. The amnesia was complete. At the present time the patient presents this peculiar condition. By a process of mental analysis he has arrived at the conclusion that probably he is composed of two beings. There is No. 1 and No. 2, and No. 2 is independent of No. 1. He gave a number of instances in which he heard No. 2 ordering him to do a certain act.

Now the question is in all these cases: What is the nature of the disease which is responsible for this peculiar condition? Dr. Gordon believed the case which he reported to be one of epilepsy. Dr. Angell's case

he believed to be a case of epilepsy. The attacks of motor aphasia are very suggestive of epilepsy. Dr. Dewey's case, it seemed to Dr. Gordon, is a clear case of hysteria.

Dr. Angell said he appreciated the possibility of masked epilepsy as being the cause of this condition. However, careful investigation failed to reveal any indication of the motor symptoms of epilepsy, or even any symptoms suggestive of petit mal.

On the Clinical Differentiation of the Various Forms of Ambulatory Automatism. By Dr. J. W. Courtney. (Read by title.) The psychologic distinction between the so-called epileptic *fugues* and the hysteric ambulatory automatisms presents great difficulties. This paper is a brief discussion of the psychopathology of these two types of phenomena and an exposition of the writer's views upon the clinical symptoms which appear to mark the line of cleavage.

Report of a Case of Huntington's Chorea in which Four Members of the Family Were Affected.—By Dr. E. D. Fisher. (Read by title.) Father first affected at forty-five. First son (who committed suicide) at thirty. Second son at thirty-five. Third son at thirty.

Migraine and Hemianopsia.—By Dr. J. J. Thomas. (To be published in this journal.)

Dr. Dana remembered distinctly the case of a woman thirty-three years of age who had migraine and hemianopsia, and after one of the attacks the hemianopsia continued. She came to his clinic and the headaches grew better, but the hemianopsia continued. They had her examined by an oculist, but examinations were negative. Dr. Dana believed with the doctor that persistent hemianopsia is extremely rare, but even in young people there is a permanent hemianopsia which shows no organic lesion.

Four Cases of Landry's Paralysis.—By Dr. J. W. Putnam. (Read by title.) Case I. Male, aged thirty-four. Suddenly and without pain or loss of consciousness paralyzed in the upper extremities. In forty-eight hours the trunk muscles and muscles of lower extremities were paralyzed. Case II. Child, aged five. Total paralysis of all voluntary muscles in forty-eight hours. Death in fifty-five hours. Case III. Girl, aged sixteen. Onset rapid; total paralysis in forty-eight hours. Death in five days. Case IV. Male, aged fifty. Total paralysis in forty-eight hours, including muscles of the throat. Recovery. In all cases the reflexes were absent. There was bladder and rectum control. Sensation normal.

Peripheral Facial Diplegia and Palatal Involvement.—By Dr. George W. Jacoby. (Read by title. To be published in this journal.)

A Family Form of Progressive Muscular Atrophy Beginning Late in Life.—By Dr. William Browning. (Read by title.) A series of five cases, three personally observed, in which the myelogenic form of atrophy began at about fifty years of age and ran a fatal course in about two years.

A Study of the Sensory Symptoms of a Case of Pott's Disease of the Cervical Spine.—By Dr. Frank R. Fry. (Read by title. To be published in this journal.)

The Progression of the Sensory Symptoms in a Case of Pott's Disease of the Spine.—By Dr. Frank R. Fry. (Read by title.) The pressure on the cervical cord in this case was entirely from the front and very symmetrical and gradual, furnishing a good opportunity to study the relative times of appearance of the various motor and sensory phenomena. The paper is a partial record of these observations.

Multiple Miliary Metastatic Carcinomatosis of the Cerebrospinal Meninges.—By Dr. D. J. McCarthy. (Read by title.) A case of primary sarcoma of the liver with a secondary nodule in the pancreas; a local carcinoma in the lung, with cavity formation. Large numbers of small pinhead tumors scattered over the cerebral and spinal meninges. The lumbar spinal ganglia presented secondary carcinoma. There was also a small tumor on one of the dorsal roots. The gross appearance presented a picture resembling certain types of syphilitic and tuberculous meningitis. The

clinical course of the disease was that of cerebrospinal syphilis.

Traumatic, Hemilingual Atrophy.—By Dr. Smith Ely Jelliffe. (Read by title.) Hemilingual atrophy, due to differing causes, is not unknown. Ascoli as late as 1894 gave a résumé of 79 cases, since which time no less than 15 or 20 more have been reported. Atrophy from trauma, however, is much rarer, representing about fifteen per cent. of the entire number reported. The present case is one of considerable medico-legal interest, as it represents a hemilingual atrophy in a perfectly sound man following an accident. X-ray examination of the cervical vertebræ reveals a partial dislocation in the upper vertebræ with fracture of the spinous process of the third. The case is interesting as one of hemilingual atrophy associated with a lesion of the hypoglossal nerve at or about its exit at the anterior condyloid foramen. Instances of at least five or six similar cases are reported.

Stereoscopic Radiographs of the Skull.—By Dr. Edward B. Angell. (Read by title.) A suggestion in X-ray study of the brain.

Syphilis and Disseminated Sclerosis.—By Dr. B. Sachs. (Read by title.)

A Contribution to the Pathology of Refrigeration Facial Palsy (Bell's Palsy.)—By Dr. L. Pierce Clark. (Read by title.) The author has studied two cases of this type. Dr. Taylor performed the operation of facio-hypoglossal anastomosis for the relief of the condition.

Case I. was typical, complete refrigeration palsy of the right side in a woman aged 45. No family or personal history of rheumatism or syphilis. Reaction of degeneration complete. Anastomosis performed at end of four months. Piece of nerve excised for microscopical study showed incomplete degeneration, with slight evidences of reparative process.

Case II. was also typical and complete in a woman aged 42, without history of rheumatism, etc., but was of twelve years standing. There had been no attempt at regeneration, and there was complete atrophy of the muscles involved above the angle of the mouth, while below the latter the muscles were partly atrophied and contractured. The miscroscopic findings comprised complete degeneration of nerve fibers, without any attempt of regeneration. As far as is known, but three similar cases have been studied microscopically. Minkowski's case was believed by him to be one of Bell's refrigeration type, but the changes reported indicate chronic inflammation rather than primary degeneration. The case of Déjerine and Theohari was clearly one of infectious neuritis in a cancerous patient dead of pneumonia. Finally, Alexander's case was practically a duplicate of the preceding. It thus appears that these three cases differ only in degree and origin from the six recorded cases of neuritis, secondary to mastoiditis, the latter being much more severe and extensive.

The two cases of the author, probably the first to be studied microscopically, represent a purely refrigerative process; the action of cold as a trauma upon a nerve enclosed in rigid bony walls. The congestion arising from the cold brings about a secondary compression of all the nerve structures in the bony canals, and more or less primary degeneration in the periphery of the nerve is the consequence.

Circumscribed Hemorrhagic Cortical Encephalitis, with the Report of a Case in Which the Lesion Was Limited to the Motor Zone, the Chief Clinical Manifestation Being Jacksonian Epilepsy.—By Dr. Charles K. Mills. (Read by title.) Circumscribed cortical encephalitis studied clinically and its existence demonstrated by necropsy and microscopical examination, while not unique, is still so rare as to give value to the report of a new case. The patient was an aged woman, who, during a few days before her death, had a number of attacks of Jacksonian epilepsy of the arm-face type, consciousness being retained. She was paretic in the arm and face. No impairment of sensation was present. Necropsy and microscopical examination showed an area of hemorrhagic polio-encephalitis circumscribed to the cortex of the arm and face areas in front of the central fissure.

Lesions elsewhere in the brain were responsible for other symptoms than the Jacksonian spasm and monoplegias.

The deduction of Strümpell that certain of the infantile palsies of children were due to a circumscribed cerebral polio-encephalitis comparable to the poliomyelitis causing the well-known form of spinal infantile paralysis has been corroborated in a very few recorded cases with necropsy. A summary of the history and literature of the subject is given.

Two Cases of Spinal Cord Surgery.—By Dr. W. C. Krauss. (Read by title.) (1) Cyst of the spinal cord; operation, removal, incomplete recovery. (2) Compression of the spinal cord due to injury; operation; return of tendon reflexes. Incomplete recovery.

Herpetic Inflammation of the Geniculate Ganglion.—By Dr. J. Ramsay Hunt. (To be published in this journal.)

Dr. Dana said he had heard Dr. Hunt discuss these points before, and was familiar with the facts that he was to present, and he simply rose to express his conviction that Dr. Hunt had made out a clinical entity. The syndrome there is no question of, and the clinical type is almost absolutely established.

He thought we could not quite do away with the ideas of the anatomists who still look upon the geniculate ganglion as concerned with taste.

Dr. Mills said that Dr. Hunt's paper was one of the most valuable that had been presented at this meeting, and we shall look hereafter with interest for reports of cases of Hunt's disease.

Functional Simulation of Sensory Jacksonian Equivalent.—By Dr. Howell T. Pershing. (Read by title.) Paroxysms of sensation like that of faradic current beginning in left great toe and passing in regular order to the groin; in later paroxysms extending to left side of trunk; still later to shoulder, elbow and hand; then to lower face, causing metallic taste; finally to upper face. Sensation followed by tonic rigidity of corresponding muscles ending in closure of left eye. No clonic spasm. Right side never affected. Consciousness never impaired. Attacks many times daily for nine years. Under observation four years. Diagnosis of functional disease. Reasons.

Spastic Paraplegia Complicated with Pregnancy.—By Dr. John Punton. (Read by title.) Etiology. Report of a case with differential diagnosis. Post-mortem findings.

Report of an Extraordinary Case of Hysterical Mutism.—Dr. John K. Mitchell. (Read by title. To be published in this journal.)

Report of a Case of Brain Tumor with Autopsy.—By Dr. William M. Leszynsky. (Read by title.) The patient was a man of twenty-five years of age, who received a blow over the right temporal region two years before admission to the hospital. The symptoms were paroxysmal headache, vomiting, bilateral papillitis with blindness, anosmia, diminished hearing, and irritative symptoms affecting the right trigeminus. The motor tract was not involved. A large tumor (endothelioma) was found compressing the right temporal lobe.

Symptoms Simulating Brain Tumor Due to the Obliteration of the Longitudinal, Lateral and Occipital Sinuses. A Clinical Case.—By Dr. C. Eugene Riggs. (To be published in this journal.)

A Case of Anemia, with Peculiar Changes in the Nervous System.—By Dr. Charles S. Potts. (Read by title.) The patient was a man of fifty-nine. Nothing of note in the family or previous history. Ill seven years. Vertigo, general weakness, dyspnea, and pains through the body. Marked pallor of the skin and mucous membranes. Characteristic blood changes of pernicious anemia. Marked weakness of both legs, the right weaker than the left. Knee jerks and Achilles jerks increased on both sides. Babinski reflex present in the right; doubtful in the left. Death five days after admission to the hospital. In the spinal cord and cortex of the brain numerous minute cavities, not causing secondary degeneration and probably representing pathological changes. This form of degeneration without any

neurogliac proliferation unusual in anemic cords. Another unusual finding in anemic cords was degenerative changes in the cells of the anterior horns.

On Pathogenesis of Reflexes Apropos of a Case of Meningeal Tuberculoma of the Spinal Cord.—By Dr. Alfred Gordon. Colored girl of eighteen. Noticed six months prior to her admission to Philadelphia Hospital a weakness in the right arm. Two weeks later weakness appeared in the left arm and shoulder. A month later the lower extremities became weak. Loss of control of bladder and rectum accompanied the complete paralysis of the extremities. The paralysis was flaccid from beginning to the end. The patellar reflexes were abolished. No ankle clonus, no Babinski. There were multiple bedsores. At no time patient complained of pain. Typical signs of tubercular lungs were present. Post-mortem record shows: Tuberculosis of lungs and pleura. On the right side of the seventh cervical vertebra there is a tuberculous mass. A similar mass is found in the spinal canal, and on the antero-lateral surface of the dura between the fifth cervical to the second thoracic vertebræ. The dura was much thickened. Microscopical examination shows great destruction of the cord between the lower cervical and upper dorsal segments. Above this level degeneration affects the posterior columns, direct cerebellar and Gowers' tracts, as far as the first cervical segment. The lower dorsal and lumbar segments show degeneration in the crossed pyramidal tracts. The most interesting changes are found in the meninges; viz., a very pronounced tuberculous pachymeningitis invading both surfaces of the dura, the roots, the pia-arachnoid, and in some places also the tissue of the cord. Besides many interesting points concerning the absence of pain, the localization of the tumor, the extension of the pathological process, the details of microscopical findings, the present case is important from the standpoint of reflexes. According to some neurologists the lumbar cord possesses an autonomous center, so that the patellar reflexes must have for its existence the integrity of the lumbar cord. If this is suddenly severed by a section of cervical or thoracic segments the patellar reflex will disappear immediately after the shock, but will be re-established later, together with spasticity. According to others, not only the integrity of the lumbar cord, but also of certain cerebral centers is indispensable for obtaining a patellar reflex, so that a permanent flaccid palsy, with loss of patellar reflex, may ensue after a complete severance of the spinal cord. In cases with slow and progressive transverse destruction of the segment of the cord spastic paralysis exists from beginning to end. Raymond (*Rev. Neurol.*, 1902) and Senator (*Deut. Ztschr. f. Klin. Med.*, 1897) report cases of psammoma which produced complete destruction of a cervical segment and spastic paralysis existed during the entire course of the affection. In the present case flaccidity existed from the beginning. The duration of the affection was six months, a sufficient time for a descending lesion to produce a spastic paralysis either by suppressing the inhibitory influence of the brain or by stimulating the cells of the anterior cornua. Assuming the existence of an autonomous center in the lumbar cord, which in this case was totally severed from the encephalon, why did spasticity not develop? Is not this case an illustration of the dependency of the cord center upon cerebral centers? We must conclude that the reflex mechanism has multiple relations and the analysis of the accumulated observations tends more and more to show that the exclusivism of Charcot and Vulpian on one hand, of Bastian, Crocq, Collier and Buzzard on the other, cannot be accepted, but that Grasset's views are more in conformity with facts; namely, that spinal, basal and cortical regions contain centers for both reflexes and tonicity.

Periscope

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10. A Case with the Symptom-Complex of Cerebral Tumor, with Termination in Cure (Pseudo-Tumor Cerebri). Cases of Pseudo-Tumor Cerebri with Autopsy Findings, Terminating Fatally. NONNE.
11. Do the Parathyroid Glands Play a Rôle in Human Pathology? LUNDBORG.
12. Tetany, Pseudo-Tetany, and Their Mixed Forms in Hysteria. CURSCHMANN.
13. Two Cases of So-Called Chronic Anterior Poliomyelitis in Father and Son. BRUINING.
14. Primary Lateral Sclerosis (Spastic Spinal Paralysis). STRÜMPELL.
15. Obituary of Carl Weigert. LICHTHEIM.

10. *Cerebral Tumor.*—A man of 32 had a slowly developing weakness in the right arm and right leg, and later, in the right half of the face. There was also headache and nausea, and spasticity of the right side. In both eyegrounds there was beginning optic neuritis, and spinal puncture showed increase of pressure in the cerebrospinal fluid. No treatment was employed, and the patient gradually improved, and eventually recovered entirely. The second case, a man of 38, had vertigo, a sensation of pressure in the frontal region, and some paresthesia in the right side of the face and righ arm. This was later associated with weakness in the right upper extremity. This increased and there was papillitis, then slow improvement, and finally complete recovery. Although there was no evidence of syphilis, the patient had been given innunctions of mercury. Third case, a woman of 43 developed diffuse, steadily increasing headache with vomiting and progressive drowsiness. There was slow pulse, bilateral choked disc, tenderness over the head, weakness in the left facial region and a cerebellar gait. She slowly began to improve, and in ten weeks she was normal. Fourth case, a woman of 44, had intense headache, and occasionally vomiting and attacks of vertigo. There was distinct choked disc, a cerebellar gait and station, and excessive intracranial pressure as revealed by spinal puncture. She was given mixed treatment, although no signs of syphilis existed, and five weeks later had entirely recovered. In fifth case, a woman of 47, had had a transient attack of blindness, followed by attacks of vertigo and then intense headache, especially in the occipital region. There were spasms in the right side of the face, weakness and uncertainty in the right arm, bilateral choked disc, right facial paresis, no pathological alterations in the reflexes, but a cerebellar gait. Later there was vomiting and apathy. No signs of syphilis were present, but syphilitic treatment was employed. The patient rapidly improved, and a year later was well, excepting that there was beginning optic atrophy in both eyes. The sixth case, a woman of twenty, had had epilepsy for four months, always beginning in the right arm. The eyes were at first normal, but later developed choked disc. There was paresis of the right facial, increase of the tendon reflexes on the right side, but the spinal fluid was normal in character and pressure. The diagnosis was uncertain, but was made as probably tumor in the region of the left facial centre. The patient had no further attacks, and rapidly returned to a normal state. The seventh case, a man of twenty, had paresis of the left facial nerve; paresis of the left upper extremity, increase in the tendon and priosteal reflexes of

the left extremity, and some slight sensory disturbance. There was a marked papillitis on both sides. Spinal puncture showed a marked increase in intracranial pressure. There was vomiting, severe headache, and rapid increase of the paresis to complete paralysis. Upon mixed treatment the patient rapidly improved, and three years later was entirely well. Nonne is disinclined to make a diagnosis of syphilitic new growth.

11. *The Parathyroids in Human Pathology.*—Lundborg, of Upsala, who has done such excellent work on convulsive disorders, here propounds the proposition that the parathyroids have a definite relation to human pathology, and particularly to the convulsive disorders, tetany, myoclonus, myotonia, myatonia, myasthenia and paralysis agitans. Studies on thyroid insufficiency have been followed by others on parathyroid insufficiency, an excellent summary of which latter is here presented. In Lundborg's opinion a heightened function of the gland brings about the condition known as myotonia congenita. Myotonia and tetany have been observed frequently as more or less accompanying affections, and the author includes this affection in his parathyroid intoxications. Occupying a close intermediate relation Lundborg places myoclonus, concerning which affection he has devoted some time. During his studies in myoclonus, being struck by the frequent appearance of paralysis agitans among the myoclonus cases, he was led to place this latter disease within the category of deficient glandular sufficiency, being described by him as a chronic, progressive, hypoparathyroidism. He constructs an interesting diagram showing how the affections of the thyroid, both as to diminished and to increased function, have their exact analogies in parathyroid disease. Thus on the side of insufficiency myxedema is made analogous to paralysis agitans, stuporous thyroid states with tetany, myoclonus has no thyroid analogue, hypothryroid chronic has myotonia from thyroid poisoning. As for hyper functioning, various psychotic states are paralleled by myatonia periodica, and Basedow's disease has its analogue in myasthenic paralysis.

12. *Tetany, Pseudo-Tetany, and Their Mixed Forms in Hysteria.*—Curschmann calls attention to the likelihood of confusing tetany with hysteria at times, since these patients with pseudo-tetany may show nearly all of the objective signs of tetany itself. He cites a number of histories. In the first patient true tetany was complicated by hysteria. The patient had a hemitetany of the left arm and side of face. There was also an otitis media and intenia. A radical operation had been performed. A second patient showed tetany with hysterical attacks. Chronic enteritis was also present. The histories of three patients with pseudotetany are then given, with a summary of the literature of other similar cases. The author concludes that practically all of the symptoms of tetany can be found in pseudotetany with the exception of the increased muscular activity to electrical stimulation (Erb's sign). This sign constitutes for Curschmann the chief differential diagnostic sign between true tetany and pseudotetany.

13. *Poliomyelitis Anterior Chronica in Father and Son.*—Bruining gives the history of the coincidental development of this disease in a father aged 45, and son 23 years of age. No etiological factor is found, and the occurrence is regarded as fortuitous. A useful discussion of the differential diagnosis of the atrophies, syringomyelia and neuritides is to be found in the article.

14. *Primary Sclerosis of the Lateral Columns.*—Strümpell returns to the consideration of this condition and expresses the opinion that a primary lateral sclerosis in the sense originally described by Charcot and later studied by Erb, is a very definite disease process notwithstanding the many recent expressions of doubt from many sides as to its true nature. He gives the complete histories with autopsy findings of three patients who had been under observation many years. It is an uncommon disease, yet not distinctly rare. The symptoms on which most weight should be laid are muscle rigidity, in the sense of hypertonia of the muscles, increase in

tendon reflexes. These are the first and most important symptoms, which though related as to their pathogenesis are not identical.

Whether a paresis can occur in the primary sclerosis under discussion is in Strümpell's mind a question. The tibialis phenomenon has been present in all of his pure cases. The Babinski reflex is not constant, but nearly so. From the etiological standpoint Strümpell groups his cases into five sets. (1) A group in which family and hereditary influences speak for some endogenous process. (2) The group of family infantile spastic paralysis. (3) A group beginning at an advanced age with a rapid progress, showing a tendency to diffusion of the process and allying itself somewhat to anyotrophic lateral sclerosis. (4) Exogenous cases, with syphilitic history. (5) A hypothetical group occurring in women as a result of pregnancy and delivery. SAILER (Philadelphia).

American Journal of Insanity

(Vol. LXII., No. 3, 1906.)

1. Melancholia. The Psychical Expression of Organic Fear. WHERRY.
2. Gynecological Surgery in the Manhattan State Hospital West. BROUN.
3. Surgery for the Relief of Insane Conditions. WITTE.
4. Observations on Some Recent Surgical Cases in the Manhattan State Hospital East. KNAPP.
5. The Making of Clinical Records. FARRAR.

1. *Melancholia.*—Reasoning that the emotions proceed from visceral sensations and do not originate in the brain, the author considers melancholia, a disease of the emotional sphere, to arise from abnormal visceral conditions. He lays down the following propositions: 1. That there are organic as well as ethical emotions. 2. That the relation of the body to the mind is that of master to servant. 3. That the influence of the brain has been overestimated in the production of abnormal mental states. 4. That the organic emotion of fear has its origin in visceral conditions. 5. That organic fear is a primitive instinct and necessary to the preservation of the individual. 6. That abnormal organic fear is the basis of melancholia. 7. That melancholia is but the expression of abnormal visceral conditions. 1. The organic emotions are primary, dating back to the time of unicellular existence, being an attribute of the cell itself. They may be called instinctive and have to do with self-preservation and reproduction and are primarily desire and fear. In the higher forms of life they are tempered by experience, through which has been evolved the faculties of reason and judgment. 2. The individual must, he thinks, be considered as a whole, and instead of the mind dominating the body, the mental content is made up very largely of the organic sensations, especially being influenced by those arising from the viscera. The consciousness of self which is made up of these organic sensations can hardly be separated from the general mental state at any time. 3. Taking as an example the action of alcohol or of morphin, the author thinks it incorrect to assume that the symptoms observed after overdoses of these drugs are due to their action upon the brain especially, but holds that their effect upon other cells, as those of the liver, etc., with resulting bodily sensations, are equally responsible. 4. In support of this proposition, he alludes to the depressing effect of disorders of the stomach, the intense apprehension of heart disease, especially of angina pectoris, etc. The author believes that the feeling of fear always arises from conditions of the thoracic or abdominal viscera. 5. Organic fear has to do with self-preservation, and is primitive, being traceable back to the cell itself. "A continuous feeling of fear can only come from abnormal bodily cells which are reacting to a disease whose toxicity is neither so overwhelming as to prohibit the reception of sensory stimuli by the brain, nor so acute as to awaken the attention of consciousness to the fact of its presence." 6. Analyzing the symptoms of melancholia, the author finds that they all

cluster around the feeling of fear. In pure melancholia there is no intellectual impairment. Such impairment when present points to the incident of dementia which he holds is due to concomitant brain involvement, and cannot correctly be considered as a result of melancholia. 7. That melancholia is an expression of abnormal visceral conditions he thinks is shown by the fact that it has always been known as the most, if not the only, curable of mental disorders. Empirically it was found that to effect a cure, treatment had to be addressed to the visceral disorder or to abnormal nutritional state nearly always present. On the other hand, in the presence of disease leading to definite changes in the brain, we in the main stand powerless.

2. *Gynecological Surgery.*—The author proceeding upon the very reasonable assumption that the same indications for surgical intervention exist in the insane as in the sane; namely, that conditions interfering with the patient's comfort or health, or threatening life, when possible should be relieved by operation, has operated upon 242 women patients in the Manhattan State Hospital West. Among these there were 62 abdominal sections, 51 operations for displaced uterus and 129 minor plastic operations. His results were: One hundred and twelve patients markedly benefitted physically, and 107 patients notably improved. Five patients died, but in only two of these could death be directly attributed to the operation, 138 of these patients still remain in the institution, 104 have been discharged. Of the latter number, 43 are recorded as having recovered mentally, and in twenty of these recovery was materially hastened as a result of the physical improvement following the operation. In no case did the author attempt the removal of healthy ovaries and tubes with a view of curing the insanity. On the contrary, he strongly opposes this procedure. From his own experience, and from the statistics of other operators, he thinks that insanity rarely occurs as a direct result of an operation legitimately called for, though he is apparently of the opinion that the removal of healthy pelvic organs on whatever ground is likely to have a disastrous effect upon the nervous system. His various operations with results are exposed in tabular form.

3. *Surgery for the Relief of Insane Conditions.*—In the estimation of the value of surgical procedures in the insane, the author thinks that most reporters have laid too little stress upon the element of time on the one hand, and on the effect of the operative shock upon the innervation and metabolism on the other. Operations for the relief of insanity, group themselves mainly under those practiced upon the head, and those upon the female pelvic organs. In the former class of cases, the trouble is unfortunately generally of too long standing for much likelihood of permanent relief. Early operations offer distinctly more hope. The author illustrates this by the histories of some cases, in most of which the outcome was disappointing. He formulates his opinion as to the prospects of relief of insanity by operations upon the female pelvic organs as follows: 1. Relief of the mental condition is not to be expected from operative interference where there is no actual disease of the pelvic organs. 2. In pelvic diseases complicating insanity often much good in improving general health and comfort, and thereby aiding mental restoration may be accomplished by the less heroic measures. 3. In the insane, as in the sane, pelvic disease which is interfering with health or comfort, and is remediable by surgical intervention should be treated by such measures as are appropriate. 4. Surgical measures intended to annul procreative power are indicated and justified in certain types of insanity.

4. *Some Recent Surgical Cases.*—Note on certain operations performed at the above institution mainly by Drs. Guiteras and Lusk. Of interest from a psychiatric point of view is the following: A man forty-two years old having melancholia of four years duration (which had followed an operation for appendicitis, with subsequent peritonitis), was noticed to be failing mentally and to have some loss of power in the muscles of the

throat. He next became excited and had epileptiform convulsions, during one of which he fell striking his head in the left frontal region. Remaining unconscious after the injury for two days and presenting symptoms of cerebral pressure, he was trephined over the seat of injury. No fracture was found, but the dura buldging through the opening was incised, and six drachms of fluid evacuated. The patient gradually improved after the operation, though twelve days later he had another convulsion (attributed by the author to an external cause). After some weeks he brightened up, and eventually made a complete recovery both mentally and physically.

5. *Clinical Records.*—The author introduces the new department of "Clinical Psychiatry" in this journal with some suggestions as to the methods of making and keeping the clinical records of the insane, and gives a provisional schema. (ALLEN) Trenton.

Journal de Neurologie

(Vol. XI., No. 5, 1906.)

1. Spasmodic Laughing and Weeping. DEROUBAIX.
2. The Patellar Reflex is Independent of the Surface of the Percussor. COSTEX.
3. The Pathogeny of Hematoma Auris. DARCANNE.
4. Kernig's Sign in General Paresis. DARCANNE.

1. *Spasmodic Laughing and Weeping.*—The majority of authors have located the lesion causing the above symptoms in the basal ganglia, respectively in the optic thalamus, and in the corpus striatum. The author reports two cases in which it was observed, and in one of these an autopsy was obtained. In both cases there had been an apoplectic attack. The first patient had general feebleness of the muscles of the face, tongue and limbs, with mental impairment, which improved up to a certain point, no definite speech trouble, but accesses of spasmodic weeping, not due to a depressive affect, but which the patient himself declared to be purely involuntary. A sort of spasmodic laugh could be provoked, but it was immediately followed by weeping. The second patient, a man thirty-eight years old, after a "stroke" presented left hemiplegia affecting especially the arm, no aphasia or dysarthria, spasmodic laughter followed by weeping, and accompanied by associated movements in the paralyzed left arm. Death from another stroke. The autopsy showed atheroma of the cerebral arteries, with a large area of softening, which had destroyed the lenticular nucleus, the anterior limb of the internal capsule, and all the white matter of the frontal lobe, and of the central region, to the cortex, on the right side. The optic thalamus, genu and posterior limb of the internal capsule were not affected, and on the left side of the brain there was no macroscopic lesion. The author draws the following conclusions: 1. The function of emotive expression probably passes outside of the pyramidal fibres, like the functions of coördination and tonus. 2. The optic thalamus is the seat of the automatic movements of emotive expression (especially of laughing and crying). 3. Laughing and crying are under the control of the cortex. 4. The cortico-thalamic fibres pass in front of the knee of the internal capsule, probably by the lenticular nucleus. 5. A lesion of the cortico-thalamic fibres in question suffices to provoke the exaggeration of the emotive psycho-reflex; that is to say, spasmodic laughing and crying, either isolated or associated. The simplest explanation consists in admitting the presence of crossed fibres in the thalamo-facial fasciculus. 6. Spasmodic laughing and crying are not in relation with the "conscious emotive constellation" of the patient. 7. The substitution of crying for laughing in the same emotive convulsion tends to prove that the thalamo-bulbar conduction of laughing and crying is effected through a single fasciculus whose fibers govern antagonistic muscular groups. 8. There appears to be a clinical and anatomo-pathological relation between the pseudo-bulbar, and the Parkinsonian syndrome.

9. The corpus striatum has a relation to the innervation of the sphincters.

2. *The Patellar Reflex.*—The author making some experiments with his "reflexometer" has found that the force of the patellar reflex is independant of the size of the percussion hammer, provided it is kept small enough to impinge upon the tendon only. He also reports some experiments upon the relation between the period of latency of the muscular contraction and the intensity of the excitation. For these the myograph in connection with his reflexometer was used. His subjects were mainly precocious dements. He finds that the period of latency varies inversely with the intensity of the stimulus, this intensity depending upon the force of the percussion shock and the muscular tonus.

3. *Hematoma Auris.*—The author, after careful bacteriological examination of the blood from hematomata in five general paretics failed to find bacteria in any case. He concludes that hematoma is always of traumatic origin. He points out its infrequency in the women's wards where roughness of attendants is less apt to manifest itself in boxing the ears.

4. *Kernig's Sign in General Paresis.*—In twenty-six female paretics examined by the author, in ten Kernig's sign was present. Of these ten, two were in the second and eight in the final stage of the disease. He also found it in four males. Of the ten women exhibiting it, six had exaggerated reflexes, six ankle chlonus, and five Babinski's symptom. He thinks that Kernig's sign may have considerable importance, especially in doubtful cases. Prognostically it would indicate progressive involvement of the spinal cord.

(Vol. XI., No. 6, 1906.)

1. Second Note on the False Reminiscence. FÉRE.
2. Infantile Paraplegia, Insidious Onset, Stationary Condition, Then Exaggeration. BOUCHAUD.

1. *False Reminiscence.*—Note on two cases of neurasthenia in which, among other troubles, there were false reminiscences; *i. e.*, the idea of having previously seen persons or things which really presented themselves for the first time. Cure under treatment of the underlying condition.

2. *Infantile Paraplegia.*—A child at the age of one year had a severe infection (presumably pneumonia), which left her very weak, and at twenty-two months of age, she could not walk except to make a few steps when supported, her legs bending under her. Her condition remained stationary until the age of six years, when the legs gradually grew weaker until she could no longer retain the erect position. The author examining her about two years later, found a flaccid paralysis of the legs, with some atrophy and altered electrical reactions, loss of reflexes, no sensory trouble or bladder disturbance, arms normal, mental condition good, and no growth anomalies. He is inclined, by process of exclusion, to regard the case as one of poliomyelitis with a recurrence, and discusses the various theories put forward to account for such recurrences. Apparently he leans toward the idea of a reinfection.

(Vol. XI., No. 7, 1906.)

1. Atypical Forms of Dementia Præcox. CROCQ.
2. Location in the Cranial and Spinal Nerve Nuclei, in Man and in the Dog. PARHON and NADEJDE.

1. *Dementia Præcox.*—The author considers a class of cases which, while evidently examples of dementia præcox, seem to represent a milder form of this disease and are capable of recovery to such an extent as to be able to again fill a place in the world, providing that their duties are mainly of an automatic character, and do not require much judgment. A later remission with ultimate termination in dementia is, however, to be feared. These cases coincide in the main with the heboidophrenia of Becker and Kahlbaum. The author recognizes several forms, the first consisting chiefly of mental deterioration, with loss of affective sentiments,

while memory and inferior acquirements may be retained, but without excitement, depression or delusional ideas. A more severe condition consists of the above symptoms with vague delusions, while in third form the delusions are more marked, as is also the tendency to dementia. The last variety, especially, tends after apparent recovery to recur and eventually to pass into the typical picture of dementia præcox, though for years such patients may play a certain and even conspicuous rôle in the world though recognized as "peculiar." He gives the histories of two typical cases.

2. *Location in the Cranial and Spinal Nerve Nuclei.*—Since the question of the muscular localization in the spinal and bulbar nuclei is by no means settled, the authors urge that no opportunity to examine a case likely to shed light on this subject be neglected. Such cases are likely to be found among the inmates of alms houses and asylums; for instance, such persons as have lost a limb, or segment of one, have diseases destroying one or more muscles, or poliomyelitis with atrophy sharply localized in one or more muscles. They give here the results of examination in the case of a man in whom a cancer had destroyed nearly all the tongue, the muscles of the floor of the mouth, the anterior belly of the digastric on both sides, while on the left, the sterno-mastoid, the posterior belly of the digastric, the stylo-hyoid and the platysma were affected. From the degeneration of the cells found in the upper cervical, the bulbar and the pontine nuclei, they make the following localizations. The sterno-mastoid in the central group of cells of the first and second cervical segments.

The muscles at the base of the tongue, in the external group of the upper part of the hypoglossal nucleus; the genio-hyoid in the anterior group of the same region; the posterior belly of the digastric, the stylo-hyoid, and stylo-glossus in the second ventral group of the facial nucleus, the mylo-hyoid, in the lower part of the motor nucleus of the trigeminus (probably the anterior belly of the digastric is also innervated from this nucleus). It has been asserted that the mylohyoid is innervated from the facial nucleus, but the results obtained in this case, and also those from some further experimental work on dogs would indicate that it is represented in the trigeminus nucleus. These dog experiments also would seem to show that the masseter, and probably the pterygoid is represented in the posterior group of the motor trigeminus nucleus.

(Vol. XI., No. 8, 1906.)

1. Physical Troubles in States of Stupor. SOUKHANOFF and PETROFF.
2. Penal Reform from the Anthropological and Psychiatric Point of View. VAN HAMEL.

1. *Physical Troubles in States of Stupor.*—Description of the case of a man of thirty-eight years of age, who on two occasions, while a political prisoner, had attacks of mental disturbance characterized by hallucinations, illusions and delusions of persecution. Recovery from the first attack, but the second has persisted. Attempt at suicide by biting the radial arteries.

The physical troubles consist in a peculiar retardation of respiration, the number of respiratory movements varying from six to eight per minute, rising to ten upon excitement, weakness of the pulse, and cyanosis and edema of the lower limbs even when kept in bed, and a leg ulcer possibly of trophic origin. Towards the latter part of the time there was slight febrile movement and chronic inflammatory trouble of the lungs was suspected, though on account of the shallow breathing of the patient, satisfactory physical examination was impossible. The case is considered as one of chronic confusion, or as probably belonging to the dementia præcox group.

2. *Penal Reform.*—A report made at the recent Lisbon Congress of Medecine, which exposes the author's views as to the aims which should guide the administration of penal reform. Summarized they are as follows: Penal reform should have as its aim, the diminution of crime.

This is an end at once practical and realistic, and should not have as its point of departure any dogmatic religious, philosophical or ethical system. The object should be to decrease the number of criminals, not to increase the number of those punished. The combat against crime should be regulated by scientific study of its causes—criminal etiology. These causes are general and personal. The general causes are mainly social, the personal causes, anthropological. Penal justice should dispense measures of social education to corrigible, measures of social protection to incorrigible criminals. The distinction between young and adult criminals is of less value than one based upon psychological state, social needs, and to what extent they are a danger to the community. Much latitude should be left to the judge in the choice between a number of educational and protective measures. In order to assist him to make a proper choice, a psychiatric service should be attached to each tribunal and to each correctional establishment. A great stumbling block is the divergence between legal and medical views as to what constitutes criminal responsibility. The legal division of criminals into the two classes of responsible and irresponsible is fallacious and should be abolished. In the study of the individual there should be noted especially psychical state, dangerousness and whether probably corrigible or incorrigible. The testimony of the trained physician should form a part of each criminal procedure, though examination by him is not necessarily indicated in every case. Two elements should decide whether his services are needed or not: 1. The nature of the act of the individual, and (2) the nature of the correctional measures whose application is under consideration by the tribunal.

(ALLEN) Trenton.

Archiv für Psychiatrie und Nervenkrankheiten

(Vol. 40, Part 3.)

25. Frequency and Causes of Mental Disorder in the German Navy, with a Comparison of Army Statistics. PODESTÀ.
26. Globus and Aura. MAX BUCH.
27. Disease of the Ventral Horns in Tabes Dorsalis. (Conclusion.) MICHAEL LAPINSKY.
28. Acute Juvenile Dementia. M. FUHRMANN
29. Three Cases of Organic Disease of the Brain with Psychosis. OTTO KERN.
30. Self-Accusing Insane. E. MEYER.
31. Spinal Cord Lesions and Progressive Paralysis and Their Significance in the Explanation of Argyll-Robertson Pupil. KINICHI NAKA
32. Paralysis and Trauma. C. GIESELER
33. Physiological and Pathological Sleepiness. HANS GUDDEN.

25. *Mental Disease in Army and Navy.*—Podestà, from the point of view of a physician in the navy, calls attention to the increasing amount of mental disorder in the army and draws comparisons between this situation and the occurrence of like disorders in the navy. The discussion of the various types of disorder met with is extremely detailed and based on much careful statistical study. He finds that in the later terms of service general paralysis, epileptic insanity, paranoia and mania are frequent in the army, but less so than in the navy, where especially paranoia, general paralysis and alcoholic insanity are particularly pronounced. Neurasthenic and hysterical conditions are likewise frequent in the navy, together with a considerable amount of general weak-mindedness. The greater amount of such difficulty in the navy is attributed to the peculiarity of the service. A large proportion of the cases occur in the later years of service among the officers. Service in the tropics is also held in a measure responsible. As a study of secondary causes the article is of much interest.

26. *Globus and Aura.*—This paper is a comprehensive attempt to explain on an anatomical and physiological basis the well recognized symp-

tom of globus, often, as the writer maintains, erroneously termed globus hystericus. Buch is of the opinion that the popular conception that globus is a pathognomonic symptom of hysteria is wrong, and that it very frequently occurs in conditions quite distinct from this neurosis. Globus hystericus, he thinks, is simply neuralgia of the sympathetic, and occurs only in disorder of that nervous mechanism. That it occurs with hysteria does not justify its acceptance as a symptom of that disorder. Among twenty cases examined by him not one showed evidence of hysteria. In two other cases in which hysteria was a possibility there was no globus. As a basis for globus he finds chlorotic and anemic conditions conspicuous. In general, the symptom is due not to a central nervous disturbance, but rather to a local hyperexcitability of the sympathetic. Treatment by iron and arsenic is successful.

27. *Ventral Horn Disease in Tabes.*—Lapinsky reaches the following general conclusions in his exhaustive paper: That the statement of Dejerine and his pupils that the atrophies and paralyses of tabes depend upon peripheral neuritic alterations primarily has a limited application; that cases occur in which a primary degeneration of the ventral horn cells takes place; that in those cases in which the muscular atrophy depends upon disease of the ventral horn cells the disease develops sub-acutely and is not inflammatory; that the affection of the ventral horns appears in two forms, either diffuse or in the form of localized areas of degeneration; that in the localized form of disease the affection bears no resemblance to poliomyelitis because of the lack of evidence of inflammation; that both localized and diffuse processes may coexist in the same case; that alterations of vessels, degenerated collaterals of dorsal roots, alterations in the pyramidal tract, and atrophied ventral horn cells stand in relation to each other; that the various pathological alterations determine the character of the disturbance in the horn, and finally, that the paralyses, pareses, atrophies and changes in the muscular system of spinal origin, are to be regarded as the result of a localized disorder of the spinal cord. An extensive bibliography follows this article.

28. *Acute Juvenile Dementia.*—After commenting on Kraepelin's service to psychiatry in bringing into prominence the symptom group of dementia præcox, Fuhrmann proceeds to report three cases which he regards as belonging to the group of paranoid dementia præcox. The cases are reported at great length, and lead, in general, to the following conclusions: An acute psychosis going on to dementia was observed in three young adults, in all of whom there was a family history of alcohol in the father. The initial stage in one instance resembled a delirium tremens, in the others, an acute alcoholic hallucinosis. The interesting question is raised as to whether such a psychosis in the descendants could result from excessive alcohol indulgence in the parents, and the opinion is ventured that the alcoholism of the father gave the psychoses of the sons their peculiar alcoholic stamp, and further that there is reason to suppose that the psychoses were directly caused by the alcoholism of the fathers. The writer is not content with the term dementia præcox for these cases, and gives the name acute juvenile dementia, with the possible qualification on the basis of a degenerative alcoholic taint.

29. *Brain Disease and Psychoses.*—Kern reports three cases of structural disease of the brain associated with a psychosis. The first case during life gave the general picture of catatonia; a preliminary melancholic stage, sudden condition of agitation, later impulsive acts. The autopsy showed a brain abscess in the left cerebrellar hemisphere. The second case showed at the age of twenty-nine certain mental alterations associated with sleeplessness, followed by apathy, abulia, refusal of food, and suicidal ideas, together with headache and twitchings of the extremities. She was later depressed, but suicide was never a prominent feature. The autopsy showed a large glioma of the left parietal lobe and central ganglia. The third case developed an acute psychosis at the age of twenty-eight, with con-

fusion and occasional violence, associated with excitement and motor unrest, which went on to a marked mental alteration. The autopsy showed a rather small sarcoma in the white matter of the right frontal lobe, and a large hemorrhage in the right lateral ventricle. A discussion of the relation of mental distubances to new growths and remarks on operative interference conclude the article.

30. *Self-Accusation.*—Meyer discusses that type of mental disorder, prevalent in the sixteenth and seventeenth centuries, and also in later times, wherein the prevailing mental state is one of self-accusation. Cases are reported and the medico-legal bearings of the psychosis are considered.

31. *General Paresis and Cord Changes.*—In this paper Naka gives an exhaustive study of the now generally recognized alterations in the spinal cord in general paralysis, together with a discussion of the Argyll-Robertson pupil. A careful historical consideration of the subject precedes the writer's personal investigation, which covered the study of forty-three spinal cords in progressive paralysis. In these forty-three cords he found only once isolated degeneration of the pyramidal tracts, six times isolated degeneration of the dorsal tracts, and thirty-five times combined disease of the dorsal and lateral tracts. In but one case was the cord normal. In twenty-three of the cases there was total failure of the pupils to react to light, and in twenty-six increased knee jerks. In thirteen cases the knee jerks were lost on both sides, in two normal and in two unequal. A certain number of the cases are reported in detail, and the paper, in general, is a valuable statistical study of the spinal cord alterations in general paralysis.

32. *General Paresis and Trauma.*—Giesler in this paper discusses the difficult question of the relation between general paralysis and trauma. Such an association has been assumed in a certain proportion of cases by a large number of writers. Geisler reports six cases relative to this discussion. In these cases syphilitic infection did not definitely occur, although there was a possible suspicion of its existence in two. It was also shown that alterations of the skull or brain could not be brought into association with previous trauma. This, however, is naturally maintained not to prove that trauma may not have been a positive factor. It is, however, concluded that it is unlikely that even a very considerable trauma can be the sole cause of general paralysis, although from the practical standpoint it may be regarded as a contributive cause. Caution must, however, be observed in estimating the etiological significance of trauma.

33. *Pathological Sleep.*—Gudden discusses the question of pathological sleep on the basis of many carefully formulated cases. He concludes that the most conspicuous sign of pathological sleep is a shifting in the return of consciousness and the capacity for action; that the development of the disturbance is often favored by the weakness or the failure of definite impressions before going to sleep, which are of significance for the quick return of consciousness on awakening; that in a similar way long persistence of states of anxiety before sleeping favors its development; that the feeling of discomfort normally associated with early awakening plays a part in the thoughts and actions of those overcome with sleepiness, and that pathological sleep is often continued over a long period of time with certain complications. E. W. Taylor (Boston).

Miscellany

Treatment of Selected Cases of Cerebral, Spinal and Peripheral Nerve Palsies and Athetoses by Nerve Transplantation, with Report of a Case of Athetosis Benefited by Operation. By William G. Spiller, M.D.; Charles W. Frazier, M.D., and J. J. A. Van Kaathoven, M.D. (The Amer. Jour. of the Med. Sciences, February, 1906).

Dr. Spiller discusses the treatment of anterior poliomyelitis by nerve transplantation, the results of which in selected cases have been very encouraging. The chief dangers in operating on cases of anterior poliomyelitis

are delayed union and overgrowth of connective tissue in the nerve at the seat of the operation. It is hardly advisable to cut a healthy nerve entirely across, as Kilvington suggests, on account of these dangers, but where the fibers are small, as are those supplying the anterior tibial muscle, probably a sufficient number of nerve fibers are cut in the healthy nerve by the longitudinal slitting and the insertion of the diseased fibers within the slit. In a number of cases there is a distinct difference in the degree of paralysis of the flexor and extensor muscles in a hemiplegia developing in early life. Athetosis only occurs when the paralysis is incomplete, but in severe athetosis the patient has little or no voluntary control of his limbs, and the flexors are usually much more powerful than the extensors in the upper limbs. It was suggested by Dr. Spiller that there could be switched off, so to speak, some of this excessive innervation of the flexors into the extensors by nerve transplantation, in this way establishing a more nearly normal relation between certain groups of muscles and their opponents, and by the division of the nerves lessen the athetoid movements probably permanently. This operation was tried on a man nineteen years old, who had had athetosis all his life, except possibly during his first year. His upper limbs were jerked about most violently, but the flexor muscles were far more powerful than the extensors. The first operation was done on the left extremity—lateral anastomosis of the divided median and ulnar nerves to the musculo-spiral nerve. After this operation the movements were almost entirely confined to the muscles of the shoulder. At a second operation on the left upper extremity the circumflex and musculo-cutaneous nerves were divided and an end to end anastomosis effected between the central ends of the one and the distal ends of the other. Seven and a half months after the operation the results were encouraging, the patient is far more comfortable than before, and there is nothing to indicate that the athetosis will return. It is important to have established that, with partial paralysis produced by operation on nerves, athetosis disappears. Though the musculo-spiral nerve was not cut transversely, yet a partial return of function occured in the distributions of the median and ulnar nerves. This result justifies the end-to-side anastomosis which has been advised for the treatment of anterior poliomyelitis when the paralysis is confined to one or two muscles.

C. D. Camp (Philadelphia).

Tabes Dorsalis. By David Ferrier (The British Medical Journal, March 31, April 7 and 14, 1906).

After discussing the different theories of the pathogenesis of the disease, the author adopts the hypothesis of Turner and Hauser that the essential lesion is a dystrophy, similar to that, induced by certain toxic agents, affecting the sensory protoneurone as a whole and manifesting itself by degeneration of both the central and peripheral terminations of which the intramedullary are the most vulnerable and are usually the first to exhibit anatomical change. The author is in favor of ascribing a syphilitic origin to the disease. Concerning a diptheroid bacillus as the cause, as expounded by Ford Robertson, the author has made some supplementary researches and gives as his verdict "not proven." The physiological pathology of tabes is discussed with especial reference to the ataxy and the tabetic pupil, the latter being explained by the degeneration of the ciliary ganglion.

C. D. Camp (Philadelphia).

Tetany. A Report of Nine Cases. By Campbell P. Howard, M.D. (The Amer. Jour. of the Med. Sciences, February, 1906).

Nine cases are reported in detail, seven in adults. Six of the cases were of gastric origin, one of purely intestinal origin, is "the only adult case in the American literature of the past decade." The infantile cases both showed signs of rickets. None of the accesory phenomena, Erb's, Trous-

scau's, Chvostek's or Hoffman's were constantly present. The author adopts the etiological classification of Frankl-Hochwart. The pathogenesis of tetany is still conjectural, but in one of the cases in this series there was found evidence of abnormal activity of the cells of the parathyroid gland, a suggestive finding. C. D. Camp (Philadelphia).

Researches on the Blood of Epileptics. By B. Onuf, M.D., and Horace Lograsso, M.D. (The Amer. Jour. of the Med. Sciences, February, 1906).

The study of the formed elements of the blood was carried out on one patient, a hystero-epileptic colored man. It was found that a leucocytosis may be present directly before a seizure and is then, of course, not a purely secondary phenomenon produced by the seizure. A grand mal seizure need not necessarily be preceded or ushered in by a leucocytosis. There is no absolute parallelism between seizure and leucocytosis in so far as, even when a distinct leucocytosis is present, such may reach its height at different periods in different seizures. The leucocytosis is in part at least independent of the seizures. C. D. Camp (Philadelphia).

Abscess of the Brain, with a Report of Five Cases. By H. F. Stoll, M. D. (The Amer. Jour. of the Med. Sciences, February, 1906).

Five cases are reported in detail and a general review is given of the course, symptoms, diagnosis and differential diagnosis of the disease. The interesting feature of the first case is the etiology, which is considered to have been a slight trauma to the head. The second was metastatic, the primary focus being in the lung. In the fourth case, abscess of the right tempero-sphenoidal lobe, the patient lost the use of English and was able to understand only Swedish, his mother tongue. The treatment recommended is a prompt operation when the diagnosis is "reasonably certain."

C. D. Camp (Philadelphia).

Genius and Degeneration. By H. Edwin Lewis (The Alienist and Neurologist, February, 1906).

Genius—stated to be the capacity for spontaneous imagination, or imagination *de novo*, therefore unreal.

Talent—is skilful technique, applied to material or pre-existing things, and is essentially real. As geniuses, Poe, Whitman, Rembrandt and Wagner are contrasted with Shakespeare, Tennyson, Goethe, Holmes, Bonheur, Alma Tadema, Sargent, Reynolds, Beethoven, Gounod, considered talented. Genius is thought to be evidence of a degenerate and unhealthy mental state, exhibited by the former ones mentioned, in certain of their productions, also by their varied erratic or insane characteristics, and manners of life. As a preventative measure of possible mental retrogression, the cultivation of more healthful tastes in literature, art and music is urged, particularly in the training of young minds.

J. E: Clark (New York).

General Conditions and Insanity. By H. A. Tomlinson (Journal A. M. A., March 17).

The author emphasizes the importance of general pathologic conditions, especially of general metabolism, in insanity. "Mental aberration," he says, "in its clinical and pathologic aspects, has to do primarily with the potentiality of the nervous organization of the individual, and secondarily, with the perverted or defective processes of metabolism, as they affect the nutrition of the nervous system. In other words, we have to recognize that the degenerative process which makes mental aberration apparent is primarily a general one, affecting the vegetative functions." An analysis of the record of the patients received at the St. Peter's Hospital during the past nine years is used to illustrate his argument, and he concludes from all the data

that: "In dealing with insanity and its manifestations we are concerned with the cerebral potentiality of the individual in considering its nature; with heredity and environment in determining its form and sequence; while the evidence of the involvement of the general organism in the degenerative process must be our guide in anticipating its progress and termination."

Spinal Atrophy and Juvenile Dystrophy. By L. H. Mettler (Journal A. M. A., June 16).

Dr. Mettler reports a case of amyotrophic lateral sclerosis, notable because associated with pupillary inequality, and one of juvenile dystrophy in a lad of nineteen, which is discussed at some length. The cause was obscure and Mettler thinks it possible that a transient infectious polymyositis may have been present as its antecedent. The evidence is against a primary cord or nerve degeneration and rather in favor of a primary muscular affection. He discusses the differentiating points between spinal and muscular atrophies and believes that localization of the atrophied areas is an insufficient guide for distinguishing different types of this disease. Typical cases are in fact very rare, and he agrees with Gowers that it is undesirable to make a separate variety of juvenile muscular atrophy, as Erb has proposed. Spinal atrophies may also be juvenile and so may myositic atrophies.

Hypertrophy of the Brain. By J. H. Haberlin (Journal A. M. A., June 30).

The author reports the case of a child, aged two, dying in convulsions, in which the apparently symmetrically enlarged brain weighed 1,712 grams (53½ ounces). The membranes were not adherent, there was no flattening of the convolutions, no disproportionate increase in the size of the ventricles and the gray and white matters were developed proportionately Clinically, the case could not be differentiated from hydrocephalus.

Prognosis in Mental Disease. By Robert Jones (The British Medical Journal, Dec. 16, 1905).

The average age of admission to the London County Asylum is forty-two years. At this age the expectation of life in the sane is twenty-four years, but the average age of those dying in the asylum is 50.7 years, or about 15 years less. It is proven by statistics that there is always some mental weakness after an attack of insanity. In cases of insanity under twenty years of age relapses occur with much greater frequency than after this age. The author finds the average duration of general paralysis of the insane to be two years. Favorable factors in any case of insanity are: Normal sleep; gain in weight, accompanied by lessening of mental symptoms, with no lessening of mental symptoms it is a bad sign, especially in adolescents indicating dementia; a restoration of natural facial expression and affection for friends; and increased interest in his surroundings and appearance. The cause of the insanity has a direct bearing on the prognosis.

C. D. Camp (Philadelphia).

Drug Addictions.

In the preliminary report of the Committee on Drug Addictions of the Section on Nervous and Mental Diseases of the American Medical Association (Journal A. M. A., March 3), Dr. Smith Ely Jelliffe, the chairman, states that it was not thought best to consider the whole enormous subject of drug habits at this time, but rather to confine the inquiry to the subject of opium addiction. All the committee can do at present is to formulate a series of suggestions concerning lines of fruitful inquiry, and, therefore, they have limited themselves to certain problems that seem at present most promising for solution. The first of these concerns the spread and distribution of the habit, and it is suggested that valuable data may be obtained

through the pharmacists of a state or a community, through prison and asylum physicians and through a study of the cures advertised in the public press, etc. The second problem concerns proportion of the various forms of addictions, and the third is that of the etiology, the causes that have led to the habit, the factor of pain, the influence of environment, the reasons of its prevalence among certain classes, etc. A psychologic study of the euphoria of opium is also desirable. The pharmacology of opium, he states, is in need of further investigation and the suggestion is offered that its further chemical study is not without its possible revelations The treatment is naturally the topic of greatest interest, and this should be considered from the social as well as from the individual point of view. From the personal therapy side, the committee feels that the study of the abstinence symptoms is of great importance, and that its study will certainly afford very fruitful results. The questions of the well-known phenomena of tolerance raise that of a possible immunity. Are antibodies formed or does the body take on greater oxidating powers with increasing use of the drug, thus producing the greater tolerance observed? These questions are still undetermined, but their study is offered as a fruitful task for the future.

Retrobulbar Optic Neuritis Following Childbirth. By C. J. Kipp (Journal A. M. A., June 30).

Dr. Kipp reports a case of retrobulbar optic neuritis of one eye, recurring after successive labors, and ending in atrophy of the optic nerve with whitening of part of the eyelashes and eyebrow of the same side. No heart or kidney disease could be detected and there was pain in the right affected eye and supraorbital neuralgia. The left eye is normal, and in other respects the patient continued to enjoy good health. It seems to him probable that the pregnancies caused a vascular disturbance, a congestion at or near the apex of the orbit, and that this produced pressure on the optic nerve and its sheaths and also on the branches of the ophthalmic division of the fifth nerve. Others observing somewhat similar cases have attributed the optic disease to an autointoxication. For a full account of the different views held, Kipp refers to an article by Dr. George S. Derby (*Arch. of Ophthalmology*, xxxix., p. 9). So far as he knows, the case is the only one in which the whitening of the eyebrows and lashes has been observed in connection with retrobulbar optic neuritis. Only those portions in the course of the supraorbital nerve were whitened and the change occurred within a very few days, following the appearance of neuralgic symptoms. A second case, similar to the first, but occurring after a first labor, and with recovery of vision, is also reported by the author.

Tropical Neurasthenia. By W. W. King (Journal A. M. A., May 19).

The author describes neurasthenia as he observed it in Porto Rico, using the term tropical rather to indicate local influences than as signifying a particular type of the disorder. Comparatively few individuals, he thinks, escape certain symptoms of nervous exhaustion if they live in the tropics for a long period. Women are greater sufferers than men. While climate does have a certain relaxing effect, he thinks its influence is overestimated and that much should be attributed to the monotony of life, to the defective diet and to the habit of individuals. The variety of symptoms is as great as elsewhere, but he has noticed a lack of energy, a tendency to hypochondria and digestive disorders, loss of weight, disturbed sleep and dull occipital headache as prominent features. The symptoms may disappear gradually as the patient becomes acclimated, so to speak. Change of scene and occupation and moderate exercise, not overdone, especially if combined with amusement, are beneficial. Special symptoms may call for medicines, but he has seen little good from any general medication directed to the nervous system, aside from hygienic measures.

Book Reviews

CRIMINAL RESPONSIBILITY. By Charles Mercier, M. D. Clarendon Press, Oxford, England, page 288.

The author in the preface quotes the dictum of an eminent jurist who lays it down that all crimes consist of two parts, "the outward act, and the state of mind which accompanies it." This clearly indicates that from one point of view all crime is a problem in psychology. While the subject of criminal responsibility has been treated exhaustively by Sir Fitz James Stephen, still he was not a trained psychologist, and took rather the legal view point. The time seems, therefore, ripe for a review of the subject and from the point of view of the psychologist.

In entering into the question of responsibility the author considers punishment from the three standpoints of retribution, determent, and reform, and concludes that punishment as administered to-day is overwhelmingly retributory, containing little if any of the elements of the other two. As a result of these conclusions he comes to consider the problem of responsibility from a point of view different from that ordinarily taken. "A person is held responsible when the enlightened public opinion of his age and country demands that he shall be made to sufferer in return for pain that he has inflicted. Responsibility is, therefore, not a quality of the person who has inflicted the pain, but a demand on the part of others that he shall suffer."

From this rather extraordinary statement the author proceeds to examine the question of responsibility in detail in answer to the question, "Whom ought we to punish?"

In general terms the answer to this question is that those ought to be punished who do acts that are wrong (wrong being used in a general sense and not in the legal sense of tort). This answer necessitates an analysis of what is meant by act and by wrong. Acts are divided into reflex, instinctive, automatic, habitual and voluntary. Voluntary acts are the only acts in regard to which the question of responsibility can arise, and they must be wrong acts, wrong being defined as the seeking of gratification by "an unprovoked act of intentional harm." This statement naturally leads to a discussion of the relation of volition to responsibility. The actual act of volition is considered to be the last link in a chain, the preceding links of which are respectively "intention, or desire to do the act; choice; immediate motive or desire for the immediate consequences of the act; and, in succession, more and more distant motives. * * *"

The author sums up this discussion by saying:

"Responsibility attaches to acts that are wrong, and to no others. A wrong act is a voluntary act in which the actor seeks gratification by inflicting unprovoked harm upon others. Responsibility is the more undoubted, the more closely, more deliberately, the more frequently, the will is concerned in the act.

"Therefore, to incur responsibility by a harmful act, the actor must *will* the act; *intends* the harm; *desire* primarily his own gratification. Furthermore the act must be unprovoked, and the actor must *know* and appreciate the circumstances in which the act is done."

The author, by these conclusions and by his argument, commits himself by implication to the doctrine of graded responsibility. He acknowledges that a person may be irresponsible who could not be certified as insane, but clearly believes that an act, the ultimate volition of which is preceded at any stage by a morbid state of mind, should entitle the actor to relief from full responsibility therefor. The author also commits himself to the alternate proposition that insane persons are not always necessarily wholly

irresponsible. On this point he says: "The majority of insane persons are sane in a considerable proportion of their conduct; and when, in this part of their conduct, they commit offenses, they are rightly punishable," but further, "Since the limits between the sane and the insane areas of conduct of insane persons, are ill-defined, no insane person should be punished with the same severity that would be awarded to a sane person for the same offense."

The book is a learned discussion of the subject of criminal responsibility written in the author's happiest vein, but naturally contains much that will not be universally accepted. Especially is this true of the author's position on the propriety of punishing the insane, a position he has held for many years without, however, gaining many adherents. It is well worth a careful reading, especially by all engaged in court practice in cases involving the issue of insanity. WHITE.

DIE GESCHWÜLSTE DES RECHTEN UND LINKEN SCHLÄFELAPPENS. EINE KLINISCHE STUDIE. Von Dr. Albert Knapp, Oberarzt an der Kgl. Universitäts Psychiatrischen und Nervenklinik in Halle. J. F. Bergmann, Wiesbaden. 3.60m.

The study of tumors of the temporal lobes offers a most fruitful field to the neurologist as well as to the surgeon, both from the standpoint of exact localization and of operative interference. Tumors of the temporal lobes, particularly of the right side, have been considered by many authorities as particularly difficult of diagnosis. Bruns has said that tumors of the right temporal lobes are among those which show the least of localizing symptoms, and only when the pyramidal tracts are pressed upon is it possible to tell which hemisphere is involved, and Oppenheim has recently declared that we never have the right to make a localizing diagnosis of tumor of the right temporal lobe. Hence the need for a revision of older observations and the introduction of newer methods, and particularly the keener analysis of the mental signs of brain tumor. This has been done by Knapp in the present monograph of 150 pages.

He first discusses the general historical aspect of the subject, then passes to a review of the known general symptoms of temporal lobe tumors of both the right and the left sides. Amnestic and optic aphasia and their significance with the symptoms of asymbolia, apraxia and related disturbances are next considered. Knapp points out that asymbolic, apraxic, perserveration and echolalia symptoms are almost invariably associated in his patients with temporal lobe tumors with the classical picture of Korsakoff's psychosis. It is recalled, however, that this syndrome has been observed in patients with tumors in other brain areas.

Sensory aphasia, Knapp says, is the only positive localizing symptom on which one can count with reasonable certainty in left-sided tumors. In right-sided tumors one must depend on the accompanying and accidental symptoms of contiguity. Some of the more important of these indirect or related symptoms consist in involvement of the optic tracts, the pyramidal tracts, the sensory tracts, central ganglia, corpora quadrigemina, cerebellum, the five last cranial nerves, the facial, the trigeminus and the oculomotorius. These are all illustrated in the recital of the case histories of ten patients.

Final notes on the differential diagnosis close this painstaking and valuable monograph. JELLIFFE.

News and Notes

Announcement of $500 Prize by the Association for the Best Essay on the Etiology of Epilepsy.—Dr. W. P. Spratling announced a prize of $500 offered by the Association for the Study of Epilepsy for the best essay on the etiology of epilepsy. This prize is given by persons interested heart and soul in the work of the Association, and the conditions governing the award are as follows:

All essays submitted must be in English, written in a clear, legible hand or on the typewriter, on one side of the paper only, and they must not contain more than 15,000 words. Essays must be in the possession of Dr. W. P. Spratling, at Sonyea, N. Y., not later than Sept. 1, 1907.

The name of the person submitting the essay *must not appear on the same*, but be put in a sealed envelope on which is written a motto, and which motto should also appear at the top of the first page of the essay.

All essays received will be placed in the hands of three physicians to determine their merit. Two of these physicians are members of this Association; the third a member of the American Neurological Association.

Announcement of the award will be made at the November, 1907, meeting of the Association. The Association will not feel bound to award the prize should no essay submitted be deemed of sufficient value to merit it. *Original research work into the etiology of epilepsy will be a leading factor in fixing the award.*

The Psychiatrical Society of New York has arranged for a series of four lectures on problems of insanity, to be held under the auspices of the Academy of Medicine, at 17 West 43d St., on Saturdays, Jan. 19, Feb. 2, Feb. 16, and March 2, 1907, at 8.30 P. M. The purpose of these lectures is to put within the reach of the medical profession, and also of the non-professional leaders of sociological interests, a programme of work and facts for orientation, with a view to the organization of a movement toward prophylaxis and the development of sound interests in this eminently important topic. The first lecture will be given by Dr. Adolph Meyer, on "Modern Psychiatry, Its Possibilities and Opportunities;" the second lecture, by Dr. August Hoch, discusses the "Manageable causes of Insanity, Exclusive of Heredity;" the third lecture, by Dr. C. L. Dana, "The Data of Heredity and Their Application in Psychiatry," and the fourth lecture, by Dr. Allan McLane Hamilton, "The development of the Legal Regulations Concerning the Insane." The medical profession and non-medical persons interested in a movement towards prophylaxis and the best management of mental disorders are cordially invited.

Professor Wollenberg, in Tübingen, has accepted the call to the chair of psychiatry made vacant by the death of Professor Fürstner at Strassburg. He entered upon his duties with the beginning of the winter semester.

Dr. Robert Gaupp has been made Professor at Tübingen to fill the position made vacant by the call of Professor Wollenberg.

Dr. Ossipoff, of St. Petersburg, has been made A. O. Professor of Psychiatry in Kasan.

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Original Articles

ON HERPETIC INFLAMMATIONS OF THE GENICULATE GANGLION. A NEW SYNDROME AND ITS COMPLICATIONS.*

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(FROM THE PATHOLOGICAL LABORATORY OF THE CORNELL UNIVERSITY MEDICAL COLLEGE.)

Under the general heading of herpetic inflammations of the geniculate ganglion of the facial nerve, I have brought together for the first time three separate groups of cases; each group presenting distinct and clearly defined characteristics, but showing various combinations and transition forms. Their union I believe constitutes a new and distinct clinical entity.

The pathology of this affection is identical with that of herpes zoster, of which it forms a part, the distinguishing features of the clinical picture depending entirely upon the ganglion involved and the nature of the structures surrounding it. Heretofore the only recognized seat of an herpetic inflammation on a cranial nerve was that of the Gasserian ganglion of the trifacial. Herpes zoster in the distribution of one or more of its branches was the result. I believe, however, that the geniculate ganglion situated in the depths of the internal auditory canal at the entrance to the Fallopian aqueduct may be the seat of this specific inflammation. The peculiar situation of the ganglion within the confines of a bony canal and its immediate relationship to the facial and the

*Presented at the meeting of the American Neurological Association, June 4 and 5, 1906.

close proximity of the auditory nerve are responsible for the characteristic complex of symptoms which results.

As was long ago pointed out by Bärensprung and is now definitely established by the elaborate clinical and pathological researches of Head and Campbell, the primary or infectious form of herpes zoster is dependent upon a specific inflammation of one or more of the posterior spinal ganglia. Head suggested the name, posterior poliomyelitis for the affection, and certain points of resemblance were drawn between it and acute anterior poliomyelitis. The ganglia involved are swollen by the products of inflammation and by extravasation of blood, and in some cases even the sheath and nerve roots may be involved in the inflammatory process. In very rare instances the anterior or motor root, resting upon the sheath of the ganglion may be implicated and paralysis result.

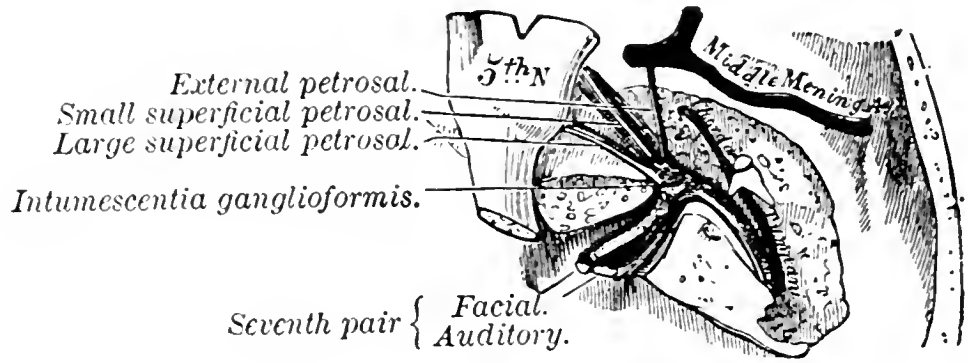

Fig. I.
The course and connections of the facial nerve in the temporal bone. (Gray's anatomy.)

Such paralytic complications are, however, extremely uncommon, and the total number of recorded cases is so small that they may be given in a few words. Herpetic inflammation of the Gasserian ganglion has given rise to palsies of the neighboring ocular nerves in 15 cases; in 12 cases the oculo-motor was the nerve affected, in two cases the trochlear and in one case the abducens. Paralytic complications of spinal zoster are recorded even less frequently. Arm palsies were observed in eight cases, to which I can add a personal observation, and a localized paralysis of the abdominal muscles in one case. Where the herpetic inflammation attacks the geniculate ganglion, palsies are of much

more frequent occurrence than in any other localization of the disease. I have collected 56 cases from the literature to which I can add 4 personal observations, making a total of 60 cases in which palsies accompanied the inflammation in this situation. This I would attribute to the peculiar location and relations of the ganglion involved.

Clinically the cases of geniculate herpes resolve themselves into three groups. The simplest expression of the disease is a herpes of the auricle and external auditory canal. *Within this skin area is to be found the zoster zone for the geniculate ganglion.* In another group of cases there is added to the aural herpes a paralysis of the facial nerve. This I explain by pressure of the inflamed ganglion or in some cases by a direct extension of the inflammation to the nerve. The most interesting, as well as the most severe, type of the disease occurs when the acoustic nerve is also involved. In this form there are with herpes auricularis and facial palsy, various auditory symptoms, ranging in severity from tinnitus aurium and diminution of hearing to the more severe forms of acoustic involvement as seen in Ménière's syndrome. In these cases I assume that the inflammatory process has extended to the auditory nerve which is enveloped in the same sheath, and courses in the same canal as the facial nerve.

Each of these groups has separately been the subject of careful study by many observers; but their intimate clinical relationship to one another, their common pathology and their common seat of origin, the geniculate ganglion of the facial nerve, has not heretofore been recognized.

I have already expressed my belief that the geniculate ganglion has its cutaneous representation and zoster zone in the auricle and external auditory canal, and that herpes zoster in this region may have facial and auditory complications. In regard to the distribution of the zoster I wish to lay especial stress upon this fact, that while these neural complications occur in auricular herpes, they also accompany herpes facialis, herpes occipitalis and cervicalis. It will be observed that in these forms of zoster, the herpes facialis and herpes occipito-collaris, the zoster zones lie immediately in front of and behind that which I have indicated as the geniculate zoster zone. Thus the Gasserian, geniculate and 2d and 3d cervical ganglia may be regarded as forming a

ganglionic series or chain, their cutaneous zones corresponding to the face, ear, head and neck.

It is not difficult to find an explanation for the occurrence of neural complications (facial and auditory) in those forms of zoster in which the eruption is not in the auricle and therefore is not in the geniculate area. The pathology of herpes zoster is such that while the inflammation predominates or is centered chiefly in one ganglion, other ganglia immediately above and below this central focus may also show inflammatory changes, but in a lesser degree. In fact, a series of ganglia may show milder degrees of inflammation. So that while one ganglion may be regarded as the central focus, evidences of inflammation are not infrequently present in recent cases, in the ganglia immediately above and below, diminishing in intensity from the central lesion.

As the Gasserian, geniculate, and upper cervical ganglia form a continuous system of cutaneous zones, so they may be regarded as forming anatomically a ganglionic series. It is therefore quite natural that if the chief focus of inflammation is centered in the Gasserian, subsidiary inflammatory changes may occur in the other ganglia of this group, the geniculate, and the upper cervical. In such an event the presence of facial and auditory symptoms with herpes occipito-collaris or herpes facialis would be readily explained. As a further evidence of multiple involvement of this group of ganglia may be cited cases in which the zoster eruption covers two distinct and separate zoster zones.

An idea of the relative frequency of these neural complications and their associated herpetic eruptions may be obtained from the following statistics. In the 60 cases at my disposal for analysis, all had a facial palsy of the peripheral type. In 19 of these cases irritative or paralytic symptoms of acoustic origin were present. In 32 cases the cutaneous manifestation was herpes occipito-collaris, in 12 cases herpes facialis, in 12 cases herpes auricularis, in 3 cases a combined herpes auricularis and occipito-collaris, in one case herpes facialis and occipito-collaris combined. I may also add that I have found but one case of herpes zoster with an associated facial palsy in which the eruption was not facial, auricular, or occipito-cervical.

With these introductory remarks I will proceed to a more detailed description of the anatomy, pathology and symptomatology of the affection.

Anatomical Considerations.—Before proceeding to the clinical aspect of my subject, it is of great importance that certain facts pertaining to the anatomy of the facial nerve and its ganglion should be known. The facial nerve clinically is generally regarded as a motor nerve. This from the anatomical standpoint is not the case. Histological and embryological investigations during the past ten years have shown conclusively that the facial is a mixed nerve, possessing an afferent or sensory portion, which is the nerve of Wrisberg, and a ganglionic structure, the geniculate, analogous in structure to the spinal ganglia of the posterior

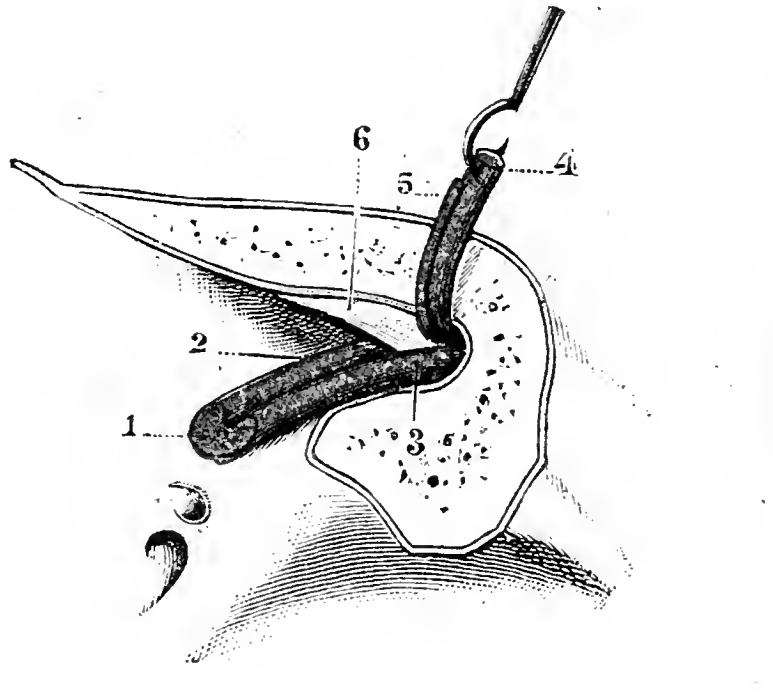

Fig. 2.

TESTUT.—The auditory nerve in the internal auditory canal. 1. Auditory nerve. 2. Cochlear-branch. 3. Vestibular-branch. 4. Facial. 5. Nerve of Wrisberg.

roots, the cell type of the geniculate corresponding exactly to that of the spinal and Gasserian ganglia.

The facial enters the internal auditory meatus in company with the acoustic and the nerve of Wrisberg. Within the auditory canal the 7th which is above rests in a slight concavity formed by the acoustic, the *pars intermedia* or nerve of Wrisberg lying between. All these nerve trunks are united and held together by a common sheath of connective tissue. At the bottom of the canal the acoustic divides into its cochlear and vestibular branches,

the nerve of Wrisberg and the facial entering the aqueduct of Fallopius. Immediately after its entrance the facial swells into the *intumescentia ganglioformis,* in which the nerve of Wrisberg takes its origin. From this point the facial nerve proper is continued to its peripheral distribution through the Fallopian aqueduct. As it lies in the entrance of the Fallopian aqueduct and the *Hiatus Fallopii,* the geniculate has important connections with other ganglia. It is connected with the spheno-palatine ganglion (Meckel's) through the great superficial petrosal nerve, and with

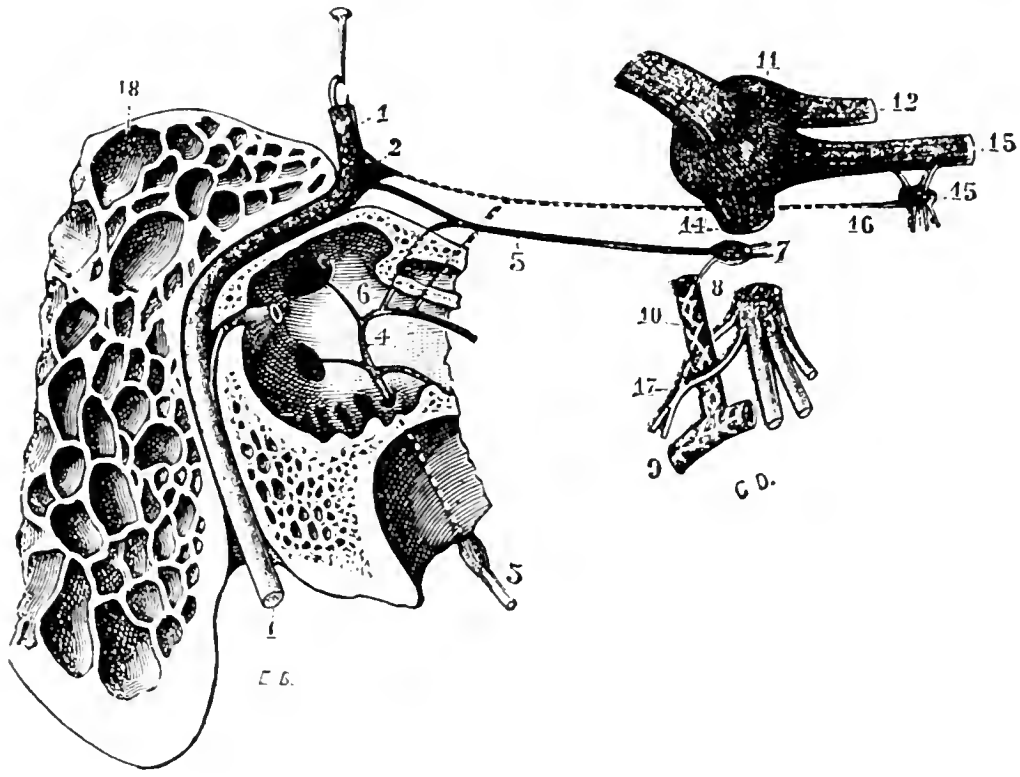

Fig. 3.

TESTUT'S ANATOMY.—The facial nerve, geniculate ganglion and relations with the otic. 1. Facial nerve. 2. Geniculate. 3. Glossopharyngeal. 4. Jacobson's nerve. 5. Small superficial petrosal. 6. Small deep petrosal. 7. Otic ganglion. 8. Sympathetic ramus. 10. Middle meningeal artery. 11. Gasserian ganglion. 12. Ophthalmic branch. 13. Superior maxillary. 14. Inferior maxillary. 15. Spheno-palatine ganglion. 16. Vidian nerve. 17. Auriculo-temporal nerve.

the otic ganglion (Arnold's) through the small superficial petrosal nerve. At their entrance into the medulla oblongata the 7th, 8th and *pars intermedia* have the following relations; the *pars intermedia* lies between the facial and the internal root of the acoustic. It sometimes joins one, sometimes the other of these two nerves as it enters the bulb, terminating in the *fasciculus solitarius* and the central gray column of the medulla. (Fig. 3.)

From this brief résumé of recognized and well founded facts it will be observed that my views regarding the affection under discussion finds only encouragement and support.

As the specific infection of herpes attacks only cells of the spinal ganglion type, the geniculate may very properly be brought within the sphere of its influence. Furthermore the intimate relations existing between the facial, the geniculate ganglion, and the terminal division of the acoustic would render all these structures liable to involvement when the seat of an inflammatory process; all the more because they are lodged in the depths of an osseous canal, within a common sheath, which would tend to resist expansion and increase the effect of pressure. (See Fig. 4.)

It may be added that the geniculate ganglion varies in size.

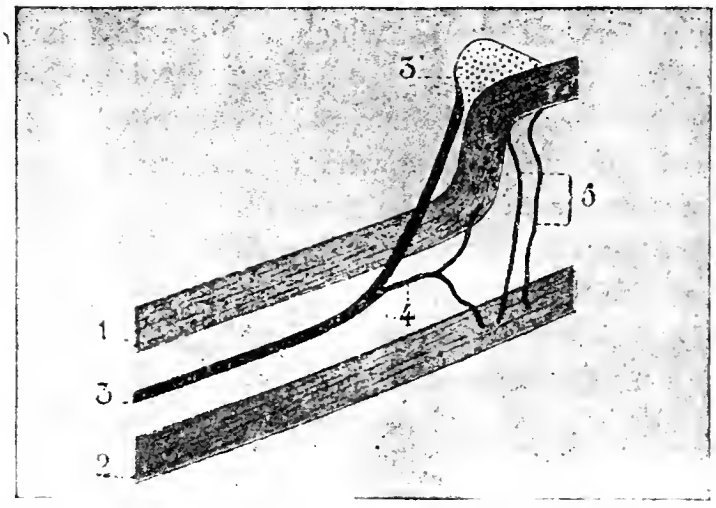

Fig. 4.

TESTUT.—Anastomoses of the facial and auditory nerves. 1. Facial. 2. Auditory. 3. Nerve of Wrisberg. 3'. Geniculate ganglion. 4. Internal anastomoses. 5. External anastomoses.

In some subjects it is scarcely visible to the naked eye; in others the swelling is double the caliber of the nerve. This may well have a certain influence in determining the severity of the case, anatomical peculiarities of the canal contributing.

The Zoster Zone for the Geniculate Ganglion.—Admitting the sensory nature of this ganglion, its anology to a spinal ganglion and its probable involvement in cases of herpes zoster, it is still necessary to demonstrate the existence of a cutaneous area on the head or face to represent it. This area must be independent of other recognized zoster zones and in it should be found the zoster zone of the geniculate. This zone, I believe, is situated

within the auricle and external auditory canal. My argument bearing on this part of the subject is briefly as follows: The peripheral innervation of the external ear is effected through the fifth nerve, branches of the cervical plexus, and *the auricular branch of the vagus.* The anterior half of the auricle and the superior and anterior walls of the external auditory canal are innervated by the auriculo-temporal branch of the trigeminus nerve. This nerve is a branch of the inferior division of the trigeminus, and I wish particularly to emphasize the neural connections existing between it and the geniculate ganglion through the otic ganglion and the small superficial petrosal nerve. The otic ganglion rests upon the inferior maxillary division of the fifth, just below its origin from the Gasserian. The posterior surface of the auricle receives its sensory innervation through the auricular branches of the superficial cervical plexus, which also overlap the rim and supply a posterior marginal area on its external surface. The inferior and posterior walls of the canal are supplied by the small auricular branch of the vagus, which also sends filaments to the interior of the concha. The ganglionic representations of sensation on the auricle and external auditory canal have been divided between the Gasserian in front and the second and third cervical ganglia behind. The anterior half of this region has been referred to the Gasserian and the posterior half to the cervical ganglion. An eruption of herpes in this area has been regarded as emanating from disease of one or other of these ganglia.

The error of the prevailing views will be shown by a study of the anesthesia produced by the extirpation of these ganglia for the relief of tic douloureux. In all of Krause's cases following extirpation of the Gasserian the sensation of the skin of the auricle and external auditory canal was found to be preserved and normal. In these operations during the tearing out of the ganglion, the connections existing between the fifth nerve, Meckel's ganglion, the otic ganglion, and the geniculate ganglion through the superficial petrosal nerves may be separated. This might cause confusion, by adding a geniculate anesthesia to that produced by removal of the Gasserian. This may have happened in some of Cushing's cases in which parts of the external auditory canal were found anesthetic after extirpation of the ganglion. The method of operation as practised by Frazier and

Spiller is free from this disadvantage. It consists in cutting the sensory root of the fifth on the central side of the ganglion. In such a procedure as this there is no undue tension or tearing the neural connections between the geniculate, and the second and third divisions of the fifth are not implicated. No procedure could be more exact for protecting the ganglionic area of the Gasserian. Following this operation the sensation of the auricle and auditory canal was found to be normal. The ganglionic innervation of the second and third cervical ganglia has been studied in a case of extirpation for the relief of obstinate occipito-cervical neuralgia by Harvey Cushing. In outlining the anesthesia in this case Cushing found the posterior marginal area on its external surface anesthetic. The interior of the auricle and external auditory canal had normal sensation.

If we now bring together and carefully adjust these respective areas of anesthesia, produced by extirpation of the Gasserian and the cervical ganglia, there still remains the interior of the auricle, and the external auditory canal in which sensation is preserved. In this area, I believe, it is to be found the cutaneous representation of the geniculate ganglion and its zoster zone.

It will be recalled that the peripheral innervation of this skin area is furnished by the auriculo-temporal branch of the third division of the fifth, and the auricular branch of the vagus. That these fibers do not pass through the inferior division of the fifth to the Gasserian is demonstrated by the anesthesia resulting from section of the sensory root of the fifth, this area retaining its sensation. If their afferent course is not through the trigeminus, how do they reach the brain? The relation of these sensory fibers to the geniculate ganglion is established if we accept the occurrence of geniculate herpes with a zoster zone in the auricle. So that it seems probable that these afferent fibers passing from the auricle on their way to the geniculate follow one or other of two routes, *i. e.,* from the skin of the auricle through the auriculo-temporal branch of the fifth, or the auricular branch of the vagus to the seventh nerve, the afferent fibers passing to the geniculate in the trunk of the facial nerve; or they may possibly be continued in the arriculo-temporal branch of the fifth to the otic ganglion and thence via the lesser petrosal nerve to the geniculate. Of these two routes, that through the facial is, in my opinion, the more likely one.

Clinical Types of the Disease.

Herpes Auricularis.—The simplest manifestation is to be found in the characteristic and well-known picture of herpes zoster of the auricle. There are the usual slight prodromes in the initial stage of the infection, followed by fever and mild general symptoms. Then sharp, darting pains are felt in the ear, the preherpetic pains, sometimes reaching a high degree of intensity. The skin of the ear may assume a red, swollen, somewhat erysipelatous appearance, until on the third or fourth day typical patches of herpetic vesicles make their appearance. These are situated in the concha, on the lobule, the tragus, the marginal portion of the auricle (helix and antihelix), and within the auditory canal, indeed, as rarely happens, on the *membrana tympani* itself. With the appearance of the eruption the acute pains usually subside, the ear still remaining swollen and tender.

At this stage the orfice of the external auditory canal may become constricted by the swollen soft parts, so as to interfere with the proper drainage and cleansing of the canal. The defect in hearing which may result from this temporary occlusion of the meatus, is purely mechanical and is not in any sense related to the disturbances of audition which accompany another group of cases and which is dependent upon involvement of the auditory nerve. In a few days the vesicles dessicate, the swelling and edema of the parts subside until at the end of a fortnight only a few scattered zoster scars remain to tell the tale. The sensory symptoms may, however, persist for a considerable time; burning pains, itching, paresthesia with impairment of the cutaneous sensation of the parts. In old people more especially the sharp neuralgic pains in the ear, the post-herpetic pains, may persist for a considerable time. (Herpetic otalgia.)

In this class of cases it will be observed that the herpetic pains and the herpetic eruption are localized within that skin area which retains its sensation after extirpation of the Gasserian and the second and third cervical ganglia. It was this area which I assigned to the geniculate ganglion as its cutaneous representation and zoster zone.

Remarks.—Idiopathic herpes zoster of the auricle has long been a recognized manifestation of zona. It is the herpes auricularis and herpes oticus of systematic writers. Dr. Anstie who was personally afflicted with the disease gave a very vivid de-

scription of his own case in "The Practitioner" of 1871. This localization of zoster has always been regarded as belonging to the trigeminal area, and due to disease of this nerve or its ganglion. If the observer favored the neuritic theory of herpes zoster, the skin lesions on the auricle were ascribed to a neuritis of the auriculo-temporal branch of the fifth nerve; if the ganglionic theory was accepted, the lesion was placed in the Gasserian. Some authors also speak of the auricular branches of the cervical nerves as playing a rôle in aural herpes, but no mention is made of the geniculate ganglion and its possible relation to this affection.

Compared with other manifestations of zona, the ear is an infrequent localization. Gruber records five typical cases as occurring in a series of 20,000 cases of ear disease.

To determine, if possible, the relative frequency of these cases, I examined the annual reports of several of our large hospitals for the eye and ear, with rather varying results. The total number of cases recorded is surprisingly small, so small indeed that it seems very possible that the affection not infrequently escapes recognition. It may be that when seen in the early stage of intense inflammation cases are regarded as perichondritis or inflammation of the auricle, or when seen later after dessication of the vesicles, as cases of eczema of the auricle. In the Manhattan Eye and Ear Dispensary during the past ten years, with a total of 47,600 cases, the diagnosis herpes of the auricle was made in only two cases. In the Brooklyn Eye and Ear Hospital, during the past five years, with a total of 15,000 cases, the diagnosis was made but once. The New York Eye and Ear Infirmary averages at the present time 10,000 out-patients a year, and is one of the largest institutions of its kind in the world. During the past twenty-three years this diagnosis was recorded but six times. The reports of the Massachusetts Eye and Ear Infirmary show a much larger proportion of these cases. In the past ten years with a total of 65,000 cases, the diagnosis was made 33 times. In these tabulations it was not possible to determine whether the cases were of the true infectious type, or merely of secondary origin, but the infrequency with which the diagnosis was made is worthy of note.

Herpes Auricularis with Facial Palsy.—In this manifestation of the affection, there is superadded to the herpes auricularis, as just described a peripheral facial palsy, which appears on the

same side as the zoster. The time of the appearance of this palsy varies, coming on in some cases simultaneously with the eruption, in others it may be delayed a week or even longer. In the majority of instances it appears on the second or third day. Too much stress should not be given to the patient's statements in this respect as the onset is often insidious and unobserved.

The paralysis is complete and involves all three branches of the nerve, and has certain peculiarities. A conspicuous feature is the frequent evanescence of the symptom, evidences of paralysis lasting only a few days or a fortnight. Many of the palsies clear up within three weeks or a month. There still remains, however, a large group of cases in which the palsy is of a severe type, reactions of degeneration persisting for a long time, leaving permanent weakness and contractures of the face. It is also a striking fact that in an unusually large number of these cases the sense of taste is lost or altered. This is not surprising when one considers that the seat of the lesion is in the geniculate, a level where the taste fibers are still coursing with the facial.

I would explain the involvement of the nerve in this group of cases by the pressure of an inflamed and swollen ganglion or by the direct extension of the inflammation to the sheath and connective tissue structures of the nerve. In light palsies probably inflammatory edema and pressure are the factors at play, whereas in the more severe forms inflammation and structural changes probably take place.

Remarks.—As has already been emphasized, a similar palsy may complicate herpes facialis and herpes occipito-collaris. I would explain the occurrence of the palsy in such cases by an herpetic inflammation of the geniculate ganglion, based on the well recognized tendency of this affection to produce inflammatory changes in a series of spinal ganglia. The Gasserian, geniculate and upper cervical ganglia constitute such a serial chain.

These cases of facial palsy complicating herpes of the ear, face, and neck, have long been the subject of study and controversy.

The old theories as to the origin of these palsies are as follows: The prevailing opinion is that the same exposure to cold produces both the herpes and the palsy, in which case the latter is regarded as rheumatic in nature, the common form of Bell's palsy. Another favorite theory was based on the infectious origin, the poison or

toxin concerned in herpes zoster also producing a neuritis of toxic origin. A somewhat fantastic hypothesis which found great favor with certain observers was the following: The herpetic inflammation is supposed to have extended along the peripheral fiiaments of the trifacial nerve, this nerve having numerous points of inosculation with the terminals of the facial. The inflammatory process then passes by continuity of structure, directly from the peripheral filaments of the trifacial to the termination of the facial, in this way producing an ascending neuritis.

Herpes Auricularis with Facial Palsy and Auditory Symptoms.—This is the most interesting as well as the severest type of the affection. In this group to the herpetic eruption on the ear, face or neck and facial palsy, are added symptoms pointing to involvement of the auditory nerve. The proximity of the terminal divisions of the auditory nerve to the facial and its ganglia, the common sheath and narrow osseous canal in which they lie would render such an auditory complication not only possible but probable. Contributing factors may be severe forms of the inflammation or certain anatomical peculiarities such as a large ganglion or a narrow bony canal.

The auditory symptoms may be both irritative and paralytic in character and make their appearance about the same time as the facial palsy. First there is tinnitus aurium followed by progressive diminution of hearing. In the more severe cases the symptoms of Ménière's disease are also present. Disturbances of equilibrium, vertigo, nausea and vomiting, nystagmus. In the course of a few weeks the acute symptoms subside, the vertigo and disturbances of the gait and equilibrium disappear, but the tinnitus often persists for a considerable time and the hearing may be permanently impaired.

Remarks.—In this group of cases as in that previously described the auditory symptoms may complicate herpes on the neck and face as well as on the auricle. In my series of 60 cases, auditory symptoms of various degrees of severity occurred in 19 cases. Of this number the zoster was in the occipito-cervical distribution in 9 cases, on the face in 4 cases, and on the auricle and auditory canal in 6.

The uncertainty which has attended the classification of this auditory group of cases and the doubtful nature of the affection, may be gathered from a perusal of the titles of the more important com-

munications. "Ueber ein Fall von gleichseitigen, akut aufgetretene Erkrankung des Acusticus, Facialis und Trigeminus" (Kaufmann, 1896).—"Ueber Polyneuritis Cerebralis ménièriformis" (Frankl-Hochwart, 1899, and Berger, 1905).—"Zur Lehre von der peripherischen Facialis Lähmung" (Hoffmann, 1899).—"Beitrag zur Casuistik der multiplen Hirn Nerven Erkrankung" (Hammerschlag, 1898).—"Herpes Zoster Oticus" (Korner, 1904).—"Trouble Auditive dans le Zona" (Lannois, 1904).

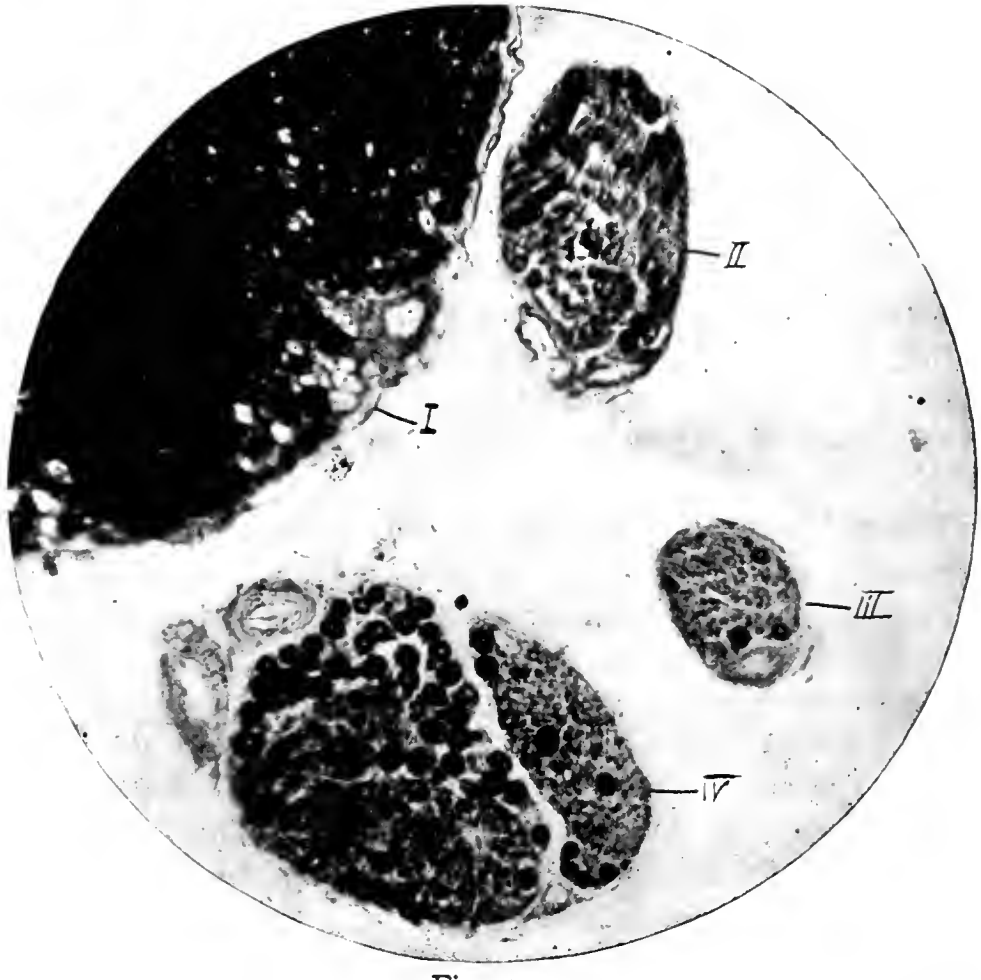

Fig. 5.
Osmic Acid Preparation, Counterstained with Rubin. I. Facial Nerve. II., III., IV. Bundles of the Nerve of Wrisberg, Showing Loss of Nerve Fibers and Sclerosis.

Pathological Evidence.—Recorded below under Case II is the pathological report of a case of herpes occipito-collaris in which a complete facial palsy supervened on the fourth day. There were no symptoms referable to the auditory nerve. Evidences of facial paralysis and objective sensory disturbances in the occipito-cervical region were still present at the time of death,

which occurred 87 days after the onset of the disease. Corresponding to the cervical distribution of the herpes, old inflammatory changes were found in the tip of the third cervical ganglion, with loss of nerve fibers and islets of sclerosis in the corresponding posterior root of the spinal cord. These could be traced through the 1st, 2d and 3d cervical segments, with evidences of myelin degeneration (granule cells and myelin droplets) in that

Fig. 6.

Marchi Method Intra-Medullary Degenerations in the Medial Root of the Acoustic. Entrance of the Nerve of Wrisberg.

portion of the posterior column, immediately adjacent to the posterior horns. Evidences of regeneration were also found in the branches of the superficial cervical plexus. Such pathological changes both in character and distribution are in accord with the findings in similar cases.

It is, however, to the facial nerve that I would particularly direct attention. The facial nerve, including the nerve of Wrisberg, from its exit at the medulla to its entrance at the internal

auditory canal, was treated by the osmic acid method and cut in transverse sections. By this method the nerve of Wrisberg was found to have lost a large number of its nerve fibers, with a compensatory increase of connective tissue. (See Fig. 5.) Fortunately that portion of the medulla which was treated by the Marchi method corresponded to the roots of the facial, auditory and nerve of Wrisberg, all of these structures taking their origin at the same level.

The nerve of Wrisberg (*pars intermedia*) lies between the origin of the other two nerves, sometimes it joins one, sometimes the other, as it enters the substance of the medulla. Sections through this level show distinct evidences of degeneration (myelin droplets and granule cells) along the course of the internal root of the auditory nerve, after its entrance into the medulla. See Fig 6. In other words, we find in the nerve of Wrisberg, which is the sensory root of the 7th and having its trophic centre in the geniculate ganglion, the same extra- and intra-medullary changes as were found in the spinal cord and posterior nerve root corresponding to the 3d cervical ganglia.

Unfortunately that portion of the facial nerve removed and which was supposed to contain the intumescentia ganglioformis consisted only of membrane, so that the changes in the ganglion itself cannot be given. But even in the absence of the geniculate ganglion, the existence of well-markd degenerations in the nerve of Wrisberg and its intrabulbar root is sufficient proof that this structure was involved.

CASE I. *Herpes facialis and occipito-cervicalis with facial palsy of short duration.*

A woman, aged 66, on March 20, 1904, was exposed for one hour to a sharp cold wind. It was snowing at the time and the effects of the intensely cutting cold were keenly felt on the left side of the face and neck. Two days later sharp darting pains and tingling sensations developed in the left face, neck and occipital regions. These pains were soon followed by an eruption of herpes in the same distribution. Patches of vesicles were sparsely scattered over the skin area of all three branches of the trigeminus nerve, as well as in the occipital and cervical regions. The darting pains continued after the development of the eruption, until seven days after exposure and five days after the appearance of the vesicles, a complete left facial palsy was observed on awaking in the morning.

I saw the patient April 3, 1904, with Dr. Robert Denniston of Dobb's Ferry. At the time there was total paralysis of the left facial nerve in all its branches, with considerable sagging of the parts, lachrymation and dribbling of saliva from the corner of the mouth. Pressure over the 7th nerve in front of the ear caused pain. There were no demonstrable objective sensory disturbances of the left face or neck, and no tender points over the foramina of exit of the trigeminus nerve. Hearing and taste were not affected. There were no general cerebral symptoms and the pupils reacted normally. The other cranial nerves as well as the motility, sensation and reflexes of the extremities were entirely normal. The urine contained a slight trace of albumin but was otherwise negative. Heart sounds of good quality, no murmurs. One of the superficial cervical glands on the left side is slightly enlarged.

The sharp shooting pains continued for a week from this date, coming on usually at 1 A.M. and lasting 3 or 4 hours, and were very severe. They diminished in their intensity during the next fortnight and soon after disappeared. Within one month after the onset of the facial paralysis all trace of it had disappeared, and the patient has since enjoyed her usual robust health.

CASE II. *Herpes occipito-cervicalis with severe facial palsy Autopsy and Histological Examination.*

The patient, admitted to the City Hospital February 15, 1905, was 48 years of age and a laborer. Periodic drinker. No venereal infections. The onset of the disease took place November 26, 1904, with sharp stabbing pains in the distribution of the right occipitalis major and immediately behind and in the right ear. With the sharp lancinating pains there was also present a burning sensation on the right side of the neck. These prodromal pains continued about four days and were followed by a vesicular eruption on the right side of the neck and the lower portion of the face above the ramus of the jaw. On the 3d of December, three days after the outbreak of the herpes a right facial paralysis was first observed. The vesicles dried up leaving many small pigmented scars. Paresthesias and burning sensations persisted with occasional sharp darting pains in the affected area.

Status Praesens, February 18, 1905.—Complete right 7th paralysis of the peripheral type. Behind the right ear, along the ramus of the inferior maxilla and on the right side of the neck are scattered, pigmented post-herpetic scars. These are in the distribution of the 2d and 3d cervical segments. (Herpes occipitocollaris.) In this same area, sensations of touch, pain and temperature are diminished, especially over the angle of the jaw. Burning sensations and sharp neuralgic pains still persist in the scarred area. Taste and hearing are unaffected. The other cranial nerves, the pupillary and tendon reflexes are normal. Urine normal.

Electrical.—To Faradism there is no response of the facial muscles on the paralyzed side when stimulated through the nerve.

Directly applied to the muscles of the face the response is slow and very much diminished.

On March 12, 1905, developed fever with evidences of pulmonary consolidation of the upper and middle lobes of the right lung. Died March 26, 1905, at 5 P.M. Clinical diagnosis, tuberculous pneumonia.

Autopsy, March 28, 1905, at 3 P.M. A complete autopsy was performed by Dr. Oertel, pathologist to the City Hospital. The right lung was found to be the seat of an extensive tuberculous pneumonia. Gross examination of the brain and spinal cord was negative. Some of the cervical and dorsal spinal ganglia, branches of the superficial cervical plexus at its emergence from behind the sterno-cleido-mastoid muscle were removed for histological examination. The entire facial nerve from its exit at the medulla to its entrance at the porus acousticus, the facial and its geniculate ganglion in the Fallopian canal, both Gasserian ganglia and portions of the *pes anserinus* of the facial were removed. The methods used were the Nissl, Osmic acid, Marchi-hematoxylin and eosin, Van Gieson and Weigert.

The Facial Nerve.—Teased osmicised preparations of the *pes anserinus* show the presence of numerous fine pale fibers, with rosary-like nodulations and swellings of the myelin sheath. In many other fibers the myelin covering is thin and poorly stained (pale), showing intervals and interruptions in its continuity. There are no myelin droplets or granule cells and the whole picture is regenerative rather than degenerative. The tissue removed from the Fallopian canal and presumably consisting of the intumescentia ganglioformis, unfortunately did not contain this organ, so that a microscopical study of the geniculate was not possible.

The whole trunk of the facial and including the nerve of Wrisberg from its exit at the medulla to its entrance into the auditory canal was treated by the osmic acid method. Transverse sections were then made and counter-stained with acid-rubin. By this method the fibers of the facial nerve proper were found normal. In the nerve of Wrisberg, however, there was demonstrated a very distinct and well marked loss of nerve fibers with a resulting sclerosis and increase of connective tissue. See Fig. 5.

Medulla Oblongata and Pons.—The cells of the facial nucleus on the affected side under a low power are smaller, paler and more rounded than normal, with absence or only slight indications of the tigroid appearance of the Nissl bodies. Under a high power the nucleolus is but faintly stained, and the nucleus is small, irregular in outline and shrunken in appearance. In many cells the nucleus shows a partial or complete dislocation to the periphery. The Nissl bodies are broken up, the cell body presenting a finely granular appearance. In some cells there is the typical

powdery appearance of axonal degeneration. The cell body is small, rounded and the processes broken off. The quantity and quality of the cell pigment does not seem to vary in the two sides. By the Weigert method the ascending root of the facial and the root fibers in the pons show no variation on the two sides.

Transverse sections of the medulla at the exit of the facial, the nerve of Wrisberg and the acoustic, show distinct evidences of old myelin degenerations, granule cells and myelin droplets in and along the course of the median root of the auditory nerve (Fig. 6). The root of the facial and the external root of the auditory are free from such evidences of degeneration.

Cord and Nerve Roots.—The examination of the 1st, 2d, 3d, and 4th cervical nerve roots on the affected side, shows distinct evidences of old degenerations in the posterior root of the 3d cervical segment. Delicate islets of sclerosis are demonstrable. These are quite destitute of nerve elements or contain only naked and swollen axis cylinders. No remnants of myelin degeneration were present in the nerve roots. The central tip only of the right 3d cervical ganglion was removed with the attached roots which showed some thickening and proliferation of the connective tissue of the sheath on its internal surface. In the spinal cord distinct evidences of old myelin degeneration, myelin fragments, droplets and granule cells, were found scattered along the mesial side of the posterior horn and the adjacent white substance. These evidences of degeneration could be readily traced through the 1st, 2d and 3d cervical segments. The right Gasserian ganglion showed no evidences of inflammation or degeneration. The teased osmic preparations of the branches of the superficial cervical plexus, show the same regenerative changes as were described in the *pes anserinus* of the facial nerve.

Concluding Remarks.—Briefly summarized my conclusions are as follows: The facial nerve like the trifacial is a mixed nerve. Its sensory ganglion is the geniculate. The motor root of the geniculate is the facial nerve proper and its sensory root is the nerve of Wrisberg. Below the ganglion the peripheral divisions are the facial nerve proper, the great and lesser superficial petrosal nerves, the external petrosal and the chorda tympani. This ganglion is of the spinal ganglion type and therefore in common with other ganglia of this type, comes within the realm of true herpes zoster.

The zoster zone for the geniculate is found in the interior of the auricle and in the external auditory canal.

The only neural connections existing between the geniculate ganglion and this cutaneous area are the auriculo-temporal branch

of the 5th through the medium of the small superficial petrosal nerve and otic ganglion and through the facial nerve proper. One or both of these routes may be taken by the afferent fibers from the auricle in their central course; in my opinion the facial route is the more probable one.

The ear-zone of the geniculate is intercalated between the zone for the Gasserian in front and the cervical ganglion behind, so that the zoster zones of the cephalic extremity are represented by the Gasserian (face and forehead), the geniculate (ear), the 2d and 3d cervical ganglia (occiput and neck). The zoster inflammations while attacking chiefly one, not infrequently involve more than one ganglion, milder changes showing in a series of ganglia above and below, diminishing in intensity from the central focus. For the same reason zoster in any of the zones of the cephalic extremity, may be accompanied by inflammatory reaction in the other ganglia of this group.

The pathology underlying the affection is the specific hemorrhagic inflammation of the ganglion as found in zona. As the geniculate is lodged in a narrow bony canal and stands in close relation to the 7th and 8th nerves, the characteristic syndrome is produced.

This syndrome may be divided into three clinical groups:

1. Herpes zoster auricularis.

2. Herpes zoster in any of the zoster zones of the cephalic extremity (Herpes auricularis, herpes facialis, and herpes occipitocollaris) with facial palsy.

3. Herpes zoster of the cephalic extremity with facial palsy and auditory symptoms (Tinnitus, deafness, vertigo, vomiting, nystagmus and disturbances of equilibrium).

In the foregoing pages I have endeavored to outline as briefly as possible the anatomical, pathological and clinical facts upon which I have based the syndrome. For the sake of conciseness and clearness, I have eliminated as far as possible all material not absolutely necessary for a convincing argument. For the same reason but few direct personal references have been made to the work of the long list of able investigators, who were my predecessors in this field. In a subsequent communication I hope to be able to give the subject broader and more elaborate treatment.

The abundant literature which has grown up in relation to

this subject will be appreciated on scanning the bibliography which is appended in full.

Neuralgic Affections of the Geniculate Ganglion and its Divisions. (Otalgia.) A Preliminary Report.

As the facial is a mixed nerve, having a sensory-ganglion, a cutaneous representation and zoster zone, the thought naturally arises, may it not be the seat of purely functional derangements similar to those occurring in the trigeminal area. May there not exist otalgia referable to the sensory mechanism of the facial, as there is prosopalgia originating in the sensory mechanism of the trifacial.

That herpes zoster of the auricle may be followed by severe neuralgic pains in the ear is well known. These herpetic otalgias are secondary, and in accordance with my views on this subject are dependent upon organic changes in the geniculate ganglion of the facial nerve.

On considering the extreme sensitiveness and delicacy of the organ of hearing, the high degree of its innervation, its exposed situation through the external auditory canal, the predisposition of such a mechanism to neuralgic disturbances would seem very probable.

This is the case. Otalgia, earache of non-inflammatory origin in its various forms is by no means an uncommon affection.

I would here mention briefly the branches and neural connections of the facial nerve and ganglion.

I. The ganglion stands in relation to the interior of the auricle and external auditory canal, its zoster zone (through the medium of the facial nerve or the small superficial petrosal nerve?).

II. The ganglion is intimately connected with the tympanic plexus. This is effected through the medium of the small and large deep petrosal nerves which arise in the tympanic plexus and join the small and large superficial petrosal nerves on their way to the geniculate.

III. The ganglion has also direct relations with the 2d and 3d divisions of the 5th, through the medium of the great and small superficial petrosal nerves which pass to Meckel's and the Otic ganglia respectively.

IV. It has reflex connections with the glosso-pharyngeal

nerve through Jacobson nerve, which enters into the formation of the tympanic plexus.

V. Another reflex connection is with the vagus through its small auricular branch. This nerve sends an ascending and descending filament to the facial as it crosses the Fallopian canal.

From these brief statements an idea may be obtained of the intimate neural relation existing between the facial nerve and ganglion and the tympanic cavity, auricle and external auditory canal; as well as the reflex neural connections with the trigeminus, glosso-pharyngeal and vagus nerves. A great many of the otalgias are of reflex origin. In these forms there occur with caries of the teeth and ulcerative affections of the tongue, pharynx and larynx, neuralgic pains, localized in the ear. These cases are quite common and the relation between the nerves supplying the region of the mouth and naso-pharynx and larynx with sensation and the tympanic cavity and auricle furnish an anatomical basis for the reflected pain.

The primary otalgia, idiopathic neuralgic affections of the ear, are recognized by nearly all systematic writers. This group of cases, however, as is the case with auricular herpes is divided between the trigeminal and occipito-cervical nerves. If the pain predominates in the anterior half of the auricle, they are usually relegated to the trigeminal, or if in the posterior to the occipito-cervical neuralgia. I believe, however, that there exists an otalgia, an independent primary and idiopathic affection of the sensory system of the facial nerve, and that this group will occupy the same place and have the same significance as the other time-honored clinical group of neuralgias..

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BRAIN TUMOR SYMPTOM-COMPLEX WITH TERMINATION IN RECOVERY.*

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The title of this paper is not at all misleading to the neurologist. All of us are at times confronted with cases which present the general symptoms of a brain tumor, together with focal signs which seem to render not only the general diagnosis of a cerebral neoplasm probable, but the focal signs are more or less marked, even to such an extent that we have little hesitation in designating the seat of the tumor. After a while, however, perhaps after several remissions and exacerbations extending over a period of years, these cases recover, showing, however, some defect, usually on the part of the eyes. Then death is caused by some other disease, and the brain is found perfectly normal.

Both the cases which recover and those in which a negative condition of the brain is found after death are of sufficient importance to merit our attention.

I will briefly report three cases of this kind, two of which are still alive, and one which died of tuberculosis, and in which an autopsy was made.

Case No. 1.—Cause of death, pulmonary tuberculosis. Under observation 10 years.

Nov. 1, 1895. Alice W., age 23, single, servant. Parents are well, father very nervous. Has sisters and brothers. One sister a confirmed neurasthenic with obsessions. No history of syphilis nor any evidence whatever of it to be found on examination. Patient has been well with the exception of an attack of la grippe two years ago. Has had attacks of headache for 4 years with occasional nausea. These attacks came on at night especially. Two years ago headaches became very severe, had enlarged glands in the neck at this time which were very painful. At this time the pain was constant, located in the occipital region, associated with pain in and behind both eye-balls. Has attacks of jerking in the neck and back.

The headaches are subject to acute exacerbations, during

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which patient walks the floor, tears her hair, screams and "seems to lose her mind." During this time patient's character changed. She became cross, querulous, was often unmanageable, subject to fits of violent temper. There was some weakness of both legs and staggering gait. The vision was diminished. These violent headaches and other symptoms would last two or three months. Then there would be a period of freedom from pain followed by another siege of headache, etc. Seven months ago she began to lose sight in the left eye. Could not distinguish objects. Legs were often numb and cold. During entire summer the legs were weak. Staggering gait came on at this time and has continued ever since, being worse on arising in the morning and when fatigued.

Examination.—Patient is of medium height, well built, 120 lbs. in weight. Intelligence good, memory good. Has violent headaches. Vertigo when she leans forward. Typical choked disk. Pupils equal and react to light, marked choked disk. No derangement of external muscles of eyes. Vision of left eye almost gone, has only light perception. Slight weakness of right facial. Has typical staggering gait (cerebellar). Muscular power and sensation normal. Left patellar reflex absent, right present.

Diagnosis.—Cerebellar Tumor(?).

Patient was treated with iodide of patassium and later on with proto-iodide of mercury.

The symptoms gradually disappeared after six months. Has headache about once a week. Choked disks had disappeared, was able to read large print with left eye, good sized scotoma in upper and outer part of field. General weakness of arms and legs but can walk well, no staggering gait.

Left patellar reflex absent, right normal.

A few months later had slight horizontal nystagmus, knee reflex returning on left side. Complains of buzzing in the head.

After a total illness of three years, and about 14 months after beginning of treatment, patient returned to her occupation, being entirely well with the exception of some defect of vision in the left eye.

Patient remained entirely well for six years.

July 9, 1902, aet 30. Has been at work 7 years. Patient returned with statement that she occasionally had headache, insomnia, vertigo, but no vomiting, these symptoms would last but a short time and would disappear.

Lately the headaches had again become more violent, they were frontal, lateral and occipital, with occasional vomiting. Staggering gait, states that she cannot see with left eye at all and only very little with the right. General weakness, can come to office.

Examination.—Some mental hebetude, peculiar inclination of head to right side probably due to defect of vision.

Marked choked disks of both eyes. The examination is otherwise negative. Patient was again placed on iodide of potassium.

In six or eight weeks all the above signs and symptoms disappeared again. About one year later patient developed an ordinary case of pulmonary tuberculosis.

I saw her once during the three years preceding her death. She became mentally unbalanced. Mental confusion, became violent at times because of suspicions of food being poisoned. This period lasted for several months and gradually passed away. The examination carefully made showed that there was an absence of all signs and symptoms pointing to any organic brain lesion.

Died Dec. 20. 1905. The autopsy was limited to an examination of the brain. The brain was perfectly normal, normal quantity of cerebro-spinal fluid. Dura mater and pia mater normal. Nowhere any adhesions. Cortex carefully examined microscopically was normal in every convolution. The ventricles were absolutely *not* dilated, the basal ganglia normal. Cerebellum was most carefully examined, no cicatrices, no atrophy, no adhesions on surface of the cerebellum, fourth ventricle, peduncles, pons, and medulla are all normal. The canal of Sylvius was open. The foramen of Magendic was also normal.

Hence no signs of a meningitis, hydrocephalus or brain tumor or abscess anywhere to be found.

CASE II. Under observation 13½ years.

Kate E., aet. 16, Buffalo, W. Va., was first examined by me on Dec. 30, 1892.

Family History.—Father alive and well. Mother dead, cause pulmonary tuberculosis. Has two sisters and one brother, all of whom are well. Collateral family history negative.

Present trouble began in 1888, four years ago. Previous had typical attacks of migraine or which were supposed to be such, viz., headache, vomiting and general prostration, the attack lasting usually 24 hours. Since onset of present trouble she has not had any sick headaches.

Present trouble began with difficulty in hearing and seeing. The difficulty in hearing manifested itself in gradually increasing deafness, which began four year ago. At the same time there was failing eye-sight in the right eye.

Menstruation, previously regular, has ceased for past ten months.

Vision has gradually diminished in right eye until patient is unable to see at all for past three years.

Turning of head to left causes pain in head and below the ears, complete turning of the eyes upward also causes pain in head.

Has had vertigo and vomiting for the past three years. Somnolence is very marked, is sleepy, sleeps often during the day.

Sleeps soundly at night. Never loss of consciousness, frequent attacks of staggering with the vertigo. Has been deaf in right ear for past three years:

About one year ago noticed that she could not turn right eye outward, lately has had daily headaches.

Physical examination.—Mental condition good, facial expression good.

Vision, right eye 0.7; left eye 1.0. Pupils equal in size and react to light.

Bilateral optic neuritis, choked disk, unable to close right eye completely. Paresis marked of right abducens, slight weakness of left abducens.

Head turns to left, face is inclined to left. Entire body inclines to left side. No tenderness of calvarium on percussion. Paresis of right side of face. Tongue is protruded in median line.

No disturbance of sensation anywhere.

Arms normal.

Legs, muscular power and reflexes are normal.

Marked static ataxia, cannot stand on right foot with closed eyes, very difficult to stand on left foot. Staggers to left when walking.

Dr. Ayres' examination.—Right eye, V = 0.7, left eye, V = 1.0.

Right ear loud tones 2 inches. Left ear hearing normal. Loud tinnitus.

Right optic disk presents a rounded mass which extends over and obscures the edge of disc. The veins are large, arteries obscured by the swelling.

Left optic disk is enormously swollen, no hemorrhage into the retina.

Patient was placed under treatment of iodide of potassium. Returned home in January, 1893, and remained entirely well for a year (see letter). She had an attack of fever in February, 1894. In May, 1894, again became very ill with a headache and returned to Cincinnati in June, 1894.

Examination, June, 1894.

Paralysis of external rectus of right eye. Vision, right eye 0.2, left eye 1.0. Marked choked disk both eyes.

Great difficulty in walking, cannot walk unassisted, staggers and falls to the left.

Has severe pain in the left side of head and discharge from the left ear, no evidences of mastoid disease.

Has severe pain in left occipital region.

In a letter dated March 9, 1906, patient writes that she has recovered entirely. She is strong and well and capable of personally doing the housework for her father. There remained only some defect of vision of the right eye and a partial paralysis of

the right external rectus. For the relief of the latter she had performed a tenotomy in April, 1905, which was completely successful, relieving the strabismus which had been present since 1894.

Viewing this case critically, in 1894 we have a history of some cerebral process gradual in its onset, gradually increasing in intensity, gradually compressing the cranial contents producing headache, vertigo, vomiting, somnolence, choked disks as general manifestations of a brain tumor and amblyopia of right eye, paresis of right external rectus, paresis of left facial, typical staggering gait, falling to left and static ataxia especially when standing on left foot.

A diagnosis of cerebellar tumor was certainly justified considering the slow gradual onset in a healthy robust girl, together with the absence of any signs of tuberculosis anywhere, or any focus of pus which might have produced a cerebellar abscess. (The otorrhea did not develop until 5 years after onset of first cerebral symptoms.)

CASE III. Under observation 2 years, sent by Dr. B., Georgetown, Ky. Examination August 22, 1904.

J. W. B., male, farmer, aet. 47, American, married, 4 children, all healthy. Patient denies all venereal diseases. Has always been well and worked hard. During past four or five years has had stomach ache with frequent attacks of vomiting. Has not been a drinker. Has had violent headaches for past four months, occipital in character. He had two attacks of violent vertigo in both of which he fell to the ground with momentary loss of consciousness. Has double vision very often. Cannot recognize objects one hundred feet off. Is unable to read ordinary print. Vomits occasionally. Has occasional attacks of staggering, as if he were drunk.

Examination.—Facial expression heavy, mental hebetude well marked. Intellect good. Memory good.

Pupils equal in size and respond to light. Marked choked disk in both eyes. External muscles of eyes normal. Reflexes all normal. Examination in every other respect negative.

Résumé: Headache, four months in duration, vomiting, violent vertigo with attacks of momentary unconsciousness. Staggering gait, double vision, great diminution of vision and bilateral choked disk.

Diagnosis.—Probable cerebellar tumor.

Examination May 1, 1906 (has not been examined for twenty months). Patient has gradually improved especially so in the past eight weeks. But made no improvement at all until six months ago. Headache has disappeared.

Has still occasional attacks of vertigo, slight staggering but very mild compared to formerly. Double vision is still present, objects being side by side. Can read large print.

Examination.—Mental condition bright, considers himself practically well. Has done some work this spring. Pupils equal in size and react to light.

Slight atrophy of left optic nerve. Right seems normal.

Right patellar reflex diminished, left normal.

Examination in other respects negative.

This patient has been taking iodine in some form all this time. There seems but little doubt that this case belongs in the same category as the other two.

The diagnosis of brain tumor is based upon the presence of certain symptoms and signs which point to a gradually increasing local and general compression of the cranial contents.

Of these the general signs and symptoms together with choked disk are of greatest importance in making the diagnosis of the presence of a tumor. The chief factors in estimating the value of these signs and symptoms are:

First—That of Time. Second—Compression.

As to the factor of time we must determine that from slight and insignificant beginning the symptoms gradually in the course of months grew in severity, duration and number, perhaps subject to periods of remissions and exacerbations, but nevertheless steadily growing worse as time passed by. The second point is equally important. All the symptoms must be the result of a gradually increasing compression of the cranial contents with localized destruction of brain tissue. When those general and local symptoms occur in sufficient number we can be reasonably certain of the presence of a tumor, and ordinarily the diagnosis and localization are not difficult. The only outcome of the brain tumor is relief by surgery or death, and then the diagnosis is usually verified. Leaving out of consideration possible errors of diagnosis which are shown up by autopsy, when abscess, hydrocephalus or some other gross lesion of the brain has simulated a brain tumor; there remain a few cases, as undoubtedly has happened in the practice of us all, in which, after careful examination, when all the signs and symptoms pointed to the presence of a brain tumor, the patients either recovered with perhaps only a slight defect or, death being caused by some other intercurrent disease, the autopsy revealed a negative condition of the brain.

Cases of this character are interesting enough, and of sufficient importance to demand our attention. While every one of us has undoubtedly seen them, the literature is exceedingly scanty. There are but two papers which deal extensively with this subject. I refer to the articles of Nonne and Oppenheim. Nonne's article gives in detail 8 cases occuring in adults and Oppenheim's deals with a similar condition in children.

It is needless to say that cases such as these cannot be attributed to a functional derangement of the nervous system. I may say that we can rule out hysteria at once. Such an array of general and local symptoms can be due only to some organic change either in the brain itself or in its membranes, and this lesion must be capable of complete retrograde change, restoring the brain to its original normal condition.

In considering the possible pathological condition underlying these cases we must divide them into two groups:

(a) Those cases in which death has occurred from some accidental cause and in which the autopsy revealed no brain lesion whatsoever.

(b) Those cases in which recovery occurred and the patients are still alive or having died, no autopsy was held.

There are very few cases of the first group on record. Nonne publishes three cases of this kind with autopsy in which the brain examination, including microscopic and bacteriological examination was entirely negative. Since Nonne's publication (1890) I have found no others and I wish to put my case with negative autopsy on record. While Nonne declines to consider the possibility of hydrocephalus acquisitus or serous meningitis, but inclines to the theory that some unknown and hitherto undiscovered organic lesion which is perfectly curable must be looked upon as the cause of the trouble, it seems to me that we must consider one of the following conditions as the underlying cause, I refer:

1. To serous meningitis producing acquired internal hydrocephalus, and

2. Hemorrhagic non-purulent encephalitis with serous effusion.

3. Chlorosis.

4. Chronic cerebritis.

In the purely clinical cases, which have recovered after all

the signs and symptoms of brain tumor were present, we must consider in addition to the above condition, the possibility of

5. Brain tumors or tubercles which have either undergone a retrograde metamorphosis or have become arrested in their development and to which the brain has accommodated itself.

It has been shown that all of the above pathological conditions except cerebritis, may produce a symptom-complex not unlike that of brain tumor and terminate in recovery.

Let us take up internal hydrocephalus first. There can be no doubt to-day, first, that internal hydrocephalus or serous meningitis occurs quite frequently in the adult, and in the second place it can hardly be doubted that although most of these cases terminate fatally, recovery may and does take place. Quite a number of acute mastoid inflammations are associated with a meningitis which disappears in a day or two after the operation, and which is in all probability serous in character.

It is possible that the majority of cases with brain tumor-symptom complex which terminate in recovery are really cases of serous meningitis of a subacute or chronic character, leading to internal hydrocephalus. We know now, that acquired hydrocephalus not only produces the general signs of a brain tumor, such as headache, vomiting, vertigo and choked disk, but also focal signs, such as staggering gait, hemiplegia, aphasia and paralysis of the various cranial nerves.

Oppenheim calls attention to the fact, that it is difficult, and often impossible, to differentiate between a tumor of the occipital fossa and meningitis serosa, and that the latter condition is the most frequent cause of a mistaken diagnosis of a cerebellar tumor. He says further, that these cases usually occur in women who present the following symptom-complex: Headache, vertigo, vomiting, choked disk, paresis of the external recti muscles, more especially of the right eye, nystagmus, more especially when the eyes are turned outward, especially toward the paretic muscle, areflexie or hyporeflexie of the cornea, tinnitus and diminished hearing on the same side, and cerebellar ataxia. He looks upon this symptom-complex as typical, and says that whereas in the beginning he always made the diagnosis of cerebellar tumor on the affected side, he now knows that these are cases of acquired

internal hydrocephalus or serous meningitis. The apparently focal signs of internal hydrocephalus are partly due to distal pressure, and this would explain the paralytic signs on part of the cranial nerves at the base of the brain. Oppenheim says that a unilateral predominance of pressure in the labyrinth of one side may so affect the cochlearis and the nervus vestibularis as to cause staggering to one side, tinnitus, as well as the nystagmus, and the abnormal holding of the head.

As a rule, gross unilateral symptoms on part of the extremities are absent in acquired hydrocephalus, but they may be present, as we know from reported cases, prominent among which is the case of unilateral hydrocephalus reported by Spiller. These, however, are the exceptions, and when they do occur they are not progressive nor permanent (Oppenheim). The focal signs may also be explained by the fact that serous meningitis may be complicated by a mild localized encephalitis or, what may also happen, the encephalitis may be the cause of the serous meningitis. Monoparesis, hemiparesis and hemianopsia are rare, still they have been observed (Annuske, Quinke and Kupferberg).

Moreover, we may have attacks of convulsions with periods of somnolence and coma, lasting perhaps a few days and then disappearing, perhaps not to return in months. I have seen this in a case of hydrocephalus which was associated with a brain tumor, and in which these attacks occurred six or eight times in a period of two or three years. Remissions are more common in hydrocephalus than in brain tumor. Herzfeld calls attention to these attacks coming on with almost apoplectic suddenness after long periods of comparative well being. Oppenheim attributes these attacks to a sudden increase of intraventricular pressure, or to a sudden compression of the medulla or vasomotor centers caused by a change in the position of the head.

We see, therefore, that there is a marked similarity not only between acquired hydrocephalus and cerebellar tumors, but also tumors of the brain in general. As a rule time will clear up the diagnosis, and even during life we can say with considerable certainty whether we have tumor or hydrocephalus. If the patient dies, then as a rule, the condition becomes certain. But what of those cases in which the autopsy

is entirely negative or in which the patients recover and remain well? Nonne concludes that in the eight cases of brain tumor complex with recovery which he reports in his article, there could not have been an acquired internal hydrocephalus or miningitis serosa, because of the absence of the etiological factors of the latter condition, namely: alcoholism, physical or psychical shock, insolation and infection.

It is possible, however, to have acquired hydrocephalus without any cause known or discoverable during life, and I personally am inclined to think that at least some of these cases are due to acquired hydrocephalus taking its origin from some cause unknown during life, which is either favorably affected by the treatment or disappears spontaneously, the exudation being resorbed.

Let us consider briefly the etiology of acquired internal hydrocephalus in regard to its pathology.

Spiller reports a case occurring in the service of Dr. Mills in which during life the diagnosis of cerebellar tumor was made. The autopsy however revealed an internal hydrocephalus caused by closure of the aqueduct of Sylvius by proliferation of the neuroglia. Byrom Bramwell reports a similar case caused by a closure of the foramen of Magendie. J. Parkes Weber, reports a case of acquired hydrocephalus with marked ependymitis of the fourth ventricles. He draws an analogy between acquired hydrocephalus and serous pleurisy and peritonitis. In both of these conditions we may have a large serous effusion caused by a small local area of inflammation, and he asks whether a large acquired hydrocephalus may not be caused in the same way. This conclusion would seem to be borne out by an autopsy which I recently made on a young boy, in which a marked hydrocephalus complicated a case of brain tumor. The tumor, an endothelioma, grew in the thalamus and extended into the white matter. Covering the serous surface of the lateral ventricle just over the tumor was a small area of tough fibrous exudate about the size of a silver half dollar which could be peeled off. In this case the symptoms were typical for acquired internal hydrocephalus, and antedated the focal signs of the brain tumor for years. It is not improbable to conclude that this hydrocephalus may have had

its origin from this small area of inflammation which was found located over the tumor in the lateral ventricle.

We also know that autopsies do not always show a closure of the various foramina in internal hydrocephalus.

From the reports of well examined cases we know that acquired hydrocephalus may occur from an inflammation of the tela choroidea, the choroid plexus, from pressure upon the vena Galeni, or a localized inflammation around the opening of the fourth ventricle. Any of these conditions might occur during life, without our knowledge, and terminate in recovery. Anton says that a frequent cause of chronic acquired internal hydrocephalus is a localized meningitis at the base of the brain. Quinke and Bonninghaus say that this meningitis spreads to the tela choroidea and the choroid plexus, and in this way causes internal hydrocephalus. Any localized swelling or inflammation around the foramen of Magendie or along the course of the canal of Sylvius may lead to a closure of these passages and thus cause an internal hydrocephalus. These various localized inflammations may yield to treatment, or heal spontaneously. The passages may reopen and the accumulated fluid become resorbed and the brain may return to a normal condition.

I refer to Anton, Quinke, Gowers and Oppenheim as authorities, that we may have recovery in typical cases of internal hydrocephalus acquisitus. The symptom-complex which is at times seen in myxedema of adults is probably due to a temporary meningitis serosa or internal hydrocephalus. Thus, I have a patient who has had myxedema for many years. She is very intelligent and highly educated. For years she has had attacks which begin with violent headache, vertigo, irritability, mental and emotional depression, marked staggering gait, delirium, mental confusion and, finally after some days, inability to walk, terminating in great somnolence, apathy and apparent dementia. These attacks may last from three to five days, to as many weeks, and then terminate rather quickly when the thyroid extract is pushed. She has had from one to four attacks per year for the past five or six years, and I look upon them as being due to an internal hydrocephalus, caused perhaps by the same toxin which causes the general myxedema. I therefore feel that a large percentage of the

cases of so-called brain tumor ending in recovery are really subacute cases of internal hydrocephalus which terminate in resolution, I include among these, those cases in which death has occurred and in which the autopsy has been negative.

Have we any means of making the positive differential diagnosis between brain tumor and acquired hydrocephalus in these doubtful cases. It is needless to say to-day that the Quinke puncture, or the specific gravity of the fluid will not clear up the differential diagnosis. The factor of time itself will only enable us to say in the recovered cases, or in cases with a negative autopsy, that we did not have a tumor but may have had an internal hydrocephalus.

Polio-encephalitis hæmorrhagica usually is acute in its onset and rapid in its course and does not ordinarily cause a symptom-complex resembling brain tumor. But the reported case of Schultze shows us how a polio-encephalitis superior may simulate a tumor of the corpora quadrigemina especially if it is complicated with an ependymitis with a secondary internal hydrocephalus. Schultze's case had in addition to the focal symptoms, somnolence and optic neuritis. Oppenheim reports a similar case with recovery, and he states that he had had a number of such cases, one in which he had even advised an operation, and recovery occurred. He concludes that there was present either a nonpurulent hemorrhagic encephalitis or some hitherto unknown pathological process capable of complete recovery.

Cases of completely cured polio-encephalitis, with perhaps some slight defect, do undoubtedly occur and we have thus another condition which may cause all the signs and symptoms of a brain tumor and terminate in recovery. In these cases, according to Oppenheim, the localized encephalitis is in or near the cortex, which explains the occurrence of Jacksonian epilepsy, monoplegia, aphasia, etc.

In this connection I also wish to refer to meningitis tuberculeuse en plaque which Oppenheim looks upon as the cause of brain tumor symptom-complex occurring in a number of children who recovered, and the author looks upon the localized tubercular meningitis as a curable affection.

Therefore, as far as encephalo-meningitis is concerned, we can never be absolutely sure of the diagnosis, because we

cannot rule out with certainty either localized tubercular or localized syphilitic meningitis.

We know from the recorded cases of Crawford-Thompson, Burton-Fanny, Jollye, Gowers, that chlorosis may produce a symptom-complex which may temporarily simulate brain tumor. We have in these cases pronounced headache, vomiting, vertigo, together with optic neuritis. Oppenheim reports a case of especial interest, in which the anemia was a complication of carcinoma of the breast, and the headache, vomiting, vertigo and optic neuritis simulated a metastatic corcinoma of the brain. The importance of recognizing the fact that anemia may produce a brain tumor symptom-complex should render us more careful about vetoing the amputation of a breast which otherwise might not only prolong the life of the patient, but even end in recovery.

The consideration of pseudo-tumors of the brain, with negative autopsy cannot be complete without taking into consideration the subject of chronic cerebritis or cerebral hypertrophy. This condition is exceedingly rare, and occurs most frequently in children, but may occur in adults. In adults where the cranium cannot expand we may have all the signs of intracranial pressure with irritation, resembling those of brain tumor. The cases are acute in their course, but may be subacute. The symptoms, according to Eulenberg, resemble very much those of acute hydrocephalus, violent headache, vomiting, slowness of the pulse, general convulsions, perhaps optic neuritis.

Let me give you Rokitansky's description of this condition: If we split open the dura mater we perceive immediately that there is a swelling of the brain, so much so, that there is great difficulty in putting back the calvarium. The various membranes are remarkably thin, the dura mater is tender, pale red in appearance, the pia mater is very close on the one side to the dura, on the other to the cortex. They are abnormally dry and their vessels small, flat and unusually free from blood, the brain hemispheres appear at first sight unusually large, the convolutions are pressed close together and flattened to such an extent that the sulci are hard to recognize. The white substance of the brain which has increased in volume is pale, anemic, which differentiates this condition from hyperemic

turgescence which Rokitansky looks upon as due to an increase in the neuroglia. Anton holds that we may have also a parenchymatous hypertrophy of the brain. Rokitansky and Eulenberg look upon the condition as due to a general increase of the neuroglia.

Eulenberg says that the fatal termination is usually due to a congestive hyperemia.

The literature on this subject is exceedingly scanty, but if such a case should occur and we were on the lookout for a tumor, especially if the brain were presented to us after the removal from the calvarium, the condition might easily be overlooked and the autopsy considered negative.

Anton asserts that there may be a partial cerebral hypertrophy, which if it did occur might produce even a more decided brain-tumor symptom-complex. He gives, however, no data to substantiate this assertion.

In the cases of recovery from brain-tumor we must bear in mind that the patient may really have had a brain-tumor, and that the latter disappeared, or the brain became accustomed to its presence after it ceased to grow. Some of these cases, however, are cases of brain-tumor with unusually long remissions of the symptoms and not cases of real recovery, which like Osler's case finally end fatally. Oppenheim, Gowers, Bernhardt and Russel have reported similar cases. Psammomata, lipomata, cholesteatomata may attain a certain size and then cease to grow. Cysticerci and echinococci may die and shrink. The observations confirmed by autopsy are exceedingly rare. Nonne was able to find but four cases, those of Bruns, Oppenheim and Simeon.

Aneurysms may cease to grow or become obliterated by the formation of a clot and the brain accommodates itself to the pressure (Oppenheim, Hutchinson, Hodgson and Humble). Solitary tubercles may undergo caseation calcification and then become encapsulated causing no further damage (Wernicke, Starr, Knapp, Gowers, Babinski and Sahlberg). These observations are confirmed by the pathological observations of Simeon who reports an autopsy in which a calcified tubercle was found which must have been in the brain for 30 years. Kirschberger's case shows how a tubercle may show all the signs of a brain-tumor for a year and a half and the patient

recover and remain well for six and one-half years. The autopsy in this case showed that the tubercle had become calcified. Gummata may become absorbed and disappear.

Long remissions and even permanent disappearance of symptoms may occur in brain-tumor as numerous cases on record show. These cases, however, would hardly come under the head of pseudo-tumors with a complete disappearance of symptoms, because they had been regarded during life as cases of epilepsy of many years' duration, and after death partly or completely ossified tumors had been found as the cause of the epilepsy.

That a pathological mass may be present in the cortex of the brain for 40 years and cause no interference with the ordinary daily life of an individual the following case will show:

Lately an individual, 60 years of age, died in my service at the Cincinnati Hospital from acute meningitis. He states that at the age of 21 his left arm became paralyzed, but that it soon recovered. He has lived the life of a farmer and teamster for the past 40 years. With the exception of some weakness of the left arm and a constant headache he had always been well.

The autopsy revealed a mass, hard, indurated, cartilagenous, about the size and shape of a peanut, situated in the cortex, at the posterior end of the first frontal convolution of the right side, pressing upon the ascending frontal convolution. This mass was partly cortical and partly subcortical, was not firmly adherent, and was surrounded by recent softening. On splitting it open we find a very hard stony calcareous center and a hard cartilagenous covering.

The practical conclusion of the consideration of this subject is, in the first place we should not be too pessimistic in our prognosis of brain-tumors, especially in cerebellar cases, until a considerable period of time has elapsed; and in the second place we should not be too hasty in the recommendation of surgical interference both in children and in adults, until therapeutic measures have been given a long and complete test.

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AFTER-CARE AND PROPHYLAXIS AND THE HOSPITAL PHYSICIAN.*

By Adolf Meyer, M.D., LL.D.,

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At the request of our President I have been induced to report briefly on a movement which has been inaugurated in New York State for the purpose of after-care of needy persons discharged from hospitals for the insane. What Dr. Wise, Dr. Stedman, Dr. Dercum, Dr. Dana, and Dr. Dewey had advocated since 1893, has at last been realized and put in operation.

Largely through the efforts of Miss Louisa Lee Schuyler, the Conference of Superintendents of the State Hospitals of New York has requested the State Charities Aid Association to organize a system of after-care for the insane, and to put it into practical operation. It passed a resolution "That in the opinion of this conference it is desirable that there shall be established in this State, through private philanthropy, a system for providing temporary assistance, and friendly aid and counsel for needy persons discharged recovered, from State Hospitals for the Insane, otherwise known as 'After-Care of the Insane.'"

This inaugurates a system of co-operation between the State Hospitals and an organization of private philanthropy and makes possible a co-operation of paid agents and of volunteers; the expenses to be paid by voluntary contribution, and with the utilization of the allowance provided by the State of an amount up to $25.00 for patients discharged and in need of help to "defray his necessary expenses until he can reach relatives and friends or find employment to earn a subsistence."

The State sub-committee on after-care consists of six members from the State Charities Aid Association, and as organization progresses, each hospital district will receive its special after-care committee; Manhattan State Hospital has a committee consisting of five members of the State Charities Aid Association and two members from the board of managers of the hospital, and the superintendent. The Willard After-care Committee has seven

*Read at the meeting of the American Neurological Association, June 4 and 5, 1906.

members from the State Charities Aid Association, three members of the board of managers, and the superintendent of the hospital. There has already been an agent appointed to work in connection with Manhattan State Hospital. The formal expenses of salary, traveling and other after-care expenses are expected to amount to about $2,500 annually, and in addition to the allowance up to $25 assured by the State, private contributions will be resorted to for practical assistance of individuals.

In the main the system of the English After-care Society seems to have been the chief model of the movement inaugurated, apart from the fact that more is made of a co-operation of the hospitals with the local Committee, and that really a *co-operation* between State and private philanthropy is assured. This special fact is very gratifying. In a paper read by me at the conference at which the State Charities Aid Association was authorized to take up the co-operative work, I made a strong appeal in favor of as close as possible co-operation between the physicians who have charge of the patients and any individuals who wish to help the patients outside. The failure of the previous efforts in such a movement in this country comes, to a large extent, from a lack of conviction on the part of hospital physicians concerning the efficiency and desirability of the ordinary type of charitable movements. And I am also convinced that unless there is a hearty co-operation between physicians and philanthropists, the movement loses its greatest opportunity and its main spring which would assure its life and its development into that which we really need most of all,—namely, the breaking down of the hospital walls, the extension of responsibility of the physician to the prevention of relapses, and the responsibility of carrying useful information gained in hospital practice to our intermediators among the public—the physicians, and those who have the broader sociological problems of the community at heart. This country is fortunately not a region in which pauperism is an *inevitable* doom of a certain portion of our population. Large cities may offer numerous samples of destitution requiring monetary aid, but as a rule our people are proud of supporting themselves; they like to make their own way, and wholly friendless individuals are extremely rare. The main need is that of guidance to a field best adapted to the qualification of the patient, and especially also guidance of their environment in the direction of adequate help

and strengthening of the healthy instincts in the struggle against morbid tendencies.

In trying to do the work with the insane as conscientiously as possible, and in interesting our colleagues in the service, certain needs have become evident which ultimately will have to be taken up by the after-care associations.

In the first place, the desirability of encouraging and organizing in each community persons capable of spreading sound information concerning what the State can and will do for victims of mental disorders.

2nd: The encouragement and organization of individuals who will have enough interest in the cause to help the hospital physician get at the correct estimate of the conditions under which the patient was wrecked, and to which the patient shall be discharged, *i. e.,* persons to be appealed to in obtaining direct information.

3d: Persons who will be willing to relieve the tension between the public and hospitals, usually based on misinformation.

4th: Persons who have a sufficiently wide acquaintance with the opportunities of a locality for drawing a recovered patient as rapidly as possible into a healthy and wholesome environment, that is, persons who have contact with movements by no means exclusively looking out for persons who have been insane. Much help is obtained from churches, from charity organizations of an independent character, from abstinent societies, and from all those who are leaders of interests in the various strata of our healthy population. The issue is to make all the possibilities accessible.

In this whole movement we physicians can do a great amount of good by helping in the co-ordination of all the individuals who might be accessible to interests,—leaders among the various strata of the healthy, physicians, schoolmen, leaders of social movements, workers against alcoholic excesses, judges, clergymen; to attract their attention to the available reports, to organize some public lectures on matters which seem to be specially in need of elucidation in a community, to distribute such lectures, and to gradually replace the notions of half a century ago by the facts available at the present time.

The New York Psychiatrical Society aims to make a be-

ginning in this direction, and it is hoped that other bodies will follow.

In closing I should like to emphasize an appeal for harmonious working, for a disinterested assistance to the hospital men, who after all assume the greatest responsibility about the fate of the patient, and well considered co-operation of any after-care and prophylaxis movement in each community. But first of all, I should urge the necessity of dropping the narrow-minded habit of considering the restrictions under which hospital-physicians work as inevitably those of sequestration. Let us help the hospital-physician broaden his horizon and overcome the diffidence imposed on him by tradition and prejudice. He should be the center of work for the patient, the collector of all the information and therefore the one who will also be the central figure in the after-care steps. Give the hospital-physician the chance and you will see that the chief obstacle in the way of after-care is overcome. Twelve years have elapsed since the committee's report. Since then much has changed, inside the hospitals and outside. Civic medicine has made much practical progress and has learned and taught some good lessons in the duties and possibilities of popular education, in social and personal hygiene and prevention. Let us use what the tuberculosis struggle has taught us, and above all, let us encourage those who more than ever try to rise from the oppressed and artificially sequestrated positions of hospital-physicians. Let us help those who have the patient under their care for months or years; let us widen their sphere of interest and responsibility beyond the temporary mending; let us create links between them and the public and the places to which the patients return. Interest and responsibility grow with opportunity. If physicians in hospitals know that physicians and others outside will profit from a knowledge of facts in a patient for the purpose of directing local after-care work, a natural diffusion of information will begin, much to the advantage of all concerned. With this additional appeal I heartily endorse the recommendations made by Dr. Stedman in his admirable presidential address.

THE INSANE COMMISSION OF THE ST. LOUIS CITY JAIL, AN EXPERIMENT IN CIVIC MEDICINE.*

By Sidney I. Schwab, M.D.,

of St. Louis.

The work which this paper attempts to outline is in the nature of an experiment in the Grenzgebeit where the problems concerning law and medicine are most frequently encountered. The lamentable position which so-called expert medical testimony has reached in the courts of the United States justifies any logical attempt at improvement, and this Society furnishes an admirable place before which to present such an attempt. The special features of criminal procedure in the St. Louis Criminal Courts give to this attempt a certain unique character, although the conditions elsewhere present largely the same sort of problem. Mention should be made in this place of the indebtedness which we feel to the efforts of Dr. Stedman of Boston towards the improvement of the procedure in criminal cases in which the question of responsibility is raised. Much of the inspiration to our modest efforts in St. Louis is due to his work.

The main purposes underlying the organization of the commission of which this paper treats were the cultivation of a better standard of expert medical testimony in respect to mental questions in the Criminal Courts of St. Louis, the training of a group of experts in such questions for service in the courts, and the collecting of such data as afterwards might prove of value in respect to the problems that arise in the relation of the criminal to nervous and mental diseases.

The central idea of this whole experiment is that all expert testimony should be given on the question at issue and should have nothing whatever to do with one side or the other in the actual legal test of the case. In other words an expert exists for the sole purpose of informing the court upon questions of the proper interpretation of facts lying within the limits where expert knowledge is required.

A brief description of the organization of the criminal

*Read at the meeting of the American Neurological Society, June 4 and 5, 1906.

courts in St. Louis and the relation of the administration of the City Jail and the Circuit Attorney to them might serve to make clear the special problem which this commission had to face.

There are nine circuit courts in St. Louis presided over by nine elective circuit judges. Two of these in rotation sit in the criminal division, thus forming two criminal courts. In addition there is a third criminal court called the court of criminal correction whose jurisdiction is more limited. This latter court has its own prosecuting officer who is appointed to that position. The circuit attorney of the city is the States' representative in all criminal cases. The city jail contains all prisoners who are awaiting trial or who have been sentenced, and who for some reason are kept there until final decision in their cases are reached. It is thus seen that the prosecuting attorney determines the matter of prosecution, and it is clear likewise that the question of responsibility concerns his office first of all. By an ordinance of the city the chief dispensary physician is likewise the chief physician of the jail, and before the present commission became active he was the chief psychiatric expert of the State in all such cases in which a plea of insanity was brought forward, or in cases where insanity was clearly present in the first instance. It is obvious that the proper time to consider the question of mental responsibility is before the trial takes place, and not at the trial where the defense of insanity becomes not a medical question but partakes of the character of an *ex parte* appeal to the jury. It became the first object of the commission to have the question of a prisoner's sanity decided if possible before the formal processes and trial took place.

For many years the most common defense in major criminal cases was that of insanity, and for the organization of this defense the defendant hired such experts as the size of his purse would allow to testify to his insanity; the State opposed to this the testimony of the jail physician who was not at all trained in psychiatry nor even interested in its problems. As a result of this method expert testimony became prejudiced, illogical, and was given not upon the question but as a defense or prosecution argument. Nothing need be said concerning the average quality of the expert testimony in

regard to mental questions in the criminal courts, suffice to say that neither judge, jury nor lawyers were impressed with its weight. The idea of the present commission arose from a knowledge of these conditions and the conviction that better methods would in the long run help to create better standards which could not be disregarded. The preliminary steps in the organization of this commission will be omitted here, mention merely being made of the valuable aid given to us by the present chief dispensary physician, Dr. Scherck, whose assistance and encouragement have made possible the experiment of which this paper treats. A scheme for the improvement of expert testimony and examination of prisoners suspected of being insane was drawn up and presented to the two judges then sitting on the criminal benches. This memorial was as follows:

1. The Courts and jail physician should appoint a body of three experts who are to be specialists on nervous and mental diseases with knowledge and training sufficient to enable them to speak with authority on these subjects. The jail physician by virtue of his office and his interest in the charges under him is to be the fourth member of this body.

2. One member of the body is to be appointed a chairman. His duty will be to apportion the work to be done and to be the representative of the body whenever such representation shall be necessary.

3 Whenever in the opinion of the circuit attorney, the jail physician, or the presiding judge there is committed to the jail a prisoner in whose case there is any suspicion of mental disease either past or present which has any bearing on the case whatever, such a prisoner before coming to trial should be examined by this body of experts either singly or collectively, as the case demands, for the purpose of determining his responsibility, his irresponsibility, degree of irresponsibility, and his final disposal if his irresponsibility is established.

4. The decision of this body is to be submitted in writing to the judge before whom the prisoner is to be tried. If the decision is not unanimous a dissenting opinion can likewise be submitted.

5. This body or any part of it will be at the service of the

court to present their findings to the jury, or in any other way to assist the court to arrive at a just opinion in regard to the sanity or insanity of a prisoner.

6. Each member of this body will agree to serve without compensation.

7. The jail physician or the court can add to this examining body at any time physicians who, in their opinion, can aid them in arriving at a decision. In this way no one can be deprived of the services of experts who are not members of this body. It is recommended, however, that if such additions are made, only those who have the requisite knowledge should be appointed.

8. A room is to be set apart in the jail building in which prisoners under observation can be studied under the best possible conditions.

9. A record of the work of this body is to be kept with great care so that its services to the courts of justice and to the community may be used as an argument in favor of the permanency of such a body, if this method of arriving at a just solution of this part of criminal procedure should find favor with those who can set about making this into a law.

As a result of this memorial the commission was created embodying the provisions as set down, by the two presiding judges in the criminal courts, with the consent and approval of the circuit attorney. It is to be observed that this commission as at present constituted has no legal nor official position, and exists merely as a matter of convenience to the court. It is and remains an arm of the criminal courts in regard to the elucidation of questions pertaining to the mental states of prisoners in the jail awaiting trial. The commission consists of four members, the jail physician being the *ex-officio* member. The other members are Drs. Fry, Graves and the writer. The method of procedure is as follows: Any prisoner in the city jail before he is brought to trial and in some instances before he is arraigned, in whom is found the slightest evidence of mental abnormality is examined by the commission, and a written report of its findings is transmitted to the circuit attorney. If the commission finds evidence of insanity the prisoner is sent to an asylum either permanently or until he has so far recovered as to be in a mental condition to stand

trial. At that time then the question of his mental state at the time the crime was committed is considered. If a defense of insanity is made by the prisoner's lawyers, and if the commission's report of sanity is not accepted by the defendant's counsel, then the findings of the commission are taken to be the State's attitude in the matter, and the members of the commission are expected to present their testimony as experts at the trial which follows. Naturally, the defense has its own experts, and the old method of *ex parte* testimony must in the nature of things be made use of. The advantage over the old system lies in the fact that the findings of the commission have been arrived at from an absolutely unprejudiced standpoint and are based upon clinical evidence uninfluenced by the legal aspects of the case. If the prisoner is found insane either the prosecution is allowed to cease and the prisoner is sent to an insane asylum, or the prisoner is declared to be insane by the jury before whom he was to have been tried. Whatever the final conclusions of the commission might be, its report being without bias or prejudice gains a certain weight and dignity before the judge and jury.

In submitting its reports, some of which were to be used in actual trials before the average juries such as are commonly found in the criminal courts, the commission was confronted with the fact that its reports must be as untechnical as possible and must avoid all complicated discussions which might be concerned with the strictly scientific side of the subject, and must show nevertheless that the important questions which might be brought out at the trial were amply considered. For these reasons the reports were of two different sorts, one contained a simple opinion without explanation, and the other contained a short review of the case aiming to give the jury a clear idea of the case from the standpoint of the examining body. Of course if the case were once brought to trial the report of the commission might not be accepted as evidence, and in this instance the opinion of the commission might be used to help the circuit attorney in his work of prosecuting the case. A little experience in criminal trials in which insanity is brought forward as a defense makes it quite clear that the question really resolves itself into the consideration of responsibility, not in the psychological sense,

but in the sense of which responsibility is used in the law.

Correct diagnosis is not so important as a careful consideration of the criminal's ability to appreciate the nature of the crime committed, its consequence to himself and society, and his ability to distinguish right from wrong in the ordinary sense. From the point of view of the psychiatrist this is unfortunate, but if he is to be of service to the State he must find a way to reconcile these divergent points of view so that he may approach as near as possible to that absolute justice which the ideal of the law holds forward. The idea kept constantly before us was to make our examinations with all possible care, and to use all ordinary means to arrive at a correct clinical decision of the case, and from such data to consider the individual from the standpoint of the law. In doing this the avoidance of mooted points and the uselessness of long discussion upon the complicated psychology likely to be present in such cases was made necessary. It was a surprising experience to us to find how simple our task became when the limits of our problem were so plainly set down. By starting out without previous opinion and by trying simply to find out if a given individual is responsible for a deed committed, is not nearly so difficult a task as to be asked to aid the defense or prosecution of the same individual on some theory advanced by the lawyers of either side in regard to the question of sanity.

In the course of the year we have had to consider a number of cases of complicated mental disease, but in no single instance did we fail to satisfy our conscience on the question at issue. In one single instance we refused to intervene in a case of murder when the individual was obviously a type of degenerate criminal, for whom further examination would have done little, since he was condemned to be hung and had already been declared responsible by one jury. The recognition of our limitations and the feeling constantly before us of the responsibility in each single case kept us always within the bounds of logical inquiry, and prevented us from yielding to the temptation of viewing our problem as merely one of mental diagnosis. I need scarcely add that we always availed ourselves of all the means of physical examination at our disposal, the data derived from careful personal and

family histories, and such other aids as are commonly used in neurological and psychiatrical examination. Such data naturally were rigorously excluded from the reports submitted to the circuit attorney.

The following two reports illustrate the foregoing remarks:

The first case refers to an alleged robbery with assult performed by a nurse employed in a quack cancer cure establishment. The nurse, one Blanche Somerset, when discovered by the police was found tied with curtain ropes and considerably scratched and cut by some blunt instrument. She denied having any recollection whatsoever of the occurrences which led up to the crime for which she was arrested. This case had attracted considerable attention on account of a diary belonging to the prisoner, and which the police gave to the newspapers, and likewise because more than one of the newspaper alienists had expressed the belief that the crime was a result of some uncontrollable impulse. The report of the commission is as follows: In accordance with the instructions contained in your letter we have examined into the mental status of Blanche Somerset, a prisoner at the City Jail, on charge of grand larcency.

Realizing the importance of the case, its publicity and the fact that an opinion had already been expressed as to her sanity, we felt that any decision that we might reach must be based upon facts and deductions from them, and not upon theories which any of us might hold in regard to the mooted questions of psychology always present in cases of this kind. Our opinion as given below is therefore based upon the following sources of information.

1. A complete serial history of the life of Blanche Somerset for the five years preceding the commission of the crime, relating to the chief events in her life. Data in regard to her family history in respect especially to the question of mental and nervous heredity.

2. A careful study by each one of us of a diary kept by her and containing almost daily notes for three or four months before the commission of the crime. The last note was made the day before she was arrested. Clippings chiefly relating to suicide and murders and note books on various subjects. (These were kindly furnished by the police department).

3. Interviews by one of us and examination made by the whole commission.

4. A physical examination as complete as circumstances would allow with especial reference to the nervous system.

A careful consideration of the facts so ascertained leads us to the following opinion in regard to the mental state of Blanche Somerset:

We find first of all that the prisoner has an absolutely reasonable and normal conception of the nature of the crime she is charged with. She is perfectly aware that such a crime is punishable, and furthermore she is perfectly aware that whatever her punishment may be, that it is the logical outcome of her guilt. We find that the crime itself was deliberately planned, and that there was a well defined motive or motives for its performance, namely financial necessity and dislike and jealousy of the individual robbed.

We find that at the present moment the prisoner shows absolutely no trace of mental disease. She is logical, intelligent and remarkably self-controlled and perfectly aware of her surroundings and her part in their causation.

We find that for a period of three or four months immediately before the commission of the crime she was as normal mentally as she is now.

We feel justified, therefore, in saying that for this period before the robbery, and during the commission of the robbery, and since that time there has been no evidence weighty enough to cause us to question either the legal or the medical aspect of her sanity.

We have carefully considered the possible influence of functional diseases of the nervous system as bearing upon the prisoner's responsibility. We have found, however, no sufficient evidence for the diagnosis of either hysteria, neurasthenia or allied conditions. There have been at least two instances of what may be considered imperative acts. The addiction to the use of drugs, especially morphine and alchohol, might be used to justify the assumption that this individual was the subject of various uncontrollable actions. If, however, these facts be carefully analyzed it will be found that they have little or nothing to do with the question at

issue. They cannot in any way be so interpreted that they would weaken her responsibility before the law.

Our opinion, therefore, is that Blanche Somerset is not insane, and that she is mentally responsible for her actions, and that from a medical point of view she is responsible for the crime which is charged against her.

The second report is concerned with the study of a young man who ran amuck in the streets of St. Louis and cut a number of women in the thigh and back, inflicting only slight wounds in each case. This man was soon given the name of Jack the Stabber and attained a great deal of newspaper notoriety. The alienists who find the daily press their best public, advanced a number of theories to explain the acts of this man, and it was pretty well believed that his crime was the act of an insane individual acting under the influence of some mysterious power which the papers only hinted at.

It can be seen from this that the report of the commission would have to meet an opinion already created in the press by the alienists, socalled, who made public their ideas before the case had been seen by anyone. Inasmuch as our report would not be accepted by the prisoner's lawyer we were forced to render our decision in more technical language than usual.

The following is our opinion in regard to John Lawrence Brady, a prisoner in the City Jail, charged with feloneous wounding:

We find that John Lawrence Brady shows no evidence of insanity at the present moment. He is to be considered as mentally responsible within the meaning of the law, and he is sufficiently intelligent to undergo trial for the offense he has committed. Owing to the fact that his crime has no apparent motive we suggest the following in explanation:

The crime may have been the result of one or a combination of these four conditions: 1. Sexual perversion; 2. Imperitive act; 3. Epilepsy; 4. Alchoholic temporary excitement.

For the first there is not the necessary evidence. The second seems to us insufficiently proven by the facts obtained. There is absolutely no data upon which to base the third. The fourth either alone or as a complicating factor in the second opens the possibility of a reasonable explanation.

We suggest, therefore that John Lawrence Brady be tried as an individual mentally responsible, and that the possibility of alchohol being the exciting factor be left to be determined by the facts as elicited in the trial of the case.

These two reports are sufficient to illustrate the manner of placing our opinion before the circuit attorney.

It is difficult to set down in a definite way the results of the first year's activity of this commission, because it is highly probable that its effect, if any, will lie in directions not at present discernable. An effort like this has many ways of making itself felt, and it cannot be determined just where and how it will eventually work out. This much can be said with perfect fairness. The judges and most of the lawyers whose work brings them in contact with the criminal courts have encouraged us to persevere in the work we have started. We have not received any special encouragement from the legal profession as a whole, or, at any rate, from such of them as have been consulted about the matter. They see in the activity of such a commission an infringement of the right which is given them by law and custom of summoning such witnesses, expert or otherwise, as may prove of strength to their side of the case without reference to the strictly medical aspect. To place the decison as to the defense of insanity altogether in the hands of a group of unprejudiced experts belonging to the court itself would be a serious infringement on the pleader's prerogative.

In no single instance has the report of the commission been rejected, and in those cases in which the defense has refused to accept the judgment of the commission and have brought the case to trial, the report or rather the opinion of the commission has finally prevailed. I believe that this result has been obtained not through any remarkable work on the part of the commission, but solely because the opinion of the commission has impressed the jury and the judge as being absolutely impartial, and as being concerned only with the facts of the case as brought out in the examination of the accused.

Omitting the advantages which each individual of this commission has obtained in work of this nature, unhampered as it is, the chief effect, I believe, so far obtained can be found

in the better opinion which expert testimony on mental subjects has been able to exert in the Criminal Courts. For the first time they are in a position to hear testimony on the subject of insanity and allied questions which bears no relation to the defense or prosecution, but is concerned only with the question at issue. In this way perhaps a better standard has been set which may afterwards bear fruit. The one tangible result has been the establishment of a small observation ward in the jail hospital for the use of the commission in the study of cases suspected of insanity.

As was said before, the commission has no legal standing as at present constituted, and it can be dissolved at a moment's notice if the judge should refuse to consult with it or if the circuit attorney should decline to respect its decision. Its precarious existence has been the subject of some debate as to the possibility of passing a State law establishing such a commission legally with the proper standing before the courts. We have up to this time opposed any such effort, for we fear that if it becomes a State institution the question of politics will enter into its activities, especially if its services are to be adequately paid. We are content at present to let the work it is doing effect in as large way as possible the procedure of the courts in respect to the employment of expert testimony, and leave the solution of the permanency of such a commission to other hands.

In conclusion it may be said that the voluntary and unpaid service to the State has appealed to us as being in line with the larger view of civic responsibility which physicians feel bound to share, and from which until of late there has been considerable shrinking. It is a part of a widespread movement which is tending to remove the isolation which the profession has held to the community of which it is a part.

CONSCIOUSNESS IN THE BRUTES.[1]

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(*Continued from page* 41.)

Such are some of the considerations which tend when taken together to deprive the nervous system of its preeminence as the representative of consciousness in animals, a supposition which naturally arose from the former's peculiar function of being the chief means of associating impulses passing between the really active tissues of the body and between their parts. On the other hand certain events in the realm of chemiphysics have recently altered somewhat our view of matter itself and this alteration bears in a sense on the basal relations or unity perhaps of mind and body. Matter is proven in certain cases at least to be not something fixed and immutable but rather centers of spontaneous forces active despite all known conditions, and the tendency is undoubtedly to place force where formerly matter stood. In biology the same tendency has long been felt, for it is seen that those reactions we summarily call vital are indiscriminately composed of heat, movement, electricity, light, etc. But now that similar phenomena are seen to take place in inorganic substances as well as in protoplasm, it is obvious that little is left of the "substantial" part of matter. Just as a limited and rationally forced pan-psychism is in the philosophical air, so this pan-energism or pan-kineticism (if I may be pardoned the rude terms) is in the physical air. To the one view (the substantial rational seeing of Idealism) nervous processes as well as mental processes are psychical and the body itself is sentient experience. To the becoming physical view, matter is something akin to a vortex of kinetic energies and the brain, in the last analysis, becomes reduced to the powers of chemiphysical attraction and repulsion, of heat, of electricity, of light, of chemism, modes of movement. Both to physics and to epistemology protoplasm is a somewhat whose chief characteristics are movement, change, indescribable because unique but identical in terms of consciousness. Deny it

[1]Read before the American Philosophical Association in Philadelphia, Dec. 29, 1904.

as one may with wealth of dialectic, with statement heard so often that "force and movement are not consciousness but force and movement," still consciousness does seem more like these various forces to which protoplasm bids fair to be reduced by physics than like the lumps of white and greyish matter from out the interior of a bony skull, and to this extent the becoming view of physics and the liberal view of consciousness approach each other. If consciousness is not a force still it has some of the qualities of force as we know it,—and no matter what force is as we don't know it! Is this heresy to orthodox idealism, to claim the forces of protoplasm and the thing we call consciousness alike in some respects and to base on this similarity an increased claim for the wider locus of consciousness? At any rate, the sole contention is that the becoming conception of protoplasm including nerve as made up of molecules or of clusters of molecules which are, even physically, reducible to centers of chemic force, makes it somewhat easier to conceive of some sort of correspondence between consciousness in animals and the mechanism of their life. The difficulty is not to see this this way but rather to persist in spite of basal resemblances in maintaining a contrast based alone on a logical syllogism. Above, we urged the usefulness of this very syllogism at the basis of idealism—at present we urge its limitations when the basal paradox of our experience demands that we should do so. A few years ago most physicists would have claimed the doctrine of the conservation of energy infallible in our experience; to-day he may see hints of phenomena which relegate it perhaps to the limbo of other tottering theatrical idols! If simple metals exhibit phenomena of this sort, surely we may expect from a substance as highly elaborated as living protoplasm relations at least as aberant from the supposed laws of matter. And protoplasm finds only one of its forms, and that apparently not the most active chemically, in the protoplasm of the nervous system.

The structure of protoplasm, of late gradually made out conjecturally at least, offers a prëemiently appropriate and adequate seat or representative for consciousness—its uniqueness and the uniqueness of sentient experience correspond. Let us glance at some of what are probably the salient features of this substance bioplasm, biogen, or protoplasm, "life-stuff," "life-bearer," "primal matter." Each year enlarges our knowledge of

it as obtained through the microscope, and continually its general chemiphysical nature becomes better known. Into the theories of its morphology we need not go, for surely it is its basal chemic constitution and not its physical structure which underlie its relations to consciousness. If biochemistry has learned anything surely about the structure of biogen it is that the group or cluster corresponding to the molecule of inorganic matter is of unique complexity and of a maximum instability. Among the constituents of this substance and probably of every cluster composing it are three materials organic in nature and each of great complexity. These three, of course, are proteid, fat, and carbohydrate, and proteid constitutes nearly the whole. Besides these there are water and an unknown number and variety, very large, of salts which elsewhere are termed "inorganic," here doubtless part of the biomolecule, or life-unit. Of these five classes of constituents of this vital unit every one is a compound and three at least of them of great complexity, especially so the proteid. No two analyses of the many forms of this substance are alike and no one at all represents the original substance, for chemical analysis inevitably kills it and its structure chemically depends on its life. There is no doubt that all of the proteids contain hundreds of "atoms" in their units and many of them thousands. To account for the spatial arrangement of these thousands of atoms the imagination must be actively employed, but the problem is simplified by considering the "atoms" stresses and strains, forces, rather than as space-filling material bodies. The fats of the vital molecule and the carbohydrates are also complicated in molecular structure with scores of "atoms," and in instability only secondary to the proteids, while the inorganic salts form combinations with these in the life-unit group which are quite unknown. All of these "atoms" are continually interchanging as the vital unit anabolizes or katabolizes, and not only interchanging with themselves but with other more or less similar complexes brought to them as food. Of all the "molecules" known to chemistry, this one, probably, at the basis of protoplasm and of life is the most complex in its ever-changing construction and probably has as its essence a metabolism quite unique. This metabolism, these most intricate chemical interactions within and between these vital clusters making up protoplasm can be occasionally *imagined* by the chemist who knows the nature of chemical reac-

tions generally and the materials and end products of these vital processes, but they cannot be described by him. From them are set free motion, heat, light, electricity, power of various sorts, phenomena we call forces. Nowhere else than in the living tissues of animals are the causal or the effected conditions so complex and inter-involved, and above all so capable of permanent adjustment to varying relations. Such a uniqueness is fit concomitant for the uniqueness of consciousness. This intricacy of forceful interaction would seem to supply the only apparent basis for the intricate phenomena of the mental process. On the one side a living body made up of events like these implied, almost too complicated to be even suggested by other than a biochemist — on the other side the indescribable experience consciousness. Only these two of all finite things are reasonably adequate to mutually represent each other it may be.

And there is at hand an illustration of this relation which is more than rational fitness and probability. That protoplasm, bearing no immediate relation whatever to any nerves, is capable none the less of representing and of containing within its activity at least latent psychical characteristics is seen by us all continually in the fact of the heredity of psychical characteristics — or else consciousness is indeed the secretion of the brain and La Mettrie was right and Leibniz and Spinoza, Plato and Hegel quite beguiled. The extremely minute speck of protoplasm constituting the male element in reproduction carries in its molecular structure or activities not only the physical but also the psychical peculiarities of the father of the new individual —or else we must retire to materialism. Strength of will, temperament, affections of various sorts, even habits of thought (such, for example, as those which lead to suicide in successive generations), all are commonly transmitted, besides a host of characteristics as much mental as bodily. Twins are often nearly alike not only in psychophysical but in purely psychical respects. There must be a basis for these aspects of consciousness; no other basis is transmitted than the speck of protoplasm; hence this speck must be the basis required —the climax of human mystery. The interactions between the units and within them, kinetic relations in the last resort perhaps, stand in the spermatozoan for psychical events. If so many and so various determinants of consciousness inhere in a male pronucleus, where nerve is not as yet, why then

not a portion of consciousness in the quite comparable protoplasm of the active tissues of a developed being? No theory of heredity as yet suggested attempts to account for a relation of this nature, just as no details of a doctrine of psychophysical parallelism will be expected when the consciousness of protoplasm in general shall have become recognized as an element of its vitality, basally inherent in it.

The phenomena of sleep and of its analogues strongly implies that there is more in consciousness than the nervous system can adequately represent. Unconsciousness is not to-day a scientific any more than it is a metaphysic verity, for grant a nervous break between the waking and the sleeping consciousness (in sleep normal, in coma abnormal, in sleep easily bridged, in coma seldom or never), and with exceptions the sleeping consciousness has the extensity if not the intensity of the waking experience. These exceptions are especially the senses of seeing and hearing, which being in part closed off leaves us nearly the sleeping consciousness. Sleep is particularly the resting of the nervous system, as measurements show, while some of the bodily metabolism is likewise decreased. When the nervous system rests the person "loses consciousness," the mysterious break between the two states gradually or suddenly occurring, yet no person is awakened suddenly, unless too violently, without finding himself in the midst of some sort or other of mental experience, often with all the characters of the waking consciousness minus the overwhelming experience of flooding daylight and of universal sound. Such a mass of consciousness is not perhaps adequately represented by the metabolism of a few grams of grey nervous matter in the cortex cerebri but only by a general bioplastic metabolism in the mass of the body cells which know no rest corresponding to the sleep of the individual. An increase in the metabolic activity of any portion of the body-protoplasm is apt to at once break into the sleeping consciousness, oftentimes with vigor sufficient to recall the waking consciousness by stimulating the nervous system out of its repose. In sleep the sub-conscious mass of experience predominates, represented as it is perhaps by the somatic or protoplasmic, as distinct from the nervous, portions of metabolism.

We have now reviewed certain considerations which tend to connect the activity of animal protoplasm in general with con-

sciousness in general. In this vague relation is a "correspondence" which is not subject to the close limitations of a supposed parallelism between consciousness and neural tissue alone. If the correspondence cannot be worked out in detail neither can the details of the events themselves in either of the two correlatives concerned, while on the other hand idealism supplies a logical hypothesis of their relation if not one to satisfy psychophysiology. Now all these complex protoplasmic conditions are found in every sort of animal tissue in some degree or other, the degree depending in general on the respective activity of any tissue in an organism. Thus bone and connective tissue for example have little epithelium and nerve and muscle much. It is of these various sorts of protoplasm that the bodies of all the animals we know, brute as well as human, are composed. The protoplasm of all, so far as we know, is of equal chemical complexity, and as richly endowed with the interactions which form a reasonable basis for a material fit for accompanying consciousness. Thus for example the ameba, simplest of animals, lives on protoplasm as its food, and the enzymes which digest it and the chemical processes which make these enzymes are doubtless as complex as those which make them in a human pancreas. Again in the ciliary movements of many of the protozoa one can see (without seeing a trace of nerves) as perfect cöordination and adaptation to a complex purpose as complete as is to be found anywhere in man himself. Man but shows his egotism when he claims himself superior on any biological standard to the simplest of animals —the true measure is rather one of protoplasmic fitness, or else one quite outside the range of biology, namely in ethics. in the possession of a will free to do right or to do wrong. Looked at broadly, unbiased by our human habits of thought, what reason is there, if these statements are facts, to assume as preliminary in every discussion of the consciousness of the brutes, that the consciousness of each species and of each individual even, corresponds to the complexity of its nervous system? Back of any possible correspondence between organic process and mentality is the unique complexity of chemic protoplasmic activities and this is present in all animals so far as research has shown in equal degree. It is a matter of the existence rather than of the quality of consciousness in any form of life, the quality and the intensity as well as the extensity of the mental processes depend-

ing undeniably on the extent to which the division of bioplastic labor has gone in any particular form or individual.

We have above offered a number of presumptions, but certainly there's more than a reasonable "presumption" of the existence of consciousness in the brutes, especially from considerations like the following: Says Mill, "When two things resemble each other in one or more respects and a certain proposition is true of the one, it is probably therefore true of the other," and all the whole world so far as rational at all works and lives on this principle of analogy plus the calculus of probability. The person skeptical of brute-consciousness knows himself conscious and knows more or less of his own body's structure and the mode of that structure's life. This is the "certain proposition" which "is true of the one." Now here, instead of there being "one respect," in which the analogues brute and man are similar there are thousands of respects, there are almost as many scattered up and down the animal series as there are known facts in the physiology of animal life. The conclusion is logically, that is to right common-sense, all but a syllogistic certainty. Outside certain innate mathematic analogies there is hardly any probability greater than that the most evolved brutes are conscious, man being so. Muscles, nerves, and glands, all are constructed similarly, act on the one same plan, serve the same biologic purposes —the muscles to achieve movements, actions, the nerves correlation and adjustment, the glands bodily supply, the other tissues for more mechanical service as groundwork of the rest.

The sceptic who admits his own consciousness but denies it in the dog and elephant and lion has one resource which he is bound to make use of continually. He can deny the principle of continuity in organic evolution and claim that the protoplasm of man is essentially different from that of all other animals, and that the difference is such that consciousness may inhere in it and not at all in the brutal organism, the consciousness accruing to it suddenly as if out of Pandora's box. So arbitrary, however, is this supposition, so gratuitous in the biology of protoplasm that it may be disregarded, the burden of proof resting wholly on its claimants. So far as every evidence goes, structural and functional, the two classes of animals compared, brute and human, are built on the one same plan, live in the light of the same consciousness save probably for ratiocination which has perhaps de-

veloped with the vast complicating of the connections of the fibers of the brain and the consequent development of abstract symbols into speech and language. The burden of proof in this matter rests with the deniers of the analogic probability.

Now going along the multitude of animal forms toward the simplest animal from the most human, where can a line be drawn beyond which consciousness may be disclaimed? With wonderful morphologic variety there is striking biologic uniformity, the same use of the same mechanical principles and of chemical reactions and assuredly of protoplasmic nature generally, so that even in ameba, the simplest, indeed the logical limit of animal structure, we find the same events in type and the same means for producing these events —metabolism in irritable living protoplasm. Even in ameba is that same amazingly complex protoplasm whose chemiphysical interactions science is beginning only to unravel. Cöordination in ameba is poor and we find him sometimes trying to crawl in opposite directions for a brief space, for of nervous matter there is of course nothing. For the same reason the animal's adjustment to external conditions is imperfect, although in the main protective of its life. But all the varied metabolism is there, giving rise to the same sort of heat, motion, and probably electricity, from the same protoplasmic life, supplied by the same nutrients and giving off the same katabolic waste as does a nerve cell or any other cell of a human body. Without a single nerve-fibril the naked protoplasm conducts impulses, as one may quickly see when the whole animal nearly at once contracts after stimulation, and cöordination and even adjustments occur without anything which can be called nerve. Without muscle contraction takes place. Without separate gland-tissue secretion goes on. Without reproductive cells the marvellous so-called simple division of the animal occurs whenever its overgrowth demands, a process compared with which even mitosis is easy to understand. In ameba then, the logical limit of animal simplicity, a minute drop of uncolored streaming protoplasm, the matter is organized as forces which interact and are mutually and self-sustaining. The adjustment of relations which is its life is nearly as perfect as in other animals and because the chemiphysical process at the basis of this series of adjustments is perhaps even more complex than elsewhere (since every function nearly

inheres in every part), a modicum, a trace, a sample of consciousness must be supposed to be concerned.

What can be supposed as to the nature of this requisite consciousness of ameba? Shall we imagine it like our own? Of course not. Can we imagine it at all? Scarcely so, it must be allowed, and yet, "the dim crawling life" is part and parcel of a system of uniformities in which we ourselves and our own consciousness partake and within these limits we can imagine much. It is customary for descriptive psychology to say that the most prominent aspects of mind as we know it are feeling, will, and thought. The first, feeling, is based wholly on sensations, on modifications of consciousness which seem to have to do with that protoplasm, which in man and his congeners is made up as sense-organs. Ameba's protoplasm and that of other unicells is universally sensitive to irritating stimuli, and its irritation causes reactions exactly comparable to those of protoplasm elsewhere under the influences coming from sense-organs. A dim and simple sensation-mass would seem then to be a reasonable basis and substance of ameba's consciousness. Besides this there are obviously elements of which we know as will or action and this involves that something else, that "certain plusness," not to be defined but representing perhaps in its last analysis the life itself, action, active purposiveness that quality by which the interactions of ameba's molecules exceed in complexity and in self-adjustment those of other self-regulating materials—if indeed any such besides bioplasm exists. Another word for will is conscious spontaneity and possessing this the animal may still be as much the slave of circumstance as you please for its dependence on its environment is extreme. Sensation-mass and will, conscious spontaneity, such we may imagine are present in ameba's consciousness. In these nerves are unnecessary, and their physical basis is adequately enough the universally complex, irritable, and active protoplasm of which the whole body is composed. So much for this logical type of animal, a speck of undifferentiated bioplasm. In this form we are interested only for these very reasons —that it is the logical type and it is undifferentiated protoplasm only.

In the series of animals from ameba "up" (no scale save a very indistinct one can be made out), we find at once development in the way of a division of labor. Scarcely are we out of the

protozoa, the unicellular animals, before certain cells of the various organisms begin to take on distinctly the qualities and uses of nervous tissue, the first form being cells with a new vigor and promptness of reacting to stimulation, it being therefore the sense-organ part of the nervous system which appears first in the phylogenic development. In this simple plan these sensitive cells are also means of defense. In animals a little more complicated we find the rudiments of a nervous system entire, the first form being a mere line or lines of fibrils, with the necessary nerve-cells, connecting parts required to work in unison for some reason or other, or where adjustment of any sort is necessary, functional connection being the business of these fibers primarily and always. In forms of life more and more complicated, the nervous system's reticulum of fibers shows corresponding development, not only in actual intricacy, but in relative mass. Thus in a way the nervous system comes to be a rough general index of the complexity of any animal's life, of relations more and more involved, not only between cells and organs within the limits of the animal, but with numberless conditions outside the individual yet part of its environment and in a biologic sense inseparable from itself. Conduction, cöordination, adjustment, appreciation, become more and more complete as the vital conditions demand. Small portions of the protoplasm develop into sense organs and the corresponding sensation-mass of the animal we may suppose becomes "larger" and richer with inevitably a tinge of feeling in its nature. In very simple forms muscular tissue of a simple sort has already developed, actions thereupon are made more promptly and in a more perfectly adjusted way and therewith the will has developed also in richness and in strength, as represented by what the various animals variously *do*.

As a glance backward over the literature on the will quickly shows, the theories of its nature and of its relation to organism on the basis of the phenomenal dualism are many and complicated, for in this notion will or volition is involved the heart of the question as to any sort of correspondence. If we think of will as found in the brutes as spontaneous activity we cannot be far wrong, but if we do so, we at once express the unification of the mental and the bodily. "Will" at least, whatever "sensation" and "thought" may mean, means nothing whatever unless within it is included bodily activity. Here then is the empirical or scien-

tific corroboration of the epistemological viewpoint and for once the two views are in fact unified, as well as logically. The scarcely experienced "certain plusness" or fiat in the willing-process as we describe it psychologically appears phenomenally as the *activity,* kinetic, chemic, atomic, molecular, and molar, of the components of protoplasm. In no other way is the fiat, the relation of the two aspects, experienced, for all the rest of the volition is sensation arising from this activity.

In this way of thinking the sensation-mass of the brutal experience joins with an almost intelligible activity in the protoplasm and in the environment its sensations represent to constitute the larger part of its consciousness. It is only by this free and easy transition back and forth between physical and psychical, between protoplasmic activity and experienced will and sensation, that any sort of explanation is forthcoming as to the relations of body and soul. We must, really to explain, merge the scientific and the epistemologic, the phenomena with the sentient experience of the phenomena. To the dualist even this degree of explanation seems forever forbidden.

But if sensation and volition constitute the by far larger part of the brute's consciousness (as of man's) what of cognition, the faculty of knowing, an analytically distinct aspect of the mental continuum? Scarcely yet have we escaped the exaggeration which the associationists forced into psychology and man's pride is reluctant to admit how small the proportion of cognition and especially of constructive conception in his mental process minute by minute. Who can doubt that in the brutes the proportion of conception is not much smaller still?

If cognition, understanding, thought, etc., be a comprehending of relations (and little else when the sensation of perception are taken out), perhaps it is not extreme to suppose that on the bodily side this comprehending, or taking together, will imply the proportional development of those organs which receive, collect cöordinate, compare, and adjust. No other organ or system in animal bodies except the nervous system performs these functions, the sense-organs the nerves and the nerve-cells.

Whether the relations comprehended be derived from stored traces in the grey matter of the nervous system or be brought in anew to it from without the process is one peculiar to nerve, and only suggestions of the faculty exist in protoplasm itself,

other than nerve. If ameba collects, cöordinates, and adjusts, it does these slowly and imperfectly as compared with more evolved forms of possessing nervous systems. Whatever then our compelled notion of ameba's consciousness must, we can think of its cognition only as at the logical limit, the vanishing point of reality. As the nervous system and its various sense organs, however, become more complicated, the comparing and cöordinating powers of the animal become more adequate. The fibrils making up the system become general in the other tissues, ramify more widely, gradually connect more universally, while their knots, the nerves-cells, perhaps also distributing centers of nutrition, probably become more complicated, although less numerous relatively. The universal neural reticulum is connected not only with more sense-organs but with more kinds of sense-organs themselves always more and more complex as we look up the series of animals. Becoming progressively greater in mass in proportion to the grey matter, the conducting paths become also more complex and their interconnections ever more involved, thus representing more and more perfectly the consciousness which so far as expression and conduct indicate becomes also more and more complex. If the grey matter of the nervous system represents the switching and adjustment of impulses, besides nutrition, the white net of fibrils stands for the essential cöordination of bodily influences. In the simpler animals there is neural simplicity in every way apparently, the complications coming later being not only in connection but in structure—a condition which neither use nor observation necessitates as true as regards the body-protoplasm generally. Thus with developing nervous systems, the animals up the animal series gradually acquire the comprehension of associated relations which little by little approaches the faculty we see evinced in the most knowing of our brute friends and victims. One sees well enough that rabbits and sheep are less intelligent generally than are cats and dogs, for example, and the ant's faculty of overcoming difficulties far exceeds that of the cray fish. These differences have a physical basis in the proportional quantity of grey matter of the nervous systems and, as the physiologist knows, in the way the nervous matter is arranged in the brain cortex and in the nerve trunks sometimes. In general, in short, one can see a correspondence between the complexity and mass of the nervous system, and especially of its grey matter (the comparing part) and the cog-

nitive processes of the animal. Knowing then corresponds to the nervous system because reception of relations, and their cöordination, comparison, and adjustment, are at once the basis of cognition and the special function of this system alone.

But understanding and adjustment to simple new relations such as we see in the most knowing of the brutes does not mean necessarily conception and abstract thinking, and self-consciousness arising therefrom. The recent work of Stumpf and his colleagues on the calculating horse Hans shows these relations well. Here is an animal which probably through an unusually plastic or extensively ramifying nervous system has developed a faculty of observation especially of its questioner's face which seems to equal that of the most expert of human physiognomists. Perception and recognition form the adequate basis of this brute's unusual powers and the investigators could find no trace of any appreciation of any numerical concept not unified with the percept of the questioner's face. The orang outang, Joe, which visited this country seven or eight years ago left a quite similar impression on the minds of many of his psychologic observers. No proof then of conceptualization in the brutes is to be furnished either by the animals themselves or by their human apologist. It is not enough to be able to see in the deep eyes of a fine setter dog expressions sometimes like those of man under like conditions, for these expressions arise rather in what one might call an emotionally tinged simple cognition than in an abstract conceptual experience. Indeed, if both theory and observation may be trusted no sort of cognitional function beyond perception rendered more useful by memory, and so going on to recognition often with an affective tone is to be proven or evidenced by any brute's expression or conduct.

It was from this sort of consciousness perhaps as a beginning when speech had been acquired (perhaps by a Mendelian "chance" variation in heredity), that man in the course of millions of years has developed special limited mental processes whch have made invention and through that civilization possible. In the boundless multitude of the abstract notions humanity has thus acquired one sees represented, very possibly, the size and complexity of the hemispheres of man's brain. This difference in consciousness, probably the greatest to be found in the whole range, is sufficiently accounted for, it may be, by the development of language—but even this blessing may be over-done!

Periscope

Deutsche Zeitschrift für Nervenheilkunde.

(Band 27, Heft 5 u. 6.)

17. Contribution to the Clinical Knowledge and Anatomy of Progressive Facial Hemiatrophy. LOEBEL and WIESEL.
18. Studies Upon Oppenheim's Feeding Reflex and Some Other Reflexes. FÜRNROHR.
19. Mikropsia and Related Conditions. HEILBRONNER.
20. Contribution to the Question of Bladder Disturbances of Spinal Origin. BERGER.
21. The Anatomical Findings in a Case Described as Hemorrhage Into the Right Half of the Pons, Etc., from the "Ram. central. arter. radicular u. facialis dextri." WALLENBERG.
22. Investigations Upon the Nature of the Reflex Hyperesthesias Occurring in Disease of the Organs. PETRÉN and CARLSTRÖM.

17. *Progressive Facial Hemiatrophy.*—Hitherto 170 cases of progressive facial hemiatrophy have been published. Autopsies have been performed on only six, and of these, five apparently indicated the existence either of severe complications or of other conditions. The case reported was that of a woman who, at the age of 14 years, had had a discharge from the right ear. At the age of twenty-two she began to have pains in the head, and the atrophy of the left side of the face began. Twelve years before death Nothnagel demonstrated her as a case of progressive facial hemiatrophy. The examination showed loss of fat and muscle, and atrophy of the bones on the left side, with preservation of the hair, sensation and vision on that side. She also suffered from pulmonary tuberculosis, from which she died. The histological examination of the brain and cord was negative. Aside from the atrophic tissues definite changes were not found in the other organs. The left Gasserian ganglion showed a variety of changes, particularly focal areas of round-cell infiltration, with some proliferation of connective tissue. Changes indicating chronic neuritis were also found in some of the branches of the fifth nerve. The sympathetic nervous system was not abnormal. The muscles affected were those supplied by the left motor root of the fifth nerve. The authors believe that in all likelihood pregnancy caused an autointoxication which injured the fifth nerve on the left side.

18. *Feeding Reflex.*—Oppenheim's reflex consists of a rhythmical tasting, sucking, chewing and swallowing movements, following irritation of the lips and tongue in young children. Henneberg's hard palate reflex consists of a powerful contraction of the orbicularis oris when several vigorous strokes are made upon the hard palate. It occurs in cases of bilateral focal disease of the brain, and occasionally in cases of hemiplegia due to brain tumor. The buccal reflex of Toulouse and Vurpas consists of a pursing of the lips when a blow is struck upon the upper lip with a percussion hammer. It occurs in new-born children, in cases of anencephalia, paretic dementia, chronic alcoholism, and in idiots. Oppenheim's reflex was investigated upon a number of children. It apparently begins to disappear in the eighth month. In older children it is observed in cases of spastic diplegia, and twice in adults suffering from epileptic coma. Fürnrohr also reports some other interesting cases in which it was present either typically or atypically. The reflex is probably the result of the irritation of two special centres in the brain, one subcortical, which may be described as the true reflex center, and one in the cortex, which has

merely an inhibitory action. In regard to Henneberg's reflex, Fürnrohr found it present seven times in 124 healthy children. In 138 cases of nervous disease it was present 29 times in typical form, and 20 times atypically. Of the 29 cases, 19 were hemiplegias variously complicated, and the others presented epilepsy, multiple sclerosis, paretic dementia, encephalo-myelitis, Little's disease, infantile pseudo-bulbarparalysis, general tic, psychopathic deterioration. The histories of a number of cases are given. Frequently crossed reflex was present. Fürnrohr does not agree with Henneberg that this reflex is closely related to that of Oppenheim, but believes that it is a mucous membrane reflex whose paths are in the trigeminal and facial nerves, and is probably physiologically similar to the anal reflex. The buccal reflex occurred in 12 of 20 children during the first week of life, and of these 12 children the majority were less than five days old. It was also present in all of 28 cases of paretic dementia. It was also present in 60 of 70 cases of idiocy, or epilepsy associated with dementia. It was likewise found in cases of alcoholism, hemiplegia, pseudobulbar paralysis, Little's disease, acquired hydrocephalus, multiple sclerosis, cerebral syphilis and neurasthenia. This reflex appears to be due to some irritation of the cerebral cortex. It is probably not a true reflex action, but appears as a result of the irritation of the peripheral nerves; therefore it cannot be regarded as a true reflex.

19. *Mikropsia.*—The patient, 27 years of age, had a subjective sensation when reading that the book of paper moved away from him to an infinite distance. The type, however, remained perfectly clear, and he could continue his reading. He also suffered from dizziness in all those situations in which it is likely to occur. After a brief analysis of the case, and some discussion of the nature of the dizziness, Heilbronner concludes that, in addition to the subjective removal to a distance, with apparent diminution in the size of the object (micropsia) there may also be a subjective removal to a distance without diminution in the size of the object (porropsia); that both these may be combined with disturbance of the apparent position and movements of the object, and that they are probably due to some diseased condition of the cortex. These forms are to be carefully distinguished from those occurring in disturbance of accommodation. In epilepsy they may replace the epileptic attack.

20. *The Bladder in Spinal Diseases.*—Berger reports two cases of men who had received severe injuries to the spinal column, and who, as the chief result thereof, suffered from involuntary dribbling of urine. In the second case there were also attacks of forcible micturition. The sensation of a full bladder was not present in either case. In regard to the nature of the lesion, it seems unlikely that we can consider this symptom as due to a traumatic neurosis. In the first case it seems likely that the fifth lumbar and the first sacral segments were involved, and possibly some of the lower sacral segments. The absence of rectal disturbance indicates that the fifth sacral segment at least was free. In the second case the lesion probably involved the cauda equina, and affected not only the fibres to the bladder, but also those to the sexual organs, but not the nerve fibres controlling the rectum. These cases seem to controvert some of the theories of Müller regarding the symptomatology of lesions of the lower portions of the spinal cord.

21. *Hemorrhage in Pons.*—Wallenberg describes a case which he diagnosed as hemorrhage from the central branch of the root artery of the right facial nerve, which had caused destruction of the right half of the pons between the sensory fifth nucleus, the trapezoid body, and the root of the abducens nerve. Subsequently the patient died of pneumonia, and a lesion was found in the right half of the pons which consisted of a cyst, evidently the result of an old hemorrhage. Posteriorly this cyst was attached to the under surface of the vermiform process of the cerebellum

which had been displaced to the right. On the ventral surface it extended as far down as the inferior olive. Anteriorly extensions from the cyst reached to the lingula cerebellae dorsi, and to just in front of the entrance of the fifth nerve ventrally. Wallenberg gives a description in great detail of the secondary degenerations which could be traced to this lesion, and a most interesting table, much too long for reproduction, in which he compares the symptoms during life with the supposed lesions which caused them, and the lesions found at the autopsy. This shows how accurate his topographical diagnosis was. He also gives for comparison the supposed lesions and those actually found. Among the most interesting results are, first, that the study of the sensory disturbance in the region of the fifth nerve makes possible a diagnosis of the location and extent of the lesion. Second, the sensation of touch does not seem to be disturbed if the spinal root of the fifth nerve is destroyed. Third, complete destruction of the motor root of the fifth nerve causes the tongue to be pushed obliquely toward the opposite side. Fourth, disturbances of sensation in the opposite side of the face suggest involvement of the formatio reticularis alba. If the dorsal trapezeoid fibers going to the dorsal olive are destroyed there is labyrinthine deafness. Fifth, loss of equilibrium after lesions of the terminations of the vestibular nerves is not necessarily restricted to the side of the lesion. Sixth, in spite of complete destruction of Deiter's nucleus, the dorsal nucleus of the eighth nerve, and of the abducens nucleus, there is not necessarily persistent conjugate deviation toward the side of the lesion. The anatomical conclusions must be read in the original.

22. *Reflex Hyperesthesia.*—Petrén and Carlström have investigated seven cases of gastric disease with the algesimeter of Thunberg. They found that hyperalgesia invariably occurred in the left upper quandrant of the stomach, and over the lower ribs on the left side. The explanation of this hyperalgesia is still indefinite, but it seems certain that it is not due to any alteration in the sensory nerves of the skin in the affected region. They suggest the theory, however, that the nerves from the skin, when they come into contact with the centripetal sympathetic fibers, are either rendered more irritable or else are distinctly inhibited. It is not known where this action takes place, but they believe that it may be either in the spinal ganglia, or in the corresponding segment of the spinal cord. They then discuss the relative likelihood of these two hypotheses. In a postscript they call attention to the great value of Thunberg's algesimeter for the determination of slight defects of the pain sense.

JOSEPH SAILER (Philadelphia).

Journal de Neurologie

(Vol. XI., No. 9, 1906.)

1. Notes on a Case of Progressive Muscular Atrophy. DE BUCK and DEROUBAIX.

1. *Progressive Muscular Atrophy.*—The authors call attention to the fact that while the muscular atrophies have been divided into spinal, neural and myogenic forms, facts brought out of recent years have pointed rather to the essential pathological unity of these different forms. As a contribution to the subject, they report a case very carefully studied both clinically and anatomically. The patient, a man of middle age, had always been of weak mind, and had had difficulty in using the muscles of legs and arms, which difficulty had become more pronounced. The general symptoms of the disease would place this case among the myopathies. Death occurring from exhaustion, the pathological examination showed decided changes in muscles, nerves, spinal ganglia, and in the posterior columns and nerve cells of the anterior horns of the spinal cord. These changes, which were of chronic degenerative character, are very carefully described by the authors, and their bearing upon the pathology of the muscular atrophies in general, is discussed at some length.

(Vol. XI., Nos. 10 and 11, 1906.)

1. The Perverse. MARANDON and DE MONTYEL.

1. *The Perverse.*—The author emphasizes the distinction between the criminal lunatic, the insane criminal and the perverse. The first commits his crime on account of his insane condition, the second is criminal at the start, and later becomes insane, while the third is not properly to be considered as insane at all, his memory, reason and judgment being sound, his deficiency presenting itself in the moral sphere alone. Each of these classes of cases requires different treatment, and it is an injustice to each to keep them together, as has been done heretofore, and is to a large extent still the practice. To a discussion of the characteristics of the third class and their management the author's paper is devoted. The perverse are the terror of asylums. They know perfectly well their rights, that they are not insane, and are only through an injustice confined along with the insane, though they are ready enough to use the asylum as a place of refuge and support, especially through the unpleasant season of the year. By their egotism, their quarrelsomeness, their insubordination and ability to evade the rules, to fashion keys and weapons, and by their bad influence over the other patients, they keep the institution in constant turmoil, and through the reckless carrying out of their schemes are a source of danger, though their crimes do not so generally take the form of violence, as of swindling, malicious mischief, and mean or dirty tricks. They are not mentally deficient and are capable of education, but the defect in moral control is congenital and can rarely be overcome. Differing in all particulars from the insane, criminal or otherwise, while the good of the community demands that they should be sequestered, they should be provided for in special institutions, which should be constructed strongly enough to prevent their escape, and in which there should prevail a discipline more rigid than in an ordinary asylum, and yet less strict than in a prison. Here they should not only be cared for, but should be taught trades of some sort, and given such moral training as possible. Into these institutions they need not be committed for life, but in the interest of the community; their discharge should be hedged about with such precautions as to insure that their condition has sufficiently improved as to make it unlikely that they will continue to be a danger to the public. The author discusses at some length the general plan of such an institution, and the legislation on the subject which he thinks is needed.

(Vol. XI., No. 12, 1906.)

1. Prophylaxis and Treatment of Habitual Criminals. MOREL.

1. *Habitual Criminals.*—An earnest plea for the thorough and individualized study, by trained psychiatrists of all cases of habitual criminals, the great majority of whom are afflicted with mental defect, more or less pronounced. Also for the establishment of institutions for the medical care and training of such individuals. The author insists upon the inutility of punitive measures in cases of this character, and that better results are to be expected by medico-pedagogical methods.

(Vol. XI., No. 13, 1906.)

1. Some Considerations on Mirror Writing. BOULENGER.
2. Recent Work on the Auscultation of Muscles in Paralysis, Contractures and Reaction of Degeneration. IOTEYKO.

1. *Mirror Writing.*—Persistent involuntary mirror writing is always pathological, and the sign of some mental disturbance characterized by more or less disorientation. After quoting at some length from the writings of a number of authors, the author describes some cases of his own, particularizing specially the methods employed by an experienced writing teacher in instructing an imbecile who showed this defect, but in whom some improvement was observed under the persistent and intelligent efforts of the instructress in question. In cases of obstinate mirror writ-

ing the prognosis is unfavorable since it is most frequently observed in idiots and imbeciles.

2. *Auscultation of Muscles in Paralysis.*—After quoting freely from some previous works of her own, especially from one, "The Functional Duality of Muscle," the authoress considers the study of Link upon the muscular sound. This is the sound which is heard when listening over a contracted muscle with the stethescope, or better with the phonendoscope. This sound in the healthy muscle voluntarily contracted is found to correspond to 22-24 vibrations per second. In complete flaccid paralyses this sound cannot be produced, though when the paralysis is incomplete it is heard more or less faintly, in proportion to the number of fibers capable of functionating which are left. The sound is also more feeble in any condition giving rise to impaired muscular force. It is heard in paralysis agitans, in tremors, and is very intense in tetanus. On the contrary in muscles having undergone contracture through nutritional causes (*e. g.*, in contracture after hemiplegia) it is not heard. If the patient is able to make any voluntary movement, however, it is immediately produced.

Normal muscle excited by the faradic current gives a note whose pitch is dependent upon the number of interruptions per second of the apparatus. In muscles showing complete reaction of degeneration neither the galvanic nor the faradic current produces any sound. The authoress thinks that these observations confirm the theory which she has put forward; namely, that the muscular contraction is composed of two elements, the contraction of the anisotropic substance, which is intermittent and produces the muscular sound, and that of the sarcoplasm which is continuous and silent. She even suggests that we can take a step further in advance, assuming that these two kinds of contraction are under the influence of innervations from different points, that of the anisotropic substance coming from the higher, the voluntary centers, in the brain, that of the sarcoplasm, from the lower or medullary centers.

(Vol. XI., No. 15, 1906.)

1. A Rare Form of Pseudoesthesia. MATTIROLO.

1. *Pseudoesthesia.*—By "pseudoesthesia" the author understands a false though physiological mental perception of colors, sounds, odors, etc., which are not real, but are nevertheless evoked by a real perception in the realm of another sense. He narrates the case of a man of thirty years of age, in good health, presenting no psychical anomaly, not neurasthenic, "in the true sense of the word," though a little excitable, and easily depressed. This man claims to have had since early childhood the following anomaly: The hearing, or even the reproduction of the mental images of certain words, are invariably accompanied by the sensations of the taste of certain substances as of fruits, vegetables, meats, etc.; for instance, the word Russia calls up taste of pears; admit, tomato sauce; captain, roast fowl, etc. The man is an Italian, but speaks French as well as he does his own language, and knows German, though this he learned much later than French. The corresponding French and Italian words equally arouse the gustatory sensations, not so the German words. He has the idea that words containing a number of labial and dental consonats give rise to tastes which should be perceived on the anterior part of the tongue and palate, while those in which gutturals abound cause tastes usually perceived by the posterior part of the tongue and palate.

(ALLEN) Trenton.

Journal de Psychologie Normale et Pathologique

(Third Year, No. 3. May-June, 1906.)

1. The Sense of the Mysterious Among the Insane. G. R. D'ALLONNES.
2. Modern Witchcraft. A. MARIE and M. VIOLLET.
3. Some Time-Reactions Among the Insane. CL. CHARPENTIER.

1. *The Sense of the Mysterious Among the Insane.*—Among normal

individuals there is a sense of the mysterious. In accordance with the degree of culture, it assumes the general features of an anxiety about the unknown. Normal individuals, however, possess pre-eminently the power of discerning the real, a perceptive power or faculty of drawing inferences which, if not entirely all that could be desired, is nevertheless sufficiently practical for all human and earthly affairs. The explanation of the unknown is the province of science and of philosophy; but it is in metaphysics and the theoretical sciences especially that we confine the exercise of the sense of the mysterious. This leaves us free for the furtherance of the positive sciences and the elaboration of the practical life.

It is abnormal for the sense of the mysterious not to be thus excluded from the domain of the practical, but to manifest itself, as it were, in connection with anything and everything in life. In the doubting mania (folie de doute), for instance, question follows upon question; and as long as the doubter remains engulfed in the sense of the mysterious he is lost. The systematic psychopath organizes the mysterious and gradually builds up a false conception of the world, of society, and of himself, sufficiently strong to involve all of his utterances, his writings, his movements, and even at times to lead him into terrific outbursts of violence.

Physiological disturbances, abnormalities in connection with the proper knowledge of men and of things, and a vague consciousness of these abnormal processes constitute the material out of which is evolved the morbid sense of the mysterious; the preservation of the reasoning faculty is the active element in its evolution. According to the relative dominance of one or the other of these different factors; namely, disorder of the physio-psychological processes, confusion of the consciousness, vigor of the reasoning faculty, the patient is more or less overwhelmed in an ocean of confusion and flounders feebly about, or on the other hand reacts, interprets and forms curious deductions while carving out of the chaos, in a most summary way, certain frightful, amusing or even beautiful conceptions. And, if he is able, he organizes them and creates a perfect dream-world, elaborate in construction, monumental and absurd.

A total absence of intelligence at one end of the scale, and intact lucidity of mentalization at the other are both incompatible with a morbid sense of the mysterious. It is the mentally weak, the half-lucid, the middling-intelligent who, not fully comprehending the world or themselves, fail to recognize their own incapacity while assuming to themselves a full and perfect comprehension. If during the preoccupation of a mystic or of a victim of the persecutory obsession one converses with him, one will be struck with the fact that there is a constant intervention of the voluntary reasoning faculty. This is the cardinal trait of this psychical abnormality. The author illustrates his thesis with a detailed report of three pronounced cases.

2. *Modern Witchcraft.*—Witchcraft, sorcery and incantation are all the offspring of superstition. They are in existence to-day, though we are prone to regard them as having vanished with the fallacies of the past. The underlying cause and foundation of witchcraft are the same in all ages. In the past, as well as in the present day, it assumes various guises. To-day it masks itself under the various forms of spiritism and assumes the high-sounding name of occult science.

After presenting a brief but interesting account of ancient witchcraft and an analysis of its basic religious and phychic constituents, Marie and Viollet report in detail four striking cases. In all of them the mental instability, hysteria, ignorance and superstition of the patients or of their friends and relatives gave origin to the belief that the victims were bewitched and could only be cured, or were cured, by the well-known methods recorded in the history of mediaeval sorcery. The special danger of these modern forms of witchcraft lurks in the degenerative psychasthenia which they are prone to awaken from a latent state. They excite the predisposed and lead to disastrous results.

3. *Some Time-Reactions Among the Insane.*—According to the observations of Wundt, Hirsch, Exner, Waller, Auerbach, Binet and others the reaction period for audition, in normal individuals, varies from twelve to sixteen hundredths of a second. Buccola and Patrizzi have shown that among patients whose powers of attention are deteriorated, fatigue appears and the graphic tracings incline more quickly than among normal subjects. The general mean is more elevated, the oscillations are wider, and the subject is slower to react. These same results are obtained under general anesthesia. Janet has clearly established a characteristic reaction curve for hysteria.

In the present essay Charpentier presents the tracings obtained by him in a number of cases of mental trouble, such as mysticism, persecutory insanity, mental debility, incipient general paralysis, athysoidea, dementia præcox, melancholia, alcholic general paralysis, maniacal excitement, etc.

He concludes that these reaction curves are of some interest, but that they possess only an imperfect and approximate value. It would be an exaggeration and wholly rash to affirm that each curve has a special diagnostic value. On the other hand, when studied in conjunction with the other symptoms, such as tracing may prove, useful in indicating the patient's degree of attention. One can detect simulation and obtain thus most useful objective criteria. A simulator of hysteria, for example, produced a reaction curve wholly inconsistent with the one usually obtained in cases of genuine hysteria.

From a physiological point of view, it is of interest to note that the tracings obtained by the author are almost parallel with those obtained with the aid of the dynanometer and the ergograph of Mosso. The question is asked by the essayist, are we then to conclude that muscular phenomena and attention are identical? To answer this we need, he says, to study with greater precision than heretofore the relationship of attention, not only to muscular force and the time of reaction, but also to the circulation and the respiration in order to complete the psychophysical and psychochemical basis of attention. METTLER (Chicago).

Miscellany

DEMENTIA PRÆCOX. By W. A. White (Journal A. M. A., May 19).

Dr. White argues against the theory advanced by some authors on the basis of chemical studies, etc., that dementia præcox is caused by an autointoxication. The disease, he considers, is due to an original developmental defect, and the physical symptoms suggesting autointoxication are the result of the imperfect functioning of the nervous system.

MISLEADING SYMPTOMS IN CEREBRAL DISEASE. By J. A. Stucky (Journal A. M. A., April 28).

The author reports a case of sarcoma involving the whole right temporosphenoidal lobe, associated with and infected by chronic suppurative ethmoiditis, and asks which was the primary lesion. No macroscopic evidence existed of disease elsewhere in the brain. The local symptoms, pain and bulging of the orbit, pointed to trouble in the right fronto-orbital region, but there was aphasia (the patient was right-handed), spasm of the right arm and hand, and the tongue deviated to the right. The author asks: Could general intracranial pressure exist without motor symptoms referable to right-sided pressure and also what would have been the result had the sinus disease been eradicated months before?

PSYCHOSES RESULTING FROM COAL-GAS ASPHYXIATION. By Sanger Brown (Journal A. M. A., April 28).

The author reports a case of asphyxiation from coal gas. When the patient was found (Nov. 7) he was unconscious, his face was a deep purple and the body was entirely flaccid. He was carried to bed, and after the use

of restorative measures he regained consciousness at the end of three days, and in about three weeks he was able to be up and about. His face continued to be purple, and his expression was dull and stolid. He had no recollection of anything that transpired for from 36 to 48 hours prior to the time he was found unconscious. He recognized his old acquaintances and repeated their names and discussed correctly with them various matters relating to a period prior to the asphyxiation. He repeated incidents of early life and childhood usually well, but retained practically nothing since the afternoon of Nov. 6. He could not remember what took place from hour to hour, even the visit of an old friend whom he was very glad to see. By dint of constant practice he finally remembered the name of his nurse and his way to the toilet and dining rooms. He could read and make simple computations in arithmetic. He realized his disability and wanted to recover, but was not emotional or complaining. He would read the newspaper, but could not discuss current topics. Though he retained some few impressions in his memory enabling him to find his way about the house and when out walking, there was no material improvement while under observation, a period of fourteen weeks. On leaving the sanitarium he returned to the care of his family physician, who reported no notable change in either the bodily or mental symptoms till about the middle of December, two weeks before the man died. He suffered from severe attacks of dyspnea so that he had to be under constant surveillance. He was a little better for two days before his death, which occurred suddenly, soon after he retired and while he was alone in his room. Autopsy showed no significant changes in the brain or spinal cord. The lungs were markedly emphysematous, and the heart was considerably dilated. There was a large cyst of the left suprarenal capsule. Dr. Brown says that it may reasonably be assumed that some deleterious influence, not demonstrable by present methods of examination, had been exerted on certain cerebral neurones, so that they were no longer able to retain new impressions. He also calls attention to the fact that the impressions made on them some hours prior to the action of this influence were effaced by it, while impressions of long standing were not affected.

TREATMENT OF IDIOPATHIC EPILEPSY BY APPENDICOSTOMY FOR COLONIC IRRIGATION. By S. LaPlace (Journal A. M. A., June 2).

Dr. LaPlace considers it probable that epilepsy is due to the manifestation of a toxin accumulated in the system as a result of the intoxication of improper food or tissue metabolism and the locality of the production of this poison is the colon. Consequently, he has resorted to appendicostomy with subsequent irrigation of the colon through the fistula in one case with excellent results. He concludes that the treatment is harmless, and is based on sufficiently sound theoretical and scientific grounds; and even should improvement of the epilepsy not result it cannot fail to do good from a hygienic standpoint in affording general relief from constipation, to which most sufferers from epilepsy are subject. The repeated flushings of the colon keep it as nearly empty as possible, and almost eliminate its presence as a reservoir for undigested and putrefying substances. Fear of a permanent artificial opening in the intestine should not be entertained. The artificial fistula closes easily, and, after months of treatment, should one desire to discontinue the flushings of the colon, the natural processes of evacuation are resumed without difficulty. The method is not expected to cure all cases of idiopathic epilepsy; but only those whose existence can be traced to this particular cause. The procedure here advocated should be accompanied by the proper dietetic measures.

Book Reviews

HISTOLOGICAL STUDIES ON THE LOCALIZATION OF CEREBRAL FUNCTIONS. By ALFRED W. CAMPBELL, M.D., Pathologist to the Asylum Board of the County of Lancaster. University Press, Cambridge.

This is a work that has been urgently needed for a long time. Hammarberg's work on "Idiocy," and isolated monographs here and there have led up to the desire for a work in which one could find the chief histological features of the entire cortex accurately and adequately presented. Dr. Campbell has done this in his quarto of 360 pages. After discussing the methods and material, and general remarks on cell lamination and the arrangement of nerve fibers in the cerebral cortex, he takes up the several areas of the brain substance. In Chapter III. the motor area is studied as to its structure and function. The arrangements of cell and fibers are given and comparisons drawn between the structure of the precentral area of man and ape. Special attention to the giant Betz cells is given as well. Functional studies on the changes in the cortex in amyotrophic lateral sclerosis, post-amputative changes and the effects of lesions of this area are given in this chapter as well. The post central area is then taken up in the same exhaustive manner. Histological studies are followed by physiological considerations. Chapters V. and VI. treat of the visuosensory and visuo-psychic areas, and the temporal lobes and auditory area. The limbic lobe, the parietal area, the intermediate precentral area, the frontal and prefrontal areas and the island of Reil complete the main body of the work. In an addendum the author presents further histological studies on the localization of cerebral function, dealing particularly with the comparisons of the brain of man and those of the lion, the dog and the pig.

The more one reads the more is the impression deepened that we have here a memorable volume which will serve as a new starting point in all work on the brain, whether one is interested in neurological or psychiatric problems. Campbell has set before himself the task of going over the entire brain cortex, millimeter after millimeter, and here gives us, it might be said, for the first time the complete story of the histology of the human cortex. But he has done more, for in his hands histological investigation, supplemented by embryological and pathological considerations has opened up a well of suggestions concerning cerebral localizations which may well be designated as fundamental. The work deserves the highest praise.

JELLIFFE.

A TEXT BOOK OF PSYCHIATRY FOR PHYSICIANS AND STUDENTS. By LEONARDO BIANCHI, M. D., Professor of Psychiatry and Neuropathology in the Royal University of Naples; Minister of Public Instruction in Italy; Medical Director of the Provincial Asylum of Naples. Authorized translation by James H. Macdonald, M.B., Ch.B., Glascow, Senior Assistant Physician to the Goran District Asylum. William Wood & Co., New York.

This text book, coming from one of the leading psychiatrists of Italy, is of more than passing moment, for the works of Bianchi and Tanzi represent the leading trends in the Italian psychiatry of to-day. The latter is strongly pervaded by the influence of German psychiatry, particularly of the Kraepelin school, while Bianchi represents Bianchi, a strongly individualistic writer and thinker.

The author introduces his work in the conventional manner by summarizing the general facts of cerebral topography and minute anatomy,

which is a scholarly and interesting presentation. The main principles of mental evolution from the physiological point of view are there considered, special attention being given to the function of language.

In part II. the author discusses the psychological principles which will serve as the framework of his symptomatology. The Psysio-pathology of Perception, of Attention, of Memory, of Ideation, of the Emotions and Sentiments, of the Will and Consciousness, are the chapter headings in this part of the work. Here the method of the author is made manifest. He takes up in an easy manner the general phychological scheme and elaborates it by copious references to contemporaneous literature. This is done, however, in an interesting, though in a very disjointed, manner, yet the main facts of recent psychological research are admirably presented. Illusions are defined by Bianchi as disorders of perception by which the subject reproduces, in the object present, features that do not correspond to the reality, but which are reawakened by an altered associated mechanism and by an anomalous tension of certain nervous apparatus. Such a representation necessitates the action of an objective stimulus. Hallucinations are subjective perceptions according to our author, and are closely related to illusions, if indeed they may not be the same thing. These phenomena are elaborately and minutely illustrated by clinical examples and by a wealth of suggestive material.

Attention, Bianchi regards as a psychic fact, interposed between perception and the reaction which it tends to provoke. It is therefore one of the higher reflex processes, such as conscious and voluntary movements, and their inhibition. This chapter is a very fascinating one, and the following one on memory is no less so. In the chapter on Ideation the phenomena of delirium are taken up. Delirium is a qualitative disturbance of ideation. The formative process of thought is altered by extraneous associative relations between preformed images, drawn sometimes from the unconscious, or between already acquired ideas and the newcomers, thus giving rise to a conclusion which does not correspond to reality. The product of this abnormal process goes under the name of delirium. Bianchi's attitude toward deliria is unusual. He says that the "deliria" are always preformed mental products that have been eliminated from consciousness at an early date; and he further speaks of the consciousness offering less resistance to the invading power of these products. His illustrations are striking, but the suggestion is too diagrammatic. According to the nature of the content he distinguishes deliria of grandeur, of depression, of metamorphosis, of negation, of persecution, and of religion.

The chapter on the Emotions contains an excellent discussion of the James-Lange theory, as well as much literature, old and new, and ranging over an extensive field. It is surprising to an American reviewer to find an author who has made so much use of the work of American psychologists. In fact, no worker of any significance is neglected, notwithstanding the variations in speech.

The will is defined as "nothing more than the conscious resolution of the motor tendencies of the intellectual-emotional synthesis," and we may figure it to ourselves as the conscious motor potential tending to discharge itself through circuits of various orders, from the lowest, which must resemble the inferior and instinctive reflexes, to the highest, which are represented in the actions of the heroes of humanity. That man is a reflex animal even in his most complex intellectual acts is therefore the attitude of our author.

The author has a repugnance to classifications of the psychopathies. "Almost useless, incomplete and not vital" are his terms. He gets along without one, therefore, but presents a series of groups. In the first group he puts all those psychopathies developing on an evolutionary psychocerebral defect; a second group consists of all the mental affectations of

infective, autotoxic and toxic origin, developing in individuals regularly evolved, and a third group includes all the affections with an organic substratum, localized or diffuse, in the central organ of mind.

In his first group he arranges phrenasthenia, paraphrenias, congenital moral insanity, epilepsy, hysteria, developmental paranoia, fixed ideas, developmental neurasthenia and the sexual psychopathies. In the second group are arranged: Mania, lypemania, exalted-depressive insanity, circular and periodic insanity, sensory insanity, mental confusion, acute paranoia, late paranoia, neurasthenic insanity, choreic insanity, luetic insanity, acute delirium, and the drug and infectious insanities. His third group contains: Paralytic dementia, leutic dementia, senile dementia, post hemiplegic dementia, aphasic dementia, traumatic dementia, and dementia arising from tumors, scleroses, placques and other organic diseases of the brain.

It is very evident that the ideas of prognosis so valuable in Kraepelin's schemes are not taken into account by Bianchi, and furthermore it is difficult to understand his attitude when he says that the secondary dementias are always identical, whatever the clinical form from which the particular symptoms are derived. If there is any large fact of psychiatry that has been brought forth by the work of Kraepelin it is that the secondary dementia group is far from being hemogeneous, and as long as they were so considered little hope was offered in the unravelling of the process that led to the dementia.

The descriptive portion is admirable, particularly so from the standpoint of comparative psychiatry, although it is to be regretted that much of the newer German symptomatology is conspicuous by its absence. As a whole the work is one that in its present form should be read by psychiatrists, who cannot help but profit in so doing. JELLIFFE.

News and Notes

At a recent meeting of the New York Psychiatrical Society the following memorial notice of the death of Dr. Alexander E. Macdonald was presented by Drs. Charles F. MacDonald and William Mabon, a committee appointed for the purpose, was unanimously adopted and ordered to be spread upon the minutes of the society and a copy of the same to be sent to the family of the deceased:

Alexander E. Macdonald, L.L.B., M.D., a member of the New York Psychiatrical Society, died Dec. 10, 1906.

For thirty-five years Dr. Macdonald had been intimately associated with the insane. He commenced the study of medicine at Toronto University and graduated M. D., Medical Department, New York University, 1870; LL.B., Law School, New York University, 1881. Lecturer upon Medical Jurisprudence in 1874; subsequently, Professor of Medical Jurisprudence, Professor of Psychological Medicine and Medical Jurisprudence, and was Emeritus Professor at the time of his death. House Physician Hospital for Epileptics and Paralytics, Blackwell's Island, 1870; chief of staff, Charity and Allied Hospitals, Bleckwell's Island, 1871. Resident physician, New York City Asylum for the Insane, Ward's Island, 1874. Medical superintendent of the same from 1874 to 1904, the title of the asylum having been changed in the meantime to Manhattan State Hospital, East, Ward's Island.

In 1901 he established the tent treatment of the tuberculous insane, removing them from all communication with any unaffected patients. The

principles underlying this undertaking are now universally accepted by the medical profession here and abroad.

An article on this subject was published by the Charity Organization of New York City and the National Association for the Study and Prevention of Tuberculosis.

Dr. Macdonald was a delegate from the American Medico-Psychological Association to the Fourteenth International Medical Congress at Madrid in 1903; President of the American Medico-Psychological Association in 1904; delegate to the Congress of American Physicians and Surgeons, to be held in Washington in 1907; honorary member of the Medico-Psychological Association of Great Britain and Ireland, and of other continental medical associations.

His splendid administrative abilities made him familiar with every detail in the care of the insane, seven thousand at one time being under his direction. He possessed the rare gift of attracting to himself experienced, trusty and loyal officers and friends.

Dr. Macdonald was one of the most distinguished alienists of this country, and a man of striking force of character. He had a hatred of cant and pretense. His far-seeing powers, his unswerving integrity and great executive ability qualified him in an extraordinary degree for his responsibilities. At all prominent medical meetings his activities were conspicuous. His commanding presence and lofty sense of duty will always be remembered by those who had the privilege to be acquainted with him, and his pupils in all parts of the country will pay many tributes to his memory.

Dr. Macdonald was a cultured man of affairs, who wrote in a convincing and agreeable style, and enjoyed a well-earned reputation as an after-dinner speaker. He was a member of the Lotus Club and a Mason of Holland Lodge, New York City.

Our sincere condolences go out to the wife and children of our fellow-member in their affliction.

The Psychiatrical Society desires to spread upon the minutes this tribute to the memory of their late associate.

C. MACFIE CAMPBELL, M.B., Secretary.

P. J. Möbius died in Leipsig, Jan. 8, 1907.

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Original Articles

MIGRAINE AND HEMIANOPSIA.*

BY JOHN JENKS THOMAS, A.M., M.D.,

OF BOSTON.

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The literature of migraine is large, and few diseases have been more carefully described, and there are few in which more writers have described their own cases, many illustrious physicians and neurologists having written of their own attacks of this curious trouble. In one respect our knowledge is singularly defective, as autopsies, except in cases where some serious brain disease has coexisted, are rare. In consequence, many theories have been advanced in explanation of the symptoms, many of them more ingenious than probable.

The question broadly stated which I wish to discuss is whether attacks of migraine, in the absence of disease of the vessel walls, is capable of producing permanent changes in the nervous structures. Moebius in his exhaustive monograph on this subject in Nothnagel's *Specielle Pathologie und Therapie*, published in 1894, speaking on this point, says that the question whether hemorrhage or softening occurs, more fully stated should be—

First: may it be possible that in time, because of the repeated attacks of migraine, degeneration of the vessel walls

*Read at the meeting of the American Neurological Association, June 4 and 5, 1906.

may occur so that some attack may occasion rupture or thrombosis of the vessel?

Second: may an attack of migraine itself, without previous degeneration of the blood vessels, lead to destructive processes?

To answer these questions with our present knowledge of the subject is difficult, if not impossible. Moebius rightly calls attention to the division of migraine into what may be called the symptomatic, and the idiopathic forms. Both migraine and degeneration of vessels are frequent conditions, and may be simply coincident, especially in old persons, or where syphilis is present. He concludes that the migraine may be the exciting cause of the organic brain disease, and this may be considered to be the case especially in instances where the attacks, particularly the aura, are always unilateral and the softening occurs on the same side. As he says, the question is usually not whether a case is migraine, but whether it is migraine alone or with something else. Obviously this is a much more difficult question to decide.

In idiopathic migraine we have to do with a disease which is most often hereditary. Kovalevsky in a recent monograph collected 110 cases, in 70 of which there was a history of heredity, and in 18 of these cases for three generations. The disease generally begins in youth, and cases are to be regarded as suspicious when it begin late in life, as well as those where there is no history of migraine in the family. The next characteristic of migraine is the intermittency. Then the character of the headache must be considered, as headache is an extremely common symptom. In neurasthenia the headache is generally constant, and there is no vomiting. Headache from disease of the nose is also without free intervals and vomiting. Syphilitic headache does not occur in separate attacks like migraine, and is apt to be worse at night. Headache from malaria is somewhat questionable, while the pain of glaucoma, anemia, and of supraorbital neuralgia can usually be easily distinguished. The headache of gross brain disease, such as tumor and abscess, generally occasions no difficulty in diagnosis, though some cases of symptomatic migraine may be difficult to differentiate. Moebius thinks the cases of migraine due to gout and malaria are altogether

doubtful, the latter disease in particular usually causing neuralgias rather than migraine, and in the former case we probably have to do with alternations between the two diseases, as is seen in other diseases. Migraine occurs as a symptom, usually an early one, not uncommonly in tabes and paralytic dementia, cases having been reported by Moebius and others, but in these cases a careful examination should make the nature of the case evident, and the same is true of the cases due to tumor of the brain, though occasionally cases may arise in which the diagnosis is extremely difficult, or even impossible, as in the case of aneurism of the basal artery reported by Karplus.

A young woman, 29 years of age, all of whose nine brothers and sisters had attacks of headache, had suffered from youth with severe attacks of headache with eye symptoms such as scotoma. She had no syphilis. She was suddenly taken with a severe headache on the right side of the head. The next day there was right ptosis, and roaring in the ears with disturbance of hearing. She then improved, but three weeks later the pain returned much more severely in the right forehead and temple with paralysis of the right third nerve, and the patient died four days later. At the autopsy there was found an aneurism of the right posterior communicating artery which had burst, and adhesions of the right third nerve to the aneurism.

Karplus thought there had been first a rupture of the aneurism, and later a rupture of the spurious aneurism which had formed, and thought that the migraine had aggravated the disease of the vessels.

Moebius calls especial attention to the fact that in tumor of the brain the vomiting is not followed by relief of the pain as is usual in cases of migraine, that patients are not usually free from symptoms in the interval, are often mentally dull, and that the pain is not relieved by rest.

We should probably separate also the cases of recurrent oculomotor paralysis from migraine. In the former disease the intervals are usually longer than in migraine, and in the intervals the patients are not free from all paralysis, at least in the later course of the disease, and the headache is probably often symtomatic only. There is usually no aura, or history

of heredity, and the pain often lasts for weeks. While these differences are observed in most cases this is by no means always true. Cases of recurrent ophthalmoplegia have been reported fairly frequently in which an aura was present, such as a scintillating scotoma, where there was a distinct heredity, where the intervals were short, and most important perhaps where the disease began in youth, or migraine alternated with attacks of ophthalmoplegia. Such cases have been reported by Paderstein, Ballet, Seiffer, Suckling, Romano, Joachim, Kollaritz, Chalbert, Lappersonne and others. So that while in general a marked difference exists in these diseases there is a close relationship, but the weight of authority seems to be with Schmidt-Rimpler when he says that there is probably the same cause for the pain and the paralysis, and as Karplus says the cases form two separate groups, but in some there is a relation between some anatomical alteration which is primary and independent of the migraine, and is the cause, but which may or may not be active in producing the attack; while in the hemicrania cases there is a local defect, but other independent factors enter. Ballet thinks the ophthalmoplegia cases are related to what the French call the migraine ophthalmique, where there is a scotoma of some form, but not to the ordinary cases.

Hemianopsia as an aura of migraine attacks is too common a phenomenon to require more than mention. Occasionally a transitory aphasia is seen, as in the cases of Determann, Pick, Charcot and others. More rarely still disturbances of sensation or of motion of one side of the body appear, and sometimes aphasia with them, as in a case of Féré. A number of cases are reported where symptoms appearing during the attack similar to transitory ones in previous attacks have become permanent. The French writers in particular are incline to look upon these as the permanent effect of spasm of the blood vessels in the brain, such is assumed to be the cause of the transitory symptoms. In support of this theory certain cases are repeatedly quoted, especially those reported by Galezowski, Féré and Charcot, but a careful analysis of these cases and others shows that in most, if not all of them, we probably have a symptomatic migraine accompanying organic brain disease, vascular or otherwise, coming in per-

sons, in some cases subject to migraine attacks, and in other cases, even this much is doubtful.

Rossolimo's case of recurrent facial paralysis in migraine if more than a coincidence is more closely allied to the recurrent ophthalmoplegias, as the attacks lasted several days, and the intervals between the four attacks of paralysis varied from two years, to three and a half years.

Galezowski's cases were:

1st. A man, 67, who had had attacks every month to every week for twenty years, in which he had hemianopsia and scintillating scotoma for a few minutes, followed by pain in one-half of the head lasting for two or three hours, had in one attack a sudden loss of sight in one eye, from a thrombosis of the central artery. The heart was negative.

2nd. A girl, 15, who had had migraine with scotoma of flames and zigzag lines since the age of seven or eight, suddenly in an attack became blind in one eye, which blindness was permanent in part. The field of vision was lost in its whole lower half, the papilla pale and like the appearance seen in embolism of the central artery, the fronto-temporal and the fronto-nasal arteries were sclerosed, while the heart was normal.

3d. A woman, 29, who had had migraine without hemianopsia, but with zigzags of light and dark scotoma, nausea and vomiting with the attacks, was found to have normal vision in the right eye, while that of the left was much diminished, and there was paleness of the disc and diminution of the size of the blood vessels, and a neuro-retinitis, with vessel thrombosis.

4th. The patient, 52 years old, had had migraine with eye symptoms for four years only, having had numerous attacks, with hemianopsia in some and aphasia in some, of four or five minutes' duration. There was an attack of migraine with left hemianopsia, after which the vision of the right eye was found normal, while that in the left was poor, without affection of the field of vision, but a neuro-retinitis with capillary thrombosis.

In two of these cases we should suspect that arterial disease was present, on account of the age of the patient, and the age at which the headaches began, and they are probably to be

considered cases of symptomatic migraine. The other two cases prove pretty conclusively that thrombosis of the vessels of the optic nerve may occur in an attack of migraine, and are important as showing that this can occur in young persons. Voss reports a similar case where sudden blindness came on in a woman of forty-two during an attack of migraine to which she had been subject since youth, where there was found atrophy of the optic nerve from thrombosis of the central artery or hemorrhage into the sheath.

Other cases are reported in which an aphasia, which was permanent, came on in the course of a migraine attack. Many of these were cases where there was disease of the vessel walls, and the migraine was evidently a symptomatic one. One of the earliest of the cases I have been able to find is one reported by Féré in 1883.

A man, 53, whose father had suffered from migraine, and who himself had had migraine from childhood, the attacks coming about every eight days, accompanied by vomiting and preceded by zigzag lines of light and a scotoma, had an attack in which he developed a permanent aphasia with partial paralysis of the right arm. Later he had trouble with the left side also, and difficulty in swallowing, while later still loss of consciousness came on, and he died evidently from some vascular lesion of the brain.

Charcot mentions two cases of permanent aphasia coming on during an attack of migraine, both occurring in old people. Oppenheim's case, reported in 1890, is one of the few with autopsy on record.

The case is that of a woman who had suffered from periodical headaches, probably from childhood, certainly for a long time, which generally affected one side of the head only and lasted from twelve to twenty-four hours with photophobia, nausea, and sensitiveness to noise. She had had one healthy child one year after marriage, but none since. Three years after her marriage asphasia came on during a headache which disappeared in twenty-four hours, and there were four similar attacks. Twelve years later she suddenly lost her speech but without loss of consciousness, and there was also a right hemiplegia noticed as she lay in bed. She soon began to speak, but after an hour this was again lost and remained

so. At first she was excited and sang. In five weeks she could move the leg. There was no syphilis. Examination showed aphasia, word deafness, and complete paralysis of the right arm and partial of the right leg and face, with increased knee jerks on both sides. Hemianopsia could not be made out. The ophthalmoscopic examination was negative. The prick of a pin could be felt on the right side. Some two months later she was taken with vomiting and died, and at the autopsy there was found a thrombosis of the left internal carotid shortly before the giving off of the artery of the fossa of Sylvius. There was also endocarditis and cicatrices of the kidney.

This case again is evidently one of a combination of migraine with vascular disease, though it is difficult to explain the repeated attacks of temporary aphasia, except as circulatory disturbances after a small embolus in the neighborhood of the speech areas.

Quairolo in 1893 reported a case of asphasia with paresis of the right face and tongue, coming on suddenly, with dizziness, in a man of fifty, who had a diffuse arterio-sclerosis. He thinks the transitory symptoms of migraine may become permanent, and ascribes the disease to a spasm of the blood vessels. The arterial disease in his case makes it unnecessary to consider the migraine as more than an exiting factor for what was probably a cerebral thrombosis.

Flatau in 1902 reported a case which seems to me to be one where we have a migraine causing organic trouble in a person whose age was favorable to disease of the vessels.

The man was forty-eight years of age, and there was a history of heredity of migraine on both sides of his family. His attacks had begun at the age of sixteen, and had occurred every two to four weeks, accompanied by a scintillating scotoma in one eye followed by the pain and vomiting. He was well in the intervals. He also had paresthesia in the fingers, face and tongue on the side opposite the headache, accompanying the aura. He had typhoid and then smallpox, and at the end of this aphasia appeared, with difficulty in swallowing, but no weakness of the extremities. This lasted between four to seven weeks and then passed away. The attacks of migraine returned varying from one to ten a year.

In these attacks he had a peculiar feeling in the right hand, face and half of the tongue, and a scotoma on the right, while the headache was on the left. After one attack he found agraphia, and some difficulty in getting the word he wanted to use. This trouble lasted four days. With the next attacks of headache he had no such symptoms. Later he had a similar attack of agraphia, with aphasia, but no paralysis, which lasted ten days.

The cases in which a permanent hemianopsia came on during an attack of migraine are very similar to those in which a permanent aphasia or paralysis occurred. That is, most of them are in all probability cases in which arterial disease existed. Féré in 1881 reported a number of cases of migraine ophthalmique of which the ones that concern us are the following:

Case 6—A woman, thirty-four years of age, with no history of migraine in the family, and no previous illness, was taken one day, three years before she was seen Féré with a sudden pain in one half of the head, was stupid and then slept. Some days later she was taken with a vertigo and feeling of pressure in the head, which kept on until one month later she woke one morning unable to move the fingers of the right hand. The next day the arm was paralyzed. This passed off. Some two weeks later the leg became weak in the same way. Three weeks later she had an attack of vertigo and a convulsion involving the right arm, while the head was turned to the right and the mouth drawn to the right side, and she was unable to speak. This lasted an hour, when the pain in the head began, accompanied by nausea. In three months she was well. Then for eighteen months she had no trouble, no headaches or other symptoms appearing. Then she was taken with vertigo, lost consciousness, had pain in the head with a scintillating scotoma, and vomited. The pain lasted five or six days, and was worse on lying on the left side, and she had tinnitus and fever every evening. Six weeks later there were convulsive movements of the left arm, the mouth being drawn to the left, and she could not move the left extremities, and they were anesthetic. This lasted two hours, and was followed by violent pain in the head, and there was diplopia during the attack. Later the attacks became less frequent, and she

improved, but hemianopsia remained. Both sides showed increased tendon reflexes especially the knee jerks. Three months later there was another attack of headache with increase of the hemianopsia.

In considering this history with the absence of heredity, the onset of the disease in adult life, the long intervals between some of the attacks, the fact that convulsions were followed, instead of proceeded by the pain, I think, any neurologist of the present time would consider the case one of organic cerebral disease, and the pain purely symptomatic.

Féré's next case, case 9, was one of Charcot's. The history is given very briefiy. The patient whose age is not given, a year before in consequence of an attack of hereditary migraine with temporary aphasia was left with a hemianopsia which had not improved. The migraine attacks, however, after this were less severe, and less frequent, but continued. This case in the absence of further particulars we must consider as a possible one of organic disease from migraine, but we are without the means of judging of the condition of the blood vessels.

Féré's third case, number 10 of his series, was also one of Charcot's. It was that of a woman whose age was not stated, who had suffered from intense migraine almost all her life, sometimes on the right side, and at others on the left side of the head. It could not be learned whether she had had hemianopsia or scotoma during the attacks. Of late the attacks had become less severe. Shortly before she was seen she had an attack of migraine like her ordinary ones, only with numbness of the fingers on the right and difficulty in seeing the end of the line she was reading. She had no vomiting as she usually did. She had remaining a right hemiparesis of the face and tongue only, and aphasia and agraphia, but it could not be determined whether hemianopsia was present or not. There was no heart lesion or trouble with the stomach, and there had been no vertigo.

This case, judging from the circumstances told, a woman living alone, with no one to give information about her but servants, the precautions urged upon the physicians not to trouble her by many questions or much examination for fear of alarming her, makes us suspect that it was an old woman,

where we probably have to do again with arterial disease.

Schroeder in 1884 reports a case, which is also open to some doubt. A man of thirty had had for ten years scintillating scotoma with migraine attacks, and after they had lasted several years, had some attacks in which there was loss of consciousness. Then he had attacks with numbness of the left hand and weakness of the left leg. The headaches were very severe, lasting a week. He had a symmetrical defect of the left half of the field of vision, and diminished hearing on the left. He had acquired syphilis four years before, but after the appearance of the migraine, and after the appearance of severe symptoms, so that Schroeder did not consider they could have been caused by the syphilis. His second case of visual defect coming on with migraine attacks, presented a separation of the retina, and need not detain us.

Infeld in 1901 reported a case in a young woman twenty-nine years of age whose mother had attacks of migraine about once a month with vomiting, and one of whose brothers was similarly affected, while she herself had had headaches since she was twelve years old, coming about once a month, generally before the menstrual period, but not exclusively, lasting one day, without aura or vomiting. There was no syphilis. About seventeen months before she was seen she had one of her headaches which had lasted several hours, when she was frightened at some danger to one of her children, and while bending over him she suddenly felt something like a tremor in the left side of the head above the ear, and a sensation of heat in the right side of the face and a sensation like a flash of lightning in the right side of the body. She fell, but did not lose consciousness. The right side of the body, including the face, was paralyzed and without sensation, and there was loss of sense of position of the right extremities and paresthesias, while the mouth was drawn to the left. There was no diplopia and the speech was not affected, but the right eye felt blind. This condition passed off in about ten days. A month later she had cramps in the right arm, but not in the leg. During the period from this time till she was seen, she had few headaches which were milder than usual. On examination the internal organs were normal, as were the blood vessels, and there was no evidence of syphilis and the field of vision was

normal. The right extremities and the right side of the face were still slightly weak, with very slight spasm, and somewhat increased reflexes, but no disturbance of sensation, or sense of position and no astereognosis, but there were rather quick athetoid movements on the right. During the next nine months she had four more headaches.

Hœflmayer in 1903 reported a case of a woman of fifty-seven, who had had frequent attacks of migraine for forty years, from which her father had also suffered. She had an attack after much worry and severe long continued constipation in which she became stuporous. It was difficult to make her put out the tongue, and she could only repeat a couple of words without sense, but she showed evidence of pain. This condition lasted ten days, and then gradually cleared up, but hemianopsia remained for a month, and then gradually disappeared, at first the outline of objects being seen dimly, and then more and more clearly. Hoeflmayer thought the condition a toxic one, added to a neurasthenic exhaustion and states that he could rule out hysteria.

To these cases of organic disease coming on during an attack of migraine I am able to add three cases where a permanent hemianopsia was observed. Two of these cases were seen by me at the Boston City Hospital, and I am indebted to the Physicians for Diseases of the Nervous System for permission to report them, while the third case was seen in consultation with Dr. R. G. Loring of Boston, who has kindly placed all his notes at my disposal.

The first case was seen at the Boston City Hospital on March 28, 1894, and was that of a young woman (4630) thirty years of age, single, a nurse at the hospital. The mother died of cardiac trouble, and her father at the age of seventy-five, of some brain trouble following an attack of the grippe, and he had had an attack of apoplexy a year previously. Two brothers and two sisters were living and well.

The history states that the patient had always been perfectly well, but both Dr. Knapp and I recall that she afterwards stated that she had suffered for some time from periodical headaches, which had not been severe, and that also shortly before her illness, she had had a great deal of worry and mental strain from the illness of her father. On February 2, 1894, she had been a good deal excited at some occurrence, and had a severe headache,

during which she had a sudden attack of vertigo, accompanied by loss of consciousness, but without paralysis or aphasia. The next day there was violent frontal headache and a general numbness of the entire left side of the body, and slight paresis of this side, but no paralysis. For the first three weeks after the attack there was complete amnesia, and after that she improved rapidly. When examined at the Out Patient Department she complained of numbness of the left side of the body, and of a sensation of fulness in the head. There was still considerable mental dulness, but no vertigo, headache or digestive disturbance. There was some weakness of the left side, the grasp being 45 on the right, and 20 on the left, and there was slight ataxia of the left hand, but no muscular spasm. The knee jerks were increased, the left more than the right, and there was no ankle clonus. Sensation was normal except subjectively somewhat diminished on the left. There was left hemianopsia. The left pupil was larger than the right, but they reacted normally, and there was no hemianopic reaction. Fundus oculi normal. Ocular muscles were normal. Heart was normal except for a systolic pulmonary souffle. There was slight swaying when standing with the eyes closed, which soon disappeared. The hemianopsia was permanent.

A letter of May 28, 1906, says that her health has been of late much better than for the three or four years following this illness, but she still has headaches almost constantly although not severe, unless aggravated by excitement. With these headaches she writes she has no vomiting, and never has had.

The second case was that of a single woman, twenty-seven years of age, who was seen at the Boston City Hospital (5200) on March 11, 1895. Her mother and one sister had migraine. For ten years she had had attacks of headache always accompanied by vomiting, but she could not say whether one side of the head was affected more than the other. These attacks usually lasted three or four hours, and had not been growing more severe. Excitement was apt to bring on an attack. In them she had never lost consciousness, and neither the limbs nor the eyesight had ever been affected. She had never vomited except during an attack of headache.

Four weeks before she was seen, a sister died and the patient was much affected and had one of her ordinary attacks at this time. Two weeks later while sewing she began to feel the onset of one of her headaches. She vomited and felt faint, but there was no loss of consciousness. Then she noticed a sensation of pins and needles in the entire left side of the body with a partial loss of power in the same side. This was accompanied by a loss of sight in the left side of the field of vision of the left eye. She regained power in the extremities in a short time, but the trouble with the vision continued.

On examination there was left hemianopsia, and at the first examination it was said that there was present the hemianopic pupil sign, but at later examinations made by myself and others, both sides of the retina, when illuminated, caused a reaction of the pupils to light. The ocular muscles were normal, and sensation was normal also. The strength was fairly good. The knee jerks increased, but no ankle clonus. There was no evidence of syphilis. The urine at this time had a specific gravity of 1.022 and contained sugar, but no albumin. On June 26, 1895, it was 1.012, and contained no albumin, and no sugar. She had no headache after this attack till August 18, 1895. The fundi were found normal at repeated examinations, the last being on August 30, 1898, when the vision was found to be 20/20ths, the same as at the first examination.

On February 21, 1900, the sensation for touch, pain, and tem-

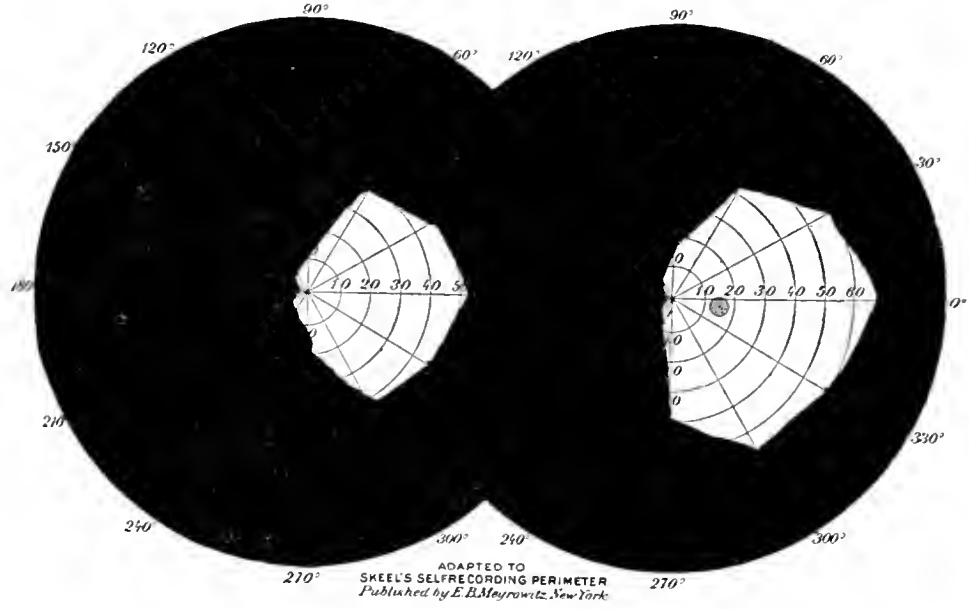

Fig. 1. Case 2.

perature was normal, and there was no astereognosis. The grasp was 42 on the right, and 27 on the left. The chest was negative, both heart and lungs being normal. Pulse 144. Complains of slight headache, and of dyspnea on exertion. Menses every three weeks, with some pain, and there is a retroversion. Headaches are more on the left side of the head. Complains of passing much urine, but the urine contains no sugar.

On March 6, 1900, she had had a headache during which she had vomited, which had not occurred for a year after the loss of vision. She was last seen on July 6, 1900, when the condition remained the same.

The third case was that of a young woman, twenty-seven years old, single, a dressmaker, who was first seen by me in consultation with Dr. Loring on May 31, 1904. Her father,

mother, seven sisters and four brothers were all living, and healthy, with the exception of her father's trouble spoken of later. One other sister had died at the age of three years, from some unknown cause. There was no nervous disease, rheumatism, tuberculosis, epilepsy or insanity in the family, except that the father had been subject to periodical headaches, beginning in youth, and continuing for many years, which were much more severe than those of the patient, but similar in character. But these had gradually become milder, and he had had none now for several years. No other member of the family had had such headaches.

The patient's headaches began at about the age of twelve years, when menstruation was established, and came about once a month, almost always about two weeks after the menses. The pain was in both temples, but apt to be more severe on the left

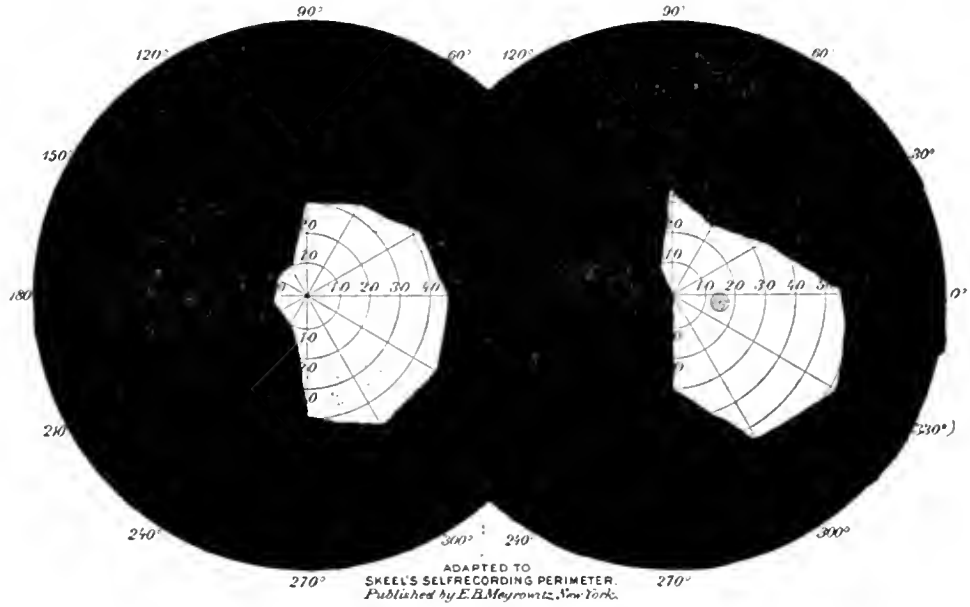

Fig. 2. Case 2.

side, and the left eye usually ached first. She had no scotoma. The pain usually lasted about two hours, when she almost invariably vomited, after which she felt relieved. The headache was throbbing in character, and was not associated with indiscretions in diet. She is pale during the attack. She had had no illness since measles at ten years of age.

She went to Dr. Loring first on April 4, 1904. Shortly before this she had had one of her headaches not very severe, and of the usual character, and at the ordinary time, two weeks after the menstrual period. She felt dizzy and had some pain, but no vomiting. The pain lasted from eight in the morning till noon, and then as it was no better she got up, when she noticed that she was dizzy. The dizziness lasted three days, and after that she felt well. She noticed no awkwardness in using the hands,

or weakness, nor numbness in the face or elsewhere. She did notice some difficulty in seeing, but thought this was only in the right eye, and finally went to the oculist, because she found that she bumped into people on the street, and then realized she had not seen them.

Dr. Loring found the fundus normal and a right hemianopsia which had not changed.

There was no constant headache, no dizziness, and no palpitation. Neither was there any vomiting, nor pain in the eyes. Sleep was good. She did not have to rise at night to urinate, and there was no dyspnea on exertion.

On examination the gait and station are normal. The grasp, right and left is 18 kgm. Strength in the extremities equal and normal. No disturbance of sensation for touch, pain, or temperature anywhere. No inco-ordination, or disturbance of the sense

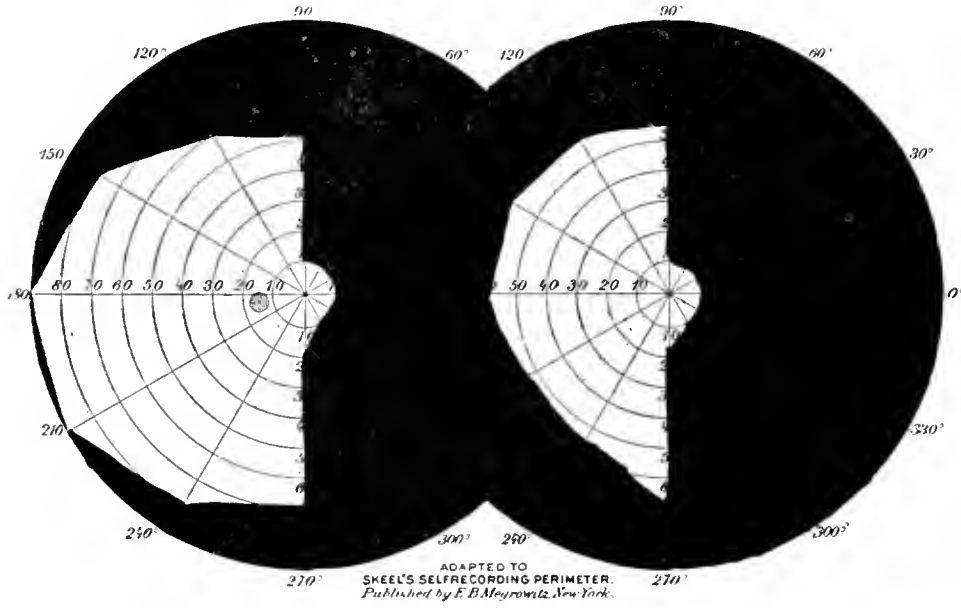

Fig. 3. Case 3.

of position, or astereognosis. External ocular muscles are normal. Pupils are equal, and respond normally to light and distance, both sides of the retina causing the response to light equally. Right hemianopsia. Triceps, biceps, and supinator reflexes equal and normal. The knee jerks are normal, equal and reinforceable. The ankle jerks are equal and normal, there being no ankle clonus, or front tap contraction. The plantar reflex shows extension of the outer toes, but no movement of the great toes, and is alike on the two feet, and there is no Babinski's or Oppenheim's sign. The abdominal and epigastric reflexes are equal and normal. The heart is normal. Blood vessels not stiff. Hearing good in each ear, and equal, air conduction being better than that by bone. Reads normally. Writes normally, both spontaneously, copying

and at dictation. No aphasia, paraphasia or other disturbance of speech. No evidence of syphilis.

The patient was again examined by me on May 27, 1906, when she said the headaches had come since the onset of the hemianopsia just the same as before that, but that perhaps the pain had not been quite as severe. The relation to the menses had not been altered, and the vomiting had remained the same. The last headache she had had at that time, had been on May 19, 1906. Examination showed the hemianopsia still existing. The blood pressure was 120mm. of mercury. The urine 1.024 without albumin or sugar. All the other details of examination remained as at the previous examination.

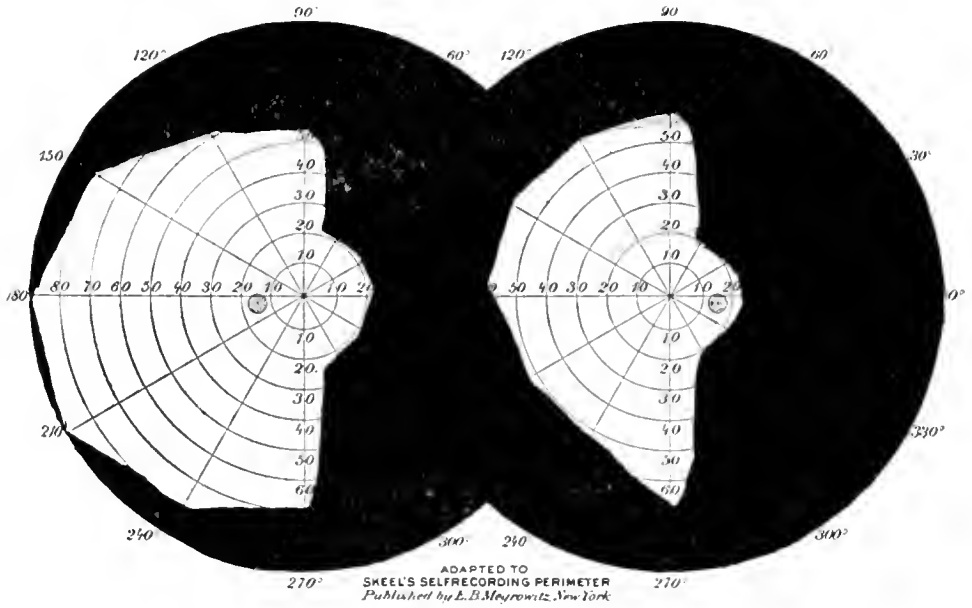

Fig. 4. Case 3.

A consideration of these cases of permanent hemianopsia, and cerebral paralyses, and aphasias reported by various writers shows conclusively that such accidents occurring during an attack of migraine are not exceedingly rare, and at least indicate that migraine may be the exciting cause of a cerebral thrombosis, or possibly a hemorrhage, but, as I have already said, in most cases there is at least strong ground for believing that the cerebral lesion was due to arterial disease, the migraine being at the most only an exciting cause and in some cases the headache was probably symptomatic only, while in other cases the presence of migraine at all, even as an independent disease, seems very doubtful. Féré's case of aphasia, reported in 1881, Oppenheim's, Queirolo's Flatau's, Féré's case 10, Schroeder's and Hoeflmayer's were probably compli-

cated by vascular disease. In Féré's case 6, I should doubt even the existence of migraine. Féré's case 9, though incomplete, and Infeld's form the only ones I have been able to find in which it seemed even possible that the migraine attack was responsible for the cerebral lesion in the absence of disease of the blood vessels.

Of the cases presented in this paper, the first one seems, from the history, the absence of heredity, and the nature of the attack itself, to have been due to arterial disease, and it is probable that the migraine attack was no more than an exciting cause for the vascular lesion, and possibly only a symptomatic migraine. The second, and particularly the third case seem to me to be clear cases where we can ascribe the cerebral softening to nothing else than an attack of migraine. In the second case the presence of sugar in the urine was only temporary, it being absent in two later examinations, and only present at the first examination, so that it was probably symptomatic only. In neither case was there any evidence of kidney, heart, or arterial disease, or of syphilis.

The general opinion of writers upon migraine is that the cause of the attacks is a vasomotor disturbance, probably dependent upon some toxic cause of unknown origin, though the former division into cases of vaso-constriction, and dilatation must undoubtedly be given up, if for no other reason than that the condition of the superficial vessels, from which the argument was drawn, often varies in the same person at various stages during a single attack. Spitzer in 1901 advanced an ingenious theory to account for migraine. He ascribes it to changes apparently supposed to be inflammatory in character about the foramen of Monroe, which produce a relative or absolute stenosis, then he also assumes a hyperemia causing an increase of fluid in the ventricles, and so a pressure which is greater upon the veins; and hence there is added a passive hyperemia, and he thinks often an actual tearing of the tissues and hemorrhages. When the subdural spaces are filled the headache begins, then the ventricles dilate, the foramen opens, and the fluid passes off. This is of course pure theory and I know of no recent theory of disease which is founded upon fewer facts, and his view has found little favor among other writers. Various objections have been urged

to the theory, such as the fact that all symptoms disappear in the intervals between the attacks, which we should hardly expect in the case of organic changes such as have been supposed. Neither does such a theory explain the frequent unilateral character of the headache, or its shifting from one side of the head to the other during an attack, nor its heredity.

Moebius thinks that in migraine there are changes in the cells in the brain. Oppenheim considers a vasomotor constriction of the vessels the most probable explanation. Stekel and Meige and most other recent writers agree with this opinion.

In considering the cases which I have collected in this paper, I think we can conclude that attacks of migraine may result in an area of softening in the brain, which shows itself by a permanent paralysis, aphasia, or hemianopsia, and that in most instances this is due to the attack favoring a vascular lesion in persons who have already disease of the walls of the blood vessels, but that in certain cases the vascular lesion may occur in young persons whose blood vessels are in all probability in a normal condition. In all cases of organic disease of the brain, coming on during an attack of migraine extreme caution is necessary before ascribing even an exciting rôle to the migraine, as it is much more common to find independent organic disease of the blood vessels which would account for the organic disturbance in persons who have suffered from migraine, yet a few cases remain which can hardly be explained in this way, and justify the statement made by Charcot and others that the transient hemianopsia or aphasia seen occasionally in this disease may become permanent. Still this accident does not seem to be only, or even more common in the cases having the temporary phenomena, then in those where it has never appeared.

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PERIPHERAL FACIAL DIPLEGIA AND PALATAL INVOLVEMENT.*

By George W. Jacoby, M.D.,

of New York.

Facial diplegia in its complete form has always excited the interest of the observer, not only on account of the comparative rarity of its occurrence, but also on account of the striking clinical picture which it presents and of the distress and inconvenience which it causes the patient.

Since Charles Bell, in 1836, first made known to us this clinical picture, the subject has been studied in its entirety by Davaine[1], Wachsmuth[2], Pierreson[3], and Sainton[4], while very many other observers have given us reports of single instances.

Indeed so many single instances have been recorded and Sainton has so recently given such a complete and excellent description, with full literary references, that it would be superfluous to publish still more personal observations were it not for the fact that observers still differ as to the presence or absence of certain symptoms. Nor would this alone induce me to report the four cases which I have observed were it not for the fact that in my opinion their study adds weight to our present position in regard to the nerve supply of the soft palate.

In such a study we must bear in mind that facial diplegia may occur in diseases of the pons and medulla oblongata, as part of a multiple palsy of cerebral nerves (Rad[5]), in the course of cephalic tetanus, as well as, and particularly in, the course of a multiple neuritis (Pierreson, Eisenlohn, Strümpell[6], Starr[7], and others), due to rheumatism (Romberg, Mott, Stintzing[8],

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[1]Davaine, C. M. Gazette médicale de Paris, Nos. 46, 48, 50, 1852; Nos. 2, 3, 1853.

[2]Wachsmuth, Adolf. Monograph. Dorpat, 1864, Part II., pp. 81-114.

[3]Pierreson. Arch. générales de médecine, August, September, 1867, Vol. X., pp. 159-296.

[4]Sainton, M. Paul. Gazette des hôpitaux, 1901, p. 1265.

[5]Rad, C. Von. Zeitschrift f. Nervenheilkunde, Vol. XVII., 1900.

[6]Strümpell, Ad. Neurol. Centralblatt, 1889, No. 21.

[7]Starr, M. Allen. Organic Nervous Diseases, 1902.

[8]Stintzing. Munehene med. Wochenschrift, January, 1893, Nos. 1, 2.

*Read by title at the meeting of the American Neurological Association, June 4 and 5, 1906.

Raymond), alcohol (Oppenheim[9]), influenza (Barkas[10]), or any other toxic or infectious cause.

Only such cases as are certainly peripheral in their origin are serviceable for casting any light upon the question, and of these, only those in which clinically the dominant symptoms are those of the facial diplegia. The supranuclear cases and the nuclear, or bulbar ones, cannot be utilized in this connection.

The nerve supply of the palate has been the cause of much discussion. In 1852 Davaine, writing upon facial diplegia, stated that the majority of anatomists believed the nerve fibers to the levator palati and azygos uvulæ muscles to be derived from the facial through the connection of its trunk with the Vidian by the petrosal nerves. Debron[11] had, about this time, by means of galvanic experiments upon dogs, shown that the nerves to the soft palate did not go from the seventh, but from the glossopharyngeus directly, and Davaine, in order to explain this divergence of views, endeavored to show that the fibers from the glossopharyngeus should be looked upon as sensory and those from the facial as motor fibers of the soft palate, and that therefore paralysis of the palate, when it occurred, was actually caused by affections of the facial.

From 1866, when Longet cast the weight of his authority into the scales, it had been accepted as a fact that the facial nerve governed the contraction of most of the muscles of the soft palate and that this motor influence was effected in the manner already mentioned.

The acceptance of this anatomical condition led to the clinical agreement that when in peripheral facial palsy the lesion was located above the geniculate ganglion there ought to be paralysis of one side of the soft palate demonstrable by relaxation and inability to raise the palate upon the paralyzed side, with a deviation of the uvula to the opposite one.

In 1873 Flint[13] summarized the state of knowledge of that time as follows: "In view of the pathological examples of paralysis of the palate and uvula in certain cases of facial palsy,

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[9]Oppenheim, H. Lehrbuch der Nervenkrankheiten, 1905, Vol., p. 405.
[10]Barkas, W. G. Lancet, Jan. 26, 1896, p. 217.
[11]Debron. Thèse de Paris, 1842.
[12]Longet. Traité de physiologie, 1866, Vol. I., p. 796.
[13]Flint, A., Jr. The Physiology of Man, Vol. on the Nervous System, N. Y., 1873, pp. 159 *et seq.*

the frequent occurrence of contraction of the muscles of those parts upon galvanization of the facial, and the reflex action through the glossopharyngeal and the facial, there can be little doubt that the muscles of the palate and uvula are animated from filaments of the seventh nerve."

Later Erb, Seeligmuller, Leube, and Chevostek lent the weight of their authority to the faciopalatal innervation theory, and until 1893 this theory met with but scant opposition. In that year Gowers[14], writing of facial palsy, said: "It has been said that the palate is sometimes paralyzed upon the same side as the face from disease of the facial nerve," etc. * * * "*but the opinion that the palate ever suffers from disease of the facial nerve seems to be erroneous.* The levator is supplied from the spinal accessory and in more than a hundred cases of facial paralysis due to disease of the nerve in various situations, carefully examined, I have never observed a corresponding defect of movement in the palate."

Later Hughlings Jackson[15], in connection with a case of paralysis of the face and palate which seemed to prove the association between peripheral facial paralysis and palatal involvement, but in which closer study demonstrated the cerebral origin of the facial palsy, said: "The uninstructed might have mistaken the case for one of Bell's palsy with paralysis of the palate, a combination of symptoms which I (Jackson) have never met with."

More recently Koester[16], examining thirty-three cases of facial palsy, could find no case with implication of the soft palate, so that he also is a sceptic as to the existence of such a combination.

Since then considerable has been written upon the subject, the majority of authors taking a position in support of Gowers, Jackson, Lermoyez[17], and Koester, while others still maintain that palatal involvement may form an integral part of the clinical symptomatology of peripheral affection of the facial nerve.

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[14]Gowers, William. Manual of Diseases of the Nervous System, Vol. II., 1893, p. 236.

[15]Jackson, Hughlings. Lancet, April 2, 1887, p. 680.

[16]Koester, George. Deutsches Archiv für klinische Medicin. 1900, Vol. LXVIII., pp. 343 and 505.

[17]Lermoyez. Presse médicale, May 7, 1898, No. 39.

Maum[18], of Dresden, writing as late as 1904, deplores the tendency to consider palatal palsy occurring in peripheral facial paralysis as being due to vagoaccessory affections, and, publishing four cases of such a combination due to acute middle ear disease, stoutly maintains that both symptoms are dependent upon an affection of one and the same nerve. He maintains that the type of palatal palsy occurring in vagus affection differs clinically from that due to facial nerve affection.

Others yet take a middle path and assert that, while palatal involvement does occur (Tomke[19]) with more or less frequency in peripheral facial palsy, it is never due to lesion of the facial, but is a result of contemporaneous affection of other nerves (the spinal accessory or its palatine branch). The arguments which have been adduced on the one side or the other are as follows:

1. From the point of view of experiments in physiology.

a. Excitation of the facial.—Béclard[20] (in 1866), Rad, Debron, and more recently Chauveau (experimenting on the horse), Beevor and Horsley (from experiments on various animals), Vulpian[22] (experimenting on the dog), and others are agreed upon the fact that electrical excitation of the facial muscles causes no movement whatsoever in the palate.

The experimental investigations of Rethi[23] have shown that the levator palati mollis is not supplied by the facial, but by the vagus, and Koester, experimenting upon animals, was never successful in observing a paralysis of the soft palate after destruction of the facial nerve as high up as the geniculate ganglion.

b. On the other hand, excitation of the tenth and eleventh pairs produces energetic contraction of the palate.

All these experimental investigations and a case of traumatism (stab wound in a man, Hoffmann[24]) show more and more conclusively that the vagoaccessory carries the chief if not the entire part of the innervation of the palate.

2. Clinical. Here the two questions must be answered.

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[18]Maum, Max. Zeitschrift für Ohrenheilkunde, Vol. XLVII., 1904, pp. 1 to 39.
[19]Tomke. Archiv f. Ohrenheilkunde, Vol. XLIX., 1900.
[20]Béclard. Traité de physiologie, 1866, p. 1010.
[21]Beevor and Horsley. Proc. of the Royal Soc., May 16, 1888.
[22]Vulpian. Acad. des sciences, Oct. 18, 1886.
[23]Rethi. Wiener med. Presse, 1893, No. 50.
[24]Hoffmann, J. D. Zeitschrift f. Nervenheilkunde, Vol. V., 1894, p. 72.

a. Do lesions of the vagoaccessory produce palatal paralysis?

b. Do lesions of the facial produce such paralysis?

The answers to *a* are unanimous and conclusive, viz., paralysis of the soft palate on one side and recurrent laryngeal paralysis upon the same side, without facial palsy, are of not infrequent occurrence. Cases are known in which paralysis of the palate and larynx was accompanied by paralysis of the sternocleidomastoid without being accompanied by facial palsy.

The answers to *b* are more divergent, being affirmative (for competent observers have recorded cases of facial paralysis in which the levator palati and azygos uvulæ were more or less completely paralyzed) as well as negative. An analysis of the affirmative cases, however, shows that certain objections have not been met by the reporters of these cases, viz.: A certain immobility of the palate is physiologically not infrequent. Local affections of the naso-pharynx which may give rise to partial or complete paralysis of the palate have not always been excluded. Congenital asymmetry may be mistaken for unilateral palsy. Sufficient care has not been taken to search for symptoms pointing to a vagoaccessory lesion in addition to the facial one.

On the other hand, it must seem strange that the mere clinical association of facial and palatal paralysis, leaving aside for the nonce the physiological relationship, should have been so infrequently observed by certain clinicians, while others have been able to note its occurrence repeatedly.

It has seemed to me that the explanation of these variations of observation must be sought in the period of time at which the cases have been examined, that cases examined very early in their course will be the ones which may show a palatal involvement, while those cases in which the facial paralysis has existed for some time will certainly show none. We understand, since Broadbent has explained it to us, that those muscles of the body which are used only in conjunction with their fellows of the opposite side have a bilateral cortical representation and can be excited to action from either hemisphere,that these same muscles are equally innervated, or nearly so, through the bulbospinal centers from each hemisphere, the impressions being conveyed freely in either direction through the commissures; and that destruction of the cerebral center of one hemisphere

does not cause paralysis of those muscles which are completely bilateral in their actions.

This is well shown when the respiratory muscles, those of phonation, and the muscles of the trunk or the abdominal muscles are involved in consequence of unilateral lesion.

As I have shown elsewhere, this law applies in a way also to peripheral palsies of the same muscles, only here there exists in the beginning a complete paralysis, which in a comparatively short period of time may be overcome by an interposition of the unaffected contralateral hemisphere, so that a certain amount of function or complete function is re-established at a time when other muscles supplied by the affected nerve are still paralyzed or paretic.

Synergic action of both palatal halves under all circumstances shows that they are excitable from either hemisphere, and that therefore if one side of the palate is paralyzed, the excitation could still take place from the opposite hemisphere and the unilateral palsy thus be minimized or perhaps overcome. It has therefore occurred to me that herein we perhaps not only need the explanation of why paralysis of the palate was infrequently or never found in facial paralysis, but also why cases of diplegia facialis, both paths from the cortex being involved, should show a persisting palatal involvement, one which clinically would keep pace with the paralysis of the rest of the muscles affected, provided the palatal muscles were innervated by the facial nerve.

A study of the published cases of facial diplegia from the point of view of palatal involvement is not very satisfactory, for in many of the cases no reference is made to the condition of the palate, and in others it is stated that speech was "nasal in character" and that "no paralysis of the palate existed," two statements which can be reconciled only with difficulty.

In a general way it is stated by Rosenthal[26] that "speech is nasal," by Grasset[27] that "the voice is nasal, liquids pass through the nose," and by Turner that "in complete bilateral facial palsy there is no palatal movement on using the vowel sound "ah."

[25]Jacoby, George W. Sign of the Obicularis, Etc. JOURNAL OF NERVOUS AND MENTAL DISEASE, October, 1903.

[26]Rosenthal, M. Lehrbuch der Nervenkrankheiten. Erlangen, 1870, p. 487.

[27]Grasset. Maladies du système nerveux.

and the pronounciation of words requiring the closure of the vaso-pharynx is rendered imperfect, so that "rub" is pronounced "run" and "egg" "eng."

Davaine speaks of the "nasal tone and the paralysis of the velum palati which causes it," and says, furthermore, "when both facial nerves are paralyzed, the velum palati cannot be moved, cannot be lifted, in consequence of which air passes into the posterior nares and thus the nasal tone is produced, and fluids and mucus easily find their way into the nasal passage.

On the other hand Sainton says that "in peripheral diplegia the soft palate is usually intact," and, "of twenty cases collected by Wachsmuth in 1864, in eighteen the palate was unaffected while, of thirteen cases published by eleven different observers since then, which I have analyzed, in only two was such paralysis described."

This infrequent association of palatal palsy and facial diplegia, occurring only four times in thirty-three cases, certainly does not lend support to the facial innervation theory. It is strange that, of the following four cases which I have personally observed, three should show palatal palsy, yet their careful study evinces that here also this palsy is explicable in another way, and that they therefore also speak against the above-mentioned theory.

My cases were as follows::

Case I.—B. S., æt., nineteen, seamstress, came under observation October 28, 1901; she had been perfectly well until October 15; then complained of general malaise, had pains in the extremities and especially in the right side of her face and in front of the meatus of the right ear. This condition continued until the 23d, when she noticed that she could not close the right eye as well as the left, and when she arose the next morning (the 24th) her face was drawn to the left side. This condition remained unaltered during that day, but when she awoke on the 25th her face *was straight again,* but she was unable to move her mouth from side to side and she was told that her eyes did not close. She also had some difficulty in swallowing and complained of pain in front of the meatus of each ear. In contrast to the distortion observed in cases of unilateral facial palsy, the patient attracted attention on account of the marked smoothness of the entire face and on account of its complete immobility and mimic rigidity.

The cheeks and angles of the mouth were flattened and the

eyes could not be closed completely and showed the usual rolling upward of the eyeball when closure of the lids was forcibly attempted. She could not pucker up her mouth to whistle, nor could she blow out a candle or retain fluids in the anterior part of her mouth. She seemed to secrete a great deal of saliva, which was constantly dribbling from her mouth. The lips were slightly separated from each other and could not be approximated.

There was some difficulty in chewing (due to weakness of the buccinator), but none in swallowing. Very noticeable was the inability to pronounce any labial letter, b, p, m, v, and f being supplanted by d, t, n, and th. Otherwise speech was in nowise interfered with except that it was decidedly nasal in tone. The tongue could be protruded perfectly straight.

Examination of the fauces showed the uvula to be relaxed in the median line, with its point resting upon the base of the tongue; there was no asymmetry of the arcades of the palate, but the *velum palati hung relaxed and motionless and did not move upon deep inspiration, upon intonation or upon tickling.* At the same time an almost complete insensitiveness of the palate was noticeable, the patient saying that she felt no touch there; very striking was the ease with which she bore all pharyngeal examinations; the tonsils, the fauces, and the base of the tongue could be freely touched without causing any retching or contraction. Her hearing was good, there was supersensitiveness to noises, and there was no disorder of taste. An electrical examination subsequently made showed reduction of electric excitability on both sides, especially marked in parts innervated by the lower facial. The palatal musculature was not tested.

The course of the case was the usual one of a facial palsy of medium severity, the improvement being steady, but one side, the right, always being somewhat in advance of the left. Ten weeks after the onset complete recovery had taken place. The palatal paralysis was noted at each examination until the fourth week, when, the patient not having been seen for a week, the palate was found to react perfectly upon phonation, *but it did not react reflexly and the entire fauces presented the same insensitiveness to manipulation which has been described.* During the entire course of the trouble no other symptoms than those noted were present—no aphonia, no palpitation, no respiratory difficulty.

Remarks.—This case certainly seems to be one which could be used in support of the faciopalatal innervation theory, as have been those already referred to and published by other observers.

Now, leaving aside all purely anatomical considerations, with the necessary deduction that this case is one with lesions of the facial nerve at or above the geniculate ganglion, we note that the patient presented certain sensory symptoms which persisted and were still present after the palatal paralysis had passed

away. Sensory symptoms have been noted in cases published by others (Stintzing[28]), and it is stated that the palate acted upon phonation, but not reflexly. Whatever value such symptoms may possess from a diagnostic point of view, this value certainly does not lie in the line of support of the faciopalatal innervation theory. In view of our knowledge that sensory fibers from the glossopharyngeus and trigeminus probably reach the palate, and certainly do reach the tonsils, posterior pillars of the fauces, and posterior parts of the tongue, while it is just as certain that the facial takes no part in this sensory innervation, we must conclude that in this case there were symptoms present which point to the involvement of other nerves than the facial, and that therefore the vagoaccessory may also have been involved and have been the cause of the palatal palsy.

Case II.—Mrs. K., æt., thirty, seen in consultation June 16, 1905. She had never been sick. She has four children, the last born about ten days ago. The labor was a perfectly normal one without rise of temperature. On the third day she complained of pains in the right leg and of numbness in the great toe of the right foot. On the following day the other toes of that foot and the toes of the left one became numb. She then had pains all over the body, and on the fifth day numbness of the fingers and of the legs set in and the speech became nasal in character.

On the sixth day she was attacked with left-sided facial paralysis, and two days later the right side became similarly affected. At this time she also complained of buzzing and roaring with pain in both ears.

Examination shows a complete bilateral facial palsy. She cannot close either eye completely, but closes the right eye better than the left. She cannot wrinkle her brows or corrugate the forehead, cannot pucker her mouth, whistle, or blow out a light. The complete paralysis of the upper lip is apparent, and she cannot pronounce a single labial letter, but linguals are pronounced perfectly. Her face is entirely expressionless and gives no evidence of emotion. According to her statement she is no longer able to cry, and *lachrymation has ceased entirely since the attack; no tears are secreted.* Her laugh is merely an inward grunt, and certainly here the words of Davaine are applicable, that "the face seems like a lifeless mask behind which the patient laughs or cries." The masseters were not involved.

The tongue was protruded straight and was freely movable in every direction. Taste was unaffected. *The palate reacted but imperfectly* to direct (reflex) and to indirect (voluntary) stimulation.

During the course of that day her speech became more decidedly nasal in tone and fluids returned through her nose upon attempts at swallowing. She also had paroxysmal attacks of pal-

[28]Stintzing. Münchener med. Wochenschrift, January, 1893, pp. 1 and 2.

pitation. On the following day (the 17th) *complete palatal paralysis was present.*. Hearing was overacute in both ears, and she was supersensitive to slight noises.

An electrical examination showed reaction of degeneration in all the facial muscles; mechanical excitability of these muscles not increased. Marked tenderness existed in the arms all over the nerve trunks; she could not button her clothes easily, and with her eyes covered she did not know whether she had dropped objects from her hands or not. There was no muscular paralysis, and there was no atrophy. Both knee jerks and both foot jerks were absent, and an extensor paresis of both feet was observable. This condition remained unaltered until the 24th, when she was attacked with lobar pneumonia and died on the third day, with complete consolidation of one lung.

Remarks.—This case is clearly one of multiple neuritis with facial diplegia and palatal involvement. The attacks of palpitation and the difficulty in swallowing indicate that the vagus was involved. The clinical deduction that the paralysis of the soft palate here was a vagoaccessory symptom has quite as much to support it as the assumption that this symptom was due to affection of the facial nerves.

Case III.—Seen in hospital service. Mr. S., æt., forty, November, 1896. There was a marked alcoholic history. The patient came into the hospital suffering from a multiple neuritis. There was drop foot on each side, with tenderness over the nerve trunks of both lower extremities, knee jerks, foot jerks, and sensory disturbances were absent in the legs. The upper extremities showed no objective symptoms, but the patient complained of paresthesia in the fingers of both hands.

In addition to these symptoms, persistent tachycardia was present: the pulse never went below 108, and paroxysmally rose to 180.

On the eighth day after his admission the patient had facial palsy of the left side with disorder of taste; two days later a similar paralysis set in upon the right side. The face then was perfectly smooth and almost devoid of wrinkles.

Chewing and swallowing were attended with difficulty, and here also the characteristic symptom of facial diplegia, the inability to utter any labial letter, was present.

On the following day his speech became nasal and he showed *unilateral palsy of the soft palate*, the palate being relaxed and immobile on phonation, but the relaxation and immobility being confined to the right side; the uvula was distinctly turned toward the paralyzed side. At the same time his voice became hoarse and rough, stridor upon deep inspiration was present, and he had slight difficulty in swallowing. Laryngoscopic examination revealed the right vocal cord standing in the median position and not taking any part in phonation and respiration.

The tongue could be protruded straight.

Ten days later, the clinical picture remaining about the same, an electrical examination showed partial reaction of degeneration in the lower facial territory of both sides and distinct reaction of degeneration in the right side of the palate.

This patient was seen at intervals for about three months, during which time all the symptoms, except the palatal palsy and the vocal cord paralysis, disappeared. These later symptoms persisted at the time of his discharge from the hospital.

Remarks.—We are here dealing with a multiple neuritis of alcoholic origin in which numerous symptoms of vagus involvement are present. These symptoms are aphonia, palpitation, and respiratory difficulty. Cases of palatal palsy with recurrent paralysis are not unknown, and a form of vagus neuritis limited to one recurrens is occasionally encountered. In multiple neuritis, especially the alcoholic form, bilateral facial palsy is not unusual (Starr), nor is vagus neuritis infrequently met with (Mays).

The simultaneous occurrence of bilateral facial palsy and unilateral recurrens paralysis has, so far as I know, not previously been described: that the palatal palsy here is also due to vagus involvement is, I think, proved by the fact that it, as well as the recurrens paralysis, is unilateral and both are upon one and the same side, while the facial palsy is bilateral. Of interest also is the degenerative change found on the right side of the palate, showing that the vagus complication was also a neuritic one.

Such electrical changes in the palate have been found by Hill[29].

Case IV.—Bilateral facial palsy without palatal involvement. A woman, æt. thirty-four, had pains in both ears for several days, when, in October, 1904, she awoke with facial palsy of the right side, especially noticeable on talking and chewing. Three days later the left side became affected. There was a marked affection of taste, but no dryness of the mouth; taste was lost on the anterior two-thirds of the tongue, the patient being unable to distinguish salt from sugar or vinegar from a solution of quinine. The ears were very sensitive to noises; sensation everywhere in the face was unaffected. There were no changes of electric excitability in the muscles or nerves.

Speech was not nasal in tone, but the interference with the formation of labials was the same as in my other cases. The palate responded perfectly to all forms of stimulation.

During sleep the lids, which otherwise could not be closed, were gradually and completely approximated, so that the entire eyeball became covered.

Recovery took place in about eight weeks.

[29]Hill. British Med. Journal, Feb. 2, 1899.

General remarks.—It has been stated that paralysis of the dilatatores narium and the compressores narium renders the alæ flaccid, so that in breathing they move to and fro and close the nostrils upon deep inspiration. Thus, in the case of Labadie Lagrave[30] and Emile Boix it is stated that the alæ nasi were passive and, instead of dilating at each inspiration, had a tendency to close the nares. This was especially noticeable in an effort to smell anything, such as cologne.

Were this a fact, then another explanation than that of weakness of the soft palate could hereby be given for the nasal tone which at times accompanies bilateral facial palsy, but in none of my cases could this statement be made, and the nostrils were neither dilated nor contracted, but remained patent throughout, being merely somewhat narrowed. It is indeed difficult to believe that this could ever be otherwise, for the inner and outer walls of each nostril are formed by lateral cartilages, so connected to the upper cartilages and the front part of the cartilage of the septum, as well as with the nasal process of the superior maxilla, that inactivity of the muscles which act upon them can never cause them to collapse.

The nasal tone is therefore always due to weakness of the velum palati, and when this symptom is present, and it has been present when the palatal movements were not decidedly interfered with during phonation, the conclusion is warranted that paresis of the palate exists. This speech disorder due to palatal weakness, being added to that caused by the labial paralysis, greatly emphasizes the characteristic speech difficulty.

Reports differ as to the position of the uvula in unilateral facial palsy, some observers saying that it is turned toward the paralyzed, others that it is turned toward the sound side, so that as far back as 1853 Debron looked upon the deviation of the uvula as an accidental occurrence in health, having no concern with the facial paralysis, and Bernhardt, in 1876, refused to draw conclusions as to the involvement of the palate from such deviations in posture. To-day the complete symptomatic insignificance of such deviation is accepted.

So also in bilateral facial palsy it would be unwarranted to infer palatal weakness from deviation of the uvula.

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[30] Archives générales de méd., 1896. Vol. I., p. 23.

In all of my cases the tongue could be protruded straight, thus showing what has been demonstrated by Hitzing[31] and since accepted by every one, that the tongue does not deviate in facial paralysis, but that the apparent deviation is due to crookedness of the face and can be corrected by straightening the mouth.

In bilateral facial palsy, there being no distortion of the mouth, the tongue is not protruded to one side.

In all of my cases the facial palsy of the two sides came on at intervals, first one and then the other side being affected. This has been so also in all reported cases, and in one of Hoffmann's[32] the palsies came on a month apart.

The classical symptom of absolute inability to utter any labial letter was present in all my cases, and in all of them it was of interest to observe how little interference with such pronunciation the unilateral palsy caused, and how marked it became when the other side became paralyzed.

In conclusion, and referring again to palatal palsy as a concomitant of bilateral facial paralysis, I would call attention to the necessity of seeking for symptoms which cannot be explained by facial nerve involvement alone, such as palpitation, sensory disturbances, hoarseness, etc. Whenever such symptoms are found to be present persistently, the conclusion is warranted that some other nerve than the facial has been implicated by the morbid process and that the co-existent palatal palsy must be due to such implication.

This conclusion agrees entirely with what we know of the associated functions of the vocal apparatus, for, as Lermoyez[33] and Rousseau[34] have so well said: "Is it not more logical to admit that the larynx and pharynx (the reed and the resonator), which are after all but two parts of the same vocal apparatus, should respond to the influence of one and the same nerve? And would it not be difficult to understand that the larynx and soft palate, destined to live in accord, should receive their orders from two nerves so foreign to each other as are the facial and the vago-accessory?"

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[31]Hitzing. Gesellschaft für Psychiatrie, Nov. 14, 1892.

[32]Hoffman, J. Deutsche Zeitschrift für Nervenheilkunde, Vol. V., 1894, p. 72.

[33]Lermoyez, L. Presse méd., May 7, 1898, No. 30.

[34]Rousseau, L. Thèse de Paris, No. 503; also Gaz. hebd. de méd et de chirurg., 1898, p. 1009.

A STUDY OF THE SENSORY SYMPTOMS OF A CASE OF POTT'S DISEASE OF THE CERVICAL SPINE.*

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of St. Louis.

I have decided to present the following record because it well illustrates the difficulties of sensory studies. I had started to say some of the difficulties. But only those who are most used to making sensory examinations know best that they all present difficulties. In this case I am not offering new sensory problems nor attempting altogether new interpretations of old ones. On the contrary I have selected a case where the problem will readily appeal to those having only an ordinary knowledge of the anatomy, physiology and disease of the spinal cord. By what is in one sense a simple case I may gain the sympathy of a larger audience among those whom we wish to impress with the necessity of careful sensory work and its difficulties. In attempting to do this I shall apply to this case, in as simple manner as possible, the most recent data.

Dr. Henry Head, whose important contributions to sensory studies have gained very wide recognition, has recently published some observations, the result of a remarkable work both in its amount and its scientific character. I shall briefly sketch certain of his conclusions which are of assistance in a study of the kind of a case now before us. The sense of touch may only be distinctly tested by the lightest touch to the skin, which he makes by using with great care little whists of "cotton wool." If this care is not used "light touch" will be confused with pressure sensibility (deep pressure sensibility), which is separate and distinct and presided over by its own end organs and fibers, which latter travel with the motor nerves. This distinction can be made clinically, and when assured by the careful methods which he describes is at times of much value. One may feel the lightest touch possible to be made with the point of a blunt lead pencil, for example, and yet be minus light touch sensibility, so delicate is the pressure sensibility. This Head proved by destroying one kind of nerves and preserving the other kind of sensibility and vice versa.

In the same manner he proved that one kind of nerves and

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their end organs recognize only warm and cool temperatures, and another kind hot and cold ones; the first, for example, temperatures between 20C and 35C. (or about these) the latter only temperatures below 20C or above 50C (or about these). He shows that spacing sensibility is also distinct in kind and intimately connected with light touch. Although we may recognize by deep pressure sense when a certain part is touched, and locate this sensation with a good deal of accuracy, we cannot recognize with this kind of sensibility the two points of the dividers (esthesiometer). This is only accomplished by the "spacing sensibility" which may be absent in the presence of the pressure sense and vice versa. Head divides the sensibilities which we may test in the skin into three groups. In the first he places:

Sensibility to light touch.
Sensibility for spacing. — Epicritic Sensibility.
Sensibility for warm and cool.
(Between 20C and 45C).

In the second group he places:
Sensibility for pain.
Sensibility for hot and cold. — Protopathic Sensibility.
(Below 20C and over 50C).

In the third group he places:
Sensibility to deep pressure.
(Located in muscles, tendons and joints).

When a nerve which has been severed has been sutured and begins to heal, the second group of sensibilities begin to return within seven or eight weeks and are rapidly restored. On the other hand the sensibilities represented in the first group do not show any sign of returning for 200 to 300 days and the full restoration of them is proportionately slow. He therefore named the second group "protopathic" sensibility; and the first one representing a much higher kind of sensibility, he named "epicritic." There is at least one advantage in these new terms, namely, they facilitate description. There are several reasons, however, why he has coined this new word "protopathic". As we have just stated the fibers which conduct this kind of sensibility are the first to heal, and when repair of them has taken place to a certain extent, trophic functions begin to return which were previously absent. For example in a complete anesthesia of all kinds of

sensibility on the hand after severance of the ulnar, or another similar nerve trunk, a certain skin area is apt to be hard and dry and trophic ulcers apt to appear. With the return of the protopathic sensibility this portion of skin regains its moisture and the ulcers heal. This group of sensibilities is evidently closely associated with the vasomotor and trophic functions. In a region with disturbed protopathic sensibility there is also present what is generally spoken of as hyperesthesia *i. e.*, the prick of a pin or the contact of heat or cold causes a suddenly diffused, badly located sensation, with an irresitible tendency for the subject to wince *e. g.* to suddenly jerk the arm away when a protopathic area upon it is irritated.

He claims, and I believe proves, that what have been taken for different degrees of sensibility are actual differences in kind of sensibility. It will readily be seen that it is more important in almost any case to know the kinds of sensory disturbance than merely the degree of the same.

It is very easy to show the advantages of these distinctions in studying lesions of nerve trunks of various sizes. In this report I have attempted to show the advantage of keeping them in mind when studying a spinal case. In our case the onset was gradual, the pressure was very symmetrical, the subject in a hospital under constant observation, and an exceptional good one for this kind of examination.

Mrs. Chas. L. M., aged 25, married two years ago.; No important items in family history, nor in the personal or clinical history prior to the fall of 1904 (probably in the month of September). At that time she had a recurring slight pain in the back of the neck which was always (or nearly always) accompanied by a decided tingling paresthesia in the left arm. She describes it as coming in the outer side of the arm and very slightly in the fore-arm. About three months later, December, 1904, her baby was born and she was then much worse. In February, 1905, two months later, it was very bad. The pain was almost constant and she had severe paroxysms, during which the pain would radiate from the back of the neck out to the shoulder tips and up behind the ears and into the lower occiput. May 9th she went to Hot Springs, Ark., and remained seven weeks with no material benefit. During the summer months there was considerable relief, but the neck was always stiff and she was subject to frequent attacks during which the stiffness and helplessness

and pain were much increased. In the fall months she steadily got worse. I first examined her December 21st, about fifteen months after the first appearance of pain and stiffness. For several weeks she had been getting very little sleep on account of the pain. The head was carried in a fixed position and she had to be very careful in her movements to prevent pain paroxysms. It was almost impossible to get a position on the pillows in bed that would relieve her. She indicated the spines of the 2d and 3 C. vertebræ as the centre or starting point of the pain. From there it radiated into the shoulder, especially the left and to the sides and back of neck and up to the lower occiput and back of the ears. She insisted that the pains had never crept high up on the back of the head. At times she had felt considerable pain shooting down the left arm as far as the middle of the fore-arm, but usually the sensation here had been a tingling paresthesia. Rotation of the head was quite limited, but better than extension and flexion, which were almost nil. Pressure over the spines was not tolerated and there were many sensitive places in the muscles of the back and sides of the neck, and a generally rigid state. No objective sensory symptoms were discovered at this time. The deep reflexes of the left arm seemed greater than those of the right. The knee jerks were plus and there was some ankle clonus on both sides. A week later she returned to the city and was put to bed at St. Luke's Hospital.

December 29, 1905, careful observations were begun.

As already stated the knee jerks and ankle jerks were exaggerated on both sides and there was a slight ankle clonus, but there was no Babinski nor Oppenheim. After sitting still for some time her legs were often somewhat stiff, and slightly spastic in starting to walk; but she could walk and ascend stairs without marked fatigue. There had been no bladder weakness at any time and she did not complain of her back at any point below the level of the shoulders. She complained of an extensive area of paresthesia, a numb feeling, not especially uncomfortable, on the anterior and external aspect of the right thigh. (Very much like a meralgia paræsthetica). Occasionally she experienced somewhat the same feeling in the toes and feet. She had a paresthesia similar to this in the upper extremities, felt more distinctly in the right arm and hand. She was never entirely free from this, but it was at times more pronounced. Of this she did not complain so much as she did of another sensation, namely, a feeling of arrestment and lack of precision in the movements of the hands, arms and shoulders. She found writing and sewing increasingly difficult and her hands and arms tired too soon, in holding a

book, for example. This condition was more pronounced on the right side. And yet an actual inco-ordination could hardly be demonstrated. There were no localized atrophies. No groups of muscles seemed especially weak.

The sensory examinations included tactile, spacing, localizing, position, pressure, temperature and pain tests, all conducted with the precautions recently indicated by Head and others. Control tests were at times made on nurses. We were careful not to make the seances too long and to work only when the conditions were conducive to reliable results.

The patient is intelligent, well educated and of a temperament to make her an exceptionally good subject for this class of work. Between December 29 and January 8 we had examined the skin carefully on all portions of the body. On the left there was an area about the size of a silver dollar which embraced the edge of the trapezius half way between the acromion and the mastoid, where there was slight but evident hyperesthesia to pin pricks and to hot and cold. When this area was thus irritated to a certain extent she could not resist a jerking or shrugging of the shoulders. She could not here recognize warm and cool normally, only hot and cold, and light touch (cotton wool test) was slighty reduced, not lost in any portion of this small area. There was another spot not so large, on the anterior aspect of the deltoid over the head of the humerus where the conditions were similar. These two areas were so small and the deviations so slight that they could only have been established by the careful kind of an examination which we made.

In attempting to know the exact sensory state of an area of this kind it is necessary to have some tangible and conventional method of examination. I think it will readily be seen how Head's distinctions may be here applied, and the knowledge of them elucidates the description at once. But we may go farther and make certain deductions from them. We have here evidence that two classes of nerve fibers in the cord are involved. The epicritic fibers do not resist pressure as well as the protopathic. Here is an area which seems to show that both kinds of fibers have been only slightly disturbed, presumptively within the spinal canal, but so slight that it may not be taken into consideration except in a negative way in determining the condition of the cord.

These were the only objective sensory deviations present at this time. There were certain localities where there was at times a question of hyperesthesia; for example, at the bend of the elbow (*i. e.*, the plexor side), in drawing a needle upwards or downwards, as it reached this region the patient showed a tendency to flinch or slightly withdraw the arm.

But we found that this same condition obtained in normal subjects to such extent that we could not pronounce it abnormal in this case. It was the same on both sides.

The patient was kept in bed from the first, almost constantly. Within a few days the pain in the neck was rapidly diminishing, so that she was beginning to have restful nights. When she entered she could lie only with the left side of her head on the pillows and could rise from this position or change it only with much difficulty. She soon was able to sleep on the other side and turn from one side to the other with increasing freedom and to sleep all night with no medicine. She was also able to flex and extend the head with increasing freedom. Her general condition was very good in all respects. There was never any elevation of temperature. She had a good appetite and was in good condition. The function of the bladder was perfect. There was a constant constipation which was somewhat difficult to manage at times. She had all her life been of a constipated habit.

Between January 15th and 31st, routine examinations were made sufficiently to keep track of the sensory conditions, and no objective changes occurred. During this period she complained continually of a paresthesia in both hands, *e. g.*, she would say "things do not feel just natural," "I do not feel them distinctly" especially with the right hand. At the same time she could tell a penny from a dime with closed eyes and no objective defects could be established anywhere. By way of illustrating the manner in which the sensory tests were followed up we may cite a note made on January 18th. She was complaining of the paresthesia being most pronounced on the palmer surface of the thumb, index and second finger of the left hand. Tests were made as follows: light touch (cotton), position, location, pain, temperature, the latter made by spoon handles lifted from hot and cold, warm and cool water and dried before bringing them in contact with the skin. No objective changes were found.

During this period there is recorded a good deal of complaint of muscular startings in the right arm (brachial region) and right thigh. She also complained that the right foot and leg were "shaky" and "jerky" in walking (spasticity). There were at times between the 20th and 31st the suspicion of a Babinski and occasionally of an Oppenheim on this right side; none on the left side. The right knee jerk and ankle jerk seemed slightly greater than those of the left. February 6th we noticed for the first time that the spacing sense at the tips of the fingers on both hands, and especially the right index, was somewhat defective. Although very slight this obtundity seemed greater in the thumbs and the

first two fingers than in other digits. There was no question that the distal phalanx of the right index was most involved. In all other respects sensation seemed objectively normal. The strength of the right arm was less than that of the left, *e. g.*, in lifting a good sized pitcher of water she could manage it better with the left than with the right hand. She could not at this time detect any differences in the feeling or in the strength of the lower extremities. On the 16th, however, she called our attention to the fact that the right leg was less reliable than the left and that there was a constantly increasing paresthesia in the thigh of this side. The knee jerks seemed equal. Sensory tests of all kinds were made at frequent intervals especially over this area on the front and external aspect of the thigh. We here estimated light touch 75% as good as on the opposite thigh. (This was with a most delicate cotton test.) There was only slight loss of spacing sense and temperature seemed equal to the opposite side. She noticed that the toes of this side were objectively and subjectively cooler than the opposite side, yet the temperature sensibility of them was so nearly that of the opposite side that it would be very difficult if at all possible to establish the fact of a difference.

Between the last of February and 10th of March we could notice a very gradual falling off of the sensory acuteness of the fingers, hands and fore-arms and to a less extent of the feet and legs. This obtundity was slightly more pronounced in the right hand, and in the index more than elsewhere. Light touch and spacing sensibility were the most affected in all localities on the hands at this time, and other kinds of sensibility so slightly so that delicate tests were necessary to make the conditions present evident. She could recognize almost everywhere the difference between 30C and 40C and sometimes even closer. The pain sense seemed perfectly acute.

By the 20th of March there was a great deal of paresthesia in the hands and feet (but no pain), and the paralysis was rapidly increasing and all kinds of sensory losses were evident. Although the sensory conditions still varied from day to day it was now evident that light touch and spacing sense were most affected and next to these position, and temperature and pain still less. There were small, variable but distinct areas of slight protopathic and epicritic disturbance all the way up the arms, especially on the right. The anesthesia of all kinds in the hands was more pronounced in the fingers and shaded off upwards, *i. e.*, somewhat of the glove character, being more pronounced on the radial sides,

however. The hands were sweating a great deal and often cold and examinations were not easily made.

On the 26th of March she first experienced difficulty in emptying the bladder. Within a few days the catheter was constantly necessary. On April 1st the conditions were, briefly stated, as follows: R. upper exteremity; can flex and extend digits feebly except the index which she cannot extend, flexion and extension of wrist also very feeble. Pronation and supination complete but very feeble. Flexion of fore-arm almost lost, very feeble. Abduction and aduction of arm well preserved. The distribution of the paralysis was exactly the same in the opposite extremity but it was a shade less in all segments than that of the right.

The right foot, leg and thigh were all weaker than the respective segments of the left, and the right was more spastic. Babinski and Oppenheim reflexes could not be obtained on the right but both obtainable on the left. In other words they had disappeared from the right and appeared on the left. This was possibly due to the lowered sensibility of the right, or possibly to an increase spasticity which masked them.

At this time finer sensibility, *i. e.*, light touch, spacing and intermediate temperature sensibility were absent in the digits and the greater portions of both hands, but to a less extent, apparently, in the toes and feet. Position sense was lost in the digits, but not at the wrists until a little later date. Deep pressure, high and low temperature and pain sensibility were never altogether lost at any time, although much obtunded. A better idea of the conditions will be shown by the notes of several examinations made about this time, taking the right arm as an example: On the hypothenar margin an area two inches long and one and a half inch wide was hyperesthetic to heat, cold and pin pricks, sensitive to deep pressure, but insensible to finer sensation. Areas that were particularly hyperesthetic to pricks, heat and cold, were found just above the wrist, just below the elbow, about half way up the arm and just below the axilla on the ulnar side. Similar areas were found on the radial side, especially over the belly of the supinator longus and the belly of the deltoid. On both sides of the arm all the way up, areas were found where light touch was still well preserved. These areas were less difficult to outline than the protopathic ones but there were more of them over the size of a silver dollar. The same phenomena were present in all the other extremities and upon the trunk. The different kinds of areas were not disposed in the arms in a perfectly symmetrical manner but nearly enough so to show that they had a distinctly axial distribution, and were of spinal cord origin.

In this case the pressure was evidently from the front and so symmetrical and gradual that the different kinds of tracts in the cord all felt the effects of it and almost equally from before backwards. There were no symptoms pointing to focal lesions in either the anterior or posterior gray matter, hence no confusion in either motor or sensory signs from this source. There were no posterior root symptoms. The pains in the early history of the case although severe were not root pains. This is evident from the subsequent history. They were the kind of pains that have often been described as cervical neuralgia and were of peripheral origin.

There were no pupillary changes, and there were no respiratory symptoms that could be assigned to involvement of the phrenic nerves.

The uniform manner in which the distal portions of all extremities were most affected is explained by the well-known fact that the longest nerve fibers, or those which travel the greatest distance from a lesion, are most affected.

An X-ray examination made soon after the patient entered the hospital showed an absorption of the contiguous portions of the bodies of the 2d and 3d C. vertebræ and a tilting upwards of the spine of the 2d. Since the last report herein recorded there has been a gradual improvement, the patient having been kept under continuous extension.

Two years ago I reported to the American Neurological Association two cases of stiff neck with great pain, some paresthesia, with no paralysis but with deep jerks suspiciously increased. In these cases I found no objective sensory signs. The present case much resembled those cases when she first came under my observation and I began at once the application of these more recent tests, making a close personal study of it. Dr. Philip Newcomb kindly assisted me by making separate examinations for control and comparison.

I believe that after this kind of an examination we not only know better the conditions present but we can more definitely translate or express them. It is interesting to note the length of time the paralytic or motor signs and the subjective sensory symptoms preceeded the objective sensory signs, and it is a source of satisfaction to reveal this fact in a more precise way. The very gradual falling off of sensory function which occurred in this case is recorded in terms that are readily referred to for purposes of comparison and description.

HEMILINGUAL ATROPHY OF TRAUMATIC ORIGIN.*

By Smith Ely Jelliffe, M.D., Ph.D.,

ATTENDING NEUROLOGIST CITY HOSPITAL, CLINICAL ASSISTANT DEPARTMENT OF NEUROLOGY, VANDERBILT CLINIC.

Instances of hemilingual atrophy are not common, and in these the etiological element of trauma is rare. In Ascoli's[1] able summary now some ten years old, in 79 instances trauma was the cause in some 10 or 12 cases, and injury to the hypoglossal at its exit from the skull due to an injury of the cervical vertebræ was noted in about 4 instances.

The following history is reported as contributing another instance to this form of affection originating in this rare manner. I am indebted to Dr. J. Sherman Wight for the privilege of reporting it at this time.[2]

History of Patient.—A. B. W., 27 years of age (June 1, 1905), a confectioner, telegrapher, nurse, by occupation. His father, aged 62, and his mother, aged 52, were both healthy—both living and in good health. It is asserted that father had syphilis, but when, it is impossible to learn. One brother died of diphtheria; one sister is living and well at the age of 15.

Personal History.—Patient has worked since he was 12 years of age. He has had scarlet fever and measles and denies syphilis. Was a strong, healthy man. Moderate drinker. Not exposed to exogenous poisons.

History of Accident.—May 31, 1904, at 10 A. M., while a passenger on a DeKalb Ave. car on Brooklyn Bridge, he was thrown suddenly and forcibly backwards by reason of a rear collision. He was not rendered unconscious but was able to walk to a neighboring office of a patient who was about to make arrangements to hire him as a nurse. He then went home and went to bed. He called his family physician, Dr. Hoag, who said his spine was injured.

He suffered from severe pains at the end of his spine and also in the back of his head. He was shaky and tremulous. Any movement of his head resulted in severe pain. He remained in bed.

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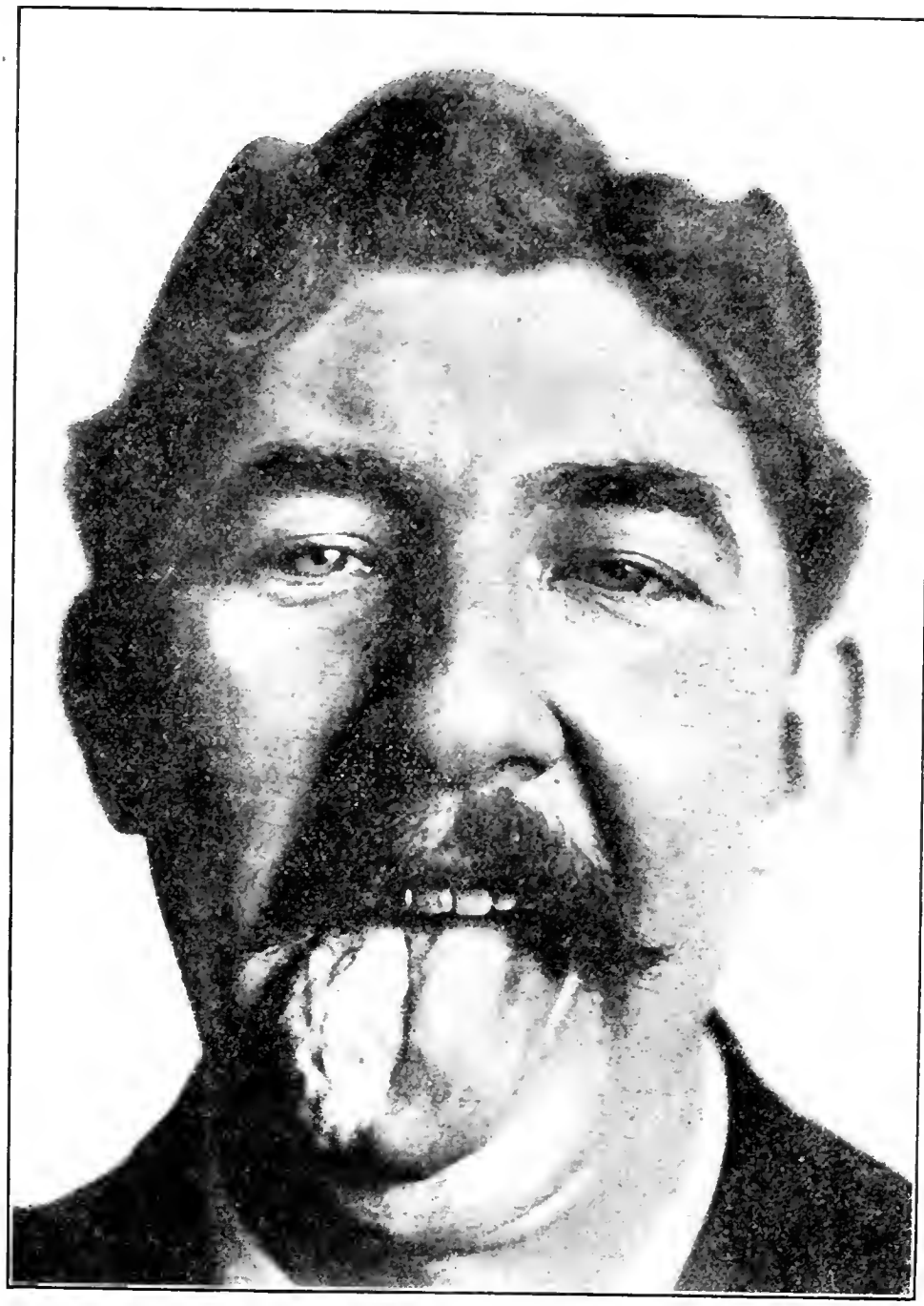
[1] Il Policlinico, 1, 1894, pp. 14, 51, 159.

[2] A fuller report of this and other similar cases by Dr. Wight and myself is in preparation.

*Read by title at the meeting of the American Neurological Association, June 4 and 5, 1906.

On June 9, 1904, nine days after the accident, he was examined by Dr. Sherman Wight in consultation with Dr. Hoag. The patient was in bed. He held his head rigid with



Hemiatrophy of Tongue.

his chin forward in the position typical of cervical vertebral trauma. There was severe pain on the slightest movement and attempts at forced movement were impossible. There were no evidences of severe nerve injury at this time and patient's

spine was strapped with adhesive plaster and a plaster jacket was applied. No positive diagnosis could be made at the time but absence of blood in the ear, or conjunctival ecchymoses or symptoms of involvements of cranial nerves seemed to indicate that there was no fracture of the skull. The idea was entertained that some severe injury to the cervical vertebræ was present.

The patient remained in bed seven weeks and wore the plaster jacket a period of six weeks; after which time it was removed. He stated he was unable to work, but as the accident suit against the railroad had been begun this is not unusual. He continued to complain of pain about the injured parts, held his head forward and stiff and was unable to rotate it as formerly.

From this time the history is not very distinct. The patient says that in about a month or so after the accident he noted that he did not speak as clearly as he had done previously. He thought his speech was becoming thicker, but it did not inconvenience him very much. This became worse, however, and he noted a change in his tongue, that it was unsymmetrical and quivered a great deal.

I saw him first on June 1, 1905, with Dr. Cecil MacCoy of Brooklyn. He was a moderately muscular and well built man. He carried his head forward in a stiff and strained position. Attempts at forcible movement were painful, although moderate, conscious motion was not attended with much discomfort. The only anomaly was found in the tongue. This as shown by the accompanying photograph is atrophied. It was broadened, moist, thrown up with numerous furrows, and there were very marked constant fibrillary contractions sharply limited to the atrophic side. Pinching the tongue showed a loss of muscular substance. The motion imparted by the atrophied muscles was distinctly weaker than the action of the well side.

Thus on protrusion the tongue tip was forced to the atrophied side and a typical though slight bending of the lingual raphe was observed. By strongly pulling the tongue within the mouth a certain amount of bending in the opposite direction could be induced. The movements of the tongue, however, were not strongly interfered with. A slight but distinct slurring of speech could be detected for certain labials.

There were no detectable changes in taste perception. Electrical tests were not satisfactory, as a typical reaction of degeneration was not obtained.

There were no observable changes in the innervation of the palatal muscles and none in the larynx. Slight vasomotor disturbances were noted. The patient usually had a

flushed face and a slight inequality of the pupils (right dilated) was noted at my last examination. It had either been overlooked or was not present at a previous examination. The patient's general attitude was hopeful and his personality was buoyant and inclined to be boastful. His intelligence was of a medium grade only.

So far as we could ascertain no other exciting cause for the atrophy was probable. It was not a congenital affair, since it was definitely not present at the time of Dr. Wight's first examination following the accident. Tabes and syringomyelia were definitely excluded. There were no indications of either trouble. We believe that we can exclude syphilis, both from his own statement as to his non-infection, and also from the fact that as ordinarily observed the lesions incident to syphilitic involvement, whether medullary, or extra-medullary, are in the great majority of cases not so clearly confined to the hypoglossal. Isolated nuclear involvement of syphilitic origin has not yet been reported, so far as I have been able to learn, and a syphilitic exudate either meningeal, or occurring outside of the foramen, usually implicates other nerve structures and thus gives rise to a more complex syndrome.

There is ample justification for the view that a traumatic luxation of the upper cervical vertebræ may give rise to this uncomplicated picture, and the case is presented as one of simple hemilingual atrophy due to injury to the hypoglossal nerve at its exit from the skull. A Roentgen photograph shows a distinctly anomalous shadow in the region of the third cervical vertebra. It is not distinct enough, however, to be offered as conclusive evidence of the injury. In view of the cases of Uhde, Hagemann and Boettger (*Arch. f. kl. Chirurgie* 22, 1878, p. 217, Brasch, *Arch. f. Psychiatrie*, 32 1899, p. 105, Morison *Br. Med. Jl.* 1, 1888, p. 75, Parry, *Lancet* 1, 1900, p. 537, Trevelyan, *Brain* 13, 1900, p. 102), and others quoted in Ascoli's[1] list, the etiological factor is considered highly probable.

Society Proceedings

PHILADELPHIA NEUROLOGICAL SOCIETY.

October 23, 1906.

The President, Dr. D. J. McCarthy, in the Chair.

MOTOR PARALYSIS AS AN EARLY SYMPTOM OF TABES DORSALIS.

By Dr. C. D. Camp.

First Case: Male, 56 years old, history of syphilis thirty years before and alcoholism for past three years. The first symptom was a unilateral paresis of the extensors of the foot. Examination showed loss of the knee jerks and of the Achilles jerks, sensory symptoms, Romberg's sign and myotic pupils with loss of the reaction to light. There was no pain or tenderness over nerve trunks.

Second Case: Clerk, 65 years old, no history of syphilis or alcoholism, but he had been a painter 20 years previous to the onset of his symptoms. First symptoms sharp, shooting pains in the legs followed by gastric and vesical crises, unilateral foot drop of recent development and ataxia. Examination showed Westphal's sign, Romberg's sign, ataxia of all four extremities, sensory changes, and optic atrophy. Dr. Camp said that the motor nerves of the eyes are frequently affected in the beginning of a case of tabes, but that other motor nerves may be affected is not well recognized. The pathogenesis is the same as in other cases of tabes, but the toxin acts more extensively. The affection may be due to degeneration of the anterior horn cells, owing to a default of the habitual excitations upon which the vitality of the cells depend.

Dr. F. X. Dercum said that it is a well known fact that motor involvement may occur in well advanced tabes, but it is not, as Dr. Camp notes, a well known fact that these palsies may occur early in tabes. It is a very common thing to see palsies in connection with the eye muscles in tabes; there is nothing more common than a transient diplopia, nothing more common than a ptosis. Sometimes these palsies are temporary, at other times permanent and persistent. The speaker said that one thought was suggested to him in both of Dr. Camp's cases, namely, that the ordinary etiology of tabes was wanting. Both syphilis and alcohol were present in one case, and the part possibly played by alcohol had to be considered; in the second case there was no history of specific disease. Dr. Dercum also called to mind the palsies met with in primary neurotic atrophy, in which disease we may also have organic changes in the cord, such for instance as degeneration of the posterior columns of the cord. Dr. Dercum, however, agreed with Dr. Camp that the cases shown were true tabes.

Dr. Alfred Gordon stated that a year and a half ago he reported a case in *American Medicine* in which he discussed the pathogenesis of lead intoxication. The patient presented during his life the picture of tabes, but questioning closer he found the patient had been a painter. He examined the peripheral nerves of all the extremities, also the cord. He found posterior sclerosis as well as degeneration of the nerve trunks.

Dr. J. Hendrie Lloyd thought it hardly fair to criticize these cases after only a brief reference to the notes. He was skeptical about the diagnosis of locomotor ataxia in the first case presented by Dr. Camp, and considered it more suggestive of alcoholic neuritis. One point that raised a doubt, however, was the condition of the eyes. Dr. Lloyd stated that his ideas have been considerably enlarged and modified on the subject of the possibility of the ravages of alcohol on the nervous system. He had seen a number of cases in which the classical symptoms were not all present, but a clear and distinct history of alcohol

was obtained. Two years ago he had a young man under his care, an undoubted case of alcoholic multiple neuritis, in which the symptoms were confined entirely to the lower extremities. In such a case it might have been difficult for some observers to differentiate it from a beginning case of locomotor ataxia. The patient made a good recovery.

The second case of Dr. Camp's he thought had a suspicious history of exposure to lead, although he had not been thus exposed for a good many years. One of the most marked cases of pseudo-tabes that Dr. Lloyd had ever seen, was reported from his Blockley clinic ten or twelve years ago. On making a careful microscopic examination he found no trace of degenerative changes in the cord. The man had followed painting for many years, but also had a history of alcoholism. This he thinks is the only matter of doubt about such cases, and raises a very interesting question. Another interesting question, is the possibility of locomotor ataxia beginning as a multiple neuritis. He had seen cases which suggested this possibility. There is nothing inconceivable in it.

In closing Dr. Camp replied that in reference to the case in which there was a history of alcoholism being one of alcoholic neuritis, the patient had only been drinking for the last two or three years, and the symptoms dated back nine years.

As regards the etiology in the second case where there was no history of syphilis nor alcoholism, there was only the one fact of the man having been a painter 20 years before. That is a long time to go back, but he supposed that if we have to theorize, there might be some connection between the two. Lead intoxication has been considered to be one of the causes of tabes.

As to Dr. Spiller's reference to a statement of Cole's, that he (Dr. Spiller) did not believe the posterior columns of the cord are degenerated in every case of multiple neuritis, Cole's exact words are, that he "could find no record of any case of multiple neuritis in which the spinal cord was examined by the Marchi method, in which it was stated that the posterior columns were free from degeneration."

TRAUMA PRECEDING PROBABLE SYRINGOMYELIA AND TABES.

Dr. S. D. Ludlum presented these cases:

Dr. Dercum thought one important point should be insisted on in these cases; that is, the relation of trauma should be clearly defined. He did not think too much stress should be laid on the fact of trauma in either of the cases presented by Dr. Ludlum. The relation of trauma to tabes has been gone over a great many times and none has ever been shown. He said that he understood that Dr. Ludlum disclaimed an etiological relationship with trauma in the cases presented.

Dr. Gordon said that the cases did not impress him as in any way extraordinary. Traumatic syringomyelia, as understood in its broadest sense, that is a hemorrhage or anything which involves in the spinal cord Gowers' tract, is a possibility. Dr. Gordon recalled that he presented before the Neurological Society a few years ago a woman who had a distinct history of trauma and developed distinct syringomyelic symptoms and atrophic disturbances. As far as the tabes is concerned, he thought this a question of some importance. At the last Congress of the French Neurologists and Alienists the question of traumatic paresis came up and observations were brought forward showing that the symptoms of paresis developed immediately after trauma. The general opinion nevertheless was that the trauma was simply an accidental cause. We know nothing of the previous condition of the patient in regard to his knee jerks, his ocular symptoms, so it is difficult to draw even the slightest inference as to trauma as a factor in the causation of tabes.

Dr. Spiller said he did not believe trauma had been the cause in

either of the cases presented, and Dr. Ludlum was of the same opinion. He did not believe that trauma is ever a cause of syringomyelia, unless the trauma directly affects the spinal column and spinal cord. Many writers have tried to show that trauma of the peripheral part of a limb, *i. e.*, the hand, as in the boy presented, may be the cause of syringomyelia. He thought the case of the boy very interesting from the fact that his symptoms developed after the giving of a blow. He was not injured by the blow although immediately after giving it his hand commenced to swell and continued swollen for two years. The boy probably had syringomyelia before the trauma occurred. He presented the Brown-Séquard type of paralysis, *i. e.*, he had disturbance of sensation in the left lower limb and of motion in his right upper and lower limbs.

Dr. A. R. Allen stated that the boy had given him, upon questioning, a history of excessive work for two years prior to this condition, carrying as much as a ton of coal a day in hods up to the third story of a building. Dr. Allen mentioned this point as possibly having something to do with his condition.

A CASE OF ADIPOSIS DOLOROSA.

By Dr. G. E. Price.

The patient was a female; white; widow; aged 54. She complained of severe pain about the knees, rarely spontaneous, but induced by motion, palpation, or by contact of her flesh with any object. She had marked paresthesias (numbness, burning, tingling and crawling sensations), variously distributed. Her flesh would bruise without adequate cause and she manifested extreme fatigue upon slight exertion. The patient was nervous, irritable and anxious. Weight 225 lbs. Symptoms developed 1½ years ago. Previous history: married when 25. Had never been pregnant, catarrhal jaundice when 37, syphilis when 39, sciatica. No history of alcoholism.

Family History.—Father died of consumption. Rest negative.

Examination.—Nodular, lobulated masses of adipose tissue, very painful when palpated, about knees, elbows and back of arms, face, hands and feet unaffected, and trunk but slightly involved.

Thyroid gland not palpable. No muscular atrophy about hands, but had marked deformity of both little fingers and nodular deposits about many of the joints. Large varicose veins were present upon both legs. The skin was dry and the reflexes diminished.

The eyes were negative except for hyperemia of the discs. A few granular casts were found in the urine which was otherwise normal. The patient had shown distinct improvement following 5½ months' treatment with thyroid extract. Attention was called to the common history of antecedent syphilis or alcoholism in adiposis dolorosa.

Dr. Dercum thought the case a typical instance of adiposis dolorosa. The pathology of this disease is an interesting matter. Unfortunately it is one largely of speculation still, although in a number of cases at autopsy changes have been found in the thyroid gland, pituitary body and in the suprarenal capsules. There is probably some disturbance of the internal secretions. It is not improbable that changes of the thyroid secretion leads the way, and that the disturbances of the other glands are probably secondary in character. However, whatever the original cause is, cases are benefited by the use of the thyroid extract, though Dr. Dercum thought it going too far to say they are cured. He has seen several cases greatly and persistently relieved.

Dr. D. J. McCarthy called attention to the fact that the case of adiposis dolorosa reported by Dr. Dercum and himself revealed later a very marked hypertrophy of the suprarenal capsules. He also alluded to an interesting case recently brought to post-mortem at the Philadelphia General Hospital. A German with multiple adipose tumors scattered mainly over the upper extremities, although there were a few over the lower, shortly before death had areas of painful swollen fat in the legs with what appeared to be forming tumors. This was of

interest because the other tumors had not been painful. It may have been that the previous tumors developed many years before as areas of painful fat.

Another matter Dr. McCarthy mentioned was in connection with syphilis where widespread changes in the lymphatic system occur as an etiological factor in adiposis dolorosa. In the case studied by Dr. Dercum and himself there were extensive hemolymph tissues, not only scattered throughout the body, but in the adipose tumors, and he thought perhaps it represented an attempt by nature at compensation for disturbance of lymph tissue elsewhere in the body.

A CASE OF PROBABLE TUMOR OF THE BRAIN OF PARIETO-OCCIPITAL LOCATION.

By Dr. J. W. McConnell.

H., aged 53 years, a plasterer, was admitted to the Philadelphia General Hospital, complaining of loss of power in the left side and staggering.

He gave a history of an attack of unconsciousness occurring seven years ago, preceded by dizziness and an indescribable illness, accompanied by twitching of the left face and followed by transient loss of power in the left upper and lower extremities and persistent numbness of the left hand and forearm. At irregular intervals subsequently he had twitching of the left face, up to one year ago when he had a violent convulsive attack involving the left half of his body without loss of consciousness and without modification of the symptoms residual from the previous attack.

His family history was negative. His personal history contained alcohol, tobacco and probably syphilis. He stated that he suffered for a long time from headache, recently growing worse, especially after the convulsive attacks, from dizziness and occasional nausea without adequate gastric cause.

Examination shows a hemilateral ataxic gait, better brought out by sudden turning. He sways some with eyes open and considerably with eyes closed.

Mentality is excellent, memory good, both for past and recent events. He has not aphasia, word, letter or number blindness, word deafness or agraphia.

There is no difference in the pupils which respond to light, convergence and accommodation. Extraocular muscles are normal. There is no nystagmus, no loss of any associated movement. Examination of vision shows left lateral homonymous hemianopsia without pupillary inaction, with contracted fields, without optic neuritis or atrophy. The motor fifth, the seventh, in fact all the cranial nerves seem to be normal.

The movements of the left upper extremity are weaker than the right, but are of good power, they are slow and ataxic. The reflexes are increased. The movements of the left lower extremity are similar to the upper. The reflexes are increased. Ankle clonus is not obtained. Plantar stimulation gives no response.

The right upper and lower extremities are normal in all respects. Plantar stimulation of the right foot gives plantar flexion.

A convulsive attack observed by the resident physician had for its features a preceding numbness of the left arm. The left hand, arm, face and leg in sequence were tonically convulsed and the patient asked to be laid down. He became unconscious, and the convulsion drew the head upward and to the left, the face was drawn to the left, eyes up and to the left, the body in left pleurosthotonus. Later the convulsion became clonic and general.

Sensation: On the right side is normal to all forms of stimulation. On the left side tactile sense is diminished over the arm and leg, it being almost lost on the hand and forearm. Hypalgesia is found in the same areas. Temperature sense is not disturbed. There is marked ataxia in all movements of the left upper and lower extremities. Pas-

sive movement is not always recognized, especially is this true of the fingers. There is loss of stereognostic conception in the left hand.

The general features of the case are those of a growth, probably of specific origin, located in the parieto-occipital region of the right brain. His previous improvement under treatment and his present betterment under mercury and iodides both suggest the syphilitic nature of the lesion.

ASTEREOGNOSIS WITHOUT MOTOR OR SENSORY INVOLVEMENT.

By Dr. T. H. Weisenburg.

The patient said he was under the care of Dr. R. S. Dorsett, of Philadelphia, with whom he was seen in consultation by Dr. Weisenburg, and subsequently by Dr. Mills.

The man was thirty-two years old, with no history either of alcoholism or syphilis, was perfectly well until three weeks before coming under observation, when he awoke during the night with a pain in the left side of the back of the head and in the same side of the neck, this pain disappearing the following morning. Two days after this, he began to complain of a numb, dead feeling in his left arm, followed in a day by similar sensations over the left chest and abdomen and the left leg. These sensations have persisted. About two weeks after the onset of these sensations he noticed that the grip in his left hand was not as good as before when his attention and eyes were directed elsewhere than to the object grasped. He has never had headache, nausea, vomiting or any disturbance in his eyes.

When examined his eyes and cranial nerves were found to be in a normal condition. The grip of the left hand, when his attention was called away, was not as good as when he was looking directly at his hand, in which case it was normal. The left leg, like the face and arm showed no weakness. The reflexes were somewhat prompt, especially on the left side, but no Babinski was present. Sensation for touch, pain and temperature, and tone sensation were normal over the left side and everywhere. The senses of position and movement were lost or greatly impaired in the fingers of the left hand, the loss becoming less as the thumb and forefinger were approached. To a less extent the sense of location was disturbed, more so, as the radial side of the hand was approached. The sense of pressure was normal. He could not recognize any object placed in or manipulated by his left hand, the astereognosis being absolute. The hardness or softness of an object, or its surface contour could, however, be recognized, but only by the tips of his forefinger and thumb, especially the latter.

The patient was placed upon daily mercurial inunctions with increasing doses of iodides. He seemed to improve almost immediately and in the course of six weeks all of the symptoms above detailed disappeared. The numbness of which the patient first complained disappeared first, and as the patient improved he was first able to recognize objects placed between the thumb and forefinger, and later on in the other fingers. At the present time, several months after the patient was put upon treatment, no neurological symptoms of any sort can be found.

Dr. Mills said that with regard to the case presented by Dr. Weisenburg, which came under his observation, this man was carefully studied by him. He had taken him into the lecture room and demonstrated the facts spoken of by Dr. Weisenburg, namely, the presence of astereognosis and the absence of motor paralysis and of all sensory symptoms. He said that he had studied the patient on several occasions and the condition remains the same, with the exception that the astereognosis gradually receded until he now is practically normal. He thought the case important from the standpoint which Dr. Weisenburg referred to, that which he had personally held and taught, namely, that there were separate cortical centers for movements for cutaneous and muscular sensibility and for stereognostic conception.

With regard to the first case, that presented by Dr. McConnell, Dr. Mills stated that he had also lectured on this man at his clinic at the Philadelphia General Hospital, having previously studied him in the nervous wards. The man's symptoms when first seen by him were much as they are now, with one possible slight exception. He believed that the patient at first had some slight retention of tactile sense. He now has loss of tactile and pain sense, not equally in the entire extremity, but with receding intensity as you pass from the distal to the proximal portion of the limbs. The reverse, as is well known is frequently seen in hysterical cases. He has no motor paralysis. (This Dr. Mills showed in examining him before the Society). If you eliminate the awkwardness which results from impairment of sensation, the muscular sense and stereognosis there is slight if any true motor weakness. The absence of motor paralysis is interesting in connection with the fact that he has had typical Jacksonian spasms. He has lateral homonymous hemianopsia. Dr. Mills thought it might be a case of tumor of the parieto-temporo-occipital region. It might, however, be a case of arterio-sclerosis with gradual necrosis of brain tissue. He did not believe that the case could be explained as one of hysteria, possibly the patient had some hysterical epiphenomena. Hemianopsia in Dr. Mills experience is extremely rare, if it ever occurred, in hysteria.

Dr. F. X. Dercum asked whether the sense of position of the fingers had been tested. Astereognosis is made up of a great number of factors—not only cutaneous impressions, but also muscle sense impressions and impressions received from the joints. As Dr. Mills says, there may be entire loss of the tactile sense, and notwithstanding preservation of the stereognostic sense.

Dr. C. W. Burr thought it very difficult to determine in Dr. McConnell's case whether the inability to distinguish objects by handling them as due to astereognosis or anesthesia. Though a man may be able to recognize objects in the presence of slight tactile anesthesia, yet if there be complete anesthesia to touch and deep pressure, he would be unable to tell what he had in his hand. Dr. Burr also thought that a distinction should be made between the inability to recognize objects because of the loss of sensibility, whether it be of space sense, the sense of the position of the hand itself or any other sensory disturbance, and inability on account of loss of memory of tactile sensations. This last condition, tactile amnesia, is comparable to word-deafness and mind-blindness.

Dr. Mills thought with regard to the relations between stereognostic conception, sensation and movement, he believed that the first was an independent function, although there is a sensory pseudo-astereognosis and a motor pseudo-astereognosis. It is possible for a patient to have entire loss of cutaneous sensibility (for touch, pain and temperature), and yet retain stereognostic power; in other words, to retain the ability to recognize objects by manipulation. This had been demonstrated by himself and others in a well known case at the Philadelphia General Hospital.

Dr. McConnell, in closing, said that he had brought his patient before the society more particularly for diagnosis. Upon the question as to whether or not the case was originally thrombosis or arterio-sclerosis, these were matters on which he wanted the opinion of the society. The man had an attack seven years ago, and he states that he had loss of power following that attack. Whether what he took for loss of power was ataxia or peculiar sensory disturbances, which he now has, is the question which comes to Dr. McConnell's mind. The fact that he has improved must be given due weight. He says he was under treatment for a long time previous to coming into the hospital. Perhaps the treatment was the same as he is now obtaining. Since his last Jacksonian attack he has distinctly improved. He is still on mercury and iodides.

Dr. Weisenburg thought all agreed that a man may have astereognosis without sensory or motor involvement. In regard to Dr. Der-

cum's query as to sense of position: the patient had presented involvement of sense of position, but less of the sense of localization. The question arises whether one can have a case of pure astereognosis without involvement of the senses of position, pressure, movement, and localization. The fact that in this case there was involvement of all of these senses seems to show that in astereognosis there is involvement of the senses of position, movement, pressure and less so of localization.

FACIAL DIPLEGIA ASSOCIATED WITH LABIO-GLOSSO-LARYNGEAL PARALYSIS.

By Dr. Alfred Gordon.

The patient was exhibited, and Dr. Gordon stated that she had bulbar palsy and presented some unusual features. In addition to the involvement of the muscles of the power part of the face there was complete paralysis of the muscles of the upper part and of the forehead with RD in the latter. The orbicularis palpebrarum, also the external recti muscles were equally affected. This points to a probable association of the nucleus of the upper facial nerve (which as is well known is separate from the nucleus of the lower) with that of the 6th. Another interesting feature of the case is the total motor aphasia, which is quite unusual for the classical bulbar palsy where dysarthria or anarthria are only present. Finally, the patient presents a total suppression of salivary functions: her mouth is unusually dry. The most important point about the case is the involvement of both superior facial nerves, as such an occurrence in association with the typical bulbar palsy has been reported only in family bulbar palsy of children.

Dr. Spiller said he had seen several cases of palsy following diphtheria. He had never seen a case of bulbar palsy occurring in an adult with paralysis of the upper part of the face. Cases of muscular dystrophy involving the muscles innervated from the medulla oblongata and pons and cases of multiple cranial neuritis causing bulbar symptoms have been observed. This patient presented by Dr. Gordon has the ability to lower her eyelids when she looks downward, but she cannot close her lids when she tries to do so voluntarily.

Dr. Spiller thought the hypesthesia of the face and reaction of degeneration a strong evidence of multiple neuritis. Another point of importance was in regard to vision. The woman talked like a person with bulbar palsy, and not like one with aphasia. He regarded the case as one of multiple neuritis of cranial nerves.

Dr. McCarthy stated that two months ago he saw a case five weeks after an attack of diphtheria. The symptom group of cranial nerve involvement was rather irregular, the first symptoms were those following usually an attack of diphtheria with paralysis of the soft palate, with regurgitation of fluids through the nose, and followed after several weeks by a paralysis of the 7th on one side and later both sixth nerves, and later by involvement of the third nerve, and then paralysis of the ninth and tenth with disturbance of the diaphragm and very shallow respirations. The patient, however, finally recovered, but it seemed to Dr. McCarthy that there was some latent or post-intoxication indirectly connected with the previous attack of diphtheria. He thinks the case Dr. Gordon presented would correspond in a way to the case just narrated—some time after an attack of diphtheria or throat infection there has been involvement of the cranial nerves, multiple and more or less bilateral.

Dr. Gordon, in closing, stated that the "yes" that she says is very indistinct and the "no" is absolutely indistinct. As to the lesion in this case, he does not think it is of a cortical nature. He believes it is a case of polioencephalitis; the nuclei involved are those of 6th, 7th, 9th, 10th and 12th nerves.

THE SECOND ANATOMICAL PROOF OF THE VALUE OF THE PARADOXICAL REFLEX.

By Dr. Alfred Gordon.

He reviewed Dr. Dercum's communication made at the February meeting concerning a patient with a hemorrhagic pachymeningitis who, during life, presented the paradoxical reflex on the side opposite the lesion. Dr. Gordon reported another case in which the paradoxical sign existed on one side without Babinski's or Oppenheim's sign. The operation was based exclusively upon the existence of this reflex and when careful decompression was done, the reflex totally disappeared. The patient recovered completely. This was verified by Drs. Mills and Dercum at the Jefferson Hospital where the patient was placed. Incidentally Dr. Gordon mentioned another case of epilepsy which is now under his care at the same hospital. Upon admission the patient presented no abnormal reflexes. While in the hospital he developed convulsions. Immediately after the knee jerk became increased and a distinct paradoxical reflex appeared on both sides, Babinski's sign was slight on one side, but there was no Oppenheim's sign. Six days after the seizure all abnormal reflexes disappeared completely. Dr. Gordon draws the conclusion that his reflex is a sign, to say the least, of cerebral irritation (motor area) or of a beginning lesion of the motor pathway, while Babinski's is a sign of a well established lesion of the same tract.

Dr. Mills said he had seen the first case Dr. Gordon referred to, and Dr. Gordon demonstrated before him the condition as stated in his paper.

Dr. Mills further stated that it might be interesting to Dr. Gordon to know that a few days ago in his office he had a case in which this paradoxical reflex was present on one side when neither the Babinski nor the Oppenheim phenomenon could be elicited. Curiously enough this was a case of multiple neuritis of acute but not very severe type, the woman was still able to walk and got to Dr. Mills' office with a member of her family from somewhere out of town. She had not lost her knee jerks. Dr. Mills expressed his pleasure in being able to testify to the two cases. He has in many cases examined patients or had them examined by his assistants in his presence for this paradoxical reflex, at the same time that the Oppenheim and Babinski reflex were tested for. He had never seen the paradoxical reflex demonstrable when the Oppenheim and Babinski reflexes were absent excepting in these two cases. The great value of a sign of this kind is shown when you can elicit it in the absence of other signs. If it can be elicited in the absence of other signs it has certainly some value.

Dr. Dercum stated that he has seen Dr. Gordon's sign a number of times independently. He demonstrated it in his clinic only a week ago, in which it was the only symptom present, the Oppenheim and Babinski both being absent, a case of mild hemiplegia, with slight exaggeration of the knee jerk on the paralyzed side. Deep pressure upon the gastrocnemius and soleus near the origin of their tendon gave extension of the toe as a marked clean-cut reaction.

In regard to the first case Dr. Gordon spoke of, the man in whom it occurred was an assistant in the clinic at Jefferson. Dr. Dercum had studied him very carefully. He had a Gordon sign and no other sign excepting plus knee jerk. The case was one of hemorrhagic pachymeningitis. Dr. Keen operated upon the opposite side of the head, and as soon as the skull was opened there was a tremendous gush of bloody and serous fluid; immediately afterward the Gordon reflex disappeared. A few days later the man became restless, and Dr. Dercum again tested him for the Gordon reflex; it was again present. The wound was reopened, retained discharges allowed to escape, and again the Gordon reflex disappeared. To Dr. Dercum this was a clear demon-

stration of the value of this reflex. He regards this reflex as a distinct addition to our clinical knowledge. It will certainly often enable us, in the absence of other signs, to determine the proper side in the case of operation.

Dr. McCarthy stated that when Dr. Gordon brought his reflex to the attention of the society some months since, he thought it was identical with, or a modification of, the Oppenheim and the Babinski signs. Since that time he has made extensive trials for it, and found it in a case diagnosed as a prefrontal tumor transferred from the insane wards to his service at the Philadelphia General Hospital. In this case it was the only sign apart from some mental phenomena. He has found the Babinski reflex present in several cases in which the Gordon reflex was not present. If the condition is to be considered as a modification of Oppenheim's reflex he is sorry to hear Dr. Mills give this case of multiple neuritis as an example of the reflex. If there is anything in the case, it lessens the value of Dr. Gordon's reflex as a symptom of disease or disturbance of the central motor tract. At the same time the case quoted by Dr. Dercum seemed to show that the reflex is certainly an addition to methods of clinical diagnosis. From all the investigations he has carried on, Dr. McCarthy is still confused as to its exact value. The cases in which it occurs in which the Babinski is also present, the cases in which it does not occur and the Babinski occurs, these cases have not come to autopsy—or if they have, Dr. McCarthy has not followed them; that is the difficulty with the work at Blockley where the next service comes along, and in the absence of the chief making the original observation, the cases are not followed up. He thinks if a larger number were followed to autopsy they would prove of much value in establishing this sign as a means of clinical diagnosis.

Dr. Gordon, in closing, said concerning Dr. Mills' report he wished to relate the following fact: A patient came to Jefferson Hospital with a supposed sciatica on one side, he was examined as usual very carefully and Dr. Gordon found a distinct paradoxical reflex on the diseased side, with an exaggerated knee jerk. A month later the patient developed weakness on the opposite side, difficulty in micturition, and finally the case turned out to be one of myelitis with difficulty in walking and paraplegic symptoms.

In regard to Dr. McCarthy's remarks, it is true that we do not have many autopsies; but what about Dr. Dercum's case reported in the September issue of this journal, also what about Dr. Gordon's case verified by Dr. Mills and Dr. Dercum, where we had not only before the operation the reflex demonstrated in a clean-cut manner, but also disappearance of it after the patient recovered from the immediate effect? As to the reflex being a modification of the other two reflexes, Dr. Gordon said he did not know. We cannot give the proper explanation for any of these reflexes, but simply strong inferences.

Besides these two anatomical proofs, Dr. Gordon is in possession of a number of clinical facts showing the value of this sign. As to its exact significance he states that he can only repeat what he has stated in his first clinical contribution, viz., the paradoxical reflex is a delicate sign of an early stage of a lesion or only of irritation of the motor tract. The latter particularly is seen from the case examined by Dr. Mills and Dr. Dercum; Babinski's sign shows a definitely established lesion of the motor system. Dr. Gordon stated that he has examined two-hundred and fifty normal cases, and has never found the paradoxical reflex present. It was always in conjunction with some of the classical symptoms pointing to the involvement of the motor tract. The demonstration of the reflex depends a great deal upon the method. If the rules laid down by Dr. Gordon in his original contribution are adhered to, he believes the reflex will be demonstrated in a larger number of cases.

Periscope

Journal de Psychologie Normale et Pathologique

(Third Year, No. 4. July-August, 1906.)

1. Disorders of Voluntary Pantomime Among the Insane. DROMARD.
2. Note Upon the Nature of the Subconscious and Unconscious Elements. G. L. DUPRAT.
3. Attempted Suicide as a Result of Suggestion. A. LEMAITRE (of Geneva).

1. *Disorders of Voluntary Pantomime Among the Insane.*—This is the first of the essays promised by the author in his earlier article (JOURNAL NERVOUS AND MENTAL DISEASE, October, 1906, p. 675), upon the classification of the pantomimic manifestations of the insane. Herein he studies the first of the two grand divisions into which he divides these manifestations; namely, those of the *voluntary* or active expression (ideative pantomimia in its relationship to the intellectual life). Theoretically, these disturbances are the result of a rupture in the associations which, in the normal state, connect thought with its appropriate motor exhibition. There are three general types of these particular, abnormal manifestations; namely, (*a*) those that result from a bad or vicious adaptation of the gesture to the thought, (*b*) those that are the product of a conventional adaptation, and (*c*) those that reveal a total absence of adaptation.

(*a*) The *vicious adaptation* of gesticulation to the thought is shown in *paramimic asemia* and the various forms of *mannerism*. In *asemia* the patients are incapable of portraying an idea by a corresponding motor symbol; hence the disorder is sometimes called *asymbolia*. The patients, for example, open the mouth when told to close their eyes; or they close their eyes when told to put out their tongues. The trouble is observed in cases of senile dementia, general paralysis, and many diseases with circumscribed or disseminated lesions of the brain. A kind of inverse manifestation is seen sometimes in the exaggeration or multiplication of the gesture, made to supply a deficiency of speech. This might be called a *hypermimia of supply*. It is noticeable, especially in hysterical mutism. This hypermimia of supply is to be contra-distinguished from paramimic asemia by reason of the fact that in the former there is an ideo-pantomimic hyperfunction complementary to the ideo-verbal, associational deficiency, whereas in the latter there is an insufficiency of the ideo-pantomimic associations.

Mannerism is a form of paramimic manifestation which is characterized chiefly by outlandishness and artificiality. It is outlandish because it is not in consonance with the triviality, simplicity, and poverty of the thought which it accompanies. At times it is inco-ordinate and void of all uniformity, whereas at other times it assumes a most systematized and predetermined character. It thus takes on at all times the appearance of vulgar affectation, simpering childishness, or theatricality. Mannerism is common among hysterics. The affectation so noticeable in the speech of these patients is equally noticeable in their attitudes and gestures. A mannerism of attitude is also observable among weak-minded people generally. It is particularly so among the precocious dements. In the latter the mannerisms are monotonous, whereas in hysteria they are characterized by a high degree of richness and mobility. In a large numbe of cases the mannerisms represent merely a survival of the motor element in the psychic complexus handed down to the individual by his ancestors. It is then a kind of atavistic stereomimia. *Puerilism* is a form of mannerism, and usually represents, in most of the cases, an infantile or atavistic regression. Pitres has indicated this symptom under

the name of ecmnesic delirium. The case of Garnier and Dupré, detailed by the author, affords a most striking and interesting illustration; so also do a couple of fully reported cases of his own.

(*b*) In the disorders manifesting a *conventional adaptation* and *pantomimic neologism*, the adaptation of the gesture and facial expression to the underlying thought is not, properly speaking, bad or vicious, but is merely conventional, without any value except to the patient himself. The ideo-motor association is, in a way, arbitrary and the significance of the pantomimic expression is incomprehensible to the spectator who has not been foretold of it. Degenerates, subject to obsessions, often attach special importance to certain gestures and movements. They may regard them as favorable or unfavorable, protective or antagonistic. Thus one patient felt as if he were falling into a pit every time he closed his eyes; while another, a victim of tic, always performed some particular movements in order to prevent some worse or more annoying gesticulations.

Pantomimic neologism is analogous to verbal neologism and hieroglyphic writing. Cabalistic signs are often made by the persecutional insane that resemble veritable formulae of exorcism, incantation, and conjuration. For example, one patient made circles in the air with one hand while he kept striking his abdomen with the other in order to rid himself of the spirit of his brother who he thought was seeking to dwell in his body. Among the pantomimic neologisms may be classed also certain gestures which correspond to rudimentary representations in a backward brain. Such exhibitions are not rare among idiots and agenesics generally. They indicate, as a rule, an atavistic regression. The patient claps his hands, sucks his lips, etc., which in the normal state indicate the sense of pleasure, hunger, etc., but which in this condition are performed merely in a senseless, conventional manner.

(*c*) When the *adaptation of the gesture and facial expression to the underlying thought are wholly at fault*, the higher mind has lost all control over the motor activities and the latter exhibit themselves automatically, without reason or purpose. In normal individuals there is an automatism of gesture, as, for instance, the extension of the hand unconsciously when bidding a friend good-bye. In abnormal individuals this automatism shows itself more particularly in *stereomimia* and *echomimia*.

Stereomimia is only a mode of stereotypy. In it the festures and play of facial expression are repeated to the point of satiety, always in the same manner, and without any real purpose in view. The manifestation is pre-eminently characterized by its fixity and uselessness. The movements are incongruous and are absolutely not in the least adapted to the conditions actually present. They are intensely monotonous. In another essay the author has studied this form of pantomime in detail (JOURNAL OF NERVOUS AND MENTAL DISEASE, January, 1906, p. 56). In a large number of cases, especially in the different varieties of dementia, and above all, in insane dementia, this stereomimia is a form of *secondary automatism*. The movements originally accompanied an idea, which idea has gradually disappeared, leaving the movements as an acquired habit. Inexplicable at present, the movements are perfectly comprehensible in the light of the past when they had their beginning. This secondary stereotypy of the gesture and facial expressions indicates an intellectual enfeeblement, and up to a certain point measures the degree of that enfeeblement.

In many other cases these stereotyped movements indicate a *primary automatism*. Such are the stereomimic manifestations of the catatonics. The preservation of fixed attitudes and the incessant repetition of the same monotonous movements are but exaggerated manifestations and ultimate witnesses of that mental state, of which the milder phases are seen in the simple hesitancy of the psychomotor processes.

Stereotyped pantomimia is observed not only in cases of mental en-

feeblement, but also in cases of simple suspension of inhibition provoked by a morbid process affecting directly but temporarily the psychomotor apparatus. Here the phenomena represent an active process and not merely a residental function. It is usually accompanied by catatonic traits such as sluggishness, waxy flexibility and negativism; but it in no way indicates what will be the outcome of the disease process causing it.

To agencies, such as imbeciles and idiots, also belong this primitive automatism as seen in stereomimia. Their stereomimia should be carefully distinguished from that of the catatonics and dements on the ground of its psychological mechanism. It is characterized by an imitative or atavistic formalism, which is very often rhythmical in expression and contains an emotional element based upon a hunger for activity with satisfaction in its accomplishment. It ought not to be regarded as a mere crystallized relique, so to speak, of a rich motor past, but rather as the immediate manifestation of a congenitally miserable activity.

Echomimia consists of the impulsive imitation of the facial expressions, and gestures of another; an imitation which occurs immediately, brusquely, and with the promptness of a reflex function, without the slightest possible inhibitory intervention on the part of the will. It is a particular form of a much more general phenomenon; namely, echokinesia. The author has studied this symptom in an earlier article (JOURNAL OF NERVOUS AND MENTAL DISEASE, August, 1906, p. 546). Echomimia is devoid of any emotional element, is involuntary and unconscious. It is the result of a failure of inhibition. Like stereomimia, the echomimia of the demented indicates an integral disintegration of the personality. It is not uncommon in congenital weaklings, and it is pronounced in idiots. In the latter it is not so perfectly autonomous and independent of the entire aggregation of the personality as it is in the demented, but it is rather the direct manifestation of a wretched personality which thus exhibits its activity as well as it can.

Incontinence of the lower brain centers, these centers being set free by the dethronement or separation or absence of the higher directing faculties, is in all cases of disorder of the voluntary pantomime the general substratum of the phenomena. By reason of the etiology, as herein explained by the author, these psychomotor manifestations are seen to accompany various psychic troubles which spring from the same general soil, and which for the most part depend upon automatism.

2. *The Nature of the Subconscious and Unconscious Elements.*—Claude Bernard, Durand (de Gros), Grasset, Janet and others attribute to the subconscious or "inferior psychism" the same faculties or modes of psychic action that are recognized as belonging to the conscious or "superior psychism." As Grasset puts it, the inferior psychism is endowed with the power of memory and of volition; is capable of perceiving, judging and reasoning; and is to all intents and purposes to be held as responsible.

Duprat thinks that certain psycho-physiological facts seem to show that beneath the intellectual and representative psychic syntheses, involving such objects as the ego and the external world (superior psychism), there are certain unconscious psychic syntheses or elements which are only the *substitutes* of the neuro-muscular phenomena of all sorts constituting essentially the sensations, the emotions and various tendencies. The acts of the inferior psychism are not intellectual operations; they do not even involve or comprehend the images of objects; they concern themselves only with the "images" of the affective or sensorial states and states of motricity. Between them and the phenomena of the clear, personal consciousness take place the acts of the superior psychism, of which some are not assigned by the individual to his own ego, but are perceived by him merely while others are wholly subconscious. Hence Duprat recognizes (1) certain unconscious psychic acts which are very close to mere physiological states; and (2) certain acts that are (a) subconscious, (b) con-

scious, and (c) of the personal consciousness which alone is capable of comprehending or including in genuinety intellectual processes the perception of objects, ideas, reasoning, and the formation of judgments.

3. *Attempted Suicide as a Result of Suggestion.*—Lemaitre reports the interesting case of a young man having a weak, hysteroid temperament who was so dominated by his intellectually stronger and more vicious companion that upon two occasions he deliberately attempted self-destruction for no other reason than that of the direct suggestion of the latter. The case is not unique, and the moral of it is obvious. The power of suggestion employed by a criminal to secure the commission of a crime by his dupe is and always will be a question of much medicolegal importance.

(Third Year, No. 5. September-October, 1906.)

1. The Personality. W. Bechterew (St. Petersburg).
2. Attenuated Responsibility. J. Grasset (Montpellier).

1. *The Personality.*—This is not so much of a psychological or even psycho-physiological study as it is a diffuse sociological and political critique of the individual and communal personality observed in Russia under present conditions, and of the adverse elements in the government of that unfortunate country that are thwarting the growth of the nation and its constituents.

2. *Attenuated Responsibility.*—Grasset, like all advanced students of normal and abnormal psychology, recognizes that there are varying degrees of responsibility in human affairs depending upon many conditions that involve individual mental capacity. Men are not, as the law too often assumes, either responsible or irresponsible. There is such a thing as an attenuated responsibility, which, however, must be distinguished from mere partial responsibility. Hysterics and epileptics furnish good examples of attenuated responsibility. In court the medical expert is often taken to task by the legal fraternity for asserting this diminished responsibility exists. Many of the highest legal authorities hold that it is both irrational and harmful to society to regard a man as anything but responsible or irresponsible. Grasset has gathered together in this long and interesting medico-legal paper some of these adverse judiciary opinions, and in a clear, well-knit and forcible argument shows that the medical and legal writers are concerning themselves with two separate and very distinct questions; namely, the condition of the patient mentally (medical question) and the best interests of society (legal question). The practical conclusions of the article are that in court the expert is justified most emphatically, legal opinion to the contrary notwithstanding, in maintaining that there is such a thing as attenuated responsibility and that this sort of responsibility should be taken into full consideration in the awarding of punishment.

For the weakly responsible criminals, Grasset argues that corrective infirmaries should be established, which infirmaries should not partake wholly of the nature of prisons on the one hand nor hospitals upon the other, but of the nature of sharp, training schools wherein the mind diseased may be helped medically while the individual is made to feel at the same time that he is undergoing punishment. In some countries such institutions have already been established by law, but in France, Grasset says, there is still a crying need for them. The improvement of the criminal rather than his mere punishment is what society needs; hence such institutions as above indicated will be most apt to foster harmony between the medical and legal authorities anent this great question of responsibility, and in the end accomplish the highest aims of both medicine and law.

Mettler (Chicago).

Allgemeine Zeitschrift für Psychiatrie

(Vol. 63, 1906. Heft. 2.)

1. The Weight of the Cerebellum in Normal and Pathological Conditions. M. Reichardt
2. A Case of General Paresis with Accumulated Convulsions, with Some Observations on the Blood Pressure. W. Plaskuda.
3. Contributions to Clinical Knowledge of the Puerperal Psychoses. G. Herzer.
4. Etiology and Symptomatology of Katatonia. Pfister.
5. Psychical Condition of Insane When Dying. W. Albrand.
6. Commitment on Account of Partial Insanity. H. Kornfeld.

1. *Weight of the Cerebellum in Normal and Pathological Conditions.*—Reichardt gives a very complete account of his studies on the weight of the cerebellum as found in 122 patients. From the weight of the cerebellum alone, it cannot usually be stated whether it is atrophied or not. What is wanted is the proportion which this weight bears to that of the cerebrum, also the capacity of the skull. The highest and lowest cerebellar weights found in the author's cases were respectively 185 and 80 grammes. They usually ran from 110 to 150 grammes, averaging 130 grammes in normal cases. The skull capacity was measured in most cases, making use of the author's method of determining the quantity of water which the cranial cavity would contain (already described in this journal). A brain was considered normal, whose weight in grammes was from 10 to 16 per cent. less than the capacity in cubic centimetres of the skull. The brain was first weighed as a whole, then stripped of pia, the ventricles opened and allowed to drain, and weighed again. The crura cerebri were next cut through, the cerebellum separated, by cutting its peduncles close to their entrance into it, the cerebral hemispheres separated, and each piece weighed singly. The quotient obtained by dividing the weight of the cerebrum by that of the cerebellum, was found to be normally from 7 to 8.5. Figures below 6.5 or above 9 should be regarded with suspicion, as probably indicating a pathological condition. There is no absolute relation between brain weight and size of the body except that in general, large people tend to have larger brains than small people. Neither does brain weight as a rule diminish with emaciation of the body. The 122 cases are arranged in twelve tables, in which the most important facts with regard to each case are given. These tables are classified according to the cerebro-cerebellar quotient normal or abnormal, as related to skull capacity, and as found in functional psychoses, in general paresis and senile dementia with and without brain atrophy, in microcephaly, in brain tumor, in cerebellar atrophy, and in congenital smallness of the cerebellum. As indicating that the cerebellum has to do with coördination of movements, it has been found that at birth, the cerebro-cerebellar quotient is considerably higher than in the adult, but since the cerebellum grows faster than the cerebrum in the early months of life, at the end of the first year, the quotient does not differ from that found in the adult. Also the author found in a 25-year-old cretin, who could neither stand nor walk, a quotient of 9.5. In the lower animals, those which immediately after birth can run about readily, have at that time heavier cerebella than those which are awkward and slow in their movements for some time, the difference being later equalized. In old age, low cerebellar weight is not seldom found, and the author thinks that this may account for such symptoms as tremor and ataxia often observed in old people. As an appendix, he discusses the relation between the cerebellum and sexual instinct, asserted by Gall, and to some extent supported by Moebius, though pretty effectually demolished by Rieger. It happened that among his cases there were two males, each of whom had lost a testicle, and one female aged 29 years, in whom the sexual organs had not developed beyond the infantile stage. In none of these cases was there any change in the cerebellum,

whose weight and general appearance in each instance was normal. There was no evidence of atrophy of the opposite half of the cerebellum in the cases of lost testicle, such as according to Gall should occur.

2. *General Paresis, Accumulated Convulsions and Blood Pressure.*—An account of a case of general paresis in which 568 convulsive attacks occurred in twenty-two days, there being 100 attacks in one day alone. Death at the end of this period, of exhaustion. Measurements of the blood pressure by the Riva-Rocci sphygmomanometer during one series of attacks showed a state of hypertension rising always during the convulsion, though of course during this stage no accurate reading could be made. The author exhibits his measurements in tabular form.

3. *Puerperal Psychoses.*—The author among 1896 patients admitted to the Basel psychiatric clinic during the last twenty-five years found 221 cases of puerperal, or as he prefers to call them, "generation" psychoses. He agrees with other authors that there is no special generation psychosis, but he divides his cases—after Kraepelin's classification—as follows: Dementia præcox, 107 cases; manic-depressive insanity, 32 cases; hysteria, 18 cases; acute confusion (amentia), 15 cases; alcoholic insanity, 12 cases; epilepsy, 11 cases; neurasthenia, 5 cases; general paresis, 4 cases; eclampsia, 2 cases; chorea imbecility, 2 cases; diagnosis uncertain, 13 cases. According to time of appearance, the generation psychosis may be divided into : (1) Those of pregnancy. (2) True puerperal cases (those coming on at any time within the first two months after delivery), and the lactation psychoses. Of the 221 cases, 46 belonged to class 1, 102 to class 2, 69 to class 3, while in the remainder, the exact time of onset could not be ascertained. He next takes up the cases according to clinical form, and discusses their relation to hereditary predisposition, personal history, age, whether occurring before or after delivery, the number of the pregnancy in which they appeared, the nature of the labor and complications, with the percentage of recoveries under each head, and after history as far as could be learned. In general, recovery was more frequent in the older patients, though of the primipara, those under twenty-five years more frequently got well. The general percentage of recovery for the dementia præcox cases was fifty-seven, which is much better than that found in dementia præcox unassociated with reproduction. Heredity did not appear to influence recovery in these cases.

Among the manic-depressive cases, 81.2 per cent. had hereditary predisposition, and 9 of the 32 patients had shown traces of this psychosis before marriage. All except two who died of intercurrent diseases recovered, but had other attacks sooner or later, four, however, not until from eighteen to twenty-four years later. This psychosis seems to run its course in the main, but slightly influenced by the act of reproduction as does also hysteria. Ten of the eighteen cases of hysteria had shown previous symptoms of this disease, and in 71.4 per cent. of the cases there was hereditary predisposition. By acute confusion (or amentia) the author understands the "exhaustion psychosis" of Kraepelin. Its frequency has been much decreased by the placing of most of the cases formerly diagnosed as such under the heads respectively of dementia præcox, and of manic-depressive insanity. Ten of the fifteen cases of this psychosis were puerperal, five lactational. All were associated with difficult labor, puerperal infection or some complication. All recovered usually within three and a half months except one case, which was taken away after one month, and was lost sight of. Five of these women bore one or more times after recovery from a first attack without recurrence. Six of the epileptics had their first attack in one or other period of the reproductive act. In the other cases the influence of the child-beaing could hardly be considered as more than helping along an existing trouble. In fact, it must be considered in the main, as chiefly an exciting cause acting upon an already present predisposition.

4. *Etiology and Symptomatology of Katatonia.*—A male during his six-

teenth year suffered a severe injury to the head, causing fracture of the skull with concussion of the brain, necessitating an operation for the removal of depressed bone. From this there resulted alteration of character, headaches, tremor of the hands, and later intolerance toward alcohol. Nine years later, there began a mental disturbance characterized by hallucinations, illusions and delusions of persecution, with later the characteristic symptoms of negativism, verbigeration and stereotypy, which proceeded to moderate dementia. Discussing this case as to its peculiarities, the author discovers some features in the speech and handwriting with dermographism, etc., which suggests a traumatic neurosis, and makes him think that there may be here a combination of this condition with dementia præcox. He believes that trauma is never the sole cause of a neurosis or psychosis, but always plays the rôle of an exciting cause acting upon an already present predisposition. This he thinks well illustrated in this case, and while he inclines to the view that dementia præcox in general is due to an autointoxication of some sort, trauma, he suggests, may have an influence in so disturbing the normal metabolism as to favor the production of harmful substances.

5. *Psychical Condition of the Insane When Dying.*—Walter Albrand, while making certain observations upon the iris movements in the dying, at the Sachsenberg Asylum (already published in another journal), was led at the same time to note the mental condition of his patients. After a review of the subject, with some illustrative cases, he concludes that: In insane persons the improvement in mental condition just before death over that observed in the same individual in bodily health is inconsiderable as a rule. People in general, upon the approach of somatic death, may conduct themselves differently, depending upon the nature of the death bringing disease, but nevertheless their mental processes are apt to move more or less in their formerly accustomed channels. The same thing is usually to be found in the dying insane, the method of death, and the more or less mobile mental condition having a similar effect upon the psychical state as it would have in the sane. A complete alteration in the mental personality of an insane person on his deathbed is never found, but a profound change of mood, both on the approach of death and during severe bodily illness, is undoubtedly sometimes observed, though we can in no way explain this fact. That an insane person, just before death, occasionally abandons his latest idea in favor of earlier ones, and begins, as it were, a new psychical life, is also from time to time noted, but it is impossible to construct a definite antemortal psychopathology. The author makes a digression to discuss the influence of acute febrile diseases upon the mental condition of the insane, and the proposal to utilize therapeutically the occasionally observed curative effect of such processes. With regard to this last, however, he confesses great skepticism, and thinks that in general, infectious diseases are more likely to have an unfavorable than a favorable influence upon the mental condition.

6. *Commitment on Account of Partial Insanity.*—Report of the judgment of an Austrian upper court in a case of this character.

ALLEN (Trenton).

Brain

(Vol. 28. Parts 3 and 4.)

1. Cerebral Sclerosis. ALFRED W. CAMPBELL.
2. The Clinical History and Post-Mortem Examination of Five Cases of Myasthenia Gravis. E. FARQUHAR BUZZARD.
3. Ataxia in Childhood. FREDERICK E. BATTEN.
4. On the Metabolism and Action of Nerve Cells. F. H. SCOTT.
5. The Onset of Hemiplegia in Vascular Lesions. A. ERNEST JONES.

1. *Cerebral Sclerosis.*—Dr. A. Campbell contributes a lengthy article on the general pathological aspects of cerebral sclerosis. After some general

notes on neuroglia, its development and growth he takes up tuberose sclerosis, megalocephaly, cerebral hemiagenesis and hemisclerosis, lobar agenesis with sclerosis and microgyria, cerebral arteriosclerosis, giant cell sclerosis and other forms of sclerosis in general paresis, senility, epilepsy, idiocy and that following cerebral softening and syphilis.

He recognizes five neuroglia elements, (1) the large neuroglia cells of Bevan Lewis, Kurzstrahler, of German writers; (2) the stellate cell of Golgi, small spider cell, Langstrahler; (3) glia cells of Retzius; (4) small cell of Bevan Lewis, nucleus; (5) neuroglia cell of Ford Robertson. In reference to the sclerotic processes he holds that for the commoner forms of sclerosis they are brought about by the deposition and metamorphosis of new elements and not by the proliferation of pre-existent cells. The most important of these new cells is the large mononuclear leucocyte or phagocyte. Speaking of tuberose sclerosis he notes that it is almost always confined to idiots and imbeciles, and this suggests to him an intrauterine origin for the cerebral disease which underlies it, which he believes is the ultimate manifestation of some evolutionary aberration or disturbance.

Hypertrophy of the cerebrum is included although sclerosis is usually accompanied by shrinkage. Here, however, there is expansion with marked proliferation in neuroglia elements. The induration and enlargement are general rather than local, and inasmuch as there is little evidence of irritative conditions the process is a unique one. Although comparatively rare, it is common enough to have good material for comparison. There is also a marked hereditary element in these cases, and Campbell inclines to the view that the process leading to the hypertrophy probably was an active one during intrauterine life. Its essential factors are not yet clear. The increase in neuroglia is situated in the white tissues only, and probably took place at the time of the neuronal development of the great systems.

Hemisclerosis, or hemiagenesis, is a rare affection. He believes it to be due to an arrest of growth of one-half of the brain, and the histological evidence tends to show the structures of a foetal brain. The cessation of development probably took place just before or at the time of birth. The elements subserving primary essential functions being developed persist, while those elements subserving higher evolutionary functions, being only partially developed, suffer decay, and hence the neuroglia proliferation and sclerosis. Lobar agenesis is another rare condition, probably similar in its origin, but not so widespread; moreover it is usually bilaterally situated. The author makes a distinction between this process and that of the contraction and induration about an old patch of cerebral softening, and to postmeningitic atrophy and sclerosis. Cerebral arteriosclerosis is next discussed. He believes it not so common among the insane as heretofore thought. He is speaking of gyral attenuation and sclerosis apart from that attendant on gross lesions such as embolism and thrombosis. Gyral arteriosclerosis, he concludes, with atrophy and pitting, is a disease chiefly confined to aged dements with diseased arteries. It attacks the cortex in patches, and apparently is the outcome of a discrete occlusion of cortical arterioles. Colloid sclerosis is an affection of the blood vessels, with a minor participation of glial proliferation. The colloidal material results, the author believes, from a proliferation rather than degeneration. One patient showed a giant cell sclerosis, or infiltration. It might properly be termed a "glioma gangliocellular." Cerebral sclerosis in paresis, in senility, in epilepsy, in hemorrhage, in syphilis, idiocy and multiple sclerosis are briefly considered.

2. *Myasthenia Gravis.*—Buzzard contributes an illustrated paper on this affection based on the pathological findings in five patients. He would maintain from the clinical side that sensory disturbances may be present in this disease, although heretofore they have been considered absent, and he has had one patient with complicating mental symptoms. It is in all probability a disease with a constant morbid anatomy consisting of widely distributed cellular, and sometimes serous, exudations (lymphorrhages) in

the tissues and organs of the body. Slight muscle fiber changes are frequent and severe, muscular atrophy is rare. Proliferative and degenerative changes are frequently, but not constantly, met within the thymus gland. The symptoms, he believes, are best explained on the basis of some toxin having special influence in voluntary muscle and its relations to the thymus are suggested.

3. *Ataxia in Children.*—Batten deals here with some irregular and little known forms of ataxia not included in the well-known groups of Friedreich, and tumor and lesions of the cerebellum and midbrain. He favors the term cerebellar diplegia for his cases, grouping them in three series. (1) Case in which ataxia has been noted early in life, and in which there is a tendency to gradual improvement (congenital cerebellar ataxia). (2) Cases in which ataxia has developed suddenly after some acute illness (acute ataxia, encephalitis cerebelli). (3) Cases in which the child has been healthy until a certain age and then has developed ataxia gradually. These cases resemble Friedreich's, but differ in development (progressive cerebellar ataxia). Particular attention in differential diagnosis must be given to multiple sclerosis, to quiescent cerebellar tumor and to past meningitis and hydrocephalus.

4. *Metabolism and Action of Nerve Cells.*—In this paper Scott tries to solve some of the problems of nerve function by trying to find similar chemical substances in other organs of the body. He finds that a substance of the same nature as Nissl's substance occurs in the cells of the pancreas and in the chief cells of the fundus glands of the stomach. The neurosomes of Held, he holds, are morphologically homologous with the zymogen granules of gland cells, and there is an interdependence between the amount of Nissl substance and the number of neurosomes, exactly as there is between the prozymogen of Macallum and the number of zymogen granules. The nuclei of these three cells also resemble one another. There is also much resemblance in the action of these three cells in that they all are concerned in controlling the changes in proteids. On these similarities the author frames the hypothesis that nerve cells also act by a kind of proteolytic ferment.

5. *The Onset of Hemiplegia in Vascular Lesions.*—The author contributes an extremely interesting paper, which must be read in extenso. Among those conclusions which seem to offer the greatest probability he gives:

(1) Rest in bed, and especially sleep; protect to some extent against cerebral hemorrhage. (2) Severe exertion and time of day appear to have had too much stress laid on them in the past. Time of day is of interest only when the habits of the patient's blood pressure at different hours are known. (3) Consciousness is lost at the onset in half of the cases of occluding lesions and three-quarters of hemorrhage lesions. (4) The immediate prognosis is much graver when the onset is apoplectiform; especially is this so in cases of hemorrhage. Contrariwise, cases of late hemiplegia due to hemorrhage are less likely to have suffered from loss of consciousness at the onset than hemorrhage cases taken as a whole, and are therefore the more likely to be attributed to thrombosis. (5) Intraventricular hemorrhage, which is nearly always secondary, may not cause loss of consciousness. On the other hand, immediate loss of consciousness as the initial symptom may be due to extraventricular hemorrhage. The immediate prognosis is much graver in cases of hemorrhage than in occluding lesions. In 828 cases of hemorrhage, 158 of thrombosis and 273 of embolism the results are as follows: Over 30 per cent. of the cases of hemorrhage were fatal within twenty-four hours; half as many of thrombosis and a quarter as many of embolism; almost two-thirds of the hemorrhage patients are dead in a week, and more than a third of the patients with occluding lesions. Of twenty cases of each lesion four hemorrhagic ones would survive a month; five thrombosis ones and nine embolism ones. Most of those that survive two years are patients with

thrombosis. (7) Of the patients in whom blood is found in the ventricles, 60 per cent. die in the first twenty-four hours and 90 per cent. in the first week. It is not very rare, however, for such cases to live a few weeks. (8) The mortality incident is heaviest on the first day amongst the younger men; in the next few weeks it is the aged women who are most likely to die. (9) There is no indication that hemorrhage affects the right side of the brain more often than the left. Occluding lesions may affect the left side more than the right for all that is known to the contrary, but the statements usually made are not warranted in the present state of our knowledge. JELLIFFE.

Psychiatrisch-Neurologische Wochenschrift

(Feb. 3, 1906.)

1. *Simulation of dementia by Weakmindedness.*—BRESLER. The patient, a woman, had been several times convicted of crime, but the last time she was apprehended she was sent to the asylum. During her examination by the magistrate, and afterward on her admission to the asylum she showed marked mental symptoms, although all those that knew her testified that she was an accomplished rogue. One of the principal features was her marked memory defects. These defects, however, did not follow any type and when she was observed and did not appreciate she was being examined they largely disappeared. Her memory was still poor for recent events though, and this the author thought evidence of weakmindedness, as was also her great tendency to lie. In general her simulation was overdone. The amount of mental defect was too great, as for example, at one time she replied, "I don't know" to all questions.

(Feb. 10-17, 1906.)

1. *Decisions of the Prussian Court in Regard to Caring for the Dangerous Insane.*—Continued.

(Feb. 24, 1906.)

1. Delusion and Error. P. NÄCKE. (Continued.)
2. The Lack of Physicians in Asylums for the Insane and a Hitherto Unobserved Cause for Same. H. HOPPE.
3. Decisions of the Prussian Court in Regard to Caring for the Dangerous Insane. (Continued.)

1. *Delusion and Error.*—Will be abstracted when finished.
2. *Lack of Physicians in Asylums.*—Deals with local conditions only.
3. *Decisions of Prussian Court.*—Not of interest to American readers.

(March 3, 1906.)

1. The Question of the Psychiatric State Asylum. DANNEMANN.
2. Delusion and Error. P. NÄCKE.

1. *Psychiatric State Asylum.*—Continued.

2. *Delusion and Error.*—The article seems to be suggested by a recollection of a fanatical Russian sect, the Duchoborzen, who started out to find Jesus. They were a half clothed, ill-fed, poor, and more or less alcoholic people easily influenced and led. Russia seems to be the land above all others where all sort of possible and impossible sects thrive. Löwenstimm mentions a sect that instead of seeking Jesus sought the devil, and other sects are the Skopzen, Flagellants, Chlyster, Wanderers, Deniers, etc. The belief in witches and demons is not yet dead. All these ideas fixed by suggestion, delusions in the psychiatric sense, are errors only. The distinction is by no means clear. It is said that delusion must have its own special soil and error not, but epidemics of false ideas show this often not to be the case. In both cases there is a special affect state, mood, endogenous or exogenous, conditioned by the surroundings and the time. The delusion develops gradually, the error often suddenly, when the affect reaches a certain tension. The foundation of both may be quite similar. Either may take its origin in the same way—from the Bible or

from a speech—a primary or secondary affect arises, then falsification of memory and the border between delusion and error, already vague, is still more encroached upon when hallucinations, illusions, and convulsions occur as they frequently do in epidemics.

The author calls attention to the following distinctions. The delusion is seldom single. Cases of hyperquantivalent ideas in the sense of Wernicke, however, he thinks do occur. The delusion is usually fixed, cannot be changed by argument, has a strong affect-tone and leads to action. In delusional states the personality in its innermost parts is altered, while in error this is not so. The individual remains the same. The delusion grows and develops on a pathological foundation, the error on a foundation of health. The idea in itself is characterized neither by delusion or by error. We know how often the nucleus of the ideas in litigous insanity may be true, and on the other hand how the most foolish ideas may be held under certain conditions of time and surroundings. The author is of the opinion that an error under favorable circumstances of soil and strong outer influences may develop into a delusion, while an insane person may have an erroneous belief which can be corrected without influencing the delusional state. As to the alteration of the personality in error it may be greatly altered by the radiation of the false ideas, while with single hyperquantivalent ideas there may be little change. Suggestion of long duration with unchanged surroundings and affects may lead gradually to a falsification of the personality. Suggestibility may thus lead to true insanity, while on the other hand the insane may become suggestible.

WHITE.

Archiv für Psychiatrie und Nervenkrankheiten

(Vol. 41, Part 1.)

1. Symptomatology of Epileptic Insanity, Especially Concerning the Relationship Between Aphasia and Perseveration. RAECKE.
2. On the History of Spasmodic Torticollis. ARMIN STEYERTHAL.
3. A Contribution to the Study of Myxedema. FREDERICH HEYN.
4. Symptomatology and Pathogenesis of Acquired Internal Hydrocephalus. L. W. WEBER.
5. Concerning Cortical Focal Symptoms in the Amnesic Phase of Polyneuritic Psychoses. REIHOLD KUTTNER.
6. Contribution to the Normal Anatomy of the Ganglion Cell. CONSTANTINE J. ECONOMO.
7. The Operative Treatment of Brain Tumors. C. FÜRSTNER.
8. The Neutral Cells of the Central Nervous System. P. KRONTHAL.
9. Simulation and Mental Disorder. A. SCHOTT.
10. Areas of Softening in the Medella Oblongata with Ascending Degeneration in the Pryamidal Tract and Fillet. O. KÖLPIN.
11. Hereditary and Predisposition or Degeneration in Progressive Paralysis of the Insane. P. NÄCKE.

1. *Epileptic Insanity.*—Raecke reports four cases in detail as a basis of his discussion of epileptic insanity. The general result of his study is that in the mental disturbances of epileptics aphasia and perseveration do not stand in close causal relationship, and that the existence of one cannot be assumed from the presence of the other. Amnesic aphasia doubtless plays a large part in the symptomatology of epileptic insanity, but cannot be regarded as an absolutely constant symptom. Certain details are added regarding the somatic signs occurring in different stages of the disease, as observed by the writer.

2. *Spasmodic Torticollis.*—In this paper the writer discusses from the historical standpoint the condition of spasmodic torticollis, which he finds described by many of the early investigators. Much of the article is a reproduction of the original Latin in which the early descriptions were made. As an addition to a previous article the author gives the subsequent

history and autopsy findings of certain cases previously reported. The pathological anatomy of the condition remains unwritten.

3. *Myxedema.*—Heyn in this paper, on the basis of considerable personal experience of the disease, both clinically and anatomically, reviews the general theories underlying the etiology of myxedema. The treatment which has been found useful is in advanced cases, one of general hygiene with an attempt to improve the nutrition. At the beginning of treatment a milk diet with vegetables is used with benefit, although the reason is not apparent. When improvement has begun iodide of potash is given on general principles. Following this the specific thyroid treatment is begun, which is administered in the form of thyroid tablets. A discussion of the blood as studied in one of the cases concludes the article. From autopsy findings the author concludes that the specific poison injures the blood vessels to a high degree as shown by the changes in the heart, aorta and coronary vessels.

4. *Internal Hydrocephalus.*—Weber offers a profusely illustrated and elaborately detailed article on acquired internal hydrocephalus. The paper is divided into four parts, which treat the subject respectively from the point of view of clinical cases, from the standpoint of symptomatology, followed by a discussion of the pathological anatomy and pathogenesis. The concluding section summarizes the general results of the study and their significance. As congenital hydrocephalus, are considered those forms in which the cause is to be found in uterine life. It is maintained that in the congenital form the mechanical prevention of the outflow of cerebrospinal fluid is more significant, whereas the increase of the fluid is less significant. As idopathic hydrocephalus only those cases are to be considered in which the mechanical conditions cannot be explained by pathological anatomical findings in the brain or its neighborhood. The writer avoids the phrase "hydrocephalus without anatomical basis." The number of such cases is naturally diminishing as knowledge grows. The increase in cerebrospinal fluid is often due to chronic inflammatory conditions. A valuable discussion of the theories and the various conditions under which hydrocephalus may occur concludes this significant contribution.

5. *Amnesic Polyneuritic Psychoses.*—Attention is called to the fact that in certain stages of the polyneuritic psychoses focal symptoms usually of the aphasic type may supervene as somewhat temporary symptoms. As more permanent symptoms are developed defects of intellect, loss of memory for recent events, difficult concentration, confabulation and a disturbance in writing, characterized by a marked difficulty in finding proper letters, and in perseveration of components of words and sentences. Cases are reported and literature bearing on the subject is cited. The writer finally concludes that the aphasic or agraphic disturbance is due to localized affection of the sensory area of speech, which represents an increase of the diffuse brain process.

6. *Ganglion Cell.*—Economo offers a critical study of the recent nerve cell histology, and draws attention to a particular intracellular structure which he regards as an original observation. The article is illustrated by fifty-three figures, and although not permitting detailed review on account of its technical character, is worthy of close study by special students of the subject.

7. *Operation in Brain Tumor.*—Fürstner offers a clinical study of brain tumors with comments on the operative treatment. In each of five cases reported operation was performed, and the existence of tumor confirmed by autopsy in three. Following an analysis of the cases from the clinical standpoint, Fürstner expresses himself in general as in favor of early operation, not only in the hope of cure, but also as a relief of symptoms, an opinion which is generally accepted by all who have given attention to this subject. In general, Fürstner would also not be in disagreement

with others in his statement that relatively small tumors in the central convolutions alone offer favorable chances of extirpation, and that the great majority lying in other parts of the brain are open rather to a palliative than to a curative operation, and that in these latter cases operation should be done soon after the appearance of choked disc.

8. *Neutral Cells.*—Kronthal states that the simplicity of a proposition is no criterion of its correctness. As an example of this truism, he argues that the proposition that the peripheral nerve is a process of a nerve cell is simple but wrong. He quotes the new fibrillar methods to substantiate this claim. Kronthal has reached the conclusion on anatomical grounds that the nerve cell is not an organism inasmuch as it is made up of small cells. He uses the word "neutral" cell to indicate cells which occur sparingly in the white substance, but frequently in the gray, of varying size, but usually small, with large nuclei and a small amount of protoplasm, having ameboid characters and furnishing material both for glia and nerve cells. The cells which wander into the central nervous system from blood and lymph channels, Kronthal includes under the terminology "neutral cells." The possibility is maintained that central nerve cells may result from such so-called neutral cells, and an experimental method is suggested of injecting into the circulation substances which may be taken up by the cells and later recovered in the nerve cells which the wandering cells have ultimately constituted. The hypothesis is of interest, but demands further experimental classification.

9. *Simulation of Insanity.*—From a wide experience relative to simulation and mental disorder Schott draws the following conclusions: That it is questionable whether pure simulation of mental disorder ever occurs in the mentally sound; that the simulation of mental disturbance is most frequent among degenerates, and is a consequence thereof; that the acknowledgement of simulation as well as the unmasking of the simulant proves nothing for the mental health of the individual, and that in such cases the expert must bring proof of the mental health of the defendant; that all difficult cases of simulation should be under expert observation in a clinic, even if it requires more than six weeks of such observation to reach a conclusion; that in consideration of the degree of simulation the underlying degeneration must be variously interpreted; that the simulation of mental disturbance does not necessarily lead to actual insanity; that there is no absolute characteristic of simulation; and finally that a thorough study of male hysteria is extremely desirable, and promises much aid in the solution of the question of simulation.

10. *Retrograde Degeneration.*—Kölpin presents an anatomical study of secondary degeneration in the pyramidal tract, and in the fillet resulting from areas of softening in the oblongata. The point to which he draws particular attention is the retrograde degeneration of certain fiber tracts, both motor and sensory.

11. *Heredity and Paresis.*—In the discussion of heredity and predisposition in general paralysis of the insane, Näcke seeks to establish the following propositions: First, that hereditary taint plays a large rôle in the genesis of paralytic dementia, and in the second place that in general paralysis an inherited, more seldom acquired, and very probably a specific brain constitution exists in such a way that the victim falls a prey to the disease more easily in the presence of syphilis or other causes. If these facts be true, general paralysis is brought into closer relations with other psychoses than formerly supposed. Admission is made that cases of general paralysis occur without hereditary taint, and that there are also exceptions to the existence of a recognizable predisposition or degeneration. This paper is followed by a bibliography of 164 references covering the mooted points in relation to the etiology of dementia paralytica.

E. W. Taylor (Boston).

Miscellany

Beträge z. Casuistik d. Myoclonie bei Epileptischen. G. Weiss (Kiel Dissertation, 1905).

In this little pamphlet we find described no less than eight hitherto unrecorded cases. That so large a number of cases of a rare disease is reported by a beginner in medical practice is accounted for by the fact that the eight cases were evidently from the material of the epileptic colony at Bethel, near Bielefeld. As we know of no other cases reported from the material of this colony, the latter may perhaps be assumed to represent the total morbidity of this associated disease in this well-known institution.

The cases in brief are as follows:

1. Male, 16, no bad inheritance, nursed normally, no teething convulsions. In his third year had encephalitis, and as a result suffered greatly up to fourteenth year with headache (often severe enough to provoke hallucinations), and enuresis. When nine years old developed epilepsy, the first attack due to fright. Seizures reappeared about once in two weeks. About two and a half years later the myoclonus appeared as a complication of epilepsy. The myoclonic contractions were of progressive and great severity, and the epileptic seizures seemed correspondingly emphasized.

When examined patient seemed healthy in body and mind, aside from the chorea-like twitchings affecting the voluntary muscles. During the observation period two epileptic attacks were noted, at night. The frequency of the epileptic attacks at this special period of observation was six to eight monthly.

This case antagonizes Unverricht's teaching that in myoclonic epilepsy the convulsions are not synergistic, while in chorea this result is the case. In this instance the convulsions were not only symmetrical, but the lightning-like rapidity ascribed to choreic cases was present here.

2. Girl, 10. Diagnosis: Epilepsy due to intra-uterine trauma. Epileptic attacks began when child was three or four months old. Father neurotic and tuberculous; otherwise no hereditary data.

When first seen the patient exhibited no marked stigmata, but had continuous myoclonic convulsions. She seemed to be below the normal in intelligence.

There was a history of certain crises which were more than myoclonic, which resembled epilepsy and which seemed to be followed closely by myoclonic attacks of such severity that patient must lie abed. Under the observation period patient had one or two attacks per month of epilepsy, which increased the violence of the myoclonus.

3. Girl seen in 1899, then three years old. Diagnosis of epilepsy and idiocy. But little history. Mother said to have died of puerperal fever, and a brother died of convulsions.

Examination showed an idiotic child with extremities, head and face in constant movement. Under observation patient was seen to have myoclonic movements which led up to general convulsions and unconsciousness. An attack of pneumonia temporarily checked both conditions, which reappeared with renewed severity during convalescence.

4. Male, 10. Heredity normal, gestation normal. On second day of life seemed to develop a case of trismus, which did not kill child, and which became chronic. At age of nine months typical epilepsy set in, although something like myoclonus or chorea had been in evidence soon after birth. A sister had died under same circumstances. Close inspection of patient showed evidences of mental weakness, while contractures of many voluntary muscles seemed to represent the result of myoclonus. Attempts to oppose these contractures led to clonic spasms, both flexors and extensors. The progress of the case showed abundant myoclonic at-

tacks, especially of the "intention" order, following attempts to speak, etc. Psychical excitement produced the same result. During the observation period no epileptic attacks are mentioned, but the myoclonus showed a periodicity and an intensity which suggested the latter. At an earlier period in the history pronounced epileptic attacks seem to have occurred.

5. Boy aged six. The author finally placed this case under the caption of chorea electrica, mentioning it only because of the doubt as to diagnosis. The boy probably suffered from encephalitis at an early age, and as a result was backward mentally. After his brain fever he had numerous convulsions of presumably encephalic origin. These crises only partially resembled epileptic seizures; they were as a rule preceded by general twitchings. The latter were in nowise due to external influences. The author, despite Unverricht's criteria, is disposed to regard this case as one of myoclonic epilepsy.

This patient died of intercurrent pneumonia, and no autopsy was permitted.

6. Girl aged 16. No hereditary taint. At three years had encephalitis and rachitis. Then epilepsy developed at intervals of fourteen days, four weeks.

When seen at age of 14, child was found normal in most respects. Had begun to menstruate. After this period various attacks had been noted, in part typically epileptic and for the rest myoclonic. With further progress the myoclonus appeared to precipitate the epileptic attacks. This patient was left with this *status quo*.

7. Male. At age of one and a half years began to stammer as result of convulsion. At the age of 14 the spasms still persist, in part as typical epileptic seizures and for the rest severe myoclonic convulsions. This patient's condition was so interesting that considerable space is devoted to the different types of convulsions. The speech disturbances correspond with those described by Unverricht.

8. Male, first came under observation at age of 26. Not much early anamnesis obtainable. Head shows many scars. From age of 17 had had both kinds of convulsions; viz.: myoclonic and epileptic.

When examined denied being an epileptic, although gave credible accounts of myoclonic seizures. While under observation had typical epileptic seizures.

In analyzing these eight cases author states that all had true epilepsy. As for precedence, epilepsy was surely primary in case first and secondary in case fourth, also in case sixth. Others not considered. The author appears to contradict himself, assuming at the outset that all the eight patients had myoclonus, but he seems to have been undecided about Case 5, which he once speaks of as electric chorea. He does not return to this case in summing up, and further states that chorea is so rare in epilepsy that not one case was recorded in 2,000 of epilepsy.

Under the head of pathologic anatomy he first gives the results of the work of Clark and Prout. He mentions Apáthy and Kronthal on the histology of brain-cells, etc., and appears to think them not compatible with findings of Clark and Prout, although he is obscure on this point.

Muni is also quoted on this point. In three autopsies he could find no alterations in Rolandic regions. Unverricht and Bresler also deny that any anatomical basis exists for epilepsy. A thesis in 1903 by Foerster, inspired by Bresler, makes a point that the muscular movements do not correspond with definite muscular co-ordinating centres in the cortex. The present author quotes about two pages more from Foerster's thesis. He closes by stating from Unverricht that a spinal origin would explain myoclonic movements much more than a cerebral one. Author states that his eight cases reveal absolutely nothing as to the cause of the disease.

L. PIERCE CLARK.

Book Reviews

In the Thirteenth Annual Report of the Craig Colony for Epileptics at Songea, N. Y., it is seen that the daily average census of the colony has increased from 992 to 1,046. About 350 cases are low grade epileptics that should be cared for elsewhere, thus giving more room for hopeful cases. The total number of epileptics in New York State is about 16,000, and 2,000 of these are of low grade. The Superintendent, Dr. Spratling, says the number of epileptics is not increasing out of proportion to the increase of population; but he estimates a higher ratio in the community than the text-book ratio of 1 to 500. As to curability he quotes Dr. Huchzemeier that 587 epileptics at Bielefeld have been cured since 1867, and states that 5 per cent. at Craig Colony is their record. He pleads for earlier admissions as affording far greater hope. The death rate from tuberculosis alone in this and four other institutions for epileptics averaged 24 per cent. The general death rate from tuberculosis in New York State is 9 per cent. One-third of the deaths at the colony are directly due to epilepsy. Forty-five cases of status epilepticus occurred, of whom six died.

The Superintendent advocates checks on the perpetuation and increase of the defective classes by laws preventing their marriage. He also suggests as a way to help check insanity, epilepsy (and perhaps drunkenness), in their incipiency, that the State appoint eight psychiatrists, one for each judicial district, whose duty it would be to visit and assist in the early treatment of such cases, subject to the call of any physician, or poor person, or overseer of the poor, and paid a salary by the State. The money thus expended would also, he thinks, be an ultimate saving to the State, presumably by shortening the time of maintenance of such cases in public hospitals or elsewhere.

The pathologist reviews the intoxication theory of epilepsy and agrees that changes are found in the toxicity of the blood and urine in relation to the attack, with changes in the substances excreted in the urine, also related to the attack. He agrees that there is less (even no toxicity in some cases), between attacks, and that an attack seems to be associated with an "unloading of stored poisons;" that there is hypertoxicity of the blood and urine preceding an attack; and that the true nature of the poison is unknown.

The existence of an organic pathology of the cortex, he thinks, is not yet proven. "The study of epilepsy as a disease of the metabolism, possibly spontaneous within the nerve cell and possibly due to the action of an intoxicant formed elsewhere in the body, is the line of work planned for the laboratory (at Craig Colony), and is one which has always seemed to promise the greatest results in the end." C. E. A.

Ueber Störungen des Handeln bei Gehirnkranken. Von Prof. Dr. H. Liepmann, Privatdozent an der Universität Berlin, Assistenzarzt an der städt. Irrenanstalt in Dalldorf-Berlin. S. Karger, Berlin.

Liepmann here contributes a short but highly suggestive monograph on disturbances of action as a result of brain disease. He pays particular attention to the subject of motor apraxia concerning which he has written so extensively.

Summarizing his study he says that disturbances of action as local signs following the destruction of definite portions of brain substance may be referred. (1) To paralysis or paresis: Abolition of movement or loss of strength of same. (2) To ataxia: Mistakes in the measure of the

strength of the excursion, etc., resulting from the loss of the peripheral kinesthetic derivations. (3) To loss of the kinesthetic ideas: A condition not sharply differentiable from cortical ataxia-soul paralysis. (4) To Agnosia ("sensory" Assymboly-Apraxia in the old sense): A loss of perception or recognition by intact sensations. The identification of the fresh impressions with the memory pictures ceases, either by reason of the loss of the letter (Wernicke) or by reason of a delayed combination of both (Lissauer). (5) An indirect cause of disturbed action is the qualitative or eventually localized lapse of confined sensations: Cortical blindness, etc. The corresponding lapse for the kinesthetic areas is included under 2 (Ataxia).

Between 1, 2, 3, on the one hand, and 4 and 5 on the other there lies still (6) a motor (innervated) apraxia: The movement is not in accord with the ideatory process: the cortico-muscular apparatus functionates well, but is not in the service of the entire psychical processes. Not causing true focal symptoms, but conditioned by diffuse processes, eventually as general symptoms in large lesions is (7) ideatory apraxia: The movement is in accordance with the ideational process, but this in the end, disturbs the design of the movement series, by transposing the main goal idea to a secondary or partial goal idea. Ideatory apraxia is probably a part of a general ideational disturbance. (Memory, attention, etc), and allies itself mostly with Agnosia, eventually only with an ideatory agnosia. He makes a differentiation in this work between motor apraxia, ataxia and soul pralysis, and also enters more deeply into the consideration of the symptom of perseveration. His main contribution consists in the separation of a motor and an ideatory apraxia.

The work is one of great originality—schematic in the Wernicke sense—but no one can afford to overlook it. JELLIFFE.

CHRISTIANITY AND SEX PROBLEMS. By HUGH NORTHCOTE, M. A. F. A. Davis Co., Philadelphia.

The wide extension of the discussion of sexual matters betokens the demand of a curious public, whose erotic sensibilities have been much stimulated in recent years by the press, the pulpit and the stage. Northcote's contribution contains much that is new and little that is untrue, and for many readers it will prove interesting. It has the advantage of being modest and non-prurient, and its honesty of motive is unassailable. This much cannot be said for all such similar volumes. Its chief interest lies in its analogies and commentaries drawn from the Hebrew writings concerning the relations of the sexes. On the whole, it is an excellent work.
JELLIFFE.

STUDIES IN THE PSYCHOLOGY OF SEX. By HAVELOCK ELLIS. F. A. Davis Co., Philadelphia.

Ellis makes another contribution to his growing list of studies. He here deals with Erotic Symbolism, The Unchanism of Detumescence and The Psychic State of Pregnancy. This terminates his series of studies on the usual phenomena of the sexual process, and he promises a concluding volume—one on the general problem in its social relations or the psychology of sex as interpreted in the light of social hygiene.

He includes under erotic symbolism all of the aberrations of the sexual instinct, although many of the phenomena have been already discussed by him in other volumes.

Exhibitionism-fetichism and like phenomena make up the early portion of the volume. Here are discussed a number of well known phenomena, and attempts are made at general explanations.

The phenomena of detumescence are taken up for the male and female in the second portion. The variations in detumescence are inadequately yet for the purposes satisfactorily handled. The third portion is an ex-

tremely interesting summary of the psychology of the female during child-bearing. Here are collected a mass of the strange psychical phenomena attendant on pregnancy. They are rich in suggestion, although not critically presented. The volume is on a par with the author's previous contributions, showing his zeal and discrimination and his scientific training. JELLIFFE.

The Second Biennial Report of the Parsons State Hospital for Epileptics, at Parsons, Kan., claims for Kansas that it is the only State in the Union which has entirely separated the epileptic and non-epileptic insane. The total number of epileptics in Kansas in estimated at 3,000. The number cared for in the epileptic hospital has increased in two years from 170 to 355. Very young children, low grade imbeciles and idiots, are not received. Insane epileptics are committed by the courts. "Sane" epileptics are received on voluntary commitment; the latter cannot be detained against their will; and the Superintendent comments that the form of commitment is not always an index to the patient's mental condition. As regards marriage of patients prior to admission, he found that 30.7 per cent. of the men and 42.8 per cent. of the women had been married, and that 12.6 per cent. of the men and 25.7 per cent. of the women had married after the development of epilepsy. The "most important causes of epilepsy" as tabulated, were "bad heredity, infantile palsy, peripheral irritation, head trauma and alcoholism." The most frequent causes of death found were organic heart disease, exhaustion, pneumonia and status epilepticus. C. E. A.

PHYSICIAN'S VISITING LIST, FOR 1907. P. Blakiston's Son & Co., Philadelphia.

The members of the medical profession—and they are many—who have learned to look for the issue of this small volume about this time of year will not be disappointed in its annual appearance. The completeness, compactness and simplicity of arrangement which have been its boast in the past are as conspicuous as ever, and it is in every respect strictly up to date. The strong and dignified binding, the appropriate size for carrying in the pocket, the pencil always ready for memoranda, and the secure pocket for loose slips combine to make it a very desirable part of the physician's equipment. GOODALE.

UEBER ROBERT SCHUMANN'S KRANKHEIT. Von P. J. Möbius. Carl Marhold, Halle, 1906.

Möbius contributes another to his many series of biographical sketches. It was held by the physicians who treated Schumann and by those who performed the autopsy that he died of paresis, but Möbius has reviewed the entire history and shows with much wealth of detail that this is probably an incorrect deduction, and that even the pathological findings—not microscopically controlled, as is so necessary—are not conclusive.

He believes that Schumann suffered from dementia præcox, and this small monograph practically proves it. It is interesting reading.

JELLIFFE.

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Original Articles

THE DIAGNOSTIC VALUE OF LUMBAR PUNCTURE IN PSYCHIATRY.*

BY J. L. POMEROY, M.D.,

OF WARD'S ISLAND, NEW YORK CITY.

Following the work of Widal, Ravaut and others, systematic observations have been made upon the diagnostic importance of the findings obtained by lumbar puncture, in doubtful cases at the Manhattan State Hospital. We give here the results of one year's experience, and while it is too early in many cases to state positively whether or not our inferences have been correct, still there are enough in which the diagnosis now seems reasonably certain, to enable us to draw some conclusions.

While a great deal of work has been done upon this subject abroad, it has received little practical application in psychiatric work in American institutions. At our own hospital there existed a great deal of uncertainty among the members of the staff as to the exact status of the whole subject. On account of extravagant claims of the value of the method in the diagnosis of paresis, and opposing statements in many instances, as well as the admission that under certain conditions alcoholism also produced a lymphocytosis, the real value of the method was a matter of great doubt; therefore, when this investiga-

*To the memory of the late Dr. E. C. Dent is due the acknowledgment of kindly encouragement and stimulation given at the commencement of this work. To the members of the staff at the Manhattan State Hospital, Ward's Island, New York City, my sincere thanks are also tendered. I specially acknowledge the opportunities given by Dr. L. C. Pettit and the assistance of Drs. Karpas, Hamilton, Washburn and Conzelman. This paper was read in part at the annual meeting of the Eastern State Hospitals Association held at the Hudson River State Hospital, Poughkeepsie, May 18, 1906.

tion was commenced, very little weight was placed upon the presence or absence of a lymphocytosis. Other factors which contributed to the uncertainty of the evidence furnished by lumbar puncture were the lack of definite knowledge concerning the histogenesis of the spinal fluid in various states, the origin of the cellular elements, deficiencies in the technique of examination, and finally most important of all, the absence of a sufficient number of autopsy reports upon cases studied with a view to determine the bearing of the spinal fluid examination. In view of these facts, we determined to compare our results with observers abroad and gain a more definite knowledge of the practical use of the procedure. It became necessary to study the cellular content in the spinal fluid of syphilities, and we have to thank Dr. Follen Cabot for the privilege of his wards at the City Hospital. Except for these cases all of the material was under our direct observation. Nothing has been added to the histogenesis, nor have we improved upon Ravaut's technique, but it is hoped that these clinical studies with the accompanying autopsy reports will be acceptable as an endeavor to aid somewhat in the better understanding of the scope and limitations from a diagnostic standpoint, of lumbar puncture. In such a field of medicine as psychiatry, where of all others ordinary clinical methods more often fail to reveal a diagnosis, certainly it is our duty to test with greatest care any method which promises to add additional diagnostic data.

Primarily, is the question of technique. We have followed as closely as possible the method of Widal and Ravaut. Concerning the operation itself the details are well known; after obtaining the fluid, usually from 3 to 5 cc., it is centrifuged for 15 to 30 minutes in a conical shaped tube having a very fine point. This latter assists very materially in holding the sediment while the supernatant fluid is decanted. The speed of the motor should be as near 3,000 revolutions per minute as possible. Even then Nissl claims that he is not sure that all the cells have been precipitated. In our work, this is found to be a sufficient speed, and practically the decanted fluid has been thoroughly decellularized. After inverting the tube it should be allowed to drain in an upright position until only a very small droplet remains in the tip. With the tube held in

such a position, a previously prepared fine glass pipette is passed up the center until by capillary action the drop is withdrawn. Usually it rises in the pipette from 3 to 4 mm. This material is carefully divided into 3 equal parts on cover slips or glass slides, taking care not to spread the drops. The slides can be fixed either in equal parts alcohol and ether or absolute alcohol, and are then stained preferably with Unna's polychrome. Excellent pictures were obtained quite rapidly without fixation by using Nocht-Hastings' blood stain, such preparations gave very clear chromatin and nuclear pictures. Crystalline bodies frequently obscure the field, but may be in a great part removed by gently washing the slide in normal salt solution after fixation. The slides are mounted in the usual way and the specimen examined at first under a low power. This should always be done in order to see if there is a uniform distribution of the cellular elements; this acts as a control over the oil immersion. The counts should then be made with an oil immersion lens, taking Ravaut's figures as a basis, the findings are as follows, normal 2 to 3 cells; 5 to 6, a suggestive reaction; 6 to 20, a moderate increase; 20 to 150, positive. In many instances the counts in the 3 slides will differ, and the method we have followed is to take the average of 100 fields selected from all three preparations. Slides containing blood are unsatisfactory, and in this report have been rejected. Ordinarily, however, where the lymphocytosis is unmistakable, and a second puncture cannot be obtained, I see no reason why the evidence should not be admitted. The fluid should never be changed from the original receptacle before it is centrifuged. The pipettes should be prepared shortly before using by oneself, and the same technique should be observed each time.

The differentiation of the cells is an extremely difficult procedure. Artefacts are common, one finds flattened, folded and peculiarly shaped cells which sometimes stain deeply and again faintly, together with distinct types of lymphocytes, large and small apparently mononuclear elements, occasionally polyneuclear cells of the neutrophile type. Nissl makes the statement that in 100 cell elements in G. P., 80 to 90 are lymphocytes, 5 to 15 mononuclear, 5 to 10 neutrophilic leucocytes, and claims that he has never seen granulations in these cells

which compare at all to those which occur in the blood. This is certainly true in our experience, the discussion of these points, however, is foreign to the purpose of this paper, although it is hoped that with new methods the study of these cellular variations will aid greatly in the better understanding of our present difficulties. There are many things still to be explained, such as the appearance of, in one case, marked polyneucleosis without physical cause, such as fever, the peculiar types of large lymphocytes met with in syphilis, etc. In our own work, it appeared to us that the elements were poorly fixed, and in a great many instances the cells were of a transitional type. This part of the study needs further observation and work.

The albumin test should always be performed. It has been pointed out that the highest readings occur in cases of G. P., although it is not as constant as the cellular increase. The two findings are not always parallel. From a large number of observations it is suggested at the present time that the albumin content may prove as an indicator when the cellular elements are absent: this has actually occurred in Nissl's experience. A case twice punctured showed a positive albumin content and a negative cellular, at the third puncture, lymphocytosis was found.

The technique is simple. Mix equal portions of spinal fluid and saturated solution of magnesium sulphate in the cold and filter, this removes the globulin which is present normally in from two-tenths to one per cent. (Skoczinsky.) Now the filtrate should be boiled and if normal it remains clear, the appearance of opalescence indicates the presence of serum albumin, which is abnormal. Quantitative tests were not carried out systematically and in this report the varying degrees of opalescence only will be mentioned, following Nissl. Tests for the copper reducing body, cholin, etc., are not included in this report.

In consideration now of the causes of lymphocytosis in the spinal fluid, we enter upon a very difficult phase of the subject. We recognize that inflammatory changes having a known bacterial origin (meningitis, etc.) can be differentiated with due care, and it is the slowly progressing changes of uncertain causation that occupy our attention. Even concerning

these, of a surety we cannot deny a bacterial origin; as yet, however, the character of cellular content does not enable us to point out anything more than processes. Quincke is responsible for the statement that certain infectious diseases such as typhus, scarlatina, pneumonia, sepsis, and many febrile diseases, with manifestations of involvement of the central nervous system, cause a high pressure and sometimes increase cellular content. Gross changes are often absent, and the fluid is of a pure serous appearance. Such conditions are explained by stating that there have been found in certain cases a few bacteria of low virulence, but he admits in the majority of cases the conditions are produced by the irritation of the meninges and the central nervous system of a systemic chemical toxin. The lymphocytosis which has been found in herpes zoster, in chorea, heat stroke, multiple disseminated sclerosis and mumps has not been sufficiently reported upon to enable one to draw any conclusion. These conditions, however, should be kept in mind, they are explained as mentioned upon a bacterial basis or upon the theory of chemical irritation. There is still another factor which is to be considered, namely, pressure due to increased secretion. In cases of hydrocephalus, uremia, brain tumor, etc., a slight cellular increase has been observed and attributed to pressure influences. It is more than likely that there is a combination of causes. Trauma to the skull or spinal column has also been found to excite a lymphocytosis and without necessarily causing a hemorrhagic discoloration. In certain cases of mental disturbance resembling the early state of paresis where trauma has recently occurred, such a lymphocytosis is extremely difficult to interpret, especially is this true where alcoholism has been a factor. The absence of albumin increase and the degree of cell content are of assistance, but further work upon these cases is necessary. Finally, lymphocytosis bears a most direct relation to syphilitic infection, and this of all other factors plays the most important role. For a correct interpretation then, it is necessary to consider the studies upon this subject. The intimate relationship between syphilis and diseases of the nervous system requires a most thorough investigation of this disease and the spinal fluid.

SYPHILIS.

Observations have been made upon the spinal fluid in all stages of the disease. Ravaut punctured 82 cases with active secondary lesions. In 54 there was a cellular increase, in 28 the fluid was negative: in general, there was a slight albumin increase. In only 18 was the increase as large as occurs in general paralysis. There was some cellular increase in 67 per cent. None of his cases showed besides headache, any symptoms from the central nervous system. His conclusions are that the lymphocytosis in secondary syphilis varies directly with the intensity or persistence of the skin lesions. In other skin eruptions unless there were complications in the nervous system no changes in the spinal fluid were found.

Ravaut reports positive results in 26 cases of secondary syphilis; in tertiary syphilis even when the skin lesions were marked the fluid might be normal. In such cases he did not find a lymphocytosis until symptoms of involvement of the nervous system occurred.

Widal obtained negative results in 10 old syphilitic cases in which there were no specific or nervous manifestations. Fuchs and Rosenthal summarized their work in 1904 as follows: In 403 patients suffering from disease on a syphilitic basis 94 per cent. gave a lymphocytosis. In 272 other cases with nervous disease on a different basis only 6 per cent. gave a positive result.

Le Maire examined 13 cases of suspected syphilitic hemiplegia, and 12 showed a lymphocytosis. The ordinary type of apoplexy of a non-specific nature gave negative results. Chauffard and Boidin confirm these views and also claim that they are able to differentiate hemiplegia due to meningeal hemorrhage from that caused by central lesions, because of the presence in the former condition of discoloration and contamination of the spinal fluid with red blood cells.

Our findings in syphilitics are as follows: In 15 cases in the City Hospital with well marked secondary lesions, lymphocytosis was decided in 5, in 6 there was a moderate reaction, in 1 a slight increase, while in 3 the results were negative. The 5 cases with positive reaction suffered intensely with headache, but there were no other symptoms of involvement of the nervous system. In 5 of the cases also, Dr. Flex-

ner had demonstrated the Spirochitæ of Schaudin. The cases were all under active mercurial treatment and presented marked cutaneous symptoms. In 1 there was iritis. From this small series then, the lymphocytes were increased in 80 per cent. The 3 cases with negative findings showed well marked secondaries and a faint trace of opalescence, but no lymphocytosis was found.

Of the tertiary lesions, 5 cases, all of whom had symptoms of nervous involvement, were punctured; there was a case of facial paralysis, one of third nerve paralysis, another with a gummatous basilar meningitis with choked disk, a case of supposed syphilitic meningitis of the convexity of the brain, and finally a patient who was supposed to be suffering from a syphilitic endarteritis. All of these cases showed a marked increase in pressure and lymphocytes, with a slight opalescence. Two of the cases are of special interest, one because of the early nervous involvement and the second, because of a critical autopsy review.

The first case, which I wish to report, was a man of some thirty years, who entered the City Hospital with a syphilitic secondary in May, 1905. During his treatment he developed strangulation of a large hernia, and the latter part of May he was operated upon for this condition. At that time he had a fading secondary manifestation; recovery from the operation was prompt and he remained in the hospital during convalescence. During July he began to suffer with constant intense headache, vertigo became marked, there was some ataxia, and at times confused orientation. Later he had attacks of vomiting. Examination of the eyes showed a right choked disk, the right pupil dilated and irregular, but active, with equal and exaggerated knee-jerks. The spinal fluid showed enormous pressure, a very positive lymphocytosis and a trace of opalescence. The early development of the symptoms following secondary lesions brought up the possibility of a brain tumor. The finding in the spinal fluid, however, was decisive for brain syphilis, as was afterwards proven by the prompt recovery of the patient under thorough specific treatment.

In order to understand thoroughly the application of spinal lymphocytosis, investigations should be carried on to determine how often lesions of a tertiary syphilitic nature involv-

ing tissues other than the nervous system are capable of producing a lymphocytosis. We had one experience which shows the necessity of further work upon this line. A patient who was suffering from a gummatous syphilitic lesion of the skin developed convulsions and coma, accompanied by symptoms which brought up the differential diagnosis of epilepsy, uremia, spinal meningitis and cerebral syphilis. The profound stupor and absence of previous attacks were against epilepsy. The urinary findings, the absence of edema, made it appear that the renal functions were active. The febrile symptoms being of a mild character and the absence of the diplococcus in the spinal fluid were against spinal meningitis, therefore, with the presence of a tertiary syphilitic infection cerebral involvement was very probable. The spinal fluid showed an enormous lymphocytosis and the patient was improving under potassium iodide, when she died suddenly in convulsions.

The microscopical examinations failed to demonstrate any syphilitic changes in the vessels of the brain. Dr. Oertel, pathologist at the City Hospital, found no changes of an infiltrative nature in the meninges, and Dr. Rusk in the sections examined found the brain and meninges normal. There was marked chronic interstitial nephritis, edema of the lungs, chronic valvular endocarditis and atrophy at the base of the tongue. The anatomical cause of death was chronic nephritis and edema of the lungs.

As the puncture was made for diagnostic purposes and seemingly corroborated the suspicion of brain syphilis, the inability to demonstrate changes in the meninges or brain substance, leads us to a consideration of the theories regarding the causation and pathology of lymphocytosis. I am not ignoring the fact that in uremia lymphocytosis has been found and attributed to pressure, but the increase in cells is usually extremely discreet, while in this case it was enormous. In a case of chronic hydrocephalus of long standing two punctures were made. The first showing an average of 8 cells, the second of 15. In such a case the observations of Quincke go to show the presence of demonstrable chronic ependymitis. Thus pressure is not the only producing factor. I am also aware that infiltrative processes in the meninges are extremely easy

to overlook, as a prolonged search in one of Dr. Nissl's cases revealed evidences of such which at first was not apparent, nevertheless, a reasonable search was made and we must base our conclusions upon the facts as they stand.

The French authors agree that lymphocytosis is to be taken as a manifestation of meningeal irritation. By some, it is also stated that, "Every meningeal irritation produces a lymphocytosis, and that by this symptom many otherwise clinically unrecognizable meningeal changes can be diagnosed." Nissl is of the opinion that such statements are merely proof of our complete ignorance of the subject. Quincke, however, believes that such cases as mentioned form the transition into actual serous meningitis, which appears with pressure symptoms. Concerning the cases with symptoms of meningeal irritation without anatomical evidences of inflammatory changes, he regards these symptoms as resulting from a chemical toxin acting directly upon the central nervous system.

Merzbacher has contributed some valuable observations upon the question of meningeal changes. He investigated the spinal fluid in 26 patients who were beyond doubt syphilitic, but were suffering from psychoses upon a different basis. The history of infection with the manifestations is given in detail with the mental diagnosis. In none of the cases were there symptoms from the central nervous system which were not demonstrated as resulting from disease processes which are not productive of lymphocytosis. There were four such cases, in three it was evident from the autopsy, and in one from clinical examination the exact nature of the lesion.

He claimed that the French theories were entirely too hypothetical, and his conclusions were as follows: He found 23 of the cases gave an absolutely positive result; in two it was doubtful, in one only was it negative; thus in 89 per cent. an increase of lymphocytes was demonstrated. As a rule the increase was far behind that observed in paresis, and very noticeably albumin was present, in only one case.

The autopsy examinations were as follows: One case showed marked arterial sclerotic changes in the brain. In one a large internal hemorrhage was present, while in the third a deep-seated cerebral carcinoma was found. The meninges were carefully examined and were found to be perfectly nor-

mal. The following is his conclusion: "In nearly all cases syphilitic infection led to an increase in the cellular elements in the spinal fluid, and this occurred when no clinical signs of changes in the central nervous system or pathologically in the brain coverings could be demonstrated." Therefore, as a general chemical toxin syphilis is capable of producing a lymphocytosis.

Nissl endeavors to throw some light upon this part of the subject in the following manner: "One of the most important points in the whole question appears to me to be the knowledge of the exudative meningeal processes on the one side, and the hyperplastic conditions without cell exudate on the other, and the relation these conditions bear to the cerebrospinal fluid. In circumscribed meningitis when the arachnoid remains intact no changes in the spinal fluid occur."

Again, "In the hyperplastic form of meningitis with no cellular exudate, no changes in the spinal fluid was observed. Where a lymphocytosis has been present, prolonged search may reveal changes of an exudative nature." He cites a case which gave the general picture of acute nervous disease of a peculiar type—the diagnosis of tuberculous meningitis being made but was not clear. At autopsy no changes of an inflammatory nature were at first observed and the lymphocytosis could not be accounted for. Later, marked acute cortical cell changes were found microscopically. While after prolonged search the soft coverings of the brain showed exudative changes. On the other hand, in senile dementia, where the pure hyperplastic type of meningeal changes are observed, the spinal fluid is negative. Again, of no less importance it appears to me to separate the inflammatory gummatous exudative type of meningeal changes, and other circumscribed tumor formations from the non-inflammatory form of brain syphilis. In the latter type we have a syphilitic disease of the vessels at the base with results of a nature such as softening and hemorrhage. At the same time meningeal thickening of a hyperplastic type may occur.

According to Nissl's investigations, the inflammatory form of brain syphilis shows the same cytologic and chemical findings as most of the paralytics. Those singular cases of non-inflammatory brain syphilis, however, show only a slight

cellular increase and often no albumin. Whether this is correct, only a large experience with autopsy material will show.

The relatively few elements then speak more for the non-inflammatory form of brain syphilis, but will not enable us to differentiate those cases of general paralysis which show only a slight cellular increase. The usual presence of large albumin content in general paralysis, however, is of value. Thus we cannot be too cautious in interpreting small augmentations of cells, and in all cases the albumin content should be recorded. Merzbacher also, is of the opinion that by such means a quantitative differentiation is possible between brain syphilis and general paralysis. Probably a further differential study of the cells themselves may offer some help, and as yet our technique does not permit us to do this very accurately. (A persistent negative finding then is of more value for diagnostic purposes than a suggestive or large increase.)

The following case illustrates the difficulty in differentiating between cerebral syphilis and general paralysis. The patient had one attack of an alcoholic psychosis in 1904, and at that time presented the physical symptoms usually associated with acute alcoholism, besides these she suffered greatly from headache and vertigo and her pupils were small, unequal and irregular. An error in refraction was supposed to be the cause of her pupillary signs. The patient gave evidences also of a fairly recent syphilitic infection. She left the hospital in January, 1905, as alcoholic psychosis, recovered.

She was readmitted in June, 1905, showing poor memory for recent events with marked tendency to fabrication, and marked depression. During the following four months she developed progressively symptoms of involvement of the seventh, the eighth and finally the third nerve on the right side; her headache was most intense and she suffered greatly from vertigo. Besides the physical symptoms mentioned, were exaggerated knee-jerks, and slight tremor of the hands. The patient improved under iodide of potassium, and paralyses disappeared entirely save for a right internal ophthalmoplegia.

In June, when the case was first examined, and before the onset of the cranial nerve symptoms, a spinal puncture showed a positive result, and the case was regarded as one of very probable paresis. However, there was no albumin increase in

the fluid. The further course of the case makes the diagnosis of cerebral syphilis more probable. It is to be pointed out that while the lymphocytosis gave evidence of the existing syphilitic disease, and coupled with the headaches and vertigo was an indication of the developing cerebral involvement, it did not enable us to exclude the possibility of paresis. Perhaps the absence of albumin may serve as a differential point. Of this, however, we are not sure.

We have had under observation also a remarkable case of a very unusual type of cerebral syphilis, where the onset with foolish purchases, memory defect and convulsive attacks, made the diagnosis of paresis extremely plausible. The diagnosis of epileptic insanity was also considered for a time, because of the rather typical convulsive symptoms. Early in the development of the case was pronounced headache, while physical examination showed exaggerated knee jerks and unequal pupils; mentally were slight memory defect, uncertain orientation and slight speech defect, with a persecutory trend of ideas; spinal lymphocytosis was present, and the diagnosis of paresis was almost certain.

The patient developed, in the course of a few months, marked aphasia and evidences of periostitis above the left zygoma, and evidences of irritation of the motor area on the left side; double optic neuritis was present. (The details of this case will be published later and I give only the important points from the standpoint of the lumbar puncture.)

On entrance into the hospital, the first puncture gave only a slight increase in cellular elements (5-6). A month later, with the development of the aphasic symptoms and symptoms of increased cranial tension, a second puncture showed a marked lymphocytosis (20-30). The patient was then placed on active specific treatment and in a period of three weeks her improvement was most remarkable. A puncture some four weeks after commencing treatment showed that the lymphocytosis had decreased (6 cells). The patient has since been discharged from the hospital in a great measure perfectly recovered. There is still a slight speech defect which may be residual from her lesion in Broca's center, and a slight reddening of the left optic disc. There was practically no albumin increase in this case, as in the previously mentioned patient. It

is to be pointed out that unless this sign proves to be a constant one, we have no way of differentiating from the spinal fluid between paresis and cerebral syphilis. There seems to be, however, a not quite as great an increase of cellular elements in syphilis as in paresis. The diminution in cellular content in this case, following specific treatment, is noteworthy.

There is another class of difficult cases in which lumbar puncture is of diagnostic assistance. I refer to those cases of syphilis in early life with arrested development. In one case the diagnosis of acute mania excitement in an imbecile had been made. The further course of the disease, the development of the physical signs, with rapid, tremendous mental deterioration, with a spinal lymphocytosis suggests strongly the diagnosis of juvenile paresis.

In another instance, where the diagnosis of congenital inferiority was suggested, the presence of optic atrophy, loss of hearing in the left ear, exaggerated knee-jerks, double ankle clonus, right Babinski, tremor of the tongue and hands, with defective speech, together with a spinal lymphocytosis, showed that the underlying cause of the patient's condition was syphilis of the nervous system.

I have yet to mention the results of spinal puncture in several cases, with undoubted evidences of past syphilitic infection. In two cases of manic depressive insanity we obtained an absolutely clear history of syphilitic infection. One seven years previously, and the other three years.

There were no symptoms of the involvement of the central nervous system and the spinal puncture was negative in two separate examinations.

In one case of psychosis of a senile type with an indefinite hemiparesis of the face, sluggish pupils, coarse tremor of the hands, with a certain amount of ataxia and speech defect, there was absolute evidence of an old syphilitic infection. The spinal puncture was twice negative. The condition then was

*This patient has since returned to hospital because of foolish conduct. Physical signs remained as when discharged, but patient now shows very distinct grandiose ideas and memory defect. Lumbar puncture shows a large increase of cells and albumin. Case is regarded as belonging to a rare combination of tertiary syphilitic brain lesions, along with paretic changes.

of arteriosclerotic origin with syphilis merely as a passive agent.

Still another patient presented a depression of a long duration with a certain amount of deterioration—tremor of the hands and face and exaggerated knee jerks. There was an old syphilitic necrosis of the frontal bone and a specific ozena. The lumbar puncture was negative. The case has been classified as an alcoholic psychosis.

In another case of alcoholic history, there was a clear syphilitic history of some 6 years previous. The patient presented exaggerated knee jerks, tremor of the hands and tongue, with marked emotional deterioration. There was a persecutory trend, peculiar elation and active hallucinations. The lumbar puncture was twice negative.

I mention these cases because of the fact that although there had been undoubted syphilitic infection, yet lymphocytosis was absent, and this demonstrates an exception to Merzbacher's conclusions and shows that there exist patients who have suffered syphilitic lesions of tissues other than the nervous system, and still give normal reaction in the spinal fluid. This is a point of some importance, since in the cases mentioned a positive result would certainly have led us into the conclusion that the central nervous system was syphilitically affected. An autopsy has been held on one of these cases and showed no involvement of a specific nature in the central nervous system. A negative finding then in a case with a syphilitic history is almost certain evidence that there is no involvement of the central nervous tissue, and in cases where brain syphilis is clinically to be diagnosed, a persistent negative lumbar puncture should make us look for some other etiologic factor.

Another case which has some bearing upon the question of syphilitic disease of the non-nervous tissues is the following:

M. W., a woman of thirty-four, presented a psychosis commencing with an inability to hold positions formerly occupied satisfactorily, and four months ago had an attack of alcoholic delirium, following which she showed a persistent indifference, loss of memory and grasp. On entrance she showed marked irritability of the emotional tone, imperfect orientation and considerable paucity of thought. Physically there were exaggerated knee jerks, fine tremor of the tongue and hands,

slight jaw jerk; pupils irregular but active; old and recent typical specific scars on the legs, several of which had become chronically ulcerated.

There was a marked history of alcoholism; the ulcerations began seven years ago, and six years ago the patient had a necrosis of the middle turbinated bones. The diagnosis was obscure and the symptoms in favor of paresis not conclusive because of the marked alcoholic history. The presence of an old tertiary lesion also was confusing. The lumbar puncture was entirely positive both to albumin, which was very marked and cellular content. The mental deterioration and the physical signs with the results mentioned make the diagnosis of paresis undoubted. It has not occurred in my experience to obtain such a marked cellular and albumin increase in tertiary skin lesions, and in this case with the presence of physical signs I believe that we may interpret the findings as showing an active deteriorating process in the nervous system.

In connection with syphilis, I would mention here, three cases of hemiplegia. In one case there was acute apoplectic symptoms, and on account of a severe chronic endocarditis, embolism came into question. There was a suspicious history of syphilis and considerable arteriosclerosis. A positive lymphocytosis in this case points to syphilitic endarteritis. In two other cases the hemiplegia was of some years' duration and had improved under potassium iodide. Both of these cases gave positive results, these facts are of importance in enabling us to differentiate these cases from certain purely senile changes which are not directly due to syphilis. In senile dementia and changes due to non-specific arterial lesions the spinal fluid is negative. We have had several cases where this point was of value. In one case the patient was about fifty years old, of markedly alcoholic habits and following the commencement of epileptiform attacks she developed the mental deterioration with a motor aphasia. The duration of the condition at entrance was two years, her psychosis commencing about two months after the onset of the convulsion. There were no very definite neurological symptoms. It was stated, however, that she had before entrance weakness of the right side of the body; the convulsions were quite general; pupils reacted fairly well; knee jerks equally exaggerated; slight tremor

of the hands and slight Romberg. The diagnosis was to be made between pachymeningitis interna, senile sclerotic changes, paresis and a syphilitic arterial condition. The presence of the recurrent convulsions and the aphasic state with alcoholism as a factor were very much in favor of pachymeningitis. The spinal puncture showed a reaction of from 20 to 25 cells. The diagnosis remains in doubt, but certainly we can be assured of a specific arterial change. Paresis, however, cannot be absolutely ruled out, as an autopsy on such a case recently which was regarded as a senile psychosis revealed a paretic brain. (G. Y. Rusk.) In reference also to the hemiplegic episodes, lumbar puncture is of value in differentiating those cases of deterioration following apoplexy and the hemiplegic attacks which occur during the course of paresis.

A case recently entered the hospital where the first symptom of the paretic process presented two years ago with a right hemiplegia which improved greatly in two months. It was a question whether or not her psychosis could be the result of an apoplexy. Physically, her age was forty, good nutrition, weakness in the right arm and leg, tongue protruded toward right; knee jerks much exaggerated; pupils unequal, react slowly to light, right pupil larger than left, slight Romberg, tremor of the facial muscles; in walking drags her right leg. No Babinski or ankle clonus.

The mental deterioration had not become advanced enough to necessitate her commitment until a year and a half after the stroke, and was not very general; there was some loss in grasp and knowledge; the patient was dull and the memory defective for recent events; retention and orientation imperfect. There were no delusions or hallucinations. Such a picture is not uncommon in organic brain disease following hemorrhage or softening. The lumbar puncture showed a quite marked positive result, 25 to 50 cells in a field with a marked opalescence. With the presence of speech defect and hand writing defect our diagnosis of paresis is clear. We cannot rule out the arterial changes due to an active syphilitic process, especially, because of the acute onset, but the slow progress of the disease, and particularly the speech and hand writing defect, with the marked albumin content, are points

against a pseudo-paresis from tertiary syphilitic changes; there is nothing definite in the history of the patient regarding syphilis.

Another instance which shows the importance of puncture in these cases is the following. A woman of some seventy years, who had previously been normal, developed delusions of grandeur, became silly and childish in her talk and showed poor memory and orientation. Alcoholism was denied; patient had had four miscarriages. Physical examination showed a woman with marked symptoms of senile change; a fine tremor of the hands and facial muscles; spastic irregularity of the pupils and absent knee jerks; the knee joints were enlarged and there was considerable exostosis on the inner condyle of both femurs, on movement there was considerable grating and the cartilages were relaxed. There was practically no fluid accumulation and there was some external bowing.

The diagnosis of senile dementia was made as the most probable condition on account of the physical signs, however, a puncture was suggested. We were surprised to find a tremendous cellular reaction (100 to 150) and a marked opalescence. We are forced to conclude that the most certain diagnosis is that of tabes of long duration with final dementia with the presence of Charcot joints.

In this next case the question of tabo-paresis, tuberculosis of the spine and tumor of the spinal cord came into question.

An Italian woman of thirty-three presented an indefinite

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*This patient has since come to the autopsy table and shows that our conclusions were justified, at least, as to the nature of the process.

Sections examined from anterior and posterior central, first frontal and angular regions and also from cerebellum show pial and perivascular infiltration with lymphocytes and plasma cells and an occasional mast cell is observed. The cerebellar pia is similar, but with infiltration of less degree.

All the areas show a moderate increase of glia overgrowth, some few of the cells presenting pigmentation; the nerve cells themselves are not excessively pigmented, but there is great pigment accumulation in the perivascular sheaths and much evidence or phagocytosis in relation to the pigment, in which process, however, the plasma cells appear to take no part. There is general thickening of the walls of the arterioles with hyaline degeneration and occasionally small foci of rarification of brain substance with the presence of compound granular corpuscles. In a few areas in the frontal region there is an excessive number of vessels present.

Rod cells are to be seen in frontal and angular regions, especially.

Diagnosis—General paralysis with arterio-sclerosis of terminal vessels.

NOTE ON EXAMINATION OF CORTEX, AUTOPSY 279. G. Y. RUSK, PATHOLOGIST

mental disturbance with a peculiar contracture of the adductors of the thigh, fine tremor of the hands and absent knee-jerks. Insanity was present in the paternal side of the house, form unknown. Husband gives suspicious history of syphilis having had a sore eight years ago, no secondaries. Patient has had one miscarriage and four normal children. She was extremely illiterate, had earned a living as a seamstress until five yers ago, when she was married. Four years ago the patient bruised her left shin and it has been sore ever since. She refused to use any medicine and during the past year has failed in general health. Four months ago began to complain of pain and stiffness in thighs and had difficulty in walking. A month after this trouble commenced, she had one epileptiform convulsion; she took to the bed, became extremely talkative, irritable, irrational, thought she was pregnant because of a queer feeling in the abdomen, and was sent to the hospital.

P. S. C. A small, very thin Italian woman, showing peculiar spastic gait, holding thighs stiffly, bringing feet down flat, extension of thighs good, abduction limited and flexion on abdomen can be accomplished only by much pressure and then only about 120 degrees. On the left leg at middle third was a large partly healed ulcer which consisted in three adjoining indolent surfaces with thickened edges and much connective tissue infiltration. The outlines were irregular and while the lesion extended transversely across the middle third of the tibia the ulcerated surface was quite small. The scar tissue was of a bluish imperfectly formed material. At the lower third of the thigh was a small (2 x 1 cm.) puckered scar just at the inner margin of the quadriceps. The knee jerks were absent; Achilles present. The hip joints were not tender to direct insult: the adductors stood out stiff and taut; there was nothing abnormal in the pelvis, the lower abdomen was prominent and there was a little tenderness over the right iliac fossa. Examination of the chest showed a little tenderness around the right base and a few persistent moist rales. The muscles of the calves were much wasted, but perfectly equal.

Mentally the patient was garrulous, irritable, and peevish, again smiling and good-natured. Her memory was poor; grasp indifferent: orientation defective: no delusions or hallucinations.

The diagnosis of a double psoas abscess of tuberculous origin was considered. The leg condition and presence of the healed sinus were very suggestive. The absence of temperature changes, the inability to demonstrate any changes in the spinal column, and the rarity of a perfectly symmetrical condition from such a process make this diagnosis very improbable.

The symptoms could be explained on the basis of a tumor of the spinal cord, but the absence of sensory symptoms and the fact that the knee jerks were absent were against this condition. Alcoholic neuritis was another possibility, but alcohol was absolutely denied. The diagnosis of the tabetic type of paresis was made because of the absence of the knee jerks, the history of possible syphilis in the husband, the evident syphilitic infection in the patient, the occurrence of a convulsive attack, tremor of the hands, static ataxia and mental deterioration. The pupils, however, were normal; the ulceration appeared to be more tuberculous in nature than specific, the syphilitic infection in the husband was not established, and the spasticity of the thighs occurring within four months from the onset of the disease were facts which could not be entirely explained on the basis of a tabetic process. The patient has not had a repetition of the convulsive attack. The diagnosis, however, could not be absolutely ruled out. The lumbar puncture was now performed and the spinal fluid was perfectly normal. With this additional evidence then we are certainly justified in ruling out the diagnosis of paresis. The exact status of the case still remains somewhat in doubt. Patient has been under large doses of iodide of potassium for a long time without much improvement.

Closely connected with these cases are the epileptic states. In several cases where a positive result was found, Nissl was able to demonstrate at autopsy, non-inflammatory brain syphilis. The findings in epileptic cases are usually negative, and this point is of great value since we meet many cases with epileptiform attacks which render the diagnosis of paresis extremely probable. In three cases of epileptic psychosis we found two negative and one positive. In the latter case there were exaggerated knee jerks, weakness upon the right side of the face, and a history of syphilitic infection. The finding in this case then is easily explained, and points to a syphilitic

basis for the epileptic attacks. In several other doubtful cases the question of epilepsy and general paralysis was difficult to decide. A negative finding was of great value, in two punctures in a patient of twenty-three, who had suffered convulsions for two years and showed besides a memory defect, slight elation and unequal pupils. Similarly in a woman of forty, who presented deterioration, with diminished cutaneous sensibility, exaggerated knee jerks, general tremulousness, but normal pupils, with a history of suspicious miscarriages (also scars on back), a negative finding which was twice obtained, has given us the needed assurance that we were not dealing with paresis. An anamnesis recently obtained has developed facts which make the diagnosis of dementia præcox the most probable one.

Now to the consideration of the results of spinal puncture in paresis. We punctured 30 clinically clear cases, and upon 22 of these the punctures have been repeated several times. In every instance positive results were obtained. The albumin content showed a marked opalescence in every examination, and the highest amount was 5.2 by Nissl's tube. Microscopically the fields in many instances were literally covered with cells, many of which we were unable to classify. Differential counts were extremely unsatisfactory. The pressure in all cases was quite marked, the fluid literally spurting from the canal.

The Argyll-Robertson pupil was absent three times, once in a tabo-paretic and twice in the cerebral type. Syphilis could be proven absolutely in only nine cases, in seven it was suspicious, while in fourteen the evidence was entirely negative; practically then 50 per cent. were syphilitic by the records, while the results of the spinal puncture point to syphilis in all.

Regarding this point we must naturally think of the traumatic origin of certain cases. Two cases gave a history of trauma, in one, however, it was found that the psychosis developed before the fall; in the other the patient did not develop symptoms until one year after the accident. We cannot therefore conclude anything from these cases. But we should remember that trauma to the head or spine, if severe enough, may produce a cellular increase in the spinal fluid.

Lymphocytosis is one of the earliest and most constant signs of paresis. It appears before the memory, eye and ataxic symptoms (Jaffroy, Marie and Duflos). I have collected the findings in 500 cases containing the results of 14 different observers. Of these 500 punctures only 18 give negative results. Many of the cases (the latter) were punctured once only, in two (Dana) the fluid was transferred from the test tube to the centrifuge tube, and in three other cases although clinically they were considered as general paralysis, the autopsy showed them to be chronic alcoholic. These five cases then may be disregarded. The percentage then of negative results which one might expect from our present technique is 2.6. If we count the doubtful cases it is 3.5. Considering the individual equation as regards technique I believe that this percentage is a remarkably low one.

The importance of repeated puncture is emphasized by Achard, who had the following experience in a classical case of paresis. The first two punctures were negative, a third showed a slight cellular increase, and finally a fourth showed a large number of lymphocytes. An interval of ten days was allowed between punctures. Nissl had the following results in a case of similar nature. In an otherwise clear case of paresis a negative lymphocytosis was observed, but a marked opalescence was present. Twenty days later marked lymphocytosis and opalescence were found. He points out then that in such cases the albumin content should be recorded. Farrar makes the explanation of the variation in the examination thus: "Spinal lymphocytosis is simply the expression of a subacute or chronic cerebro-spinal periarteritis and pia-arachnitis, and its intensity doubtless stands in some relation to the degree of infiltration of the meninges and of the adventitial sheaths of the blood vessels in the central tissue itself. This adventitial infiltration in paresis consisting of lymphocytes and plasma cells may show the widest variation, at times subsiding or almost disappearing. In the same manner the elements in the spinal fluid may be present at times in greatly varying numbers; on occasion even practically disappearing." There is much hope that as the different types of cells which occur in the spinal fluid must have a different origin, with improvement of technique we will be able from the variations in the types of cells to ascer-

tain something of a prognostic value. At the present time we cannot here enter into a discussion of this point.

This brings us to the study of results in alcoholism. Besides syphilis and its various clinical pictures there is no more confusing problem for diagnosis from general paralysis and tabes than the various manifestations of alcohol upon the nervous system. We must recognize that there are many different types of alcoholic psychosis which closely simulate the protean pictures presented by paresis. Psychiatry holds no more complex problem even for its best observers than the differentiation between the paretic and alcoholic psychosis.

When we consider then that lymphocytosis has been shown to present an early and constant sign of paresis and tabes we realize that it should be of the greatest value in differentiating these diseases from alcoholism, if it can be proven that this peculiar sign does not occur in the latter group. I will now give the data on this point.

Nissl collected from the literature and his own cases the results of 30 observations. Twenty-three of these gave negative results, and seven were positive. Of these seven cases autopsy has shown in two of them that general paralysis was present and in three the clinical course since the puncture has been that of paresis. In the remaining two cases the autopsy showed the presence of chronic alcoholic conditions. The positive results in these two cases Nissl states must be traced back to an old syphilitic infection, as there were no physical signs of involvement of the nervous system. Recently Rehm supports this latter view in an examination of nine clearly alcoholic cases, where one only showed a positive result. In this case previous syphilis was proven. No symptoms of involvement of the nervous system were present. Joffroy has shown that acute alcoholic disturbances give negative results, in one such case with a positive result general paralysis afterwards developed. Dufour reports one such similar experience, but makes the broad statement with Dupré that in an alcoholic if the meninges are affected a positive result may be found. The weight of opinion, however, points in these cases to previous syphilitic infection. Regarding the meningeal complications the opinion of several observers has been quoted. Very few cases of Korsakoff's psychosis have been examined. In 12

cases collected from the literature, including four of our own, negative results have been found in all. E. Meyer, in two cases where the Argyll-Robertson pupils were present, lays great stress upon the negative puncture as a differential point between these cases and tabes. The further course of his cases have confirmed the assumption.

During the past 12 months 16 cases of clinically clear alcoholic psychosis have been punctured. Fourteen gave negative results, in two lymphocytosis was positive, but there was no albumin increase. On entrance one of these cases showed an acute hallucinosis with rapid improvement and perfect recovery in four months. For a long time we were in doubt as to the cause of the lymphocytosis. Since the patient's improvement she gives the fact that in 1899 her husband had a disease which was undoubtedly syphilis. At that time sexual intercourse was prohibited. Since then she has had three miscarriages.

The second case of alcoholism with a positive finding, showed on admission moderate elation, with expressions of the possession of great strength, with memory defect and exaggerated knee jerks. At times she is quite elated and again complains of intense headaches with vertigo and shows confused orientation. Thus the diagnosis of paresis is very probable.

In the following re-admissions the results of lumbar puncture have been of decided value. A patient developed a psychosis in January, 1900, was of marked alcoholic habit, had delusions of grandeur, and physically showed tremor of tongue, facial muscles and hands, defective speech, increased knee jerks and variable pupillary reflexes. She was discharged ten months after admission as paresis, improved. She remained in civil life for six years and entered the hospital in January, 1906. She expressed exactly the same ideas that dominated her six years before, and exhibited practically the same physical signs. There was, however, no deterioration in the memory, and she had good insight and was well oriented. Her hand writing was unsteady and there was slight speech defect. She had been drinking heavily. An undoubted specific papular rash of a mild type covered her entire body, and there were condylomata about the vulva. The lumbar puncture was abso-

lutely negative and the question of paresis was certainly ruled out. The puncture has been twice performed since the active development of the syphilitic infection, and a slight lymphocytosis has developed, but there has been no albumin increase. The remarkable coincidence of the early secondary manifestations, just at the time of entrance, together with the fact that in the three punctures we have had an opportunity to observe a slight increase in the cellular elements due to this infection, throws considerable light upon an otherwise doubtful case.

Similarly the diagnosis of dementia paralytica was made in an alcoholic patient who was an inmate of M. S. H. eight years ago for 16 months. There were marked physical signs of alcoholism and somewhat sluggish pupils. On re-admission, in November, 1905, the patient showed fair memory save for several periods during the past few years. Occasionally she was elated and happy and emotionally variable, but there was a strong persecutory trend extending over the entire period of her psychosis. The physical signs, however, were very suspicious of paresis, her pupils being unequal and sluggish, there was slight speech defect and increased knee jerks, with some tremor of the hands. The spinal fluid has been negative on three different punctures.

The following case is most striking because of the slow progress of the disease and illustrates the similarity which exists between chronic alcoholism and paresis. A patient, D. D., a woman of some 37 years was admitted into the hospital in 1902, where the diagnosis of dementia paralytica was made. She was markedly alcoholic; there were some memory defect, apathy, confusion of personal identity, while physically there were exaggerated knee jerks, unequal and dilated pupils with static ataxia; she had two convulsive attacks and developed some smoothness of the left side of the face. She was discharged after five months, much improved mentally. She suffered greatly from a terrifying hallucinosis from right ear only. On re-admission into hospital October, 1905, there was a marked hallucinosis of a depressing character, with considerable memory defect, with poor grasp and retention, but good orientation and preserved identity. Physically, the eyes were normal, there was no speech defect, the knee jerks were increased, there was only a slight tremor of the tongue and no

evidences of paralysis. The diagnosis was now made of chronic alcoholic psychosis. Recently the case has again come up for discussion and the patient now exhibits considerable difficulty of an aphasic nature. Her enunciation is clear, but she fails to name objects and frequently shows perseveration. She shows mentally a mild deterioration, still the diagnosis of alcoholic psychosis was upheld, but the case was deferred for lumbar puncture. The spinal fluid showed an enormous reaction, the cellular increase being 25 to 50, and there was marked opalescence on testing for albumin; with this evidence we must admit the correctness of the diagnosis in 1902. (Very recently the patient has had convulsive attacks.)

Another illustration of the diagnostic difficulties is shown in a case with a psychosis of a persecutory delusional content, with physical signs suggestive of paresis, who entered the hospital five years ago. She was discharged four months after admission, having good insight, with no sign of a delusional content and no deterioration. She has taken good care of herself during the past five years; during the latter part of her fifth year, her memory began to fail, she showed emotional instability, and on examination, at entrance, there was fine tremor of the hands and exaggerated knee jerks, but the pupils were normal. The spinal puncture, twice performed, has shown a positive lymphocytosis and a marked albumin increase. The extremely slow development of this case, with the very gradual deterioration, presented a somewhat unusual type for diagnosis, and the lymphocytosis was of considerable value.

We have the support of a recent autopsy on a case which shows very typically the class in which the lumbar puncture is of greatest value. This case entered the hospital with a markedly alcoholic history, and was much confused in orientation and grasp. There was a history of two attacks in which she suffered loss of consciousness for a few minutes, without paralytic sequelæ. These had occurred during the past six months. There was mild elation, marked memory defect and defect in judgment and comprehension. Physically there was marked speech defect, tremor of the hands and face and unequal knee jerks, right greater than left. During the early part of the her stay in the hospital the diagnosis of paresis was

very strongly suspected. But for the negative spinal fluid, the diagnosis would have rested as such. The patient had two convulsive seizures, one which was very transient and was accompanied by twitchings of the right side of the body and lasted only a few moments. Following this attack her speech became markedly affected, yet there were no paralytic symptoms in the extremities. A few days ago, while the physical examination for summary on this case was being made, and the discussion of this work was being carried on, the patient went into a convulsion and exhibited symptoms of paralysis on the left side of the body and in a few moments died. At autopsy, grossly, there were absolutely no evidences of paresis, and on section of the brain a large hemorrhage was disclosed involving the region of the internal capsule on the right side and breaking into the lateral ventricle. On the left side, in a symmetrical area, there was a small old focus of softening involving the external capsule and claustrum, together with a small acute hemorrhage at the level of the anterior tip of the caudate nucleus. The findings in this case give us much reassurance as to the accuracy of our assumptions in a very confusing clinical picture.

I would mention here also in support of the accuracy of the diagnostic inferences to be made from the results from lumbar puncture, a second case in which we have recently performed an autopsy. The clinical picture was extremely doubtful. The mental symptoms were very meagre; the very slight memory defect, emotional instability, with slight elation, were not at all characteristic. To further confuse the case, was the presence of chronic pulmonary tuberculosis. There was a delusional content of a persecutory trend, and at staff meeting the diagnosis of a paranoic condition with infective and exhaustive psychosis was suggested. Physically there was slight exaggeration of the right knee jerk, a rather uncertain tremor of the hands and face, and occasionally she failed on test phrases. The diagnosis of this case remained in doubt for some time. The deterioration was very slight, her delusions were poorly developed and her statements were extremely variable; the memory defect was difficult to demonstrate. Finally a lumbar puncture was performed and a positive lypmphocytosis was obtained. This finding was practically

the most significant symptom in the examination of the patient, and the diagnosis hinged upon this factor alone. Even at autopsy, when the brain was removed and showed no symptoms of paresis in the gross, there were members of the staff who still did not believe that the case was a paretic. Microscopical examination, however, showed areas of typical degeneration. These areas, however, were quite small, but were characteristic. (I am indebted to Dr. Karpas, of the M. S. H., for the notes in this case.)

In the differential diagnosis of Korsakoff's psychosis the lumbar puncture has been of value. The absence of the knee jerks, Romberg sign, slight nystagmus, memory defect, with a certain amount of deterioration, in these cases is very confusing; besides these symptoms, in one case there was a history of convulsive attacks, and a negative finding was of considerable help. Sluggish pupillary reactions in another case brought up the question of tabes, and lumbar puncture, twice performed, giving negative results, was of great assistance. In several other cases the further course of the disease has shown that our assumption based on a lumbar puncture, was correct. In one case which was complicated by peculiar aphasic conditions, besides a suspicious history of syphilis, the negative punctures were of great help.

The following case resembles several readmissions already described; it is another example of the puzzling conditions for diagnosis produced by chronic alcoholism. The patient suffered a neuritic process in 1899, the details of which are not known. She was brought to this hospital in 1901, remaining 18 months, recommitted in 1906. At her first commitment she was in the hospital for three months. The records were lost. In 1901 the diagnosis of general paralysis was made. She presented memory defect, marked tremors, absent knee jerks, irregular and sluggish pupils, defective orientation, had delusions of persecution and had convulsive seizures on several occasions. She was discharged as having greatly improved. She remained out of the hospital for three years. At the present writing the patient has reached the age of sixty. Physically she presents marked tremors of the face and tongue, little of the hands; slight Romberg and absent knee jerks. The pupils are a little irregular and react within a

very narrow range; she shows slight nystagmus. Mentally there is some memory defect for the period of her first and part of her second attack, but not the general defect that one finds in paresis. Orientation was correct; emotionally there is a little elation. There is some tendency to retrospective falsification and fabrication. The speech is a little thick, but not characteristic. No writing defect. How to classify this case with the previous diagnosis, the history of the convulsive attacks, and the mild degree of dementia present, was a very difficult question. There were also certain scars about her body which tended somewhat to support the diagnosis of G. P. on the basis of specific infection. In spite of the patient's age, the slow progress of the disease and the evident etiological factor, alcohol, paresis could not be ruled out on the case as it stood. The lumbar puncture has twice given negative results both to cellular and albumin tests. With this data we feel sure that the paient's condition is due to alcohol, but if it were not for the negative puncture I do not think we would be justified in assuming this.

(To be continued.)

REPORT OF A CASE OF HYSTERICAL MUTISM.*

By John K. Mitchell, M.D.,

of Philadelphia.

The following case of hysterical mutism, a disorder of great rarity, is reported at length as a matter of record and with the hope of eliciting histories of other similar instances.

G. C., aged 22 years, Kernersville, N. C., locomotive fireman, was brought to me by Dr. Ashworth on June 5, 1905, with complete loss of speech. The family history is without any instance of positive nervous disease, although it is said by the patient's physician that they are all neurotic in temperament.

On August 24, 1904, while on top of a freight car, the patient ran against a live trolley wire which struck him on the root of the nose, just below the right eyebrow. The "current passed through him," according to his account. He was knocked up 4 or 5 feet and fell on the car roof unconscious. He was taken to hospital, where he stayed 5 days, being, according to his account, unconscious the first 24 hours. When he awoke he could not speak; he could swallow, moved his tongue and mouth well; had much headache—frontal and over left side. The eyesight was not affected. The right eyelid was somewhat burned and was, of course, sore. (Dr. Bahnson, Salem, N. C., saw patient 10 minutes after accident and says he was never unconscious, only dull and dazed, and that this condition lasted 6-8 hours, not 24 as stated, and that the wound was the merest brush-burn of a trifling character.) He had the above mentioned headache almost continually from the date of the injury: it was less severe at times, seemingly changing without cause. He suffered from roaring and buzzing in his left ear. Occasionally there were sharp shooting pains through the arms, trunk and legs, sometimes very severe, and very constant from the time of the accident up to the admission to the hospital in Philadelphia. Although he had perfect memory and recognition of words and wanted to speak, it seemed impossible for him to send the necessary motor impulse. In his own phrase he "wanted to speak, but didn't know how to go about it." His mother writes: "He spoke three or four times (words) last fall (1904) in a clear, natural voice, only he did not know it, though awake. The last time was in November, and his sister occupying a room below

*Read by title at the meeting of the American Neurological Association, June 4 and 5, 1906.

his heard him talking in his sleep, loud and clear enough to waken her."

The patient was admitted to the Infirmary for Nervous Diseases June 6, 1905. Examination: Strong, healthy looking, well-developed muscular young man. Heart and lungs negative, sensation normal everywhere, externally; audition perfect: thinks clearly; writes a definite and simple statement of his feelings: C. says he cannot balance well when walking in the dark. Appetite and bowels normal; sleep poor, 3-4 hours at night; dreams much; he has headache almost constantly: no nausea or vomiting. He has lost 25 pounds in weight in the past nine or ten months. There is no cough or expectoration.

Dr. Langdon examined his eyes June 24, 1905, and reported them normal in every way except for complete reversal of the red and green color-fields.

Kjs. active; no ankle clonus or Babinski; plantar reflex normal; sensation everywhere preserved. Other reflexes everywhere good. No disturbance of sphincters. Larynx mobile, breathing easy. Smell and taste normal. Dyn. R 112-L 110. The electric reactions of pharynx, neck, tongue and external laryngeal muscles are normal.

Treatment—House diet; faradic brush to sides of neck and over larynx. General massage; to walk out daily. Strychnia nitrate gr. 1/100 hypodermically into sides of neck daily, alternating sides. On July 20th it was noted that there had been no improvement. Dr. G. H. Makuen saw him early in July and treated him regularly, but without benefit. His report of the condition of the larynx and vocal cords is given in full below. Dr. Eshner for some time hypnotized him daily, attempting by suggestion to make him speak, without success. Hypnosis never could be carried beyond the stage of slight somnolence.

The patient was anesthetized June 15th and closely watched, but beyond a few explosive noises made no distinct vocal sound.

Dr. G. Hudson Makuen kindly saw the patient and reported as follows:

"The aural condition seemed to be normal, and the patient had always had good hearing.

"The septum was deviated toward the right, and on its convex side there was a spur extending well back into the nostril, and causing a considerable pressure upon the middle turbinal bone. As a result of this structural irregularity there was a slight catarrhal process in both the nasal and nasopharyngeal cavities. The spur was removed by Dr. W. J. Freeman, leaving the nostrils entirely free and improving the

general condition, but having no effect whatever upon the restoration of speech.

"The oral cavity was in good condition, the alveolar and palatal arches regular in shape and the patient had good voluntary control over the tongue for all purposes except articulation.

"The faucial tonsils were slightly hypertrophied and they contained some old contractions and adhesions, due to several severe attacks of tonsilitis during the last few years.

"The pharynx was congested and there was some anesthesia of its mucous membrane.

"The larynx appeared to be normal, except that there was a marked anesthesia of its mucous membrane. An applicator passed beyond the epiglottis was easily tolerated without any reflex muscular contraction.

"The laryngescope revealed nothing unusual except the patient's inability to hold the cords in approximation and to make them vibrate during an attempt at phonation. He could easily approximate the cords, but they immediately flew apart at the slightest attempt toward phonation. He could give the so-called 'glottic clink' that is often practiced by vocal teachers for the purpose of developing and strengthening the muscles of the larynx, and he could produce the normal vocalized cough, but it always vanished into a faint breath sound. He could not prolong the cough for any appreciable length of time.

"It was found that he had lost the power not only of phonation but of whispering as well. He could not make any attempt to speak. He did not know how to begin. He was both aphonic and mute. He lacked the mental conception of articulative movements for vocalization and whispering. In the hypnotic state, in which I appeared to have him at one time, his attempts at vocalization were equally fruitless.

"All the known methods were used to try to bring back the faculty of speech, but no vocalized laryngeal sound could be elicited, and only very limited whispered sounds and articulative movements could be developed."

Instruction under Dr. Makuen's direction was carried on daily for several weeks with the result of the acquisition of only a few short, partially whispered words, the kinesthetic memories of which were lost almost immediately. He could be taught to whisper a short monosyllabic word, but in a few seconds he seemed not to know how to try to repeat it.

Discharged September 1, 1905, unimproved. There were no changes.

C. returned and was readmitted November 4, 1905. There had been no change of function during the two months since

leaving, except that efforts at speech were clumsier than before. He could not count nor pronounce letters so well as before. He could whistle and smile, though he could not laugh; he coughed, but could not clear his throat (larynx).

On November 6th a small area on the head, approximately over the speech centre, was shaved and a strong fly-blister applied. The blister had no effect whatever upon his speech.

Especially noticeable was the fact that the reflexes of the pharynx had been either lost or greatly impaired in the two months' absence from the hospital. The condition seemed like that of a voluntary inhibition of these reflexes, although C. was blindfolded in order to prevent his peeping, which he would do if he could. Of course he knew that attempts were being made for some purpose, and on stimulation there seemed some inhibition brought into play. No disorder of sensation in the face could be discovered. An effort was made to surprise him into speech by speaking to him in a low, firm tone while asleep. He was slowly awakened, but only turned over and then raised his head. He wrote next morning, "What was wanted?" Attempts were made, too, to surprise him into an involuntary reflex sound by a sudden blow on epigastrium when passing him in the hall or ward, but to no purpose. He complained of being frequently depressed and had much headache.

In the fourth week of his second stay in the hospital, C. suddenly recovered perfect control of his speech. The struggle was witnessed by Dr. Peet, the resident physician, who has given me the following account:

On November 26th, C. had complained of severe headache with the previously mentioned recurring pains in his limbs. He went to bed and possibly ate but little supper. He slept until about 12.30 A. M., then awoke and found the pains in the legs increased, and his head still aching. The nurse stated that her attention was first attracted at 1 A. M. by his moaning and great restlessness. She had the resident notified immediately. When seen the patient was lying on his back with head thrown far back and arms and legs in constant motion; the jaw was chattering at a rate of 150-200 per minute and the quickened respirations were each marked by an expiratory groan that was caused by the larynx; there was a little frothy saliva on his lips; the eyes were closed, with the pupils fixed in moderate dilation with no reaction to candle, nor did his eyes follow the light. There was analgesia (anesthesia?) to pin prick over entire body below neck except genitals (not tried) and nipples. Over the neck and face, and especially on the lips and nose, a pin prick caused motion and

a rather purposeless attempt at defense with the arms, by moving arms and legs faster and then turning over as if to escape. Limbs were spastic; Kj increased; no clonus; the eyelid reflex was present. All the fingers were fully extended. The patient was altogether unconscious and could not be roused by pin prick, voice, the application of ice to the face or epigastrium, nor by pressure on the supraorbital nerve. Tickling the ribs roughly caused violent movements of the limbs and ill-aimed blows, but no vocal sounds. These efforts at rousing him intensified the movements and apparently his headache became worse, as he groaned more deeply and held his arms to his head, or buried his head in the pillow. To stop the chattering, the lower jaw was depressed with the hand and after some minutes the jaw grew quiet. The movements then ceased and the limbs became relaxed and flaccid; breathing was easier. His pulse remained good throughout. Any attempt to rouse him caused a return of the movements. He was constantly addressed by name and questions as to pain asked, without getting any response; once, however, a sound suspiciously like "O pshaw!" was made as he covered his forehead with his arms, and another time he seemed indistinctly to articulate "O Lord!" Pin pricks now caused no movement unless stuck into the nipple or in the lips, nose, forehead, or scratched across the neck. There was analgesia over the rest of the body. Speaking was about despaired of. However, as a last resort, he was asked if he wanted to take ether and some aromatic spirits of ammonia was brought. He was now lying quiet but still using his larynx in expiratory moans. A bit of gauze was saturated with the ammonia and held near his nose; he pulled his head away, the gauze followed; he coughed, then as if he had never ceased articulating, he said in a firm voice, "Take the damn thing away, I don't want to smell it." The gauze was still kept near, however, and he begged to have it removed and said so petulantly. It was taken away and he was spoken to; he answered readily, but not always coherently; he was evidently not fully awake. Some medicine was ordered and he was asked if he would take it. He answered, "Yes." A small dose of bromide and valerian was fetched. He raised his head well up, smelled of the valerian, drank and swallowed it, snuggled on his pillow under the bed clothing and to the query, "Are you asleep?" said, "I will be soon, good-night." He was asked, "Are you awake? Sure you are awake?" and replied, "Yes, I'm awake." "Do you know me?" "Yes." "Sure?" "Yes." "Well, good-bye." "Good-bye, doctor," he said, closed his eyes and sank on the pillow. We went out and closed the door; we heard him get out of bed and then through the tran-

som saw the light of the candle left on his table go out and heard him get back in bed. To the nurse he now complained of the light; said he would go to the roof garden, etc., that he was perfectly conscious and found he could talk and wanted to continue talking all night. He said his arms and legs hurt and he was all tired out.

Finally he went to sleep at 3.30 A. M., and awoke perfectly clear and able to say anything, but headachy.

C. was apparently all right next morning; he was most happy and wanted to go home and was sure he was conscious before the attack began and that he was conscious when he took the medicine and knew then that he could speak and especially when he said good-bye to the doctor. He talked a great deal all day.

A week later the eyes were examined by Dr. G. E. de Schweinitz who found that the reversal and contraction of the color fields formerly noted had completely disappeared and that the fields were normal in every respect. This sudden and complete return to normal conditions of reversed color fields is very unusual, reversal and contraction often being found still present when all overt hysterical symptoms have disappeared.

Summary.—Some degree of loss of speech is a common hysterical manifestation and in marked cases a most intractable one, the patients seldom recovering full power of voice after the aphonia has lasted some months. Now and then an instance of moderate grade loss of voice is temporary and passes off as the patient improves in general nutrition.

Judging from experience in aphonia cases I felt doubtful after my first study of C. as to how the disability would end. The hysterical origin of the trouble seemed certain from the first. The physician who had seen him immediately after the accident confirmed my suspicion of the very slight character of the direct injury and of the utter impossibility of the lad's having received the full current from the electric wire he struck against, which would almost certainly have killed him, and added the suggestive point that the patient's family all showed neurotic tendencies. The final evidence needed came when the complete reversal of red and green in the color fields was discovered. Surface anesthesia or analgesia, so common with reversed color fields, was not found during C.'s first stay in hospital, but was noted as present in the throat upon his second admission two months later. This completes the picture, to which the final touch is given by the dramatic manner in which speech was recovered. The patient went through a kind of parturition,

pains, groans, writhings, and at last brought forth articulate speech, wholly unimpaired by its year of disuse.

Total loss of speech from purely psychic causes, followed by sudden and complete recovery must be hysterical, and is so rare that this is the only case I have seen. Dr. Weir Mitchell, who saw the patient and studied him with interest, has never previously observed an instance of absolute hysterical mutism and I cannot find one recorded.

It is proper to record here my thanks for the great interest taken by Dr. Makuen in this patient and the freedom with which he spent time and trouble in studying the patient's condition and in the endeavor to teach him to speak. His careful and minute report of the laryngeal condition is a valuable addition to the history of a most unusual state.

SYMPTOMS SIMULATING BRAIN TUMOR DUE TO THE OBLITERATION OF THE LONGITUDINAL, LATERAL AND OCCIPITAL SINUSES. A CLINICAL CASE.*

By C. Eugene Riggs, A.M., M. D.,

of St. Paul.

The patient, Miss L., was referred to me by Dr. Strickler, of Sleepy Eye, Minn., January 16, 1902. She was 22 years of age, a teacher, father dead, one brother a confirmed epileptic, two others had convulsions in infancy. Aside from the usual diseases of childhood, patient was perfectly well until the present illness. No syphilis, no tuberculosis. Two years before consulting me she was thrown out of a carriage striking upon the top of her head upon frozen ground. The only symptoms were vomiting and severe headache immediately after the fall. She apparently experienced no subsequent ill-effects until July, 1901, when she complained of severe headaches which were regarded as neuralgic. The pain was situated in the right frontal region. These headaches were at first periodic, usually occurring each afternoon although there would occasionally be an interval of as much as three days between the attacks. During the headache the face would be pale, there were dark circles beneath the eyes, and the lids were edematous. Some three weeks after their first appearance the pain became so severe as to confine her to her bed for a period of two weeks; the severe pain was attended by vomiting which lasted for two days. Afterward during her illness vomiting followed when the pain was very acute. She suffered always more severely during the day. While under my observation she was never entirely free from pain although she was at times quite comfortable. When I first saw her, vision was badly impaired and before her death it was almost entirely lost. She could not distinguish between one and three fingers held before the eyes. There was choked disc. The right knee jerk was exaggerated, the left normal; no ankle clonus, superficial reflexes normal; no Babinski; stereognostic sense normal; dynamometer, right hand 19 deg., left 16; no inco-ordination, pupils dilated but reacting to light and accommodation, no sensory or vasomotor disturbances; temperature normal; pulse varying from 68 to 70; no leucocytosis; urine normal; intelligence unimpaired.

On February 15, there was first observed a twitching of the right side of the neck; it was confined to the sterno-cleido-mastoid and platysma myoides muscles. Sometimes the muscles of both sides were simultaneously affected although it was very much more frequent on the right and always more marked; never on the left alone. Accompanying this twitching on the

*Read by title at the meeting of the American Neurological Association, June 4 and 5, 1906.

right side there would occasionally occur a slight tremor of the chin. February 16, along with a severe headache there was twitching not only of the muscles just mentioned on the right side, but also of the face and eyelid of the same side. This twitching did not seem to have any of the characteristics of cortical epilepsy and its appearance on both sides, although it appeared more frequently on the right side, added materially to the difficulty of making a diagnosis.

Miss L.'s condition became steadily worse and her suffering was so great that an exploratory operation seemed justifiable even though no definite localizing symptoms were present. Dr. Archibald MacLaren of St. Paul operated at my request. The result was negative aside from the fact that for several weeks afterwards she was absolutely free from pain. This, however, again appeared with a fiendish intensity and the second attempt at relief was made during which death occurred.

I am indebted to the courtesy of Dr. Alexander Colvin for the results of the post mortem findings.

There was no intercranial growth and the brain and cerebellum presented a perfectly healthy appearance. The longitudinal and two lateral sinuses were almost completely obliterated. The adhesions between the sinus walls were old and inseparable. Here and there in the course of the longitudinal sinus was found an open space which marked the entrance of a vein; the torcular Herophili was also obliterated. There were in some of the unobliterated areas of the longitudinal sinus adherent shreds of a brownish color which macroscopically had the appearance of blood clots.

Thrombosis of the sinuses is supposed to occur more frequently than any other form of venous thrombosis. This is due to their peculiar construction for, according to Macewen, their great width, the rigidity of their walls, their somewhat triangular form, the trabeculæ that occasionally cross them, the peculiar manner in which they are prevented from being emptied during inspiration, and, in the case of the longitudinal sinus, the direction in which the blood from the cerebral veins enters at an obtuse or right angle against the current, all tend to retard the flow of the blood and thus to favor coagulation.

The usual etiological causes of adhesive thrombosis were not present. There was no phthisis, no Bright's, no cardiac weakness, no exhausting diarrhea, no pneumonia, no carcinomatous marasmus, no influenza, no appendicitis, no disease of the vascular walls, no septic or gangrenous areas, no direct injury of vessels, and no middle ear disturbance. The symptoms were as characteristically negative. There was no epistaxis, no delerium or somnolence, no apparent edema, no distension of the veins of the frontal and parietal regions of the scalp, no convulsions, no temperature, no rapid or thready pulse; in fact the symptoma-

tology of either marasmic or infective thrombosis was entirely wanting, and I am free to confess that I did not suspect its presence.

The only significant symptom was the excessive hemorrhage of venous blood occurring as soon as the incision of the scalp was made.

Under favorable conditions the diagnosis of sinus thrombosis is not an easy matter; in fact "The clinical recognition of plastic or adhesive thrombosis," says Fagge and Pye-Smith, "is rarely possible."

I report this case because I should like to hear what the members of this association have to say in regard to similar experiences of their own.

Society Proceedings

NEW YORK PSYCHIATRICAL SOCIETY.

Stated Meeting, Jan. 2, 1907.

The President, Dr. Adolf Meyer, in the Chair.

THE SYMPTOMATIC-PROGNOSTIC COMPLEX OF MANIC-DEPRESSIVE INSANITY.

By Dr. George H. Kirby,

of the Pathological Institute of the State of New York.

Dr. Kirby said: The field of psychiatry is too complex for us to expect absolute or clear-cut distinctions either in disease types or nosological groups. Kraepelin did a great service by showing that most of the previously made distinctions of types could be replaced by far more valuable nosological groups if one considered the whole course and outcome of mental disorders. From this point of view the great bulk of the acute psychoses can be brought under two divisions, viz., those that pass into deterioration and those that end in recovery. It was found that in cases which terminated in either of the above mentioned ways, one was able to identify certain characteristic symptoms present from the beginning, and which thus acquired a distinct prognostic value. Two large symptomatic-prognostic groups were thus created—Dementia Præcox and Manic-depressive Insanity. At present it seems best to consider the manic-depressive complex merely as a reaction type and not a disease entity as Kraepelin proposes. It is rather a type of response which may be elicited in various ways. In some cases the constitutional disposition may be most important, but in others the exogenous causes are of great importance and give valuable hints as to prophylaxis and probability of recurrence. This peculiar kind of reaction in its pure form has the characteristics of a benign disorder and thus a favorable prognosis is implied. Various additional elements may enter to confuse the picture and the symptomatic and prognostic features may seem to diverge at various times. There are many equivalents difficult to recognize. In a few cases the principle seems to fail through transitions to other forms.

A series of cases was reported which had offered difficulties for various reasons. Some patients were also presented. A recurrent depression with retardation and prominent auditory hallucinations; another case of depression with sensory-somatic complex and only slight signs of difficulty in thinking. A third case illustrating the mixed forms of manic-depressive insanity, the so-called "manic stupor," showing inhibition and mutism with an exhilarated mood.

Dr. Charles L. Dana was inclined to think "prognostic principle" a rather unfortunate term, inasmuch as this was not all that was used in attempting to establish types. The consideration of the whole life history was the great principle to be used in making classifications, and he did not think there could be any objection found to basing groups of insanity upon this method. It was not new but the rational and accepted one in other forms of disease. The case presented by Dr. Kirby in which there was melancholia without retardation of thought was interesting, but was a type not infrequently seen outside of asylums at least, and often in connection with intense suicidal feelings. This sui-

cidal impulse was not always due to somatic feeling of inadequacy, but was sometimes also a well reasoned out point of view. A woman of intelligence, for example, realizing that she had had melancholia now three times, that she would have it again, that she would be a burden to her family, that she would never be right permanently, schemes to kill herself, obeying in a way a logical conclusion as well as a morbid impulse. Such patients did not have always retardation of thought, but they often could not do their work easily or effectively. He did not believe the first patient presented by Dr. Kirby would be able to do her work. Such patients could not play games of skill as formerly, becoming tired quickly. Dr. Kirby had neglected to touch upon chronic melancholia, melancholia of involution, a group which Dr. Dana believed to be represented in early life. He had records of cases of melancholia at the climateric who gave a history of depression earlier very much like that of later life, so that there was a melancholia of early life which did not belong to manic-depressive insanity.

Dr. L. Pierce Clark thought the symptom-complex of manic-depressive insanity would be easier of analysis if the idea were borne in mind that the complex was likely to partake of the nature of the physiological epochs at which it occurred. The surgical impulse to which Dr. Dana referred was well known to most alienists. Even in convalescence this tendency was most marked.

Dr. August Hoch said it was usually claimed that it was impossible to formulate any laws of prognosis about the different forms of manic-depressive insanity. This was not quite correct. Dr. Kirby had shown that a careful reasoning might do much in formulating a special prognosis. Dr. Hoch wished to mention a few more general points of view from which a prognosis might be made. He first referred to the depressions. The symptoms of the entire depressive complex were retardation, sadness, and the anxiety-unreality complex. In the typical manic-depressive states there were only sadness and retardation. In such cases the outlook was good. In the typical involution melancholias there was, from the beginning, uneasiness, anxiety, to which were often added later in the course symptoms of the anxiety-unreality complex. In such cases the outlook was bad; such patients got into a state of deterioration, characterized by a narrowing of the mental horizon. But the anxiety-unreality complex might also be reached by way of the manic-depressive states. This happened not infrequently in the manic-depressive depressions, which occur at the involution period, sometimes in those occurring earlier in life. These cases might, therefore, begin with a typical retardation or feeling of inadequacy and then develop an anxiety-unreality complex. In such cases the prognosis was not so bad as in the typical involution melancholias, but decidedly less good than in the simple manic-depressive depressions.

The second point of view referred to the prognosis of the manic states. All alienists knew that those cases which present the best prognosis are cases of clean-cut manias of considerable intensity, manias in which the exhilaration, the general excitement, and the flight of ideas are all of about the same degree. The hypomanic states were much less favorable prognostically. Dr. Hoch had always found that cases in which the disorder of behavior was out of proportion compared with the flight of ideas and the intellectual disorder in general, were prognostically also more favorable. Such cases were apt to have long attacks or many attacks. The prognosis in cases in which a delusional element was strikingly out of proportion to the flight of ideas was also more doubtful than in the simple classical mania. In other words, the cases of mania in which there were strong discrepancies in the intensity of the different symptoms were those which presented a less favorable outlook than those in which there existed a certain harmony in the intensity.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY.

October 18, 1906.

The President, Dr. Tuttle, in the Chair.

A CASE OF HYSTERIA.

By Dr. Henry C. Baldwin.

P. E., 9 years old, came to the Neurological Clinic of the Massachusetts General Hospital in November 1903. A few days earlier she fell down stairs, but did not hurt herself. The next day she could not walk.

Physical examination shows normal knee jerks and plantars. Sensation was normal. She walked on the toes of her right foot. The diagnosis of hysteria was made, and the patient disappeared from observation.

She came to the Neurological Clinic of the Massachusetts General Hospital a second time in July, 1906. She had constant spasm, especially of the muscles of the back, and when she was placed on the examining table on her back she assumed the position of opisthotonos. There were no spasms or choreiform movements of the muscles of her face or hands. She could walk, but her gait was very peculiar, and the right leg was held rigid and not bent at the knees. There was no disturbance of speech. The knee jerks, Achilles and plantars were normal. Sensation was normal. During the three years that had elapsed since her first visit to the hospital, she had been going to various hospitals in Boston and New York. Scars about the right knee showed that operations had been performed, and she stated that these operations were done at New York hospitals, the hysterical nature of the trouble presumably not having been recognized. The patient was given static electricity and sent to the Zander room for daily treatment under which she improved. This morning she walked almost naturally, putting both feet down squarely on the ground and bending both knees. The movements of the body have almost ceased.

Dr. Walton said that he had seen the case in the hospital when the symptoms were those of paramyoclonus, that is, bilateral convulsive movements limited to the larger muscles, particularly those of the trunk. He had regarded the trouble as of hysterical origin, and looked on paramyoclonus as generally allied to, if not a symptom of, hysteria.

Dr. Knapp said that Dr. Baldwin's patient occasionally visited the City Hospital and efforts were made to take her into the hospital for educational therapeutics, but the attempt to do so caused loud wails and she remained only a day or two. When last seen she was unable to sit in a chair, and, if placed there, would rotate until she faced the back of the chair, resting on the contracted knee with the back strongly arched backwards. Dr. Knapp thought that no one who saw her there was disposed to make any other diagnosis than hysteria. She bears the scars of a tenotomy under the knee, which was a very striking instance of misplaced surgical interference.

Dr. Courtney also saw the case which Dr. Baldwin had presented, many weeks ago at the Boston City Hospital, and at that time made the same diagnosis as that made by Dr. Walton more recently, namely, paramyoclonus multiplex.

FACIAL ATROPHY.

By Dr. Courtney.

The case is one of that rather rare disease known as idiopathic facial atrophy, and makes the third of the sort that Dr. Courtney had the opportunity of showing before this Society. The patient, a schoolboy of 17,

has nothing of significance in his family history. His personal history prior to the actual onset of the present trouble has a certain bearing on the case, and is, briefly, as follows: Patient is an only child. He is in his third year in a suburban High School and is said to be a student. He has had no bodily injuries of moment, has experienced no infection of any sort, and does not drink or smoke. Four years ago he was treated at the Massachusetts General Hospital for twitching or tremor of the left hand, which recurred a year later. During these attacks he was unable to hold things securely in the affected hand.

For the past year he has had sudden transitory attacks of numbness of the left side of the body, of short duration. These attacks are said to occur more particularly while the patient is studying. Six months ago it was noticed that the right side of the nose was beginning to shrink, and shrinkage was subsequently noted on the right side of the forehead also. The wasting has been accompanied by more or less pain limited to the right side of the face, and by headache confined to the same side of the head. For four months patient has noticed a dragging feeling in the right side of the nose and right half of the forehead, and this feeling has been emphasized for the past month by the simultaneous occurrence of transient vertigo accompanied by a peculiar alteration of consciousness. During these vertiginous attacks patient appears to know where he is and says he can speak, but his mind doesn't work normally. He becomes confused in his recitations and has difficulty in grasping what the teacher says.

Physical examination shows a hemiatrophy sharply limited to the tissues of the right side of the face. The line of demarcation is particularly noticable on the forehead. On the right side of the forehead is a contracted scar of a dull white color. No other leucodermal manifestations are present. The right naso-labial fold is flattened somewhat; the brow wrinkles normally. The cartilaginous scaffolding of the nose is markedly shrunken on the right, but smell is not affected. The intrinsic muscles of the face are spared; likewise the masseters. The tongue is tremulous and apparently slightly shrunken on the right half. Taste is completely lost on the right half of the tongue, both anteriorly and posteriorly. The palate and fauces are normal both sides. The pupils are regular, equal and normal to tests of light and accommodation; the muscular movements of the eyeballs are normal. The eyelashes, eyebrows, and hair have neither changed color or fallen out. There is diminished appreciation of heat and cold on the right side of the face, but sensation is not otherwise disturbed. There is a fine tremor of both hands greatly increased on rotation. The muscles of trunk and extremities are normal. The teeth and jaws are normal.

Dr. Courtney did not think that in this case the disease had attained to its full development. He did not propose to say anything concerning the pathology of the condition at this time, but hoped to show both this and his other two cases again, after they had been subjected to the paraffine treatment.

HYSTERICAL HYPERTHERMIA.

By Dr. G. L. Walton.

The case was one of high temperature of probable nervous origin. A boy of eight (seen in consultation with Dr. Bush), unusually studious and mentally active, became greatly excited over a coming exhibition. He was also knocked down a week before by a larger boy who is said to have sat upon the side of his head. This experience was followed by headache, lasting a few days; there were no further symptoms. During the following week the temperature rose steadily to 105° F. There was no restlessness, no disturbance of consciousness, no retraction of the head, no ocular or other cerebral symptom, no bleeding from the ear or nose, and no subconjunctival hemorrhage. The urine was normal and

there was no digestive or other general disturbance. There was history of a similar experience a year ago but without quite so high a temperature.

Careful examination showed no disturbance of motor, sensory, reflex, or other function. The skin was natural, warm; the pupils were widely dilated, but reacted perfectly to light. The boy was bright and intelligent without restlessness or sign of discomfort. The blood pressure was 70. The temperature, taken by the mouth under the direct observation of the physicians, was 104.7° F. The conditions were unchanged for a week, after which the temperature gradually lessened during several months. The boy has been well since. The absence of constitutional symptoms rule out infection, and the nervous origin of the temperature was rendered the more probable by the neurotic constitution of the child and the history of a similar previous experience. Such a case can, however, be hardly regarded as fever in the ordinary acceptation of the term, and the question suggests itself whether the high temperature in such a case may not represent a local hysterical manifestation rather than the general body temperature.

Dr. Knapp recalled a case of hysteria which came under his observation at the City Hospital nearly twenty-five years ago, which Dr. Edes doubtless remembered. She had very profound right hemianesthesia involving the special sense with right hemiplegia. At times she had a high temperature, as high as 105° F, while at other times the temperature sank to 95°. Very careful investigations were made and no source of the fever other than the hysteria could be found. Dr. Knapp thought that Dr. Walton's suggestion of a local rise of temperature is somewhat fanciful, yet in this case there was noted for a long time a difference of axillary temperature on the two sides of from one to four degrees. For a time the temperature of the anesthetic side was the lower, but later on the temperature of that side was higher. Sphygmographic tracings on the two sides showed a difference, the pulse curve on the anesthetic side showing at times a greater tension and at other times a lower tension and a more acute rise than that on the other side.

Dr. Courtney, apropos of Dr. Walton's communication, wished to mention a case which he had had under observation for a long time. It is that of a girl of about 18 years, who first came to the clinic complaining of persistent headache. Physical examination revealed nothing to account either for the headache or for the fact that the patient's daily temperature had been 100° or 100.5° for a matter of three years. The patient is an inveterate neuropath and has recently developed a form of myospasm which strongly suggests paramyoclonus multiplex. He was inclined to attribute the increased heat production in the case to nervous influences alone.

Dr. Mitchell said that he had made some observations upon the effects of exercise in healthy men that might be of indirect interest in connection with this subject. Three healthy young men, on six different occasions, had been given exercise for one hour playing baseball. The days were warm and perspiration profuse. Rectal temperatures before the exercise were never above 99.4°, and the rectal temperatures taken at the end of exercise ranged as follows: The lowest of the eighteen temperatures was 102.2°, and the highest 102.8° In each instance the temperature fell to normal, following bath and rest, and there was never any sense of elevated temperature.

GENERAL ENCEPHALOMACIA.

By Drs. Southard and Hodskins.

The speakers proposed to define a type of soft brain differing, on the one hand, from encephalomalacia, due to plugging of vessels, and on the other hand from autolytic softening of post-mortem origin. They termed the condition general encephalomalacia. The condition is characterized by (1) Diffuse axonal reactions in many types of cell; (2) diffuse fatty de-

generation demonstrated by the Marchi method; (3) absence in increase of weight (important in distinguishing grossly from edema); (4) absence of exudative changes.

Epileptics are somewhat prone to exitus with soft brain. The condition seems to be associated with a terminal exhaustion. The illustrative case was that of an epileptic, dying at forty-two, was of importance in that it showed the same histological changes in the midst of a sclerotic area, as were shown by the remainder of the brain and cord. Thus the lysis, while it appears to be a general histolysis, is actually a differential cytolysis or axonolysis. Enlargements were shown from photomicrographs of axonal reactions in various types of cell from the illustrative case.

DEMENTIA PRAECOX.

By Dr. Brownrigg.

Indications of beginning mental enfeeblement may be explained as wholly dependent upon other bodily illnesses or irregularities and incipent dementia præcox may be overlooked by general practitioners for weeks or months before a threatened insanity is suggested. Many such cases are recognized as nervous and put down under the general term, "neurasthenia" when close observation by experienced men cannot fail to detect even quite early the probable essential trouble. Thus slight attacks of dementia præcox may be recovered from at home without special treatment, and only subsequently when an attack brings commitment to insane hospital does the previous mild history disclose a former, but sufficiently, well marked attack that ought to have been recognized as such and more caution followed. These patients then follow the course of those that have more violent attacks from the beginning and by degrees going downward generally offer as poor a prognosis as those committed to asylums at first.

From analysis of New Hampshire State Hospital records of the last three years, 33 per cent. of dementia præcox patients "recovered" or were very much improved so as to undertake their former work. Six per cent. became perfectly normal as far as could be determined. Fifty-three per cent. had not improved, or showed only sight improvement. But of those that did not improve much, the duration of the illness previous to hospital treatment was about two years, and of those that "recovered" or "much improved" the duration before commitment was only two months.

From the past five years experience with psychasthenic cases that could be classed as probably undoubted cases of incipient dementia præcox the conclusion is drawn that such early cases under early treatment in private hospitals where they could have careful control and individual attention and separation from other more definitely insane cases, offered a much more favorable prognosis as to both early and more complete recovery than similar ones, that by habit and progress of the disease, had become more damaged mentally. In fact dementia præcox has probably in a large proportion of cases a more or less prolonged prodromal period with "neurasthenic" symptoms predominating, and in this period the disease is not at all the hopeless affection that it becomes later when complete loss of control has developed.

In treatment emphasis was laid on isolating the patient from friends and customary surroundings and most of all on the need of judicious moral treatment and control. To this end much personal time of the physician and especially trained nurses are essential.

Dr. Folsom said Dr. Brownrigg's experience was somewhat in the same line as his own. Looking up post-typhoid psychoses he found that the prognosis of cases treated in the insane hospitals was reported as more unfavorable than for cases treated outside the hospitals for the insane, and he thought the same is probably true of dementia præcox. The explanation is in part that the worst cases go to the hospital, but the hospital influences, with its surroundings and associations, are bad for quite young

people. The outlook is better for such patients out of the hospital. Dr. Folsom's impression is that dementia præcox is not uncommon; at least the cases like dementia præcox that go on to complete dementia are not unusual. The diagnosis is often overlooked, as Dr. Brownrigg says, as is especially true of mild cases in early stages. Some cases of dementia præcox resemble chorea so far as the mental condition is concerned; that is, the limitation of intellectual capacity is the most prominent symptom. Now and then in girls of 18 or 20 we find cases with very slight choreic movements, but with marked impairment of intellectual power which possibly might be called either chorea or dementia præcox. In the case of a girl, for instance, who was taking fencing lessons the fencing master was the first to find that she could not co-ordinate the movements of her right wrist. The subject of diagnosis is often open to doubt or dispute in individual cases and requires a good deal of careful thought.

Dr. McDonald said that since Jan. 1, 1903, there have been discharged from Butler Hospital 389 patients. Of these, 74 (19 per cent.) were diagnosed as cases of dementia præcox. In the hospital at the present time are 75 cases of dementia præcox, so that we have 149 cases of dementia præcox in which to consider the prognosis. This number does not include doubtful cases, but only those in which the diagnosis was made with a reasonable degree of certainty. Fifty-seven were males, 92 females; *i. e.*, 61.7 per cent. were women. These figures disagree somewhat with those of Prof. Kraepelin, who says that the disease is approximately equally divided in the two sexes. Of these cases, 6.6 per cent. have died; 33.5 per cent. proceeded to a very deep and apparently abiding state of dementia; *i. e.*, deep apathy with neither depression or exhileration, combined with various symptoms which have been characterized as diagnostic of dementia præcox; *i. e.*, negativism, catatonia, impulsive and foolish behavior, mannerisms, verbigeration, etc. 38.2 per cent., while not advancing to this deep dementia, remained unimproved; that is, with more or less acute symptoms, such as hallucinations, delusions, foolish behavior, etc., with more or less of the characteristics of mental defect. Four per cent. improved slightly, but not sufficiently to permit a return to the home. 16.7 per cent. made a partial recovery; that is, improved to such an extent that they were able to live at home without particular friction, or would be able to live at home if the home conditions were not extremely unfavorable. Many of these have taken up their old life to a considerable extent. Some are capable of occupation and a number are earning their own living. This 16.7 per cent. corresponds to those cases, which Kraepelin considers as recovered, though not necessarily without perceptible defect. .6 per cent.; namely, one case (female) has apparently recovered entirely so that not only the friends, but also physicians are unable to detect evidence of even a minor degree of mental deterioration. We see, therefore, that the prognosis of dementia præcox is extremely bad. The class of cases giving the best prognosis and from which the largest number of recoveries were drawn, are the catatonics. We are warned, however, from this circumstance to regard with suspicion our diagnosis of dementia præcox where catatonia is present to a marked degree in the acute stages, particularly where there is a condition of catatonic stupor. In these cases we have made many incorrect diagnoses in the past, and expect to make still more in the future. A number of patients where this diagnosis has been made, proved eventually to be probable cases of melancholia-mania, and we are of the opinion that catatonic stupor is not diagnostic of dementia præcox, though the condition, of course, occurs in that disorder as well as in other mental disturbances.

Dr. F. H. Packard said that the diagnosis of dementia præcox is apt to be difficult in many stages of the disease, and it is often especially so in those very early stages of which Dr. Brownrigg has spoken. He thought one should remember that there is, so to speak, often what might be called a normal abnormality during the period of adolescence which fades away

and does not lead to a dementia præcox. While it is usually or often at this period that dementia præcox begins, one should bear in mind these normally abnormal conditions, and be guarded about calling them recoveries from dementia præcox. What he wished to speak about more particularly are those cases in a more advanced condition as usually seen in the hospitals. From his experience with such cases he was rather sceptical when he heard people talking of recoveries from dementia præcox, and he suspected that in many such cases there was either a wrong diagnosis made or that the patients were not followed carefully enough or long enough. As to making a wrong diagnosis, he was quite sure this is often done, especially in those cases of stupor where the previous history is not well known and where the cases are not carefully analyzed. For a considerable time catalepsy has been considered by many as almost a pathonomonic sign of dementia præcox, yet he had seen this phenomenon in cases where the previous history showed that the way into the stupor and cataleptic condition was typical of a manic-depressive condition, and where the course and outcome of the case showed that without doubt it was a case of manic-depressive insanity. What he wished to emphasize is the necessity of considering more than the picture of the moment, as for example, when one sees a cataleptic condition one must not say immediately that this is a dementia præcox. As to the second point, he was likewise quite certain that many of the so-called recoveries are not followed carefully enough or long enough. Within a comparatively recent time several cases have been observed at the McLean Hospital with such definite symptoms of dementia præcox that there was no hesitation in making such a diagnosis and giving a bad prognosis. Nevertheless, these patients recovered sufficiently to go home and be pronounced cured by their relatives. He might say that it was almost with pleasure that within the last two months he saw two such cases returned to the hospital, again showing unmistakable symptoms of dementia præcox. One has progressed so rapidly that there is no longer any question of recovery. The other one likewise is in a very doubtful condition. Careful questioning showed that ever since leaving the hospital one of these patients, called well, was scarcely able to do more than simple things like feeding the chickens. He would emphasize the necessity of more careful diagnosis and of more careful and longer observation before speaking definitely about recoveries from dementia præcox.

Dr. Mitchell said that the paper opened to discussion a very difficult problem. The accurate differentiation of dementia præcox from other psychoses was often uncertain, long after the patient had shown symptoms necessitating hospital care, and this uncertainty must be even greater in the diagnosis of early cases. He thought critical care should be exercised in making the diagnosis of dementia præcox on neurasthenic or psychasthenic symptoms, because in taking histories of hospital cases, diagnosed as manic-depressive insanity, it was frequently found that the first attack was regarded as neurasthenia or "nervous prostration," and that this condition would be followed later by a well marked manic-depressive state. It is also difficult to determine that a patient suffering from dementia præcox has recovered, and this estimate could not be considered well founded unless the patient displayed normal conduct and unimpaired intellect for a long period. Hospital experience demonstrates that many patients who show temporary improvement following the first outbreaks of this disease, and who might at this period be considered as recovered, eventually show progress of symptoms that either incapacitates them for leading useful lives or again requires hospital restraint.

Dr. H. W. Miller agreed with what has been said relative to the difficulty of diagnosticating in the early stages of dementia præcox. It is by no means easy, and we are often led astray. This uncertainty often causes one to question the diagnosis of dementia præcox when the patient makes a good recovery. That some cases do recover seems unquestionable. We

have little experience with very early cases in a public institution such as Taunton Hospital is, and our recovery rate in this class is unfortunately low; the majority of the recoveries belong to those cases ushered in with acute symptoms, chiefly the katatonic type. He felt that the tendency is to use the term dementia præcox too loosely, and that we are inclined to group in this class cases which do not properly belong there because the symptoms are ill-defined, and do not seem to fit satisfactorily into any other group. He was interested in the discussion on this subject at the British Medical Association meeting in Toronto, where criticism of the name dementia præcox was quite general among the British psychiatrists.

Dr. Folsom did not mean that cases should not have proper and individual treatment. If people have resources and suitable means for treatment, young patients are, as a rule, better cared for outside of a hospital for the insane, because they are at a very receptive age, and the suggestions from hospital life are demoralizing to them. But if a hospital is necessary, a small one like that at Nashua is preferable to a large one. Proper treatment for the individual case is a necessity for the best results, and for that the insane hospital may be the only available means, even if objectionable, in some ways, except for the worst cases.

Dr. Knapp said that Dr. Folsom had rather anticipated what he was about to say. Dr. Brownrigg's cases illustrate the importance of early treatment, which is of great importance. He thought that there are a good many cases of sensory phrenosis that get entirely well and remain permanently well if they receive proper treatment early in the attack. In many cases, however, asylum treatment is not the best thing in the way of treatment, but proper treatment outside is usually a very expensive matter, so expensive as to be beyond the means of many, and the prejudice against asylums and the legal restraints that bar free admission often result in fatal delay. He was glad to hear from Dr. Miller that our English brethren have not succumbed to the perversions of the mother tongue and object to the term "dementia præcox." He had used Bianchi's term, "sensory phrenosis," in preference. On Kraepelin's own showing, "dementia præcox" is often neither "dementia" nor "præcox."

Dr. Folsom said as regards the expense of which Dr. Knapp speaks he did not think it need be great, if one is ready to run considerable risk as to suicide, as he often did, for the better chance of recovery.

Dr. Brownrigg was in agreement for advanced cases of dementia præcox, with those who are pessimistic about a possible complete recovery, but he believed that in the early stages where no dementia is present the affection seems essentially to be a curable one. Dementia symptoms come later, and appear secondary to the progress of some underlying changes, but before these have progressed far, treatment seems more effectual and the prognosis not at all bad.

Periscope

The Journal of Mental Science

(Vol. LII., No. 216, January, 1906.)

1. Amentia and Dementia; a Clinico-Pathological Study. JOSEPH SHAW BOLTON, M.D., M.R.C.P.
2. On Some Relations Between Aphasia and Mental Disease. SYDNEY J. COLE, M.A., M.D., Oxon.
3. Some Notes on the Study of Insanity. F. GRAHAM CROOKSHANK, M.D., London.
4. Multiple Lipomata in General Paralysis. CONOLLY NORMAN, F.R.C.P.I.
5. Some Clinical Notes Upon Urine-Testing and Results. ROBERT JONES, M.D.
6. A Note on Psychiatric Terminology and Classification. THOMAS DRAPES, M.B.
7. The David Lewis Manchester Epileptic Colony. ALAN MCDOUGALL, M.D.
8. Notes Upon the Incidence of Tuberculosis in Asylums. GEORGE GREENE, M.A., M.B., Cantab.
9. The Necessity for State Interference on Behalf of the Imbecile. F. E. RAINSFORD, M.D.
10. The Employment of Female Nurses in the Care of Insane Men in Asylums. GEORGE M. ROBERTSON, M.B., F.R.C.P., Edinburgh.

1. *Amentia and Dementia.*—Continued article.

2. *Relation Between Aphasia and Mental Disease.*—The author calls attention to the necessity of studying the aphasic disturbances in mental disease, a view based upon the work of Wernicke, Pick and others. The former was the first to emphasize this relation and laid stress upon the significance of "Transcortical Sensory Aphasia" as a link between aphasia and mental disease. The type of aphasia was thought by Lichtheim to be produced by a supposed interruption of the path from the auditory word center to a schematic center of concepts. The relation of these aphasic symptoms and their occurrence in mental disease is then discussed, the most important symptoms from the author's standpoint being echolalia. This symptom he regards as aphasic, especially when it occurs in senile dementia, epilepsy and general paralysis. In these cases echolalia is usually accompanied by other aphasic symptoms. Such a value to echolalia may also be given in other psychoses, including the katatonic form of dementia præcox. Echolalia may vary in degree from the slightest, the purposeful repetition or giving back of a question (the "Echolalia in Frage form" of Pick), to the severe grade—the "Automatic Echolalia" in which the patient merely repeats mechanically what is said to him, words and even sentences being thus repeated. In this series a relation between echolalia and a defect for understanding spoken language can be demonstrated. The author cites cases of Jacksonian epilepsy, and general paralysis in which the above symptoms were present during periods of confusion following seizures. Also in cases of acute insanity, confusional type. Ten cases are abstracted from the literature, showing this combination of symptoms, mostly in cases of senile dementia, organic dementia and general paralysis, a number of which came to autopsy and no focal lesions were found to account for the symptoms. Certain cases, which the author thinks might answer the description of dementia præcox, have been observed, and in the late stages echolalia was accompanied by aphasic disturbance and dementia. A very interesting case is reported in detail and the admirable manner in which it was examined, warrants its publication as well as our attention and respect. He states that the case considered from a psychiatric

point of view is certainly dementia præcox, katatonic form, but also from the result of the examination, is a case of sensory aphasia and the data certainly warrants such a statement. Along with mental impairment, katatonic mannerisms, stereotyped movements, impulsive acts, echomimia, good memory for certain things, and flight of ideas (rather word salad) in a woman 54, who had been insane since the age of 20, occur also the following aphasic symptoms; extreme verbal paraphasia, agrammatism, disorder of intonation with paraphasic logorrhea and jargon; serial and imitative speech well retained, mild degree of alexia, understanding is defective for spoken language, is lost for printed and written language, voluntary writing is abolished and paragraphia is present. The fact of these symptoms occurring in a case of dementia præcox is certainly worthy of notice, but whether this paradigm will hold for other cases is rather doubtful. The views regarding the causes and nature of echolalia are unsatisfactory. While most authors hold that it is a disturbance of the will, others, among whom is the author, claim that it is primarily a speech defect and has nothing to do with the will. Various views are cited, principally the dissolution of the inhibitory influence of the higher association centers (Hughling Jackson). According to Pick, this inhibition is accomplished by the auditory word center, but the author prefers the schema of Lichtheim, who claims that the inhibition is exercised by the center of concepts through the auditory word center. Whether these theories explain echolalia or not, the author is satisfied to state that echolalia and aphasic symptoms are closely related in mental diseases. From a clinical and psychological standpoint, such a relation is surely admissable, and such cases as the author produces tend to substantiate this theory. To establish such hypotheses on an anatomical basis is infinitely more difficult and even Wernicke could not demonstrate that such aphasic symptoms depend upon known properties of certain regions of the brain and their destruction by disease.

3. *Notes on the Study of Insanity.*—The author argues that in studying insanity, psychical and physical series should be considered separately, that physical causes are physical in the sense that physiological conditions are physical. He would not consider "moral" or mental treatment except as it applied to physical conditions. He also pleads for a systematic study of insanity, based upon acceptance of elements of our actual knowledge.

Then he proposes the following "stages" as characterizing insanity, and that these stages should be recognized as belonging to one process; namely, melancholia, mania and dementia, in the order named. This law is based upon the following statements: 1. That the psychical phenomena of insanity occur in the same order as sleep, delirium, intoxication and senility. 2. That the order is the reverse of that which obtains in individual and racial development. 3. That in recovery from insanity, the order of psychical phenomena is that of individual and racial development. According to the author we are to abandon the treating of "mental disease" as such, but we are to consider them as dissolutions of mind and nervous system. From the confusing nomenclature and the idea that we are to consider melancholia, mania and dementia as characterizing insanity and the stages of one process, those engaged in the study of psychiatry may find an easier method and a simpler label, but not much benefit could come from such a usage.

4. *Multiple Lipoma in General Paralysis.*—A case of general paralysis exhibiting multiple lipoma is considered sufficiently rare to report. The author gleans from the literature that only one mention is made of this occurrence in general paralysis, and another in tabes dorsalis. The author's case was a tabetic paralytic with other unusual features. The association of multiple lipoma and tabes and general paralysis would suggest that it belonged to the tropho-neurotic conditions occurring in these diseases, but the author regards this explanation as incomplete.

5. *Not suitable for abstracting.*

6. *Psychiatric Terminology and Classification.*—The author deplores the present state of psychiatric terminology and classification and the frequency of new classifications which distract the student of psychiatry. He is much disturbed because there is as yet no universal classification and lays the blame upon the fact that our knowledge on the subject is so imperfect, that the requisites for a proper classification are wanting, or that there is an essential difference between psychiatry and other scientific departments. He admits the great difficulties to be encountered. He leaves the classification to psychologists, but unfortunately little that is practical has come from such sources. He ignores the fact that what is needed is a classification more suited for medical purposes. He disputes the idea that we can have "so-called varieties of insanity" as dementia præcox, and from a theoretical standpoint combats the use of "dementia" in both dementia præcox and senile dementia. The stand taken that all other forms of insanity, if not cut off by intercurrent illness, end in dementia is difficult to comprehend. Also we can get little help from the proposed method of classifying insanity according to duration and intensity. For the former the terms "recent—sub-chronic—chronic" for periods of three, six and twelve months, and for the latter "acute, sub-acute and mild" are proposed.

7. *Not suitable for abstracting.*

8. *Incidence of Tuberculosis in Asylums.*—The author at the outset, combats the prevalent idea that insane asylums are hot-beds for the growth and spread of tuberculosis. From statistics it would seem that the proportion of deaths by tuberculosis to deaths due to other diseases was lower in asylums than in population outside. Poverty, unhealthy hygienic surroundings and alcoholic excesses, factors potent in producing tuberculosis in the general population, have no influence inside of hospitals.

Evidence deduced from post-mortem findings go to prove that the tubercular bacillus is not spread in asylums. The symptoms of tuberculosis in the insane were different from those in other classes of patients. Wasting, irritability at times coughing are present, but patients seldom expectorate and seldom have hemoptysis. The author also states that the physical signs in the insane are different to some extent from other classes of patients and more difficult to recognize. In the discussion of this paper which follows, many important points, such as ventilation and general hygiene in insane hospitals were brought forward, special attention being given to segregation of the tuberculous insane.

9. *Not suitable for abstracting.*

10. *Continued article.*

HENRY A. COTTON (Hathorne, Mass.).

Brain

(Vol. XXIX. No. 113, 1906.)

1. Upon the Orientation of Points in Space by the Muscular Arthrodial and Tactile Senses of the Upper Limbs in Normal Individuals and in Blind Persons. R. T. SLINGER and SIR VICTOR HORSLEY.
2. Notes on the Tænia Pontis. SIR VICTOR HORSLEY.
3. The Mental Symptoms of Cerebral Tumor. PHILIP COOMBS KNAPP.
4. A Study of the Minute Structure of the Olfactory Lobe and Cornu Ammonis, as Revealed by the Pseudo-vital Methods (with remarks on the Plan of the Nervous Structure of Vertebrates in General. JOHN TURNER.
5. The Abdominal Reflex in Typhoid Fever. J. D. ROLLESTON.
6. A Family in which Some of the Signs of Friedreich's Ataxy Appeared Discretely. ERIC GARDNER.
7. Note on Apparent Re-representation in the Cerebral Cortex of the Types of Sensory Representation as it Exists in the Spinal Cord. COLIN K. RUSSEL and SIR VICTOR HORSLEY.

1. *Orientation of Points in Space.*—This study comes to the conclusions that (1) the faculty of orientation in space, as determined by the muscular and arthrodial sense, progressively diminishes from the surface of the body outwards to the limits of the arm extended in any direction, (2) orientation knowledge increases in passing from point to point in the space around the body, beginning above the head, coming down to the front of the body, and gradually approaching the centre of gravity of the whole body, (3) orientation knowledge also increases in passing from point to point in the space around the body, beginning laterally, *e. g.*, in the plane of the shoulder, and approaching the mesial sagittal plane of the body.

2. *Note on the Tænia Pontis.*—It is first brought out that this bundle of fibers is rarely symmetrical. This is particularly so in the lower animals and the suggestion is hazarded that should this asymmetry turn out to be universal in view of recent work that has been done on the relation of asymmetry and the occurrence of rotatory courses and movements in quadrupedal as well as bipedal animals. Horsley believes it to be a constant structure. It is a ponto-cerebellar tract and is a localized bundle of association fibers, uniting a portion of the frontal border of the pontine gray matter with that of the cerebellum, apparently the nucleus dentatus. The direction of the fibers is cerebellopetal as demonstrated by degeneration methods.

3. *Mental Symptoms of Cerebral Tumor.*—Knapp presents an exhaustive and valuable study on this subject using as a basis, the clinical histories of 104 patients in whom a growth of some nature was found on autopsy. In 75 per cent. of the cases, mental symptoms were noted, a rigid analysis however, made it imperative to revise this figure since alcoholism, typhoid, or other complicating feature existed. Thus, in 64 cases considered as representing true tumor states, at least 90 per cent. were found to show mental symptoms. Knapp believes his statement of some fourteen years previous by "that in every case some change can be found" and "that there can be no gross lesion in the brain without some disturbance, greater or less, in the psychical functions" receives further support by his later experience. As for the mental changes noted Knapp agrees with Schuster. The largest number 31 in all, presented the recognized type of mental dulness and failure. Languor, somnolence, dulness, apathy, mental torpor, failure of memory, and a general failure of all the mental functions, ending usually in complete stupor and coma were exhibited. Seven patients showed confusion and disorientation with failure of memory, irrelevancy in speech, mild mental wandering, somnolence, stupidity and a dazed condition. In 15 there was delirium often with hallucinations. Neurasthenic and hysterical states were occasionally noted in the earlier stages of the disease, but nearly always developed more marked mental disturbances. The mental symptoms showed themselves early in about one-half of the cases. As to the relation of the symptoms to the site of the tumor Knapp shows that five of the six cases of tumor of the frontal lobe, or to speak more accurately, of the prefrontal portion thereof, exhibited signs of mental disturbance. In all cases this disturbance was simple mental failure with marked dulness, but in only three of the cases was it noted as being among the early symptoms. The only case where no mental symptoms were noted was a sarcoma, the size of an English walnut, at the foot of the left second frontal convolution near the precentral sulcus. It is curious, he remarks, that this is the only case of tumor of the prosencephalon or diencephalon in which no mental symptoms were recorded, but mental dulness may readily have been overlooked, as the patient was in bed for a number of weeks, complaining constantly of intense headache and therefore averse to any conversation. Of four tumors in the Rolandic region, two presented symptoms of mental dulness early in their course, a third later in the dis-

case was noisy and delirious, and the fourth finally developed hallucinations and delusions of persecution. One of the two parietal cases presented neurasthenic symptoms early in the disease; the other became dull and stupid toward the close. There were six temporal tumors. Two of these were mentally dull, two confused, one delirious, and one presented a typical picture of general paralysis. One of the dull cases did not show much change till late in the disease; in all the rest the symptoms were of early occurrence. Every one of the three callosal tumors showed mental symptoms early in the disease, and all became confused, delirious, violent and demented. One case had hallucinations. Seven out of nine cases of a growth in the optico-striate region were mentally dull, but only two showed this symptom early. Two other cases showed early mental symptoms and both became delirious, one wildly maniacal. Two cases of tumor of the corpora quadrigemina became dull early and the third also showed early mental impairment with delusions of persecution. One of them manifested early various neurasthenic symptoms, scrupulosity and a sense of unworthiness, later growing demented. The other case of tumor was mentally dull. Five out of seven cerebellar tumors were mentally dull, but only one showed this mental failure early. The sixth was delirious; in the seventh case no mental symptoms were noted, but she had headaches with intense vertigo, aggravated by any effort, so that mental symptoms may well have been overlooked. One tumor of the hypophysis showed mental confusion early in the disease; the second was delirious toward the close. Of seven tumors at the base of the brain, five were in the anterior fossa. Of these, one became dull early, three showed mental confusion later in the disease, and a fifth was delirious, showing mental disturbance early. One patient with tumor in the middle fossa became delirious later in the disease. In only one case of a tumor in the posterior fossa, a growth of the cerebello-pontine angle, not involving the brain deeply, there were no noteworthy mental symptoms.

Eleven tumors were classed as multiple, but two other cases might fairly be regarded in this class, one a multiple growth in the opticostriate region, the other a multiple growth in the pons. Three of the eleven cases showed mental failure late in the disease. One was confused early and three were delirious. One of them showed mental disturbances early; one was mentally depressed. Of the three cases where no mental symptoms were recorded, two were under observation a very short time, one dying suddenly with cysticerci in the brain, the other dying suddenly of surgical shock. A third case with multiple gliomata in the basal ganglia has already been reported. In one other case with mental failure the autopsy record of the findings in the brain was lost, the only note being "glioma of the brain."

4. *Minute Structure of Olfactory Lobe.*—The author first details the improvements in his technique which should be consulted in the original. The structures of the bulb as stained by his method show considerable similarities to the structures of the cerebellum and the homology of these organs is pointed out. In homologizing the cornu Ammonis and the fascia dentata with the pallium, Turner speaks of the stratum oriens corresponding to the fusiform or spindle cell layer; the stratum pyramidulum and stratum radiatum to the large pyramidal layer and the stratum lacunosum to the second or small pyramidal layer. Turner believes in the neurofibril and its continuity and suggests some features of the reflex arc integrations and the integrations of the nerve system in general along the general hypothesis of the continuity of the neurofibrils.

5. *The Abdominal Reflex in Typhoid Fever.*—Observations in 45 patients having typhoid fever, in 93.3 per cent. there were variations in the abdominal reflex. It was completely lost in 68.8 per cent., impaired in a greater or lesser degree in 22.2. Unaffected in three cases only. The

reflex resumes its activity as convalescence becomes established. The author summarizes his result as follows: (1) The abdominal reflex is affected in a very large number of cases of enteric fever, the percentage of cases in which it is entirely lost exceeding those in which its normal activity is diminished only. (2) From its absence under 50 by being confined to certain nervous diseases and acute abdominal disorders, notably appendicitis and enteric fever, the absence of the abdominal reflex in a given case of continued pyrexia in any patient below 50 is of considerable diagnostic value. (3) The comparatively transient nature of the affection of the abdominal reflex in enteric fever is a striking contrast to the more chronic affection of the knee and ankle-jerks in diseases associated with peripheral neuritis, *e. g.*, diphtheria. (4) Return of a lost reflex, and a *fortiori* resumption of its normal activity, are a valuable indication of commencing convalescence, and often correspond with lysis and characteristic changes in the feces and urine. (5) The objective sign of the return of the reflex is often associated with the return of the subjective feeling of ticklishness normal to the individual. (6) In re-appearance of pyrexia in convalescence, the condition of the abdominal reflex is a valuable index of the nature of the pyrexia. (7) No constant relation exists between the condition of the abdominal reflex and that of the tendon reflex. (8) The frequency, degree, and duration of impairment of the abdominal reflex are usually in proportion to the age of the patient.

6. *Friedreich's Ataxia.*—The author gives an extensive literary survey of the cases of Friedreich's occurring in more than one member of the family and reports a new family. The mother showed nystagmus, tremor, spasticity and increased knee-jerks. There were six daughters. One showed spasticity, nystagmus, clubfoot, increased knee-jerks, and changed speech; a second had scoliosis and absent knee-jerk, but is otherwise normal; a third, child is normal, as is the fourth; the fifth child, has no knee-jerk, *pes cavus* is present. The youngest child is normal. He discusses the anomalous character of the illness, but prefers to group them as a family disease showing some of the signs of Friedreich's disease without specifying just where the present cases belong.

7. *Sensory Re-representation in the Cortex.*—The authors put forward the thesis that the spinal representation of tactibility finds an echo in the arrangement of that function in the sensory cerebral cortex. They first review the three classes of views on sensory representation in the pallium so far as they relate to the kinds of sensation represented, *i. e.*, (1) Representation of sensation of any kind in the pallial kinesthetic area, (2) Representation of tactile sensation elsewhere in the pallium as well as in the kinesthetic cortex, and (3) No representation of sensation in the kinesthetic cortex. Evidence from the clinical side is adduced which the authors say up to the present time has sustained the views of Mills and Campbell that the post-central region is essentially the seat of sensory (tactile?) representation, and that all the instances of apparent lesion of the ascending frontal gyrus only are probably vitiated by associated disorders of the ascending parietal convolution, and finally that the corresponding "sensory" centers are arranged horizontally behind the "motor" centers.

The authors proceed then to draw attention to an additional type according to which tactility and topognosis appear to be also represented in the cortex cerebri, viz., a type which is apparently a re-representation of that according to which tactility is represented in the spinal cord. The representation of tactility in the spinal cord which is essentially a metamerically-divided organ is readily mapped out, and the terminology descriptive of the topographical mapping of tactility in at any rate the hand and forearm was originally laid down by Ross from clinical observations and by Paterson from consideration of embryological an-

atomy, while since that preliminary epoch the subject of spinal sensory representation has been exhaustively examined by Sherrington and by Head, with the result that the topographical areas of the different spinal roots are now well defined. The conclusion reached is that it is perfectly clear that what may properly be termed the mid-axial line and region of the hand and forearm is as definitely represented in the cerebral pallium as it distinctly is in the spinal cord. It is perhaps natural to expect that this should be so, because any contrast between the post-axial and pre-axial portions of the limbs respectively, presupposes an imaginary frontier line between them, and further, the cerebral memorialisation of the transfer of the limb to and from the mesial plane of the body demands that the cerebral pallium should have particularly localized within it the orientation of the middle point of the limb which is thus translated from or to the sagittal mesial plane. This further proof of similarity between the sensory representation in the spinal cord and cerebral pallium is confirmatory of the other phenomena described.

Jelliffe

Deutsche Zeitschrift für Nervenheilkunde.

(Band 28, Heft 1.)

1. The Symptomatology of Hemiplegia. Heilbronner.
2. The Status Hemiepilepticus Idiopathicus; Eight Clinical and Anatomical Observations. Müller.
3. A New Algesimeter, with a Critical Description of the Previous Algesimetric Methods. Thunberg.
4. Tumor of the Hypothesis Without Akromegaly. Kollarits.

1. *Hemiplegia.*—Upon the cadaver it may be observed that the outer contour of the thighs is more convex than during life. The same appearance may also be observed in the affected leg in cases of recent hemiplegia. A somewhat similar appearance may be observed in the calf and arm muscles. Heilbronner calls this condition "the broad leg." It is not present in sleep, nor in unconsciousness, nor in profound narcosis. It is present in severe acute polyneuritis, but was not found in tabes, in chorea, in Huntingdon's disease, or in hysterical flaccid hemiplegia. It is not certain that it will serve in the differential diagnosis of cerebral and spinal hypotonia. The broad leg is not dependent upon the disappearance of the patellar reflex, or even upon the return of voluntary movement; but if it persists the contractibility of the quadriceps to percussion upon its tendon is usually lost. After some discussion of the reflexes and muscle tone Heilbronner reaches the conclusion that in the course of recovery from hemiplegia the following series of events occurs. First, return of active motion; second, return of the contour-preserving tone; third, the return of the reflex muscle tone. With reference to certain other clinical phenomena he states his belief that the superior restoration of the function of the leg is merely apparent. He also discusses the hemiplegic gait, and calls attention to the importance of educating hemiplegics how to walk properly. He describes a curious form of rhythmical movements of the arm during walking, which are entirely involuntary. They consist of a flexion and lifting of the forearm, and may occur when apparently all the symptoms of hemiplegia have disappeared.

2. *Status Hemicpilepticus.*—Although it is generally supposed that partial or Jacksonian epilepsy is due to some focal lesion in the brain, a number of observations have been recorded in which such partial epilepsy has existed although examination of the brain has been entirely negative. Müller collects from the service of Nonne eight cases of partial epilepsy. The first, an alcoholic and leutic man of 26, who had had an injury to the head. There was left-sided status epilepticus not relieved by trephining. The macroscopical examination of the brain was negative; apparently a microscopical examination was not undertaken. The second case, a

chronic alcoholic, died in left-sided status epilepticus. At that autopsy there were various lesions of the body, but the brain was microscopically normal. The third case, a chronic epileptic, 38 years of age, had 306 attacks, involving only the left side of the body. At the autopsy an area of softening was found on the basilar surface of the right frontal lobe. Elsewhere the brain appeared to be normal. The fourth case, a boy of 19, had symptoms of acute meningitis, then left-sided epileptic attacks and death. There was a diffuse chronic meningitis in the base of the brain, but no alteration in the substance of the brain. The fifth case, a woman of 37, had epileptic attacks limited to the left side; there was a history of injury to the brain. There was high fever, cyanosis, and loss of pupillary reaction. The ophthalmic examination indicated disturbance of the optic nerves. At the autopsy nothing abnormal was found, either in the brain or body, and a microscopical examination of the former organ was negative also. The sixth case, a girl of 18, had albuminuria, and right-sided epileptic attacks. The brain was normal; the kidneys only slightly affected. There was hypoplasia of the aorta. The seventh case, a man of 37, in the course of diabetes mellitus developed typical Jacksonian epilepsy. At the autopsy the brain was normal, but there were changes in some of the abdominal organs. The eighth case, a boy of 6, had general epileptic attacks at the age of 4 years. Two years later he suffered from epileptic attacks, limited at first to the right side, then to the left side of the body, profound coma, gnashing of the teeth and trismus. Later he developed measles, and made a complete recovery. It seems wisest, in the absence of more definite knowledge, to regard these cases as atypical forms of general epilepsy.

3. *Algesimeter.*—Thunberg describes this algesimeter, which consists essentially of a lever, to one end of which a needle is attached, and to the other end a weighted screw, by which it can be brought into a state of equilibrium. The arm to which the needle is attached is divided into ten parts. The instrument is brought into a state of equilibrium and then weights hung upon the arm. The position and size of the weight determines the amount of pressure exerted. This pressure is increased until the patient experiences pain. The paper is concluded with a valuable critical description of the various instruments hitherto divised for the purpose of measuring sensation.

4. *Hypophysis Cerebri Without Akromegaly.*—Kollarits tabulates a series of cases in which there was tumor of the hypophysis cerebri, without akromegaly. Among these he includes two cases of his own. Usually these tumors occur in young persons, but Kollarits is of the opinion that a careful consideration of the cases suggests that tumor of the hypophysis is merely one of the symptoms, and not the cause of akromegaly.

J. Sailer (Philadelphia).

Miscellany

Tabes Dorsalis and Psychosis. M. Bornstein (Monatsschrift für Psychiatrie and Neurologie. Vol. 17, 1905).

Many authors claim that the psychical manifestations of tabes are purely of a paretic nature. Bornstein discussed the many difficulties one experiences in making a differential diagnosis between general paralysis and other mental disorders. The author makes abundant reference to the literature of tabes and psychosis, and among others he quotes the following important neurologists and psychiatrists. Cassirer differentiates between the elementary psychopathic symptoms of tabes from a fully developed psychopathic state. The tabetics as a rule manifest extraordinary quietness, even striking cheerfulness, which they exhibit in spite of the pain they endure. Other authors are more reticent in generalizing their observations. Leyden and Goldscheider assert that the emotional tone in the tabetic is variable, as characteristic of all chronic diseases. Cassirer

maintains that the patients who suffer from tabes are cheerful, but other authors disagree with him, and claim that the mood is changeable; at times they are elated, but oftentimes they are markedly depressed, even suicidal, suffer with insomnia and become very nervous. However, in many cases no such symptoms develop except an ordinary hypochondria. Simon alleges that all tabetics present a peculiar mental symptom complex, which he termed "Tabetic Dementia." Further study of Simon's patients showed that the so-called tabetic dementias were nothing but ordinary cases of paresis. Nageotte studied three cases of tabes which were devoid of mental symptoms, but post-mortem examination disclosed a distinct pathological process corresponding to that found in general paralysis. In regard to the real psychosis in tabetics, Bornstein states that in almost all cases of tabes, hallucinations of all senses play an important rôle in the clinical picture. Kirsch and Pierret and their pupils regard such a psychosis typical of tabes. According to Pierret, the fundamental symptom in such a mental derangement is melancholia, which is founded on a somatic basis. From such a state, sensory disturbances become marked and exaggerated by patients. For instance, pain may be interpreted as an imaginary foe and reduction of vision gives rise to optical false perceptions. Nageotte, Moebius and others disagree with Pierret. But Moebius, however, admits that tabes dorsalis and paranoia may occur in the same individual. Otto Meyer has made a study of 56 undoubted cases of tabes with various mental aberrations. He groups his cases as follows: Paranoia (chronic hallucinatory), 21 times; depressive psychosis (melancholia and hypochondria), 14 times; circular psychosis, 4 times; acute hallucinatory confusion (amentia), 2 times; maniacal excitement, 1 time; secondary dementia (after paranoia), 1 time; paranoia dementia præcox (Kraepelin), 1 time; primary dementia, 3 times; psychosis with hallucinations of restless character, 3 times; severe periodical excitements, 1 time; excitement in defective mental development, 2 times; dementia præcox, 1 time.

Schultze described two cases of tabes, one of which was melancholic, and the other a paranoic. The usual ratio of tabes between men and women is 3 to 1; locomotor ataxia with psychosis, 1.6 to 1. This shows that the relative frequency of psychical disturbance in female tabetics is greater than in men. Functional insanity (but not paresis) is more common in those tabetics which show an affection of the optic nerve and the nerves which regulate the pupillary accommodation; and furthermore in those patients who have marked consanguineous vesanity. Bornstein describes a patient who developed a clinical picture of tabes with optic involvement, who was subject to several attacks of mental disturbance. The psychosis was essentially of an hallucinatory nature. The patient reacted to auditory, visual, haptic hallucinations, and was disoriented, excited, confused and irritable. These mental symptoms rapidly subsided and the patient regained his normal equilibrium without insight. The author's conclusions are briefly as follows: (1) There are no specific foundations for a tabetic psychosis. (2) The most important symptoms of all functional, mental disorders, occurring in tabes, are hallucinations of all senses. (3) The psychical disturbance in tabes should not be considered as a complication of this disease process. (4) The frequent psycopathic symptoms in tabes usually arise from distinct somatic disturbances, as, for example, optic atrophy, paresthesia and other sensory anomalies. (5) Patients without a predisposition for mental disorders may have hallucinations without a true psychosis. On the other hand, those patients who have a distinct vesanic taint may develop different forms of psychoses, such as paranoia or depression.

F. J. Conzelmann (New York).

The Neuritic Type of Progressive Muscular Atrophy. A Case with Marked Heredity. By Archibald Church (Chicago Medical Recorder, November, 1906).

The writer reports a case of the true peroneal type—extending through

six, and probably eight, generations. According to the history the disease successively became more extensive and severe with each generation. The patient states that no females in the family were ever similarly affected, but two maternal aunts had optic atrophy, and a sister weakness of the ankles. J. E. CLARK (New York).

TRUE SCIATICA AND DISEASES OF THE POSTERIOR URETHRA AND ADNEXA. By G. S. Peterken (California State Journal of Medicine, August, 1906).

The author differentiates (1) sciatica neuritis, or true sciatica, the symptoms of which are those common to all inflamed nerves; (2) symptomatic or false sciatica, the symptom of which is pain in or along the sciatic nerve with absence of local objective symptoms usually accompanying neuritis. In the opinion of the writer, false sciatica is frequently due to a chronic inflammatory state of the posterior urethra and adnexa, and the thorough examination of the male sexual organs is advocated in all cases. J. E. CLARK (New York).

MIGRAINIC PSYCHOSES. Alfred Gordon (Journal A. M. A., Jan. 5, 1907).

The author reports twelve cases of migraine associated with mental symptoms, observed within the last four years, and all presenting similar types of derangement; namely, confusion, delirium, usually with hallucinations, and stupor. The hallucinations were usually visual, though gustatory and auditory hallucinations were also observed. The confusional stage predominated in all, and was frequently accompanied with illusions of identity, incoherence and disturbance of orientation. Some of the cases suggested psychic epilepsy or procursive epilepsy. In the majority of cases the mental symptoms occurred during the attacks, and in some they continued twenty-four hours after the subsidence of the migraine. In some cases, however, they occurred either before or after, and they lasted in some cases for twenty-four hours after the subsidence. He does not think that he can explain these conditions as epileptic or hysterical, though in some cases they suggested it. The special point is their association with an autotoxic condition, which is the basis of migraine.

LANDRY'S PARALYSIS. J. N. Hall and S. D. Hopkins (Journal A. M. A., Jan. 12, 1907).

The authors report on five cases of acute ascending paralysis, all fatal but one, and review and analyze other recent cases published, and discuss the condition generally They conclude that not one bacterial species can be credited with this intoxication, though there can be little doubt that it is due to bacterial products. There is no apparent occupational factor, though the predominance of the disease in males might suggest such a possibility. Their analysis of carefully reported modern cases would seem to show that sphincter involvement is more frequent than is stated in the text-books. As regards diagnosis, confusion is likely to arise with acute ascending myelitis, multiple neuritis and anterior poliomyelitis. In the former the sensory involvement, trophic disturbances, wasting of muscles and loss of faradic irritability should aid the diagnosis. In multiple neuritis the sphincters are not involved. The paralysis is usually limited to peripheral muscles, and there is marked pain, tenderness and sensory disturbance. Anterior poliomyelitis is a limited paralysis of the extremities, usually the lower extremities, with marked wasting, lacking sensory disorders and with tendency to rapid improvement. The treatment of Landry's paralysis is chiefly supportive. In the author's patient, who recovered, benefit seemed to be derived from salicylates and iodid of potash, followed later by strychnin, massage and electricity. Mercurial inunction was also applied, though there was no specific history.

HEAD TRAUMA AS A CAUSE OF INSANITY. C. W. Burr (Journal A. M. A., Jan. 5, 1907).

The author asserts that, excluding traumatic epilepsy and hemiplegia with their resulting mental disorder, injuries to the head will not cause insanity unless some predisposition exists. His reasons for this belief are that the vast majority of those suffering from brain injury do not become insane unless there is such a loss of brain substance as to produce dementia from that fact alone, and the rarity of mental disorder following extensive operations on the brain in which lesion of the organ is much greater than that of any ordinary trauma. That head injuries can be a directly exciting cause when predisposition exists or in senility, or that they may aggravate or revive an insane tendency is not to be denied. As regards the claim that there is a characteristic form of post-traumatic insanity, he says it can certainly be denied. The usual types are confusion, delusion, simple dementia and epileptic insanity. He has never seen paranoia or true melancholia following a head injury. He excludes from consideration all cases of post-traumatic delirium, transitory or rapidly followed by death from exhaustion, and all cases in which the physical injury was so trifling or the mental shock so slight that the trauma was a mere coincidence. The only cases considered are those in which the relation of the time between the injury and the onset of the insanity was so close as to make it reasonable to assume that there was something more than mere coincidence. The exact relation between early trauma and late insanity is difficult to ascertain on account of the lapse of time and the usual vagueness of the histories, but he has never seen a case in which other causes than the injury could be excluded. A number of case summaries illustrating the author's views are given in the article.

THE ETIOLOGICAL ROLE OF THE VASOMOTOR CENTERS IN CARDIAC NEUROSES. R. Pollard (Zentralblatt für Innere Medizin, Jan. 12, 1907).

Dr. Rudolf Pollard, after an interesting dissertation, gives us the following conclusions of his observations: (1) There is a group of diseases, chiefly chronic, whose symptoms may be recognized by a preponderating participation of the blood vessels, or rather the blood vessels of the nervous system. To this group belong the heart neuroses, paroxysmal tachycardia, Bassedom's disease, the angioneuroses of the skin, etc. (2) The source of these disorders is an abnormally increased irritability of the vasomotor centers in the medulla. (3) The resulting condition of the heart and the vascular manifestations are in the way of a reflex which may be elicited through peripheral as well as central (brain) irritation. (4) Thereto, further, a reflex sensory tract is necessary; the reflex processes (probably on account of retarded influences at the surface of the brain) may appear under circumstances at first, after the expiration of a certain time (late reflex) and are not strictly confined to the location of the irritation.

ATWOOD (New York).

PAROXYSMAL TACHYCARDIA. By John Hay (Edinburgh Medical Journal, January, 1907).

Symptomatic tachycardia is caused most frequently by a diminution of the normal inhibitory influences. Pyrexia, certain infective conditions, alcohol, atropine and thyroid, may produce the condition, as well as direct stimulation of the accelerator fibers or of the heart muscle. "Paroxysmal" tachycardia, on the other hand, is doubtless of nervous origin, but "during a paroxysm there is an essential and fundamental change in the mode of cardiac contraction, the heart beats in a manner specifically different from that normally found." Three cases are detailed, careful tracings of which are reproduced and explained. The author suggests that "certain nervous influences alter the condition of the heart muscle, and more especially the fibers joining the auricles and ventricles, rendering them more excitable and more capable of stimulus production." Traumatism affecting the

heart, physical stress or strain is mentioned as the most exciting factor. In Hay's first case the first paroxysm came on after a severe blow over the epigastrium and lower end of the sternum received in a game of football. Some subsequent attacks in the same case followed physical stress, and others seemed to have no assignable exciting cause. Paroxysms lasted from some hours to eight days. The symptoms were usually severe and distressing. The other cases were milder; one was in a child of six and the other in a man of forty-two. The author quotes that Hoffman believes that these attacks are of the nature of cardiac epilepsy, but that in thirteen autopsies of such cases no lesion was discovered either in the central nervous system nor in the vagus or sympathetic nerves. Hoffman agrees that abnomal cardiac contraction is a distinct feature of the paroxysm. Wenckebach is quoted as suggesting that the stimulation of the central nervous system results in a great increase of the chrontropic and bathmotropic influences playing on the heart. ATWOOD (New York).

JOINT AFFECTIONS IN NERVOUS DISEASE. By L. F. Barker (Journal A. M. A., Feb. 2, 1907).

Four types of joint disease in nervous affections are considered. The first of these, the intermittent joint effusions, he considers closely related to the angioneurotic edema of the skin and mucous membranes. The danger is in these cases that a mistake in diagnosis may lead to needless and dangerous surgery or to tedious fixation and inactivity. The sudden onset, absence of fever, short duration and periodic recurrence make the diagnosis usually easy. What the patients require in treatment is rest, encouragement and a flannel bandage. Aspiration of the joint or injections are totally unnecessary. Hygienic measures will often suffice to stop the attack. Angioneurotic remedies may be advisable for the patient's general condition and arsenic has sometimes been of service. The arthropathies of tabes and paresis are treated of at more length. The rarity of pain in these cases, the sudden firm swelling, usually rapidly extending, are characteristic in most cases, though rarely there may be pain and the swelling may be gradual. Sometimes the swelling subsides, and the joint is left but little impaired, but more frequently there is a breaking up of the joint and a mild case can be converted into a severe one by neglect and overuse. The painless character of the affection tends to lead the patient to use the joint unless strictly warned against it by the physician. Every joint of the body is liable to be affected, but the larger ones more than the smaller. Two special forms are mentioned: the tabetic foot, in which the bones of the arch are particularly involved, and the tabetic spine, differing from the other types of spondylitis deformans in its sudden onset and extensive destruction of the parts as well as in its associated tabetic symptoms. The joint lesions of syringomyelia are very similar to those of tabes, but the common occurrence of pain, the predominance of involvement of the upper extremities (80 per cent. of the cases) and the longer course of the affection are notable differences. Its treatment is limited to rest, orthopedic measures and the avoidance of trauma. Operative measures are rarely advisable. As regards the theories of these spinal arthropathies, Barker thinks that the neuritic explanation is plausible for tabes, but insufficient for syringomyelia, and that similar objections hold good in the case of the arthritis deformans theory. In conclusion, the painless arthralgias of hysteria and the traumatic neuroses are noticed, and the importance of differentiating them from cases of organic disease, especially tubercular, is pointed out. In doubtful cases the deep chloroform narcosis recommended by Charcot, should be employed for diagnosis. Isolation and psychotherapy are the sovereign remedies for this condition; hydrotherapy and electricity can be useful adjuncts. Brief details of such treatment are given and its suprising success is noted.

Book Reviews

A Treatise on the Motor Apparatus of the Eyes, Embracing an Exposition of the Anomalies of the Ocular Adjustments and Their Treatment with the Anatomy and Physiology of the Muscles and Their Accessories. George T. Stevens, M.D., Ph. D. Illustrated with 184 engravings, some in colors. F. A. Davis Company, Philadelphia, publishers, 1906.

Dr. Stevens may truly be said to have excited more comment about his work than any other American ophthalmologist; and this perhaps has been in many quarters more unfavorable than favorable, for while there can be apparently no doubts expressed regarding his knowledge of the physiology of the ocular muscles, and of his skill in devising apparatus and instruments to study the movements of the eyes, most ophthalmologists believe that his treatment of defects in the action of the eye muscles is improper, and most neurologists and clinicians are convinced that his claims that epilepsy and other neuroses may originate in eye-strain, are erroneous. The book under review is a valuable one, and will surely be so recognized by ophthalmologists, but amid all the scientific truths, deduced in a most admirable manner, by a logically thinking scientist, are conclusions regarding the effect of anomalous acting muscles so apparently and flagrantly false that the reader grows to doubt after a time, if even the scientific data upon which the premises are based are true.

As the work is largely concerned with the elucidation of optical principles, neurologists will be interested only in the phases dealing with the influence of imbalance of the ocular muscles upon the general system. Among other observations bearing upon this subject, they will doubtless be interested in the conclusion of the author that certain peculiarities in the excursions of the eyes, in the vertical deviations especially, are as a rule associated with certain types of crania, for he believes that as a consequence of atypically shaped skulls, the axis of the orbit undergoes variations, so that the visual planes change and are only adapted to a normal working standard by compensatory adjustment of the head. Thus, if the visual plane is elevated, the individual carries the head forward, so as to depress the orbital axis, and the forehead is advanced beyond the position to which it would otherwise come. On the other hand, if the plane of vision is depressed, the head is thrown back for the most natural pose. According to the author, bodily pose, gait and appearance are all modified by the direction of the orbital axis. With this observation there will probably be no lack of accord by ophthalmologists, and the field for further study and elaboration of his methods from which he obtained his data will doubtless receive careful study. But agreement with some of his clinical deductions is not so easy, and there will in all probability be but few who will support him in his inferences that trachoma is rife among subjects with tall or mesocephalic heads and that the peculiar carriage of the head, the result of anomalies in the visual planes, is beyond question a most important element in the predisposition to tuberculous disease of the lungs. To fully appreciate the author's convictions upon the subject it is perhaps only fair to quote his own words: "The bacillus of consumption finds no rest and no encouragement to indefinite multiplication in the chests of persons whose heads are habitually thrown backward, nor, indeed, in the lungs of those whose heads are not habitually thrown too far forward. The advantages of the so-called fresh air treatment and a great deal more beside can be secured by the proper carriage of the head which follows at once on a successful correction of the declination or of the anophoria." (By operation.) "In such corrections, important in them-

selves, are to be found the most effective means, not only of prevention, but of relief from the most general single cause of destruction of human life. I am quite aware that these statements will be regarded as extreme and as the outgrowth of too restricted attention to a single class of phenomena. The statements are neither extreme nor the expression of narrow views. They are well considered and based, not only on correct principles, but upon carefully observed facts in a large experience continued through many years."

Equally questionable is the author's observation that strabismus exercises a deleterious effect upon the health of the subjects. Thus, he says: "That some strabismics who acquire the art of effectually suppressing the mental appreciation of the image of the squinting eye remain in good health until a somewhat advanced period in life, does not invalidate the general rule that strabismus leads to early exhaustion of the powers of the patient, and that but a small proportion of strabismic persons reach the age known as middle life. Strabismic children are seen in much greater numbers than adults who squint, and the difference is only in part due to corrective operations."

The reviewer has not, nor as far as he is aware, has any other ophthalmologist, practiced as yet the form of operation which the author describes in his book for the correction of muscle errors, but from a knowledge of the difficulties attending operations upon the muscles of the eye, it would appear inadequate in cases of pronounced deviation of the ocular axes. While all ophthalmologists are in hearty accord with the author's assertion that the great principle which should guide in all surgical treatment of the muscles of the eyes is that all of the functions of movement should be made more perfect and more harmonious after the treatment than before, the realization of this ideal is difficult of attainment. The author's wholesale condemnation of tenotomies and advancements is surely improper, for every ophthalmologist of experience can relate many instances where these procedures have been of greatest advantage.

Notwithstanding its weaknesses, however, Dr. Stevens' book should be ready by every ophthalmologist, for his methods and instruments for examination deserve careful study and invite further elaboration. It must not be forgotten that ophthalmology is greatly indebted to Dr. Stevens for calling attention to the existence of anomalies in the ocular muscles, which had escaped detection by others, and that it owes to him in large measure the means of detecting quickly and accurately the degree of such variations. The neurologist will probably gain but little by its perusal, for in the opinion of the reviewer the deduction as to the importance played by various types of crania in the production of systemic disease is unsubstantiated and erroneous. WILLIAM CAMPBELL POSEY.

AFFEKTIVITÄT, SUGGESTIBILITÄT, PARANOIA. Von E. Bleuler, Professor der Psychiatrie in Zurich. Carl Marhold, Halle.

This is a short monograph of 144 pages, in which the author in an inimical manner discusses certain aspects of the "feelings," applying his analysis in part to the elucidation of the paranoia problem.

Feelings, Bleuler tells us, are distinctly various and a sharp differentiation should be made between the groups if we are to arrive at more fundamental conceptions. He says that at least four groups stand out more or less sharply differentiated: (1) Those due to mass of centripetal processes, of sensations, perceptions (thus the feeling of warmth—of bodily sensation); (2) those conditioned by intracentral perceptional processes related (a) to that which happens without (feeling of certainty, of probability), and (b) that which happens within us (feeling of sadness, of blindness, etc.); (3) those arising from indefinite and hazy recognition, be it a direct perception or a conclusion which is uncertain and unknown in its elements; and, finally, (4) feelings of pleasure and pain. For the first

three groups, he countenances the term "intellectual processes," and he says they have nothing to do with what he wishes us to understand by "affectivity." The feelings in the last group are closely allied with "affectivity," they are mixtures of intellectual and affective processes and contain a sensation and a feeling produced or determined by it. The intellectual processes in his series have no control over the psyche—this is only moved when an affect is added.

In order to arrive at a physiological idea of affectivity, he cites an illustration of the ameba. "The irritation of a grain of food stuff acts upon it at a given place. The portion lying in close contact sends out a pseudopod, and engulfs the spoil, digests, and throws away that which is undigested, and the individual returns to its normal shape. This whole process Bleuler designates as a localized, objective, intellectual process. By it, however, the entire amoeba must have been altered in its nourishment tone and in its entirety. During the taking in of the nourishment other portions of the body dare not flow too much in other directions; such streamings must be retarded. The reception of the nourishment works for the good of the entire body; it becomes stronger, is more inclined to divide or carry on its other functions. This general action, he says, may be arranged by the side of the affects. The affect renders a reaction general, or as he puts it, *the affect is a generalized reaction.* And affectivity is the condition determining the conduct, the pushing element in our actions; reinforcing the reaction if in the line of the affect, retarding it if opposed. A prick of a pin causes one to draw the hand back. If one is shocked by it the entire body draws away; if one reacts in anger the entire body passes on to an attack. The affect further has the interesting quality that it persists longer than the experience. Affectivity is for him the mainspring of responsiveness and activity which is not due to purely mechanical reflexes.

Blueler then takes up the subject of suggestion, which he believes is an affective process. Thus it can and does control the action of the glands, of the intestines, the beating of the heart, and can disrupt the ideas, modify judgment and even be the means of bringing about hallucinatory states. Suggestibility is more than mere imitation, it starts the affective processes, and thus can influence not only the actions of the individual, but even move an entire community.

Paranoia is then discussed at length in relation to its being an affective process as has been held by many, notably Specht. The initial experiences in most paranoics are common to all, especially the suspicions and the mistrusts. Their incorrigible fixation is the pathological element—not the ideas, since so many normally constituted individuals have them. The feature that renders them dominant and incorrigible is perhaps primarily the make-up or disposition so well known to exist in some individuals, to which is added predisposing chains of events. Further than this he does not seem to go. He concludes that paranoia is not an affective psychosis.

The reviewer finds it difficult, even with the help of Meyer's masterly summary (*Psychological Bulletin*, Aug. 15, 1906), to follow the author throughout the argument taken up in the latter part so largely with a criticism of Specht's views of paranoia as an affective psychosis. His illustrative cases are interesting, and the whole book is highly stimulating.

JELLIFFE.

EMOTIONAL VARIABILITY IN EPILEPTICS. Prof. Dr. Gustav. Aschaffenburg.

Aschaffenburg's monograph on emotional variability of epileptics is an important contribution to the literature of psychopathology. The gist of his brochure may thus be briefly stated:

Hoffman, in 1862, was the first to describe epileptic equivalents. According to this writer epileptic convulsions and coma may be substituted by delirium, confusion and hallucinations. Aschaffenburg studied fifty cases of epilepsy; forty-eight of whom were delirious and confused; twenty-

eight had convulsive seizures; twelve were subject to fainting spells; fifteen had petit mal and vertigo. The author asserts that the variability of mood in epilepsy is an important psychical manifestation, and as he expresses it: "Upon the ground of all these observations, I maintain that the fluctuation of emotional tone is a specific symptom of epilepsy; it is a symptom because of its frequency of occurrence, and the difficulty of comprehension of epilepsy, it is by all means very significant." Mood was variable; some patients were depressed, worried a great deal, and showed suicidal tendencies. Others were apprehensive and reacted to auditory hallucinations of a depressive character. Many patients complained in a verbose manner (not flighty) about their situation, confinement, and protested against former injustices which were done to them. With the disappearance of the emotional disturbance they became content and attended to their work as usual. Delusions of persecution were rare; yet one patient had marked auditory hallucinations of a threatening nature, but these disappeared in a few days. As a rule patients showed fugitive tendencies; attempts were made to escape from hospitals, opposed detention, indulged in purposeless walking, marched and travelled till they became exhausted. Especially they complained of being homesick (Heimweh). Ofttimes these attacks were either followed or preceded by a convulsive seizure. A marked clouding of consciousness was not noticed; but a feeling of insufficiency was experienced. Most of these patients showed great intolerance for alcohol, no matter how small the quantity was. Soon after taking liquor they became confused, delirious, expressed fantastic delusions and manifested impulsive and assaultive acts. The intelligence of the epileptics was divided into three classes: First, patients who showed good knowledge and sound judgment. Second, patients who showed meagre intelligence and deficient knowledge. Third, patients who exhibited evidences of intellectual reduction. During the attacks physical disturbances were manifested by the following symptoms: Headache, cardio-vascular functional irregularities, perspiration, dilated pupils; many other reactionary conditions of the central nervous system. These peculiar emotional disturbances bore a strong resemblance to periodic alcoholic intoxications. The attacks usually lasted from a few hours to several days. He classifies his cases into two tables: (1) Twenty-one criminal epileptics with convulsions; all but one showed emotional variability. (2) Twenty-nine epileptics in whom no convulsive attacks were noticed, although they could not be positively excluded.

The following is a brief summary of the fifty cases of epilepsy:

No. of cases.	Heredity.	Convulsions.	Petit mal.	Fainting spells.	Vertigo.	Nocturnal enuresis.	Confusion.	Tendency to escape.	Alcoholic intolerance.	Mood.	Character of emotional tone.			
											Depression.	Irritability.	Changeability.	Accompanying somatic symptoms.
21	3	21	13	6	14	6	9	3	13	20	15	2	3	13
29	9		16	16	23	8	9	6	14	15	8	6	1	7
50	12	21	29	22	37	14	18	9	27	35	23	8	4	20
100	24	42	58	44	74	28	36	18	54	70	46	16	8	40

Differential diagnoses between epilepsy and hysteria and epilepsy and imbecility are discussed in detail. It is worthy of note that Aschaffenburg in 1895 advanced his views on the variability of mood of the epileptics, but, unfortunately, the neurological world did not consider him very seriously. It is to be hoped that the painstaking labors of the renowned German investigator will give a sufficient stimulus for further research in this particular line.

MORRIS J. KARPAS (New York).

News and Notes

Dr. William Clendenin Pickett.—The readers of the Journal will learn with much regret of the death of Dr. William C. Pickett, which occurred at his home, at Aldan, of ulcerative endocarditis, on Feb. 5, 1907. Dr. Pickett was born at Meadville, Pa., on Nov. 16, 1870, and was educated at the Meadville High School and Allegheny College, receiving the degree of A. M. from the latter institution in 1894. He graduated from the Jefferson Medical College in 1895. Subsequently he served as surgeon in the schoolship Saratoga, and in July, 1898, was appointed assistant chief physician of the Philadelphia Hospital, a position which placed him in charge of the male department of the insane. He resigned this position in 1890, but retained official connection with the hospital as registrar. Later in 1902, he was elected examiner of the insane at the Philadelphia Hospital, and in 1904 was made a member of the Neurological Staff. For a number of years he was demonstrator of neuropathology and instructor in insanity in the Jefferson Medical College. On Oct. 7, 1904, he was elected Professor of Nervous and Mental Disease in the Medico-Chirurgical College of Philadelphia. In January, 1907, he was elected President of the Philadelphia Neurological Society, but unfortunately was already confined to his home by illness.

Dr. Pickett took an active part in the meetings of the Philadelphia Neurological Society and of the American Neurological Association. Several of his papers attracted much attention. Two of them especially should be mentioned; they were based upon statistical studies of the rich material in the insane department of the Philadelphia Hospital. One upon "Dementia Præcox," and another upon "Senile Dementia." He also, it will be remembered, made a detailed study of the von Bechterew reflex, and one of his latest studies was on pupillary reflexes in cases of extirpation of the Gasserian ganglion.

Dr. Pickett was filled with earnestness and enthusiasm, and his personal qualities were such as to make his loss seriously felt in the circle in which he moved.

The ninth biennial report of the Delaware State Hospital expresses satisfaction with the new law permitting the Board of Trustees of the hospital to appoint two physicians to pass on all patients seeking admission from the city of Wilmington, and suggests that a similar law should cover the State. Alcoholic cases and mild senile dements, it is claimed, have been weeded out in this way, and much needed space preserved for other cases. This law is, perhaps, unique in the United States. The two physicians must be of different schools and reside in Wilmington. They hold office for three years, and may be removed by the trustees at any time for cause. The physicians are not compensated for these examinations except for commitments. Since appointment they have certified 129 persons and refused certification to 30 persons, most of the rejected being alcoholics.

The thirty-third annual meeting of the American Neurological Association will be held in Washington, D. C., in conjunction with the Congress of American Physicians and Surgeons, on May 7, 8, and 9, 1907. There will be one session daily, held from 9.30 A.M. to 1 P.M., in the New Willard Hotel, which is to be the headquarters of the Association. The annual dinner will be held on Tuesday evening, May 7. The Council announces that the dues for 1907 will be five dollars, and calls the attention of members who intend to contribute papers to the following regulations: That the reading of the paper shall not exceed twenty minutes, and that no one shall consume more than five minutes in the discussion of a paper; that if possible a verbal abstract of a paper should be presented, instead of a reading in full; that no title may be printed in the program unless accompanied by an abstract; and that titles and abstracts must be sent to the Secretary at least six weeks before the meeting.

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Original Articles

A CASE OF EPILEPSY ASSOCIATED WITH ACROMEGALY.

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As the number of cases of acromegaly associated with epilepsy reported has been so small, I wish to add the following one which recently came under my observation.

L. S., thirty-one years of age, married, housekeeper. Father living and well. Mother died at age of twenty-four years, two weeks after birth of patient, assigned cause being some complication of confinement. History of grandparents negative as is also that of remainder of family. Patient was a puny infant, weighing but three pounds at birth. Had swollen cervical glands during infancy and diphtheria at seven years. Married at twenty years of age. About two years later first symptoms of acromegaly appeared. At age of twenty-six years she had her first epileptic seizure. This seizure was apparently grand mal in type. Seizures then varied in frequency, from one in two days to one in three weeks. No aura. She would not know she had had a seizure but for her lacerated tongue. Left side first involved in some seizures.

When admitted to the Colony she was well-nourished, weight being 168 pounds. Temperature, pulse, and respiration normal. Occipito-frontal circumference of head 54 cm. General massiveness of face, especially nose and inferior maxilla. Circumference neck 36 cm. Marked spacing of teeth in inferior maxilla. Tonsils moderately enlarged. Some macroglossia, patient complaining that speech is thick because of this condition. Prominent transverse rugæ on anterior portion of hard palate. Fingers are very broad. She does not know size of gloves formerly worn, but states that she is now unable to place wedding-ring on little finger of left hand, whereas said ring was at first too large for ring finger of that hand. She formerly wore No. 3½ shoes, but

now wears the largest she can buy; she wore No. 6 when admitted. Very marked kyphosis in cervico-dorsal region, slight scoliosis also present. Circumference of chest one meter.

Skin is of a brownish tinge, especially on face and neck. No flushing, cyanosis or change in hair or nails, except latter are somewhat broadened. Mucous membranes slightly pale. Blood count and percentage of hemoglobin normal.

Fig. 1. Taken two years before onset of acromegaly.

Slight enlargement of postcervical glands, no tibial nodes or other evidence of syphilis. Thyroid not palpable.

Moderate exophthalmos present. Pupils normal. Patient states that at times she is much annoyed by an involuntary twitching of eyelids. Examination of fundus shows no abnormality. Patient can readily read ordinary newspaper print. No hemianopsia, strabismus or nystagmus, except slight lateral when she looks to extreme right or left. She has never worn glasses.

At times during the last few months she has had a buzzing sound in ears, especially right. She says it resembles the sound of machinery. Hearing apparently normal as tested with voice and watch. Taste normal; smell somewhat impaired on both sides.

Breathing is mostly costal in type. Diminished expansion,

Fig. 2. Taken shortly after admission to the colony in 1906.

otherwise respiration is normal. No dullness over site of thymus.

Cardiac dullness slightly increased to left of mid-clavicular line. Blood pressure normal. Some accentuation of second pulmonic sound.

Tongue coated, some gingivitis in inferior maxilla. No increase in appetite or thirst. Bowels usually constipated. Examination of abdomen negative.

Urine normal; no polyuria, but as patient sometimes

urinates involuntarily, it is difficult to measure amount accurately.

Breasts atrophied; uterus very small. Menstruation began at age of twelve years, and was regular until onset of acromegaly, at which time it ceased, and has not reappeared.

Several years ago she had severe occipital headache, also a bursting sensation in eyeballs. At present, and for some time past, has had no headache. Indefinite history of having had vertigo at about the same time headaches occurred.

Voice moderately hoarse.

Cutaneous sensibility normal: considerable increase in perspiration with some fetid odor accompanying it. Reflexes moderately active. No paralysis of any part. Musculature good; dynamometer registers more than the average for her sex. Patient herself claims she is not as strong as formerly. Co-ordination and sense of position normal. She has difficulty in assuming an erect posture because she says it "hurts her backbone." Gait is normal except for slight awkwardness.

Mental condition is good for one in her station in life. Can write without difficulty.

Since admission she has had frequent petit mal seizures, which begin in left hand as if she were trying to approximate finger tips. Moderate dilatation of pupils, involuntary urination, marked automatism. Duration of seizure about one minute. Patient has no recollection of having had seizures. No distinctive convulsive movements observed during these seizures. A few grand mal seizures have occurred, but under such circumstances as to preclude their being satisfactorily observed.

A somewhat extended review of the literature on the subject brings to light the following cases, in which a definite history of convulsions accompanies that of acromegaly.

Grinker[1] reports a man of 45 years of age, who, eight years before, had some "psychic traumatism." Had sensations as in petit mal seizures, then after a year somnolence and marked weakness. Then screaming attacks with trembling of the legs and crying. Two years later is said to have had his first epileptic attack. He now had marked enlargement of the nose, eyebrows, inferior maxilla, hands, and feet. No disorder of the eye muscles or of the optic nerves. Larynx not enlarged. No other nervous abnormalities. Mental weakness present. This case was said to belong to the chronic form, which lasts from eight to thirty years.

Farnarier[2] reports a typical case of acromegaly associated

with epilepsy and a condition approaching dementia.

Hinsdale[3] quotes from Raymond and Souques, who describe a case of acromegaly, of many years' standing, in a man of fifty-four years, who in the last three years developed Jacksonian epilepsy.

Hinsdale also quotes from Marinesco, who describes a woman aged thirty-two years who had epilepsy beginning at twenty-two years, the attacks occurring three or four times a week. At the age of twenty-five years she had an attack of giddiness in which she fell from a second floor. Six months later she noticed that her feet were enlarged, and later her hands, face, and abdomen. Menses ceased. Strabismus, polyuria, and weakness supervened. Sensibility preserved in all forms except vision. Examination revealed bitemporal hemianopsia. Glycosuria present.

A. Napier[4] reports a case of acromegaly in a woman, with onset at twenty-five years. Later she had an epileptic seizure with albuminuria.

Hutchings[5] reports a man aged forty-four years, who had epilepsy since puberty. Acromegaly at thirty-six years. Dementia present.

In the discussion[5] is mentioned a case of acromegaly with epilepsy in a young man of twenty-eight years.

Oestreich and Slavyk[6]—acromegaly in a boy of four years. Convulsions present. Autopsy showed cystic psammo-sarcoma of pineal gland. Pituitary normal.

Jolly[7] mentions a case of acromegaly having petit mal seizures.

Gatt[8] reports a woman aged fifty-six years who had acromegaly for more than ten years. This was preceded by insomnia and headache for several years. Two years before report she had convulsive attacks. Autopsy revealed sarcoma in the sella turcica and adjoining bone. Colloid cysts in thyroid.

De Blasio[9] reports an acromegalic skull in an epileptic who had syphilic hepatitis.

Graves[10] reports a case of myoclonus epilepsy which developed acromegaly.

M. F. Moutier[11] reports a man aged 36 years. At twenty-three years of age, during military service, he had measles and

typhoid, also some indefinite genital trouble. Since then he has become fat, lost strength, and has had violent headaches. His military service was interrupted by blindness of the left eye, preceded by severe pain in eye and marked vomiting. It could not be ascertained when the acromegalic deformities commenced. The patient remembered that when seven or eight years of age he attracted attention because of his large hands and feet. Now has characteristic appearance of acromegaly. Indefinite lancinating pains in legs and jaw. A severe parieto-frontal headache, which was unrelieved by remedies. Complete left optic atrophy. Mental condition failed considerably since 1893, especially memory for recent events. Became morose.

The convulsive phenomena appeared first in 1901. These appeared every day for eighteen months, then became less frequent, but have again become very frequent, occurring two or three times daily. At present psychic equivalents appear often. No aura. Convulsive seizures are apparently grand mal in type. Psychic equivalents were first observed in 1905. He had short spells of vertigo before this time.

Moutier says epileptiform crises are more frequently observed in this class of cases than are the mental changes. During the intervals between seizures the patient is intelligent. He is inclined to believe that the cause of the condition is some cerebral tumor, hypophyseal or otherwise.

He states that these phenomena of automatism are exceptional and does not see how to indicate as analogous any but the case of Devic and Gauthier, in which there was a glioma of the frontal lobes and left sphenoidal lobes. Their patient, a woman of fifty-two years of age, had presented a slight tendency toward ambulatory automatism.

The question naturally arises as to how many of these cases have been merely symptomatic Jacksonian types and how many had typical grand mal or petit mal seizures, associated with mental symptoms characteristic of epilepsy. It would appear that some of the cases had epileptiform convulsions accompanying brain tumor.

The weight of opinion seems to indicate that acromegaly is due to an affection of the essential substance of the pituitary gland. This may be caused by some malignant growth, viz.,

sarcoma, which either involves the gland substance or else causes atrophy or change in function by pressure. Again it may be caused by a benign growth which slowly brings about changes in the gland.

A large number of cases of tumor of the pituitary gland and surrounding structures have been reported in which there have been no symptoms of acromegaly. In these cases, it was deemed very probable that the pituitary structure was not involved or at least only partially, as in many of these cases there is no mention made of a careful microscopical examination of the pituitary body.

Kollarits thinks that tumor of the hypophysis is not the fundamental cause of acromegaly, but rather one of the symptoms, as he reports 53 cases in which there was tumor of the hypophysis without acromegaly.

Many of these hypophyseal tumors are thought to originate in the infundibulum.

F. Farnarier thinks that degenerative states are favorable for the development of acromegaly and that certain changes in the hypophysis react on the nervous system already in a state of instability by the hereditary influences.

Boettiger considers a toxemia as the cause of acromegaly. The pituitary gland is thought by some authors to have an important trophic influence on the central nervous system. Interference with this function or complete ablation of the gland would bring about a state of toxemia, and so convulsions of various types. In order that convulsions may occur it seems reasonable to think that there must necessarily be a lack of stability in the cortical cells in such acromegalics. This would lead one to consider the pituitary changes as one of the many exciting causes of convulsions or other symptoms of epilepsy.

If a tumor of the pituitary is present, it is but reasonable to believe that the epileptoid symptoms may be produced by direct pressure on the cortical substance, by increase in the size of the tumor, or else by general increased intracranial tension and so bring about convulsive movements.

Various nervous phenomena may be due to the defective nutrition or the accumulation of toxic substances in the body. Brooks claims that if there is no glycosuria or hemianopsia

it is questionable as to the involvement of the pituitary body.

Heersman's theory is that the hypophysis neutralizes through its products in the blood the secretion of other blood glands. It regulates the vascular system and hinders the growth of bone. Through lack of this secretion acromegaly develops. This is the result of a tumor of the pituitary or of a secondary alteration through a chronic alteration of the blood.

Cushing states as follows: "A disturbance of the menstrual function may be one of the earliest symptoms of some intracranial tumors. Growths in most diverse situations, or indeed an increase of intracranial tension has been known to affect the regularity or even completely interrupt for long periods, previously normal catamenia. These cases may be divided into two groups: one, those in which amenorrhea accompanies tumors of the hypophysis or tumors affecting the gland by direct compression. The other, those in which the menstrual disturbance is a symptom of tumors situated elsewhere.

The occurrence of diabetes, mental disorders, epilepsy, etc., with acromegaly may be the result of a perverted pituitary secretion on unstable tissues, or such a secretion or an entire lack of secretion may produce this instability and so allow other toxins to bring about such conditions.

The case now under observation will be followed carefully so as to ascertain further eye changes and if death occur, to procure an autopsy.

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[8]Idem, 1902.

[9]Idem, 1903.

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[11]Rev. Neurologique, Nov. 30, 1906. J. Kollarits. Abstract in American Medicine, Nov. 11, 1905. British Medical Journal, 1899, Vol. 1, p. 850. Harvey Cushing. Sexual Infantilism with Optic Atrophy in Cases of Tumor Affecting the Hypophysis Cerebri. JOURNAL NERVOUS AND MENTAL DISEASE, November, 1906. Burr and Riesman. Idem, January, 1899. Sidney Kuh. Idem, Vol. 28, p. 419. Idem, Vol. 31, p. 54. Abstract of Article by Lamois and Roy. Acromegaly and Diabetic Gout. H. Brooks, Archives of Neurology and Psychopathology, Vol. 1.

GLIOMATOSIS OF THE PIA AND METASTASIS OF GLIOMA.*

By William G. Spiller, M.D.,

of Philadelphia,

Professor of Neuropathology and Associate Professor of Neurology in the University of Pennsylvania.

Various forms of malignant tumor may implicate the pia of the spinal cord and brain diffusely. The sarcomatous infiltration is the best recognized. The first case of this kind reported in America, so far as I know, is that of George K. Weaver,[1] in 1898. In 1903 I[2] reported two cases, one in association with W. F. Hendrickson, and the present communication is made in relation to one of these. Since then two cases have been recorded by F. X. Dercum.[3] The German literature contains the largest number of cases and a few may also be found in the British journals.

Sarcomatous infiltration, however, is not the only form of diffuse tumor formation of the pia. The carcinoma and glioma have been known to appear in this manner. An important paper in regard to gliomatosis of the pia has recently been published by Grund.[4] This author says that the earliest mention of glia proliferation in the pia is by Klebs, who found glia in the pia near a glioma of the brain. He cites Alzheimer as authority for the statement that glia proliferation in the pia is common in paretic dementia. Schlesinger and Saxer have seen proliferation of glia in the pia in syringomyelia. Schlesinger, in his monograph on tumors of the spinal cord, makes no mention of diffuse glioma in the pia arising from the neuroglia, but since the publication of his monograph four cases have been reported (Leusden, Fränkel and Benda, Fischer, Roux and Paviot) and Grund adds one more, but does

[1]Weaver. Journal of Experimental Medicine, 1898, Vol. III., No. 6.

[2]Spiller and Hendrickson. American Journal of the Medical Sciences, July, 1903.

[3]Dercum. Philadelphia Hospital Reports, Vol. V., 1903, and Journal of Nervous and Mental Disease, March, 1906, p. 169.

[4]Grund. Deutsche Zeitschrift für Nervenheilkunde, Vol. XXXI., Nos. 3 and 4, p. 283.

*Read before the Pathological Society of Philadelphia, Feb. 14, 1907.

not refer to my case, so that at the present time six cases of gliomatosis of the pia are on record.

The glioma, unlike the sarcoma, carcinoma, endothelioma or perithelioma, is not a sharply defined tumor of the nervous system, but infiltrates into the surrounding tissue, and it is owing to this fact that its surgical removal is so difficult. Indeed, I[5] have advocated leaving the tumor untouched if at the operation it can be shown to be a glioma. In the striking language of Walton and Paul,[6] one might as well attempt to remove the decayed spot from a fruit in order to arrest the process of destruction as attempt to remove a glioma in order to arrest the tumor formation. It may be possible, however, when the glioma is very minute, to remove surgically sufficiently surrounding brain tissue to prevent further progress of the growth.

Unlike sarcoma, glioma seldom extends into the membranes of the brain or cord, and never breaks through beyond the membranes. Glioma, unless very cellular and closely resembling sarcoma, grows very slowly. Max Borst,[7] in his work on tumors, has a very interesting chapter on glioma, and according to him cavities in a glioma lined with cylindrical cells are rare, but in this finding the embryonal relation of the glia cells to the ependyma may be recognized. Glia cells as derivatives of ependymal cells may have the power to return to the type of epithelium of the primary neural canal, or the cylindrical cells in the glioma may be regarded as separations from the ependymal cells of the ventricles from which the glia cells of the tumor are derived. Both conditions are possible.

The term gliosarcoma has fallen into some disrepute, as it implies an origin from both mesoblastic and epiblastic tissues, but Borst employs the term for the mixed tumor and calls the glioma rich in cells glioma sarcomatodes. The distinctions between glioma and sarcoma may be difficult to make. The presence of glia fibers does not make a tumor a glioma, especially if the fibers are near the border of the tumor, as

[5]Spiller and Frazier. University of Penna. Med. Bul., September, 1906.
[6]Walton and Paul. JOURNAL OF NERVOUS AND MENTAL DISEASE, August, 1905, p. 481.
[7]Borst. Die Lehre von den Geschwülsten, Vol. I.

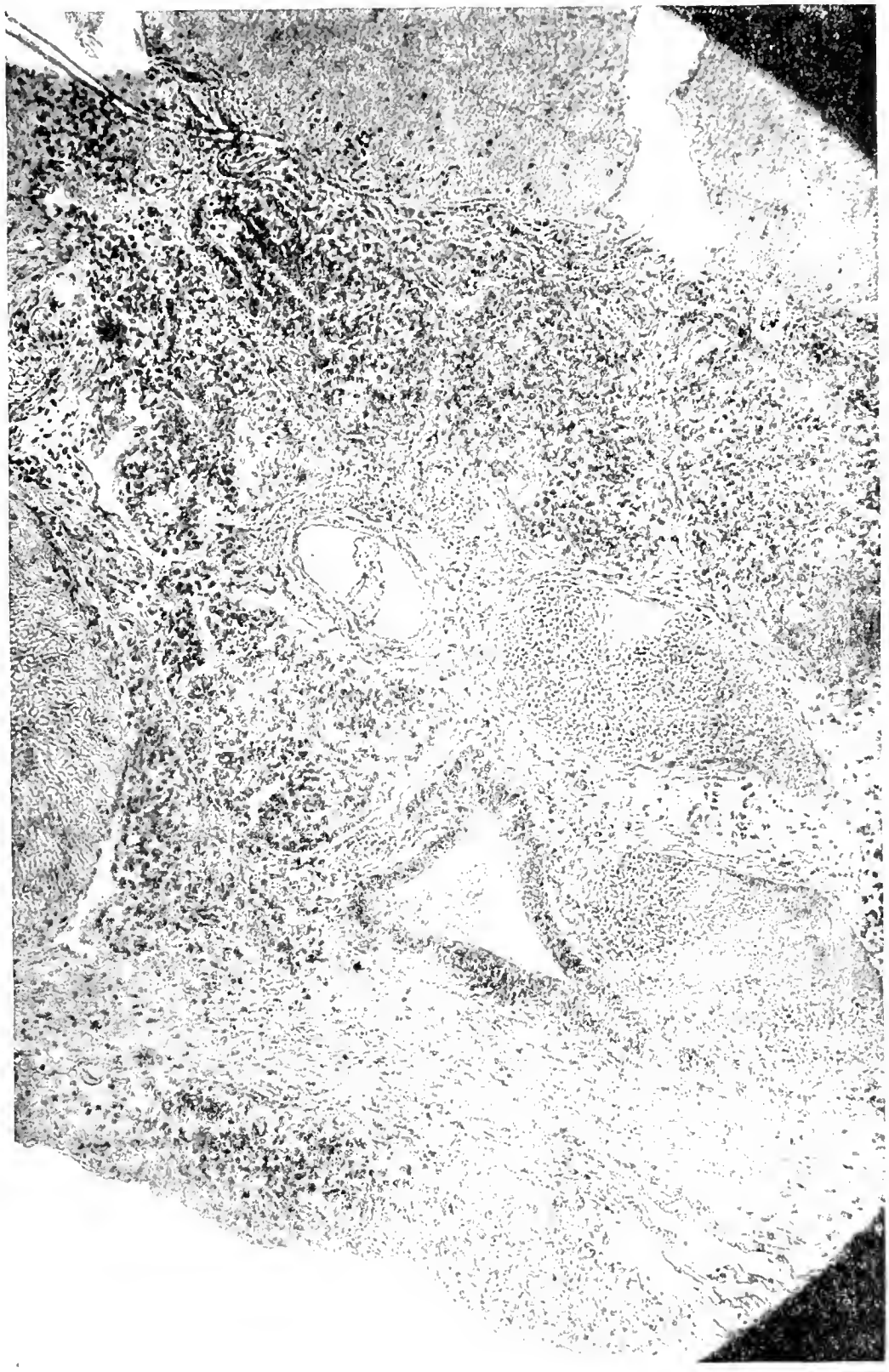

Fig. 1. Diffuse tumor formation of the pia of the spinal cord with rows of ependymal cells inclosing spaces.

they may be the remains of neuroglia caught within the tumor, and glia fibers, Borst states, occur in almost every growing tumor of the central nervous system. Sometimes what are supposed to be glia fibers are really fibrin fibers, and these may closely resemble the glia fibers, and they have no connection with the glia cells. Borst even goes so far as to say that it is uncertain whether a glioma may not be composed entirely of cells, *i. e.*, may be without fibers, and he implies that such a form of glioma may occur, the differentiation of such a tumor from a sarcoma might therefore be impossible.

In a case reported by me in 1903, to which reference already has been made, very many minute tumors and tumor infiltration were found in the pia of the spinal cord. The structure of this tumor infiltration of the pia was so extraordinary that I hardly knew what name to give to it. It resembled closely tumor arising from the ependyma. (See fig. 1.) but I could find no record at that time of gliomatous infiltration of the pia, and none of the eminent pathologists I consulted had ever heard of such an occurrence. I recorded the findings as follows:

"The tumors everywhere have much the same structure. They consist of round or somewhat elongated cells, and even by Mallory's neuroglia stain contain very little intercellular tissue. In some places the cells are distinctly columnar and resemble those of the ependyma. The columnar cells are arranged in long rows and have a large nucleus situated at one end of the cell. These rows of cells are found especially about the blood vessels, but also where there are no blood vessels. There is unquestionably a close resemblance between these cells and those of the ependyma, and the temptation, therefore, is to call the tumor an ependymoma, but it seems remarkable that an ependymoma, being a form of glioma, should give extensive metastasis to the pia of the spinal cord, which is of mesodermic origin. This, and the absence of glia fibers between the cells, and the distinct tendency to the formation of rows of cells about the blood vessels, seem to justify the classification of the tumors under the sarcomata, possibly endotheliomata or peritheliomata; and yet the close resemblance of some of these cells to those of the ependyma may permit us to regard the growth as a mixed one, and as being

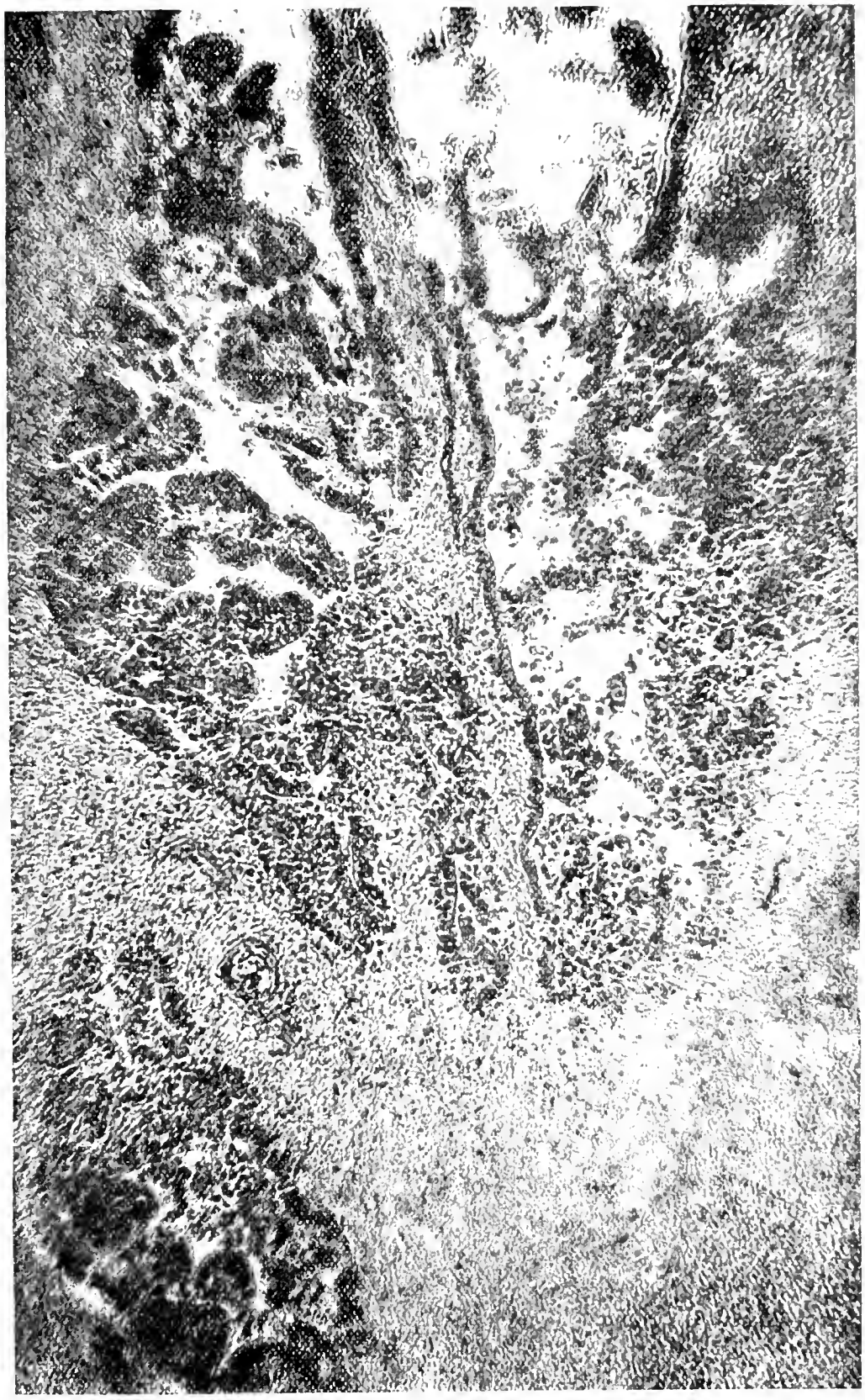

Fig. 2. Tumor filling the fourth ventricle. The proliferation of the ependymal lining is clearly shown.

partly a sarcoma and partly an ependymoma. The ependyma is known to proliferate in cases of syringomyelia when the cavity extends to the central canal and to cover in part the wall of the cavity. It is not unreasonable to suppose that a sarcoma growing from the pia may extend to the fourth ventricle, and by irritation of the ependyma lead to its proliferation, and cause in this way a mixed tumor." I referred to the fact that the tumors of the pia in this case had a resemblance to the neoplasms described by Rosenthal, Fränkel and Benda.

Since the publication of this paper the possibility of diffuse gliomatosis has been in my mind, and I have been waiting for further evidence of its occurrence, and this is now given in Grund's paper. I am now inclined to regard the sarcomatous nature of the tumor infiltration in my case as doubtful, and I look upon the growth as multiple gliomatosis. A section from the medulla oblongata shows a tumor filling the posterior part of the fourth ventricle, and the proliferation of the ependymal cells lining this ventricle is so pronounced that the ependymal origin of the diffuse process seems fully demonstrated. (See fig. 2.) These cylindrical cells no longer form a row lining the ventricle, but occur in long rows, or bordering papillomatous processes, clearly arising from the ependyma, and extending outward into the tumor.

In another case, reported by me[8] briefly from a clinical aspect and with scarcely any reference to the pathological findings, a tumor filled the posterior part of the fourth ventricle and extended downward upon the cord as low as the sixth cervical segment. This tumor also contained rows of cells resembling those of the ependyma, and forming the lining of large irregular spaces. It was an ependymoma. Another, but smaller tumor, having the same structure, was found upon the lower part of the thoracic region of the cord and was probably a metastatic growth, and therefore exceedingly interesting. Borst says that metastasis of glioma is exceedingly rare, and that Stroebe in one case saw regionary metastasis, *i. e.*, near the original tumor. Multiple gliomata, according to Borst, have not positively been observed.

[8]Spiller, Musser and Martin. University of Penna. Med. Bul., March and April, 1903.

IS EPILEPSY A DISEASE OF METABOLISM? A REVIEW OF THE LITERATURE.*

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It has been and is the hope of all that by an exact study of the vital processes of epileptics during life and of their tissues after death, some light might be thrown on the nature of the disease. Patient and accurate pathological work has yielded no changes which can be found in cases of every form and duration, and many believe that the primary nature of the changes which have been reported is open to doubt. The cause of the disease, if there be one in an organic sense, is still to be discovered. I do not wish to be understood as denying the possibility of an organic lesion. There must be such a lesion. I do, however, most seriously doubt, whether this lesion is one which can be made visible by the methods of the pathologist. I believe that the epileptogenous change affects the chemical structure of the cell—whether this change is visible or invisible, seems to me immaterial.

The common idea of the cell is a morphological one. I wish, however, to direct your attention to a chemical conception of the cell and its activities. I believe with Vaughn (under whom I had the privilege of working) that "the cell is a chemical compound of very complex, but of definite structure." This theory differs from others in the emphasis placed on the definite chemical nature of the cells. Thus function is the result of change in the chemical structure of the cell, brought about in accordance with the laws governing chemical action. Disease being a change or failure of normal function, it follows that disease is the result of interference with the normal chemical processes of the cell, either from inherent tendencies or by the action of external agents.

Such a conception of the cell and of disease, authorizes the application of chemical methods to problems which have hitherto been the exclusive province of the pathological anatomist.

Even could we point out a visible lesion in epilepsy we

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should be but little nearer an understanding of the disease. We should still have to ask how this particular lesion produced the disease, and, more important yet, we should have to seek the cause of the lesion. If the activities of the cell are carried out as I have indicated, by chemical means, bio-chemical methods can be logically applied, and offer, from their searching and accurate nature, great hope of light in what is now darkness.

The absence of a visible lesion led to the assumption that endogenous poisons or metabolic disturbances were the cause of the disease. Clinical analogies to other autointoxications have led some to accept this view, but such analogies, while presenting contributory evidence, are not direct proof,—a poison or a disturbance in metabolism must be demonstrated experimentally.

The urine being the most easily obtained of the fluids of the body, has received the most attention. While the old idea that the attack was always followed by the voiding of urine has been found wrong, it is generally stated that the epileptic is given to polyuria, especially following the attack. Krainsky reports daily amounts of two to three liters, and states that four are not unusual. Rabow, and Voisin and Petit report an increased volume after the attack. Féré speaks of crises of polyuria. Bleile and Ferrannini find no change, and Alessi and Pierri report the urine scanty. An increased specific gravity is reported, especially after the attacks, and this, with the increased volume, would seem to indicate an increase of excretory products at the time of the attacks.

Albumin in the post-paroxysmal urine was first noted by Seyfert, in 1854. Others, Rabow, Furstner, Otto, Fiori, Hallager, and Kleudgen, report its presence as rare and inconstant, and in this opinion Binswanger agrees. Voisin, however, finds it in about half the cases and other authors are in practical agreement with him. Huppert and Dewitt find that the amount of albumin depends on the time elapsed after the seizure and on the severity of the seizure. Brunninghausen finds it in the greater number of cases, but of irregular occurrence in the same case. Galenti found 0.05 to 2.0 per cent. Lanois and Mairet found it 29 times out of 50, and Klein 14 times of 23.

Albumin in the post-paroxysmal urine would appear to be of more than rare appearance. We must consider, however, the possibility of concurrent renal disease and of errors in the recognition of albumin. The fact that it is found only in the early hours after the attack, may explain some of the negative findings. The whole ground must be gone over again, using methods of unquestioned accuracy and studying a large number of cases.

Sugar has been found in epileptic urine, but the general opinion seems to be that its occurrence is not an epileptic phenomenon.

Of the inorganic constituents of the urine, the chlorides and the phosphates have been most studied. Vires finds the former increased, while Mairet and Bosc report them decreased and Agostini does not find them increased. Krainsky finds no relation of chlorine to the attack.

The phosphorus metabolism has been more extensively studied. Gibson, Ecchevera and Mendel, Voisin, Mairet, Krainsky, Agostini, Lepin, Egremont, Aubert, and Mairet and Bosc find phosphoric acid increased after the seizure. Lepin and Jaquin, and Mairet and Vires find an increase of the earthy phosphates relative to the alkaline, which may even be decreased. Féré and Herbert cannot substantiate this. Adenine and Bonelli find the earthy phosphates decreased in both urine and feces and report excellent results from the administration of calcium. Mairet associates the earthy phosphates with brain activity. Lepin, Egremont, and Aubert find an increase of organic phosphorus. This is also reported in degenerative nervous diseases by Symers. If this can be confirmed, and the phosphorus shown to be present as compounds other than neucleo-albumins, we shall have an important fact, for organic phosphorus is an important constituent of nerve tissue.

Agostini, Dide and Strenuit, Nelson Peter, Vires, Voisin, Mairet and Bosc, and Rabow report an increase of urea. Teeter finds it variable. Krainsky and Bleile find no constant relation to, and Alessi and Pierri, and Rabow find a decrease after, the attack. We must take into consideration the imperfect methods for the estimation of urea generally used. That the

majority agree on an increase is to some extent a check on the accuracy of their work.

Haig brings forward uric acid as the cause of the attack, having found a retention before and an increased excretion after the attack. Caro, Guidi, and Krainsky confirm his results. The latter does not believe that uric acid is the actual poison but that some antecedent substance, readily transformed into uric acid, by changes in the body fluids, is responsible. Bleile, Herter and Smith, Putnam and Pfaff, and Mainzer do not get results favorable to Krainsky's view. Paraxanthin was found in relation to some epilepsies by Rachford.

Galenti and Savini find the etherial sulphates increased and Galenti and Herter and Smith find indican increased. These results are in cases with indigestion.

Acetone has been found by Hoppe in 8.5 per cent. of 325 cases, but never after single attacks. Rivano found it after attacks in 30 per cent. of his cases. Hoppe observes that acetone is generally found in cases where the taking of food was interfered with; this probably accounts for its occurrence.

Benedicenti and Galdi and Tarugi find an increase of urinary acidity.

The toxicity of the urine has been much studied to determine if there was a retention or an increased excretion of poisonous matters in relation to the attack. Normal urine has a certain degree of toxicity. Deny and Chouppe, Ferrannini, Voison and Peron, Tramonti and Obriga find the interval and ante-paroxysmal urine less toxic than normal urine or the urine from after the attack. Mariet and Bose, Féré, Agostini, Galdi and Tarugi find the post-paroxysmal urine less toxic than that of other times. All except Hebold and Bratz find an increase in toxicity in relation to the time of the attack, but differ as to the exact time of maximum and minimum toxicity. The method is probably responsible for this difference of opinion. The saline concentration and the reaction of the urine, the rate of injection and the susceptibility of the experimental animal into which the intravenous injection is made are all variable factors. The method is at best a crude one.

The cause of the toxicity of normal and epileptic urines appears to be the same, the symptoms from both being alike. Osmotic action, the toxic potassium ions, the coloring

matters, uric acid perhaps, and, according to some, alkaloidal bodies, all play an important part. The toxicity is not due to one substance, but is the sum of all.

Cabitto found the sweat of epileptics more toxic just before the attacks; the toxicity afterward and during the interval is normal. Mairet and Ardin-Delteil find the highest toxicity during or after the attack. Mavrojannis finds no change. The method used is the same as that used with urine.

Belisari, Agostini, and Leubuscher report the hydrochloric acid of the gastric juice increased by the attack. Agostini finds the toxicity of the gastric juice increased just before the attack, corresponding to the transitory dyspepsia.

As regards the cellular composition of the blood no changes have been established as peculiar to the epileptic. Claus and Van Der Stricht report the density of the blood lowered, and Lui, Charon and Briche, and Pugh find the alkalinity diminished before the attack, to rise afterward.

Chiaruttini and Cololian found the toxicity of the blood greater at the time of the attack. Mairet and Vires found the blood hypotoxic in the interval. Herter found no special toxicity. Krainsky, injecting blood taken just before an attack, produced both immediate and repeated seizures in a rabbit, and Pearce and Boston performing a similar experiment on a case of pernicious anemia, got a similar result. Geni finds the blood of epileptics more teratogenic than normal blood. He also reports that the blood during the exacerbations of the disease is much more toxic than during the intervals.

Ceni's work on the serum treatment of epilepsy, if it can be confirmed, will give us absolute evidence of a toxin for the disease. But before it can be accepted, it must be verified under the most rigorous conditions, with careful controls of the hygiene and medication employed. Longer periods of observation and larger series of cases must be employed, and the theoretical side carefully studied.

Comberali and Bue and Voisin have reported staphylococci in epileptic blood. Bra found the "neurococcus" in the blood of a large proportion of the epileptics he examined, but could find it only at the time of the attacks. This fact he uses to explain previous failures. Besta, Tirelli and Bossa cannot duplicate his work, and it is quite possible that he was dealing

with a skin contamination. It is interesting to note, however, that Tirelli reports increased bacteriocidal power of epileptic blood against staphylococci.

Mott and Halliburton and Donath have found cholin in the cerebro-spinal fluid of degenerative nervous diseases and of epilepsy, and Donath believes it to be the cause of the convulsion. The presence of cholin has been doubted by several authors, and, again, others have confirmed its finding. The question centers around the accuracy of the separation and identification of cholin, those who doubt its presence claiming that the product obtained is an ammonia or potassium compound and not cholin. Its presence could be easily understood as it is a product of the cleavage of myelin, and it has been shown that after death there is an enzyme which can bring about this cleavage. One can imagine that under favorable conditions this enzyme could become active during life. Injected into the circulation, cholin is not convulsive, but according to Donath, its direct application to the brain does produce convulsions. The mechanical effect is to be taken into consideration, however. Even though cholin is not ordinarily a convulsive poison, it is to be remembered that the epileptic brain is inherently sensitive, and it responds actively to irritants which would have no action on the normally constituted brain.

While this review of the literature is an exceedingly meager one, it will be seen that there are changes in the metabolism of the epileptic and in the toxicity of his body fluids. Just what these changes are and their time of occurrence is doubtful because of contradictory findings. The fact that some variation from the normal is almost always reported, makes one hopeful that further work along bio-chemical lines, conducted with the greatest care, using the most exact mehods, carefully controlling every possible factor, and using a goodly number of cases, will in the end bring a solution of the problem.

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THE DIAGNOSTIC VALUE OF LUMBAR PUNCTURE IN PSYCHIATRY.

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(*Continued from page 252.*)

Abstract from Report of Pathological Institute.

DR. A. MEYER.

In discussion of the cases of Korsakoff's syndrome the following observation is of note. "While all the cases showed a fairly typical delirium, only two had a frank polyneuritis, in some of our cases the polyneuritis appeared to be very mild or to have disappeared entirely before the patient came under observation. Such cases may offer considerable difficulty in differentiation from general paralysis, the difficulties in diagnosis are further increased by the frequent occurrence of pupillary abnormalities, jerky tremors and speech defect, the writing frequently showed poor spelling and omissions, but we have not observed the characteristic transposition of the paralytic. We have observed in practically all of our cases a nystagmus-like twitching of the eyeball which may be slight or very well marked. Lumbar puncture has been of considerable aid in the diagnosis. In none of our cases have we found distinct lymphocytosis. Five cases, two of which were very probably syphilitic, one giving a positive history, the other presenting scars, were examined and negative results obtained."

Again, we wish to quote from the report, the results of lumbar punctures in 11 cases of G. P. In nine cases there was a marked lymphocytosis; in the other two there was a distinct, but less pronounced reaction.

Lumbar puncture was also of aid in differentiating G. P. from central neuritis. "The patient, a woman thirty-nine years old, had complained of attacks of dizziness and weakness for a few months, and had shown some change in disposition. Three weeks after a fracture of a bone in the hand she suddenly became delirious and was brought to the hospital. She then showed an intense episodic agitation with fear, talked delir-

iously, yet remained relatively clear as to her whereabouts. At this time bilateral ankle clonus was observed with some muscular twitching in the arms. The delirious episodes subsided in a month; then for a period she was averse to examination, and seemed to have a generally hazy grasp on the past and claimed to know nothing about the injury to her hand. Her mood and behavior gradually became more normal, but she was simple in manner and lacking in initiative, yet, without evidence of G. P. The ankle clonus continued and later she began to have attacks of weakness in which she would fall over. Diarrhea developed, accompanied by progressive emaciation. She became again delirious; general rigidity appeared with strong muscular twitchings, and episodes of jactitation of the arms. There was indistinct articulation and the speech was finally reduced to an almost unintelligible sound. The L. P. showed no decided lymphocytosis. The patient died from exhaustion, but no autopsy was permitted. This case is classified as central neuritis."

We have examined a number of cases where the question of dementia præcox and dementia paralytica were under discussion. First in the case of a negress, who was discharged in 1902, as dementia paralytica, improved. At that time there were increased knee jerks, tremor of the tongue and hands and facial muscles, with slight static ataxia; the pupils, however, were normal; there was defective judgment, active hallucinations and some deterioration in memory. In 1904 she was readmitted. She has deteriorated in all fields, but the physical signs are very difficult to elicit, the pupils are normal and the spinal fluid has been twice negative. Early in the development of the case were hallucinations of hearing and smell and somatic-psychic delusions, but to further confuse the picture, were several epileptiform seizures. Regarding these, however, an investigation of the subject has shown that convulsions are not infrequent in dementia præcox, and we are of the opinion that this is the correct diagnosis.

In this next case dementia præcox, paresis and alcoholism came into discussion. In favor of dementia præcox were a gradual decreasing efficiency for work, delusions of somatic origin, marked emotional instability and hallucinations of sight and hearing. In favor of paresis were unequal and slug-

gish pupils, marked tremor of the hands, face and tongue, and absent knee-jerks. For alcoholism there was a memory defect for recent events, a tendency to fabrication, delusions of a persecutory trend, confused orientation, misinterpretations and hallucinations of sight and hearing. To further make the picture unclear the patient gave a history of convulsive attacks commencing about a year before admission. The patient had been markedly alcoholic, but at staff meeting the case was viewed with great doubt. The lumbar puncture was called upon to decide, and on the grounds of two negative results we ruled out the diagnosis of paresis and classified her as an alcoholic. At this time, five months since the puncture, the patient's condition shows the correctness of our diagnosis.

In another case of diagnosis of paresis was made some four years ago, and on re-admission in October, 1905, there were many symptoms such as active hallucinations, rather fantastic delusions and variable emotional tone which were in favor of dementia præcox. The physical signs particularly were not conclusive. A positive finding in this case pointed out that the previous diagnosis was correct.

We have had several cases where the question of a purely functional basis for the psychosis had to be differentiated from certain organic conditions. In one case there were marked tremors and exaggerated knee jerks, with a peculiar unstable emotional condition. The diagnosis at staff meeting of paresis was made. The fact that there is good memory and that the patient has developed delusions of a persecutory trend, coupled with the negative lumbar puncture, make the diagnosis of paresis extremely doubtful. With a history of alcoholism this would seem to be the more fitting classification. In several cases of hysterical manifestations, such as variable anesthesias, together with exaggerated knee jerks, with a certain amount of memory defect, the differential diagnosis from paresis has been extremely difficult. In one case a positive lymphocytosis has shown the correct diagnosis. In two others negative findings have guided our observation. In this same group one meets certain cases of an anxiety type, occurring in alcoholic women about the involution period, who present serious problems for diagnosis. In two such cases, besides the appearance of extreme anxiety, there was in one, a history of convulsive at-

tacks, marked tremors of the hands and face, unequal pupils and a peculiar speech defect. The lumbar puncture gave negative results, and the patients have since improved greatly; the tremors having entirely disappeared. In the other case there were delusions of grandeur, elation, mild deterioration with tremor of the hands, fibrillary twitchings about the mouth, and irregular pupils, but on the other hand there was considerable anxiety and agitation, with ideas of self-reproach which, with her age and coincidence of the menopause, made the diagnosis of involution psychosis very probable; at the same time the presence of memory defect and ideas of wealth were strongly in favor of paresis. The spinal puncture has shown absolutely negative results on two examinations, and the patient has improved somewhat. The eye condition has been explained by the presence of an error in refraction and a choroiditis.

There are certain cases of paranoic condition where the presence of grandiose ideas with certain physical signs are difficult to differentiate from the early stage of paresis. The presence of a megalomania with elated expression, tremor of the tongue and hands, with exaggerated knee jerks in a woman of forty-three years, presented a very confusing picture. The duration was three years. Onset following death of husband, became extravagant and forgetful, gave away $11,000 to various churches because of the peculiar delusional content; she imagined that the Pope would make her queen of a certain city in Germany provided she followed a certain charitable course. A pin which had been sent her from a sister in Europe came from His Holiness. Symbolic coloring was placed on all natural events. She received communications from the man in the moon and various air ships, and she developed a strong persecutory trend against a certain bishop who had obtained possession of the magic pin. Her orientation was correct. She adapted herself rapidly to her surroundings and our examination failed to reveal any defect in memory or grasp. She had hallucinations of sight and hearing. In this case there was no alcoholic factor. On the symptoms one could not rule out the diagnosis of paresis. The lumbar puncture was now called upon to decide and the examination of the spinal fluid showed an absolutely normal condition. The patient's conduct

since that time has sustained our diagnosis of paranoic condition.

Finally I would mention a case which at first showed marked symptoms of a manic type, but presented also prominent ideas concerning money, with marked elation, exaggerated knee jerks and tremors of the hands. There was typical flight and word association. The lumbar puncture in this case gave us quite a surprise, as the finding was positive. The patient has since shown evidences of deterioration, and our diagnosis of paresis is sustained. We have punctured one case of brain tumor which, for a time, was suspected as being a paretic; the negative result ruled this out. In another case where the symptoms were similar to paresis in development, the spinal fluid was negative, and the repeated convulsive attacks with the development of a peculiar aphasia have made the diagnosis of internal pachymeningitis the most probable one.

I feel that I have shown the scope and character of the cases in which the results of lumbar puncture are of value. I would like now to speak briefly of the dangers connected with the operation. Andre Maystre collected from a large literature 18 deaths following lumbar puncture, 14 of these were punctured for therapeutic purposes, and the large quantity of fluid withdrawn, 25 to 90 cc., was responsible for the fatal issue. Two of the cases can be discarded because death did not occur until several days following the operation. Of the four remaining cases the puncture was made in the course of diseases leading to certain death, and Maystre could not establish any connection because death occurred two days following the puncture. Also 20 cc. of fluid were removed. In the last case tuberculous disease in the spinal meninges was found and at autopsy an organized hemorrhagic clot was present in the cauda equina.

Nissl, in a large experience, knows of no fatal case following lumbar puncture which has been made for diagnostic purposes when only from 3 to 5 cc. of fluid have been withdrawn. The operation is contraindicated in cerebellar tumor. Quincke does not admit of even this exception, stating that if carefully done the low pressure in such cases is a valuable diagnostic

point, and he has punctured a case six times during six weeks, causing an amelioration of symptoms and disappearnce of the choked disc. Nissl's conclusions after a study of the results in the normal and in the insane are: the patient should not be punctured unless he can be immediately put to bed. Symptoms as a rule do not appear until 8 to 12 hours after the puncture. At that time headache, nausea and sometimes vomiting may occur, as a rule there is complete incapacity for work. The symptoms are made worse, but sometimes not felt at all, except on movement. They may last from 1 to 8 days. The patient may feel perfectly well, get up, and the rapidly-appearing headache causes them to seek their bed again. So long as they remain quiet in bed they are comfortable.

Upon the insane, 48 patients out of 112 punctures had marked symptoms, the duration was usually from one to two days, several cases lasted ten days. It must be mentioned here that in 62 of his cases ether was used as an anesthetic, and he does not mention what effect this might have had. It is noteworthy that no symptoms followed puncture in general paralysis. At the time of puncture a few patients complain of transitory stinging pain in the legs. I have not used any anesthetic save a few drops of a four per cent. cocaine solution in the skin, and this was only necessary in certain cases. Chauffaurd and Boidin, in 223 punctures, report no bad results, in three cases there was vomiting, in a few others headache. In our own series of 150 punctures, I have observed mild headache, vertigo and nausea in 15. Three cases of secondary syphilis suffered from vertigo for eight days, but they would not remain quiet. In the brain tumor case death occurred three days after the puncture. The patient had been suffering from marked pressure symptoms for some time, only 3 cc. of fluid were removed, and at autopsy there was a hemorrhagic focus of softening in the pons. No connection between the puncture and the patient's death can be proven. In eight alcoholic cases headache and vomiting were quite severe, but perfect rest in bed for a few days was all that was necessary. In the majority of the cases the symptoms were of a fleeting nature and if only small quantities of fluid are removed and the patient placed in bed little discomfort follows the operation.

CONCLUSIONS.

1. Patients should not be punctured unless they can be put to bed.

2. To be of definite value the puncture must be repeated two or more times, at an interval of at least ten days.

3. A constant negative finding is of more value than a positive one, for it rules out the presence of brain syphilis and parasyphilitic conditions.

4. In general paralysis the lymphocytosis is a constant and early sign and is usually associated with a heightened albumin content. The same can be said for tabes.

5. Lymphocytosis may occur in secondary and tertiary syphilis without clinical evidences of involvement of the nervous system, also it may occur in patients who give evidences from scars or other signs of old syphilitic infection. As a rule the cellular increase in such cases is far behind that observed in paresis and there is very slight albumin increase. Where inflammatory brain syphilis exists albumin increase may also appear.

6. In arteriosclerotic insanity a positive finding points to a syphilitic process, such as softened foci following specific arterial disease. In brain tumors a negative finding is the rule. If a positive finding occurs, a syphilitic basis for the process can be taken for granted.

7. Epilepsy shows negative findings; if otherwise the suspicion of brain syphilis is justified.

8. Alcoholism in all its varieties gives negative results, if the finding is positive and there are no signs of nervous involvement an old syphilitic infection is to be taken for granted. Where symptoms of involvement of the nervous system are present general paralysis or brain syphilis is to be suspected. It is questionable in some cases even when symptoms of involvement of the nervous system are not present, in a positive finding with albumin increase, whether we are not dealing with an early paresis.

9. A differential diagnosis is to be made between brain abscess and meningitis by the presence in the latter of increased cellular material.

10. It cannot be enough emphasized that the lymphocytosis presents a singular disease sign, and only after consideration

of all other clinical symptoms of the disease, should it be used to construe the case. When the findings are considered with due care to the possibilities, the results obtained from lumbar puncture are an important and ofttimes an invaluable aid to the diagnosis of obscure nervous and mental diseases. It is of especial importance in differentiating alcoholism, general paralysis, dementia præcox, epilepsy, brain tumor and finally brain syphilis. With the advancement of our knowledge of the occurrence of lymphocytosis in syphilis of tissues other than the nervous system, with further autopsy reports, and improvement in technique, we can look forward to the solution of many, at present, doubtful phases of the subject.

TABLE.

Diag.	No. of Cases.	Pos.	Mkd.	Mod.	S. I.	Neg.	Syph.
Gen. Par.	30	30	0	0	0	0	16
Doubtful Cases	33	14	1	6	12		4 Pos. 20 Neg. 9 Susp.
Alc. Clear	16	2	0	0	14		1 Pos. 1 Susp.
including Korsakoff	5	0	0	0	5		
SYPHILIS:							
Secondary	15	5	6	1	3		15
Tertiary	5	5	0	0	0		5
Ter. C. Psy. & N. Sym.	10	7	0	0	3		10
Ter. with Psy. & no N. Sym.	4	0	0	1	3		4
Epilepsy	3	1	0	0	2		1
Dem. Præ.	6	0	0	0	6		0
Brain Tumor	1	0	0	0	1		0
Pachymeningitis	1	0	0	0	1		0
Psy. fol. C. S. M.*	1	1	0	0	0		0
Hydrocephalus	1	0	1	0	0		0
Senile Dem.	5	0	0	0	5		0
	131	65	8	8	50		66

*Cerebrospinal meningitis.

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JOINT MEETING

OF THE

NEW YORK NEUROLOGICAL SOCIETY

AND THE

PHILADELPHIA NEUROLOGICAL SOCIETY.

Held in Philadelphia, Nov. 24, 1906.

The President of the Philadelphia Neurological Society, DR. D. J. MCCARTHY, in the Chair.

A CASE OF AMNESIA.

By C. W. Burr, M.D.

A man, 55 years old, was found by the police, confused, in the street, in November, 1900. He was taken to the Philadelphia General Hospital, where he had an attack of excitement and violence lasting a couple of days. Since then he has had no recollection of his past life for some years previous, and cannot remember anything that happens now even for a few minutes. The events of his childhood, youth and early manhood and the things that he learned at school he remembers fairly well. He knows that he has been married, but does not know whether his wife is living or dead, and says he has forgotten entirely what she looks like. When he is spoken to about his wife he becomes emotional, but in a few minutes forgets all about it. He knows who he is and has never had any alteration in personality. He is neat and clean in his habits, quiet in manner and shows no moral defect. He knows that he has lost his memory and knows that it is on account of disease. His intellectual judgment is much better than is usually found in senile or presenile dementia. His loss of memory is so great that if he reads (and he reads well) a paragraph in a newspaper he forgets the first sentence before he has read the last. Though he has been told many times that he is in the Philadelphia General Hospital he can never remember it. When in the ward he can reason out that he is in a hospital on account of the number of beds and the presence of people manifestly sick. When he is in the hospital office, however, he reasons out that he must be in some business house on account of the number of desks and clerks. He cannot tell five minutes after a meal whether he has had a meal or not, but reasons that since he is not hungry he must have been fed. He does not know the year, the month or the season of the year. He remembers the year of his birth, but does not know how old he is because, as he says, "I do not know what year this is." In the last few months a few things have made some impression on him, for he now vaguely realizes all the time that he is being treated in a hospital for his sickness.

Dr. B. Sachs said the condition was an extremely interesting one, and the only cases that he has seen that have been equally puzzling have been cases entirely different from the one presented. Two years ago he saw a man who developed an amnesia which was as complete as that of the patient presented, and even more complete. He lost memory of his own personality. He developed this amnesia as an early symptom of general paresis. A few weeks later he developed general paresis with very characteristic symptoms and passed more than a year as an inmate of an

asylum. Dr. Burr's case has lasted so long that there is no question of this sort. The difficulty in the class of cases Dr. Sachs spoke of and in the one he cited as an example was to decide whether the man was simulating. In his case there was a law suit pending and he thought the patient had good reason for forgetting his personality and for simulating, but the later developments proved that he was not shamming at all. He is now very near the final stage.

Dr. Morton Prince called attention to the fact that there were two types of continuous amnesia, one the functional form and the other the organic. Charcot was the first to call attention to the functional variety in the famous case of Madame D., who as a result of an emotional shock was found to continuously lose all memory of every experience as fast as it occurred. It was found, however, that the memories were not absolutely lost, but only dissociated, for the memories of her life were rehearsed in her dreams and were completely recovered in hypnosis.

In the organic type, that is in continuous amnesia depending on organic disease, the memories seem to be completely lost, or at least it has not been shown that they were simply dissociated and could be recovered. This form of amnesia has been frequently observed in Korsakoff's psychosis and in alcoholic neuritis. The speaker had observed one very extreme case following what was believed to be from the history of the latter disease, and also in a case that clinically appeared to be one of multiple sclerosis. As to the pathology of this organic form of continuous amnesia we know practically nothing. It is curious to note how a person with a marked continuous amnesia can get along without being markedly incommoded and without allowing it to be suspected. The case of multiple sclerosis, for example, had attended the hospital clinic for three months without the amnesia being suspected.

A CASE OF COMBINED POSTERIOR AND LATERAL SCLEROSIS, WITH INVOLVEMENT OF THE CELLS OF THE ANTERIOR HORN.

By J. W. McConnell, M. D.

Dr. Joseph Collins said he did not know whether it was the intention of Dr. McConnell to present this case as a new type of disease, but, if so, he was loth to accept it as such. Sufficient had been said about its occurrence and about the character of the disease to make it clear that it was a manifestation of diffuse syphilitic disease of the cord analogous to that Nonne had described in his last contribution. He could not say at the moment whether he had seen more than two or three cases similar to this or not, but he was quite sure he had seen that number. He understood that there were some sensory disturbances in Dr. McConnell's case. There must have been widespread, more or less disseminated, lesions throughout the spinal cord which entitled it to be classed as a disseminated syphilitic lesion.

Dr. C. E. Atwood saw one case of subacute combined sclerosis when an assistant of Gowers, and it corresponded very closely to this, but it was not so far advanced. It had similar paresthesia in various parts of the body, especially about the anus and genitalia and in the extremities. The age of the patient was about the same and there was, as in this case, a good deal of anemia. He believed that in these cases one of the earliest symptoms is a very pronounced anemia. There was in his case a condition of spastic paraplegia with anesthesia, girdle sensation, increased

knee jerks, Babinski, and inability to walk, as the disease advanced, from weakness and marked spasticity. The disease runs a rapid course and on autopsy marked wasting of the white matter in the cord is found. The disease may be due to some chemical toxic substance in the blood.

Dr. McConnell, closing, said he had not stated that these patients had cranial symptoms. One of the patients showed Argyll-Robertson pupil. He had not intended to describe any new clinical type, but it had occurred to him often that the appellation cerebro-spinal syphilis is such an indefinite one that we should have a better term. Other scleroses were spoken of as posterior sclerosis, combined sclerosis, lateral sclerosis. Why not give a clinical pathological diagnosis to our cerebrospinal syphilitics?

A CASE OF TABES IN A NEGRESS.

By John K. Mitchell, M.D.

Dr. Mitchell said he was sorry to say that he had just learned that the patient whom he had hoped to show had at the last moment refused to come to the College of Physicians, and he would therefore say only a very few words about her.

The only unusual features of the case and the reason why he wanted to exhibit her lay in the rarity of the occurrence of locomotor ataxia in a black of pure blood. In spite of the frequency of occurrence of syphilis among the blacks, ataxia is so rare in his experience as to be almost unknown, except in those who obviously have a greater or less share of white blood. It is probable, of course, that there are not a great many negroes in the country who have not some percentage of white blood, but so far as one can judge by appearance this ought to be one of the certainties, and the woman herself is entirely ignorant of any trace of Caucasian in her pedigree.

She has a Charcot joint in both knees and in one ankle. She has been under Dr. Mitchell's observation with the disease for three or four years. She is pretty nearly at a standstill and presents no symptoms of any peculiar interest.

Dr. S. Weir Mitchell regretted very much that his son could not present the case. There were three disorders of the nervous system which the negro escapes. This was certainly one. He thought chorea, again, was another. Chorea among pure black children was practically unknown in this country and Cuba, and he has had occasion recently to find from inquiries sent out that this was probably the case as concerns exophthalmic goiter. He found to his surprise that in Cuba it is exceptional and rare among the negroes. Physicians wrote him from Cuba that it is scarcely heard of among the pure-blooded blacks. These were things which the negro escapes.

Dr. J. A. Booth had seen six cases of tabes in the colored race, and they were all patients in the Colored Home in New York City. Among the six there was no question of four of them being of the pure colored race. He had also seen one case of chorea in the Colored Home.

Dr. L. Pierce Clark said he had reported in his second thesis on myoclonus-epilepsy a full-blooded negro patient who died of myoclonus-epilepsy. The case had been first diagnosticated as choreic epilepsy before admission to the Craig Colony for Epileptics, but the movements were typical of myoclonus as seen in the association disease. In an analysis of two thousand cases of idiopathic epilepsy in his service at the colony there were at least six full-blooded negro patients, about the

normal ratio existing between the races in the general population of the State.

Dr. H. M. Thomas said that in Baltimore were seen a considerable number of cases of tabes in black people, but he found it very hard to prove that they were pure-blooded blacks. He was sorry his statistics were not perfectly accurate on that account. Any person who had any negro blood was classed as black, and they came in among these cases. Many of them did not know their parentage and he had to judge by looks.

Dr. J. K. Mitchell said that it was difficult to be sure in any given case of negro that he had no white blood. He supposed there were few negroes in this country without any *taint* of white blood. This woman looked as if she had none, and when asked she said: "Oh, Gawd, I don't think so, both my grandmothers was mos' 'spectable women." Considering the frequency of syphilis among the blacks, the rarity of tabes must be very great. There are 70,000 negroes of varying degrees of darkness in Philadelphia alone, it was remarkable that no more cases should turn up at the nervous clinics, the case under discussion being absolutely the unique instance known to Dr. Mitchell of ataxia in a negro of presumably undiluted African blood.

A CASE OF PROBABLE APRAXIA.

By John H. W. Rhein, M.D.

Dr. Rhein stated that the man was fifty-five years of age and had been admitted to the Philadelphia Home for Incurables on July, 1906, complaining of loss of vision. The family history, as well as the previous history, was practically negative. Three years ago he began to lose his eyesight, and this loss progressed gradually until at the end of two years he was unable to read. At present he is unable to see anything but a bright light, and is not always sure of this. He has entirely lost his ability to recognize the position of his body and limbs. He cannot tell whether he is standing or sitting, or what position his arms or legs are in. He has lost entirely his sense of localization. He recognizes a light touch in certain places, but cannot locate it. When asked to place his right hand upon his nose or ear he places his hand upon his knees or his chest, stating at the same time that he is touching his ear or nose. When he is asked to squeeze the hand of the examiner with his left hand, he grasps his right leg with his right hand and squeezes, believing that he is squeezing the examiner's hand with his left hand. At the same time he does not move the fingers of his left hand whatever. When a watch is placed in his right hand, and he is told to place it to his ear, he does it frequently correctly, but some times puts it to his mouth. He does not recognize the ticking of a watch, but when asked if he knows what the object is he replies: "Yes, it is a watch." He states that he knows this because his reason tells him that the watch would be the object which the examiner would place to his ear to test his hearing. The tactile sense in his left hand appears to be lost; in the right hand it is present. Tests of the pain sense were rather unsatisfactory because they irritated the patient very much, and obscured the accuracy of the observations. It is probable that this sense is retained in both hands. He is unable to distinguish between light and deep pressure. He recognizes the difference between heat and cold in the right hand, but does not always recognize the difference in the left hand.

Dr. Neinan, the resident, found the thermal sense markedly impaired

all over the body. The stereognostic conception is impaired; he does not recognize the nature of objects placed in his hands. His gait is "pattering," he takes small steps, first with the right foot, and usually toward the right.

In a brightly lighted room, if a hand is placed before his eyes, while he is walking, he turns away from the hand without recognizing that an object is placed before his eyes. He does not recognize objects in a brightly lighted room, and will frequently bump into a table or the wall in walking alone. Both arms and legs are rigid, especially on the left side, although not truly spastic. The knee jerks are increased; there is no clonus or Babinski. The arm jerk is present on both sides. The station with the feet together is good. The grasp is good and equal on both sides. The tongue is protruded straight in the median line, and is tremulous and tooth-indented. The taste sense is markedly altered, while the olfactory sense seems practically unimpaired. He cannot write his name, and in making this effort makes a scrawl, writing from the right to the left.

Examination of the eyes by Dr. W. C. Posey revealed the early changes of simple atrophy of the optic nerves. There was no Wernicke's pupillary inaction and no palsy of the extraocular muscles. The pupils reacted to light. Dr. W. G. B. Harland reported that there was nothing in the examination of the ears that would not be found in an old man with mild middle-ear disease.

The patient is intelligent, answers questions promptly, and his memory appears to be good. He is well nourished, his appetite is good, his digestion unimpaired, and his bowels regular. He complains of nothing excepting his blindness. He has a quick temper, and becomes frantic when the examination is pushed beyond a certain point.

The case bears some resemblance to those cases of apraxia which have been reported. However, a further study of the case must be made before a diagnosis can be ventured upon. At present this much can be said, that bilateral lesions involving the occipital and parietal lobes would explain most of the symptoms which this patient presents.

Dr. A. Fraenkel, of Haiden, Switzerland, said that he imagined the case might have been one of functional disease, but as he saw the patient coming in, he was sure it was not functional, but that it was a sort of bulbar disease, and that it interested us to know whether he had any ophthalmological history. Dr. Rhein stated that he had very early stages of optic atrophy. This would not explain his blindness. There might be a double lesion in the occiput with symptoms of apraxia.

Dr. Joseph Collins said that those who attended the American Neurological Association would recall the type of disease which he had attempted to portray as a clinical entity, a discussion of which had appeared in THE JOURNAL OF NERVOUS AND MENTAL DISEASE. They would recall that the most striking characteristic of the disease was the alteration of the patient's appearance. The individual became transformed from a person expressing grace in movement, and relaxation in repose into an immobile, inanimate replica of the normal person. The second was that the cases first impress one as being paralysis agitans, and that when you came to dissect and analyze the one symptom above all was the immobility. There was no motorial function of the body which might display the disorder. This immobolization gives a characteristic attitude and gait, and to a lesser degree a characteristic physiognomy. The gait is the most remarkable feature of the patient. The stride is short, oftentimes only a

few inches, the feet slightly lifted, the rhythm generally slow, occasionally rapid. One case he had complained of impaired vision. The disease was clean-cut in its delineation, quite as much so as Parkinson's disease. He has had several autopsies, and has had six or seven brains which show the distinctive lesions. What the lesion is it was imposible to describe in three or four minutes. Some changes were confined to the motor areas and others were distributed throughout the entire brain. The disease was that which Charcot probably had in mind when he described abasia trepidant; afterward an attempt was made by a Polish clinician Bieganski to describe it. In hospitals where many patients were undiagnosticated it had been forced upon him that there was a type of disease of which this man is the best exemplification he had ever seen, that has a definite clinical type and which must at the present time be called a definite form of general cerebral arteriosclerosis with special symptoms. The patient now exhibited seemed to him to be a fairly typical example of the disease.

Dr. Joseph Fraenkel said that the cases he had seen did not fully coincide with the description given by Dr. Collins. Those described originally by Charcot under the name of "gait stuttering and stammering" which manifested a condition like the patient presented, he had never seen except once. He thought the same patient was presented in New York. They did not make a diagnosis of his condition then.

Dr. Rhein said that he had not yet had an opportunity of looking into the literature exhaustively, but that there were three cases recorded which were in many respects similar to the one exhibited. One was a case reported by Dr. S. Weir Mitchell, a woman giving a history of gradually increasing blindness, together with loss of the stereognostic sense. She died under Dr. Pershing's care in Chicago, and an autopsy revealed the presence of cysts situated in both occipital regions, and general sclerosis of the cortex. Another case was reported by Dr. Charles W. Burr, without autopsy, in which the patient had progressive blindness and tactile amnesia.

The third case was described by Liepmann under the title of "Unilateral Amnesia." In his case there was degeneration of the occipital and parietal regions, together with portions of the corpus callosum.

Dr. Rhein thought that, as far as he had gone into the study of the case, the idea suggested by Dr. Fraenkel that the lesion was in the occipital lobes, was in part, at least, the best explanation of the case, although he himself inclines to the view that the parietal, as well as the occipital lobes, are involved.

A CASE OF PROBABLE PARALYSIS AGITANS IN A BOY OF TWELVE.

By T. H. Weisenburg, M.D.

The speaker stated that the family history and the past social history of the patient were uninteresting. The boy had the ordinary diseases of childhood, and up to a year ago was considered normal. At this time, while going to school, his teacher noticed that the boy became somewhat stupid and slow mentally, and that he was not as rapid in learning as he had been. The teacher also noticed that his speech became more or less monotonous and indistinct. About the same time the boy began to have a shuffling gait, and every once in awhile would trip over slight objects. The boy at present has a stiff attitude, he holds his body rigidly, head bent over, arms held to his side. When he walks the body is inclined forward,

the steps are short, shuffling, and he has a tendency to slip over the slightest object. His face is mask-like, suggesting strongly the facies of paralysis agitans. His speech is slow and monotonous and hard to understand. He has dribbling of saliva almost constantly. He smiles very rarely and he hardly ever cries. His mental condition is slow; he responds to questions fairly well, but it is evident that as compared to a boy of his years and opportunity for education he is undeveloped mentally. There is no apparent coarse tremor, although he has fine tremor of the upper limbs and sometimes of his head. He holds his hands in flexion and somewhat in the position of a case of paralysis agitans, but there is no pill-rolling movement. Any sudden push forward or backward will cause him to fall. There is no festination, although others have observed a tendency to it. The reflexes are increased generally, and sensation is normal.

The patient, from appearance, attitude, facies, tendency to shuffling gait and falling forwards and backwards suggests paralysis agitans. It is rather difficult to say at this early date and in such a young person whether the case is a true one of this character or whether it is a forerunner of some other disease as multiplesclerosis. There is no denying the fact, however, that it strongly suggests paralysis agitans.

Dr. Charles Dana said that about fifteen years ago a boy came into his hands with symptoms very like this case. The boy was about this age when he first came and was finally transferred to the Montefiore Home. He thought the picture had gradually changed to multiple sclerosis. It seemed to him that this was precisely what this boy's condition would be ten years from now.

Dr. Joseph Fraenkel stated that he and Dr. Sachs had had a case of paralysis agitans in which the diagnosis had been made by the children with whom the child played, long before they had made it. The boy's mother had told Dr. Fraenkel that his schoolmates called him "false face." He presented for a number of years the most characteristic picture of paralysis agitans that he had ever seen. The only feature not characteristic of paralysis agitans was nystagmus. Later the patient began to show evidences of disease of the pyramidal tracts; markedly exaggerated tendon reflexes and ankle clonus. Three years ago Dr. Turner, of London, suggested to the speaker the possibility that cases of this kind were some type of Little's disease—cerebral diplegia.

Dr. Sachs said that the case resembled that which Dr. Fraenkel referred to very strongly, but he felt great hesitation in arriving at a diagnosis in a case he had seen for only a few minutes. He thought he was one of the first to see the case that Dr. Fraenkel referred to, and suggested that it was a peculiar fact that when a condition resembling paralysis agitans occurred in a younger individual it should assume the form of multiple sclerosis. Paralysis agitans was much more common in the later periods of life than in the early. All the symptoms here suggested paralysis agitans. It would be interesting as years go on to follow developments in this case. In the boy's face there was a suggestion of scleroderma, but on examination of the hands he thought there was nothing of the sort. We should be careful about establishing new types. It was interesting to note what an unusual number of atypical cases occur which it is not easy to label one way or another.

A CASE OF HEMIATROPHY OF THE FACE.

By A. Gordon, M. D.

The gradual wasting of the muscles followed an attack of two days' duration of neuralgic pain in the infraorbital region on the right side. Now, eight years after the onset, the atrophy is complete and the reaction of degeneration is distinct. At no time were there vasomotor disturbances on the affected side, and the pupils are equal, a fact which militates against the sympathetic origin of the disease in the present case. Dr. Gordon reviewed other theories, criticizing them, and concluded that facial hemiatrophy may be caused by involvement of the lower sympathetic ganglion, by the fifth nerve, by the seventh, by the Gasserian ganglion, finally by a cerebral lesion.

Dr. M. Allen Starr asked if any one present had ever noticed any facial hemiatrophy following operations on the Gasserian ganglion. He had never seen one. In only one case of hemiatrophy had he seen neuralgia symptoms referable to the fifth. He had considered the advisability of exsecting the ganglion in hemiatrophy, but unless it is proven that the fifth ganglion has a relation to hemiatrophy he thought it would be a rather risky operation.

Dr. Spiller, in replying to a question from Dr. Starr, said he had never seen a case of atrophy resulting from operation on the Gasserian ganglion.

Dr. Dana said that this was a typical case of hemifacial atrophy. The atrophy was very slight. It was progressive; that several of the cases he had seen had been associated with scleroderma and this patient's face suggested it. He had observed a number of cases where the Gasserian ganglion had been removed for a long time without any effect upon the trophic condition of the face.

Dr. Morton Prince said that an alternative hypothesis must be considered; namely, whether it was not rather a case of muscular dystrophy rather than of so-called ganglionic hemiatrophy. In most of the cases of hemiatrophy reported the bones and other tissues were involved. In this case this did not seem to be the fact, but the atrophy was limited entirely to the facial muscles which were paretic. He believed therefore that the theory of muscular dystrophy must be entertained, or possibly the case was one of simple facial paralysis.

Dr. Knapp said that his own experience coincided with that of the other gentlemen who have spoken; that he had not found hemiatrophy following lesions of the cervical sympathetic or operations on the fifth nerve. The patients which he had seen had had no paresthesia. This case seemed to him rather peculiar on account of its general diffuse character. In the early stages of hemifacial atrophy he had noted, as others had, the involvement of bone. In one case in particular the trouble began with a marked wasting of the tissues in the forehead and bone as a depressed band right down the forehead close to the medium line with a diffuse wasting of the rest of the face. A general diffuse wasting like this without bony involvement he had not seen in a case of hemiatrophy.

Dr. Sachs said that he was impressed with the fact that this might belong rather to the class of cases spoken of as pseudo-hemiatrophy. There were marked sensory changes in this case, not of syringomyelic type. This case seemed parallel with two or three cases he had observed closely for years in private practice which began with very marked pain within the trigeminal area; they suggested syringomyelitic disorder. In

one the marked trigeminal symptoms had been followed by atrophy just as was seen in this case, an atrophy which was muscular rather than an atrophy that involved the subcutaneous tissues, the bone and so on, as was the case in progressive hemiatrophy. These cases he referred to he had not been able to label properly. He remembered that Dr. Jacoby some years ago thought that they might be incipient cases of tabes, but up to the present time no symptom of tabes had developed. They were surely cases of trigeminal disease no other symptoms following.

Dr. Adolf Meyer asked if there had been any electric reaction of degeneration in the facial nerve.

Dr. Gordon, in closing, replied that Dr. Prince's remark concerning the possibility of a muscular dystrophy cannot be accepted in this case for the reason that the duration of the disease has been eight years and further, the disease is confined to the face. His arms and the muscles of the neck are absolutely intact. The reaction of degeneration is very pronounced. Therefore the duration of the disease for eight years and the lesion being confined exclusively to the face throw out the diagnosis of muscular dystrophy.

As to another possible origin of the disease, Brissaud suggested the possibility of syringomyelia of the medulla. The patient does not present sensory symptoms corresponding with that. There is no vasomotor disturbance so as to make us think of the sympathetic system. As to the question of operation, Dr. Gordon did not see what operation could accomplish under these circumstances.

(*To be continued.*)

NEW YORK NEUROLOGICAL SOCIETY.

October 2, 1906.

The President *pro tem*, Dr. Adolf Meyer, in the Chair.

A CASE OF SUBCORTICAL TUMOR.

By Richard Weil, M.D.

The patient was a man, forty-two years old, a Russian, who, three months before he came under observation complained of severe headaches from which he could obtain no relief. About the same time, some impairment of the mental faculties was noticed, and there was impairment of vision which rapidly progressed to total blindness.

During the first two months of his illness he also had two convulsions. According to the history given by his wife, he would wake up in the night, complaining that his left arm was shaking. From the arm, the movements would progress to the corresponding leg, and finally the entire left side would become involved, and unconsciousness would supervene.

When the man entered the hospital, the optic nerves showed a secondary atrophy. The physical examination revealed very slight impairment of the motor functions. On the left side, the reflexes were slightly increased; there was no clonus in the patella nor ankle. Power was unimpaired; no ataxia; no Babinski. He had perfect thermal sensibility, and the pain sense was unimpaired. There was only one pronounced symptom, namely, astereognosis, which persisted up to the time of operation, and he was unable to

distinguish the form or character of any body that was placed in his left hand. This symptom was constant, invariable and very pronounced. The exact condition of the muscular sense could not be determined.

Upon the strength of the above symptoms, a diagnosis of cortical brain tumor was made, and localized, according to Mills, in the right superior parietal, and the skull opened in the region indicated. The result was absolutely negative. No tumor was found, and the man died two days later of shock. At the autopsy, a subcortical tumor, situated at a considerable depth, was found. It was a glioma, originating in the posterior part of the lateral ventricle. The white fibers were scarcely involved at all, and the tumor was nowhere near the cortex.

Dr. Weil said this was the only case he knew of in which a tumor situated as deeply as was this one had given rise to astereognosis; it was also remarkable inasmuch as this was the only symptom of localization.

Dr. B. Sachs said the case reported by Dr. Weil was particularly interesting on account of the diagnosis. The symptoms that were present would certainly have induced anyone to make the diagnosis of a localized cortical tumor. In addition to the astereognosis, the occurrence of convulsive seizures would point to cortical irritation; in fact, all the symptoms were those generally associated with cortical tumor. In view of the autopsy findings, Dr. Sachs said that the most plausible explanation of the cortical manifestations was that they were the result of the pressure exerted by the subcortical tumor upon the adjacent parts, thus interfering with the fibers that ordinarily conducted the stereognostic impressions from the gray centers.

The Chairman, Dr. Meyer, said that judging from a rather hurried inspection of the specimen shown by Dr. Weil, he inferred that the tumor evidently was in close proximity to the cortex on the mesial side of the hemisphere, and to the corpus callosum.

Dr. Weil, in reply to a question, said that an incision was made into the cortex at the time of operation, but nothing could be felt. In reply to Dr. Meyer he said that the corpus callosum overlaid the tumor and did not seem to be involved. There was certainly a considerable distance between the tumor and the cortex. The tumor originated in the lining epithelium of the ventricle, and destroyed only a small area of white. The most interesting feature of the case was that its effects should have been limited to the area giving rise to stereognosis. There was probably some interference with the fibers conducting the stereognostic sense, as suggested by Dr. Sachs: that explanation was more likely than to attribute the symptoms to cortical compression. On the other hand, the Jacksonian attacks certainly pointed to a cortical irritation; but the fact that the irritative motor symptoms were completely absent from the picture except for the two epileptiform explosions seems to point rather to sudden and passing changes in the relation of the tumor to the brain, as by a sudden increase in size through edema.

FUNDAMENTAL CONCEPTIONS OF DEMENTIA PRÆCOX.

By Adolf Meyer, M.D.

In this paper, the author gave an outline of the main facts of dementia præcox, which showed the fundamental importance of a consideration of the mental factors, the habits and ways of thinking of a patient, for an understanding of the disorder and prophylaxis and treatment. While

Kraepelin seemed to despair of such an explanation, and simply created a disease-entity on the ground of some final symptoms, such as negativism, mannerisms and stereotypes and disorders of volition, and appealed to the concept of autointoxication, he missed many factors which furnished a more intelligible and natural picture of the condition. As soon as we abandon the schematic use of conventional psychology and observe the actual experience and modes of mental activity of the patients, we see that most of the symptoms appear as perfectly natural results, not of abstract and so far undemonstrated auto-intoxications, or supported merely by fragments of histological knowledge, but of habits of function and mental activity which may in part open a chance for correction and should be pushed to the front, so that that danger could be recognized in time. As long as consumption was the leading concept of the dreaded condition of tuberculosis, its recognition very often came too late to make therapeutics tell; if a few symptoms and the outcome in dementia are the leading concept of a disorder, the physician will not think of it until dementia is established, and a declaration of bankruptcy drives him to fatalism. To-day, the physician thinks in terms of tuberculous infection, in terms of what favors its development or suppression, and long before "consumption" comes to one's mind, the right principle of action is at hand; the change of habits, of breathing poor air, of physical and mental ventilation, etc. In the same way, a knowledge of the working factors in dementia præcox will put us into a position of action, of *habit-training*, and of regulation of mental and physical hygiene, as long as the possible "mental consumption" is merely a perspective, and not an accomplished fact. To be sure, the conditions are not as simple as with an infectious process. The balancing of mental metabolism and its influence on the vegetative mechanisms can miscarry in many ways. *The general principle is that many individuals cannot afford to count on unlimited elasticity in the habitual use of certain habits of adjustment; that instincts will be undermined by persistent misapplication, and the delicate balance of mental adjustment and of its material substratum must largely depend on a maintenance of sound instinct and reaction-type.*

In the antecedents of cases of dementia præcox, one invariably finds where the facts are sufficiently well known, that the individual had abnormal ways of dealing with the situations of life, an inability to get square with events, and a tendency towards false adjustments. At first, perhaps, there is merely an excess of substitutive reactions, such as occur also in the normal, a shirking and scattered and distracted slurring over of the difficulties, secretiveness, instead of a frank ventilation and correction by joining the activities of the normal, a habit of excusing carelessness and lack of determination by hypocondriacal complaints or fault-finding with others, or the habit of passing over difficulties by imaginative thoughts, or mere praying, or pondering, or other expedients which as a rule help successively over an individual disappointment; or we find much more serious reactions, such as blind tantrums, or hysteroid outbreaks, or a mechanism of partial suppression, brought out so well by the studies of Breuer and Freud, and by the Zurich school; the creation of undercurrents of uncorrected false lingering attitudes, which form the foundation for the more serious developments which are almost pathognomonic of a disorder which marks the over-stepping of the elasticity; a disruption of judgment only insufficiently accounted for by any special mental or physical upset (*i. e.*, without any evidence of intoxication or other delirium, or without the manic-depressive thinking disorder, or the foundation of the hysterical or epileptic

disorder), discrepancies between the mood and the general reaction, peculiar attention disorders and feeling of interference with thinking and deterioration in matters which are largely dependent on sound instinct, such as differentiation of the real and unreal, and the critique or imaginative material; all this in the face of relative clearness, so that we are forced to think of a fundamental deterioration or defect as the only means to account for so much perversion of instinct and reasoning. In connection with this, there appear a number of symptom-pictures, also met with occasionally as more or less *adequate* excusable and corrigible reactions, such as states of puzzle, of religious and mystic fascination, of automatic and stuporous states such as can be in part obtained by hypnotic suggestion, or such as arise as psychasthenic and hysterical reactions (as we call them when they appear on sufficient and characteristic foundation). The *insufficiency* of the provoking factor, and the *oddity* and *incongruity*, rather than the mere excess of what might be the result of a sufficient cause in an average person constitutes one of the most important criteria for the estimation of the seriousness of the process.

It is striking how, in well observed cases, all the symptoms and events stand out as necessary results of small beginnings, the knowledge of which is more important to the physician than the mere recognition of irreparable end results. As long as physicians are satisfied with such terms as dementia præcox; that is, a mere statement of an end result like consumption, and as long as they disregard the factors and conditions which work towards this final result, they miss their opportunities for action, and merely satisfy their empty desire to have a name for a disorder which is to be handed over to the asylums in a spirit of fatalism. The name is unessential and only preliminary, and will have to be substituted by terms which designate specific mechanisms of work—the hysteroid reaction, the abnormal habit reaction, the type of attention-defect and of judgment-defect. The term dementia præcox will then become unnecessary, or will indicate merely the perspective, that which is likely to come about, but by no means necessarily so. Etiologically, the constitutional make-up counts for a great deal; but not in the vague sense of heredity and degeneracy merely. There is much more to be had in the *study of deterioration of the habits and undermining of instincts and their somatic components.*

Dr. Meyer warns against elusive hopes in histological explanations, in auto-intoxication guesses and the tendency of the physician to *over*-correct common-sense, to rule out mental reactions from the domain of legitimate study, and reduce all mental diseases to the paradigma of general paralysis. If he continued to believe that the only condition of mental health lies with the proper amount of indican and the ideal status of the parathyroid and a few other glands which lack of knowledge makes a home for a wealth of theories, he misses his chance with the great percentage of simple dementias. Mind, like every other function, can demoralize and undermine itself and its organ and the entire biological economy, and to study the laws of the miscarriage of its function and life, is *one* of the conditions for any true advancement in psychopathology.

Dr. B. Sachs said that a few years ago he had placed on record his opinion regarding Kraepelin's concept on dementia præcox, and he had since then seen nothing to disabuse him of the views held at that time; on the contrary, they had rather been confirmed.

In regard to what should be implied by the diagnosis of a disease, to which Dr. Meyer had referred, Dr. Sachs thought the term should necessarily cover only two factors; namely, the morbid process and the clinical

entity. Those two factors had to be included in every diagnosis; otherwise it could not be properly considered.

The speaker said the only objection he could raise to the diagnosis of dementia præcox, aside from the fact that the term was unfortunate and implied altogether too much, was that while deterioration was the central figure in a certain proportion of these cases, it was not so by all means in all of them. Kraepelin himself admitted that there was no dementia in 17 per cent. of the cases in which he made the diagnosis of dementia præcox. A serious objection to the term was the inevitable ease with which the diagnosis was made, and the presumption that the symptoms in a typical case necessarily implied a great dread of chronic and serious deterioration of the mind. This was the one feature of the entire subject that had always struck him most forcibly, and he could not admit that the term dementia was justified in a considerable number of the cases now tabulated under the title of dementia præcox. Still, he was willing to admit that a great stride in advance had been made through the influence of Kraepelin's studies, and that it was far better to accept this one general clinical term, and to recognize a certain relationship between a great many different disorders, which was formerly not properly recognized.

Dr. William B. Noyes said that the paper of Dr. Meyer was worth most careful consideration, and was the first discussion of a mental disease before the Neurological Society, where the purely psychological side of the subject had been given due weight. In New York and in America in general, neurologists and alienists have not given adequate attention to psychology. He thought that the confusion that existed in our understanding of dementia præcox was due to the fact that even while some attention had been paid to normal psychology, developmental psychology, which included a study of many defectives, queer children, etc., has been largely ignored, and it was among these that Dr. Meyer is demonstrating that dementia præcox has its inception. In the asylums, on the other hand, we had to deal with terminal cases, in whom the diagnosis was well established. In order to get a proper conception on the subject, the cases had to be studied from the beginning.

There are certain terms that serve as a catch-all for a large number of cases. Some years ago the word degenerate was used in medical and general scientific literature to include a large class of abnormal types. Out of this class of degenerates may be recognized a large class of special cases that if we know the outcome, are to be eventually called dementia præcox. It is of paramount importance to recognize these early.

Five years ago, Dr. Noyes said, he thought he had a fairly clear idea of what was meant by dementia præcox. Now he was much more uncertain in regard to the class of cases in which that diagnosis should be made. It seemed that these cases differed widely in their etiology and outcome, as well as in their clinical manifestations. The diagnosis was a very broad one, and could only be properly comprehended by a study built upon normal and abnormal psychology.

Dr. Smith Ely Jelliffe said that there were two phases of Dr. Meyer's paper which appealed to him as being of more than usual interest, and in which he heartily concurred with the presentation of the evening. These concerned themselves with Dr. Meyer's remarks on diagnosis and on prophylaxis. So far as diagnosis was concerned, he was in sympathy with Dr. Meyer's standpoint, which was philosophical, and quite apart from a mere academic presentation of the subject. Just what constitutes a

diagnosis from a biological point of view in dementia præcox was by no means a simple matter to answer off-hand. In the shifting lights of psychological tendencies, it was difficult to pick out stable features which permit of the construction of a disease entity, and those who had studied what Kraepelin has termed dementia præcox from many points of view had emphasized this difficulty. He felt that more attention could be centered on the question of what constitutes a diagnosis with advantage.

The objection that Dr. Sachs had raised to the term dementia præcox, because, according to one author, the cases temporarily classified thus did not show dementia in 17 per cent., he felt was of little weight. While it is desirable to have as good names as possible, even for shadowy entities, discussion of the name rather than the essence of the thing was fruitless. Some so-called fevers run their course without fever, but one symptom in a complex being absent in some individuals surely ought not to modify the abstract idea of the complex, as embodied in the word used for the diagnosis, or temporary grouping.

Dr. Jelliffe thought that Dr. Meyer's analogy to the prophylaxis of tuberculosis, and its detection in an early stage, if rational therapy was to be instituted, was a crucial point. He had hoped that if possible, Dr. Meyer might present some psychological foundations, if such had been observed, whereby the pre-dementia præcox stages might be recognized. Were there mental types which reacted disastrously to their environment, types that might be classified by any of the newer modes of investigation of mental character? Were there certain memory types, certain reaction types, certain association types, which in line with Dr. Meyer's idea of a habit psychosis might offer a clue as to the very early stages of a deteriorating process?

Dr. Joseph Collins said that if he had understood the speaker aright, what he desired to impress upon us was that there was great incumbency on the part of the physician to interpret small signs indicative of the future occurrence of dementia præcox, occurring remotely anterior to the apparent beginning of that disease, to weigh with care and to place in proper perspective each particle of evidence that might be construed to indicate the disordered mind, and, especially to estimate it from the psychological point of view. We probably need to have this constantly brought before us, but for his part he would say that these patients are almost invariably seen by the general practitioner, who cannot possibly have the training, even though he have the inclination, nor the perspicacity either to detect these signs, or to estimate their importance. If, when such patients displaying trifling aberrance of act or conduct which parents construe as evidences of misbehavior, are brought to the alienist or to the student of psychological medicine, then no doubt many of these cases would be detected very much earlier than they are to-day; and, moreover, the foreshadowing of the disease which we call dementia præcox and its early manifestations might become much more familiar. The speaker said that he did not understand that the validity of the term of dementia præcox was under discussion; despite this he said that he had a succinct objection to the use of the term, not based upon any academic discussion upon what the word diagnosis meant or should mean, but upon the significance of the term in the minds of physicians and the laity. It is now generally admitted that dementia præcox occurs in some cases without recognizable dementia, and that dementia præcox recovers in a very considerable proportion of cases, but despite this it is, he believed, the conviction of the general practitioner and of the laity that the disease carries with it the

stigma of mental impotency which the victim will carry to his grave, and that it is in general an incurable disease. Aside from this, he saw no particular objection to the term *dementia præcox*. Likewise, he saw no particular advantage of it over the term which was supplanted; viz., hebephrenia, and especially when the latter was used without a qualifying adjective to describe its attributed origin from factors which we now know were symptoms and not etiological factors of the disease.

Dr. Henry Rafel asked Dr. Meyer to illustrate by examples what he meant by "bad judgment," "improper reaction to life," and the other general qualities which, according to Dr. Meyer, render possible an early recognition of the disease or of the certainty or probability of its occurring later. Cases of dissolute conduct are often diagnosed as dementia præcox, but juries will not agree, as men of the world allow as normal a much greater latitude of conduct than closet-students.

Dr. Meyer, in closing the discussion, said that whether we called this condition dementia præcox or something else was of comparatively secondary importance. The important feature was the utilization of the psychological events that led to the formation of this group of cases. Kraepelin was very wise in starting from terminal dementia in shaping his general picture of dementia præcox. But our task is to study the group with more reference to etiology and to the mechanism which leads to the end result of dementia. If the term appears unsatisfactory, we can explain to the public that every physician sees a certain percentage of the cases recover, and our duty as physicians is to learn to distinguish in the individual case whether it is one doomed to dementia or one with chances of recovery; moreover, we must not allow the achievement of the diagnosis to stand in our way of establishing these facts, modification and correction of which brings the chance of recovery. We thus remove the pessimism and fatalism, and push to the front that which is to help to the practitioner and an inspiration for progressive work. A brief reference to a case pointed to the necessity of paying attention to matters usually overlooked, but the only means of prophylaxis and of avoidance of relapse that is at present available to our knowledge.

THE PSYCHIATRIC CLINIC AT MUNICH, WITH NOTES ON SOME CLINICAL PSYCHOLOGICAL METHODS.

By G. H. Kirby, M.D.

The writer gave first a brief description of Prof. Kraepelin's new clinic, and the organization of the medical work. In the wards, one is impressed with the prevailing quietness and good conduct of the patients; this Prof. Kraepelin ascribes entirely to the methods of treatment in vogue, especially to the dormitory plan for observation, the rest in bed for all new cases, and the use of continuous baths to quiet excitement. Many voluntary patients are admitted to the clinic, and a legal process for detention is resorted to only when necessary.

Each physician on the staff, besides looking after his service, carries on some definite line of investigation, he has moreover, three months in each year to devote entirely to research work.

The value of "secondary light reaction" as a pupillary sign in general paralysis was mentioned. Some of the recent work in lumbar puncture was reviewed. By the addition of formalin to the fresh cerebro-spinal fluid before centrifuging, preparations are obtained which allow a more

satisfactory differential cell count. Fischer has reported twenty cases in which the findings in the fluid during life were compared with the condition of the meninges post-mortem. When a lymphocytosis predominates in the fluid, so can a lymphocytic infiltration of the membranes be demonstrated; if the fluid is rich in plasma cells, then this type of cell predominates in the meninges.

Kraepelin was the first investigator to apply the methods of experimental psychology to the study of mental diseases. There are a few methods in use which can be applied easily and quickly enough to be practicable in any insane hospital.

As a measure of the amount of mental work which an individual is capable of doing, the method of continuous addition has been developed by Kraepelin and his pupils. With this method, we have a simple yet probably the best way of estimating fatigue and at the same time we are able to study the influence of practice, and determine the restorative action of a pause during the work. Specht studied the traumatic neuroses with this method, and thought the absence of improvement with practice was indicative of dissimulation.

Apprehension and retention are studied with an apparatus having a shutter arrangement, and by means of which a number of letters are exposed to view for a known space of time. The "writing balance" of Kraepelin has been used in the study of certain symptoms, especially inhibition and retardation; *e. g.,* in katatonia and manic-depressive insanity.

The association tests offer one of the most important aids which we have to-day in the analysis of certain mental states. Jung has especially developed this method, and has shown how it can be used to discover undercurrents of thought or hidden complexes existing in the patient's stream of mental activity, especially in hysteria and dementia præcox.

Dr. Pearce Bailey said that he had had the good fortune to spend a few months at Munich during the past spring. He had found Prof. Kraepelin extremely cordial and accommodating. In addition to the weekly visit to the wards of the hospital, which gave the visitors an opportunity to examine and discuss the various cases, Kraepelin gave regular clinics on psychiatric and medico-legal subjects. Among the cases observed there, those classified as dementia præcox were in the majority. There were also many cases of manic depressive insanity and general paresis, as well as the alcoholic forms of insanity. Other types of mental disease were not particularly frequent. Dr. Bailey said he saw no cases of involution melancholia, and only one which was pronounced paranoia. The speaker said he was struck by Kraepelin's views in regard to hysteria, to which group he gave a very much larger scope than we were accustomed to do in this country. For example, he ascribed the commission of many petty crimes to the trance states of hysteria.

In referring to the statement made by Dr. Kirby that the patients under Kraepelin's care were unusually docile and quiet, Dr. Bailey said he was inclined to partially attribute that to their low order of mentality, their intelligence having been stunted by generations of beer drinking; and also to the fact that centuries of strict military regime among the people, especially in Bavaria, have taught them to heed authority. This, he thought, rendered the patients much easier to handle than those in our country. He said that what the student in psychiatry missed in Kraepelin's clinic were the borderland cases—those cases in which it was difficult to say whether the individual was insane or not.

Dr. Jelliffe spoke of the pleasure he had had at the Munich clinic, especially with reference to the unexcelled opportunities for laboratory work. The Munich clinic had at least four times as much proportionate floor space devoted to research laboratories as any other psychiatric clinic. The equipment was particularly ample. In addition to the features mentioned by Drs. Kirby, Bailey and Gregory, he had been interested in the cinematograph in use for registering psycho-motor manifestations, and also Dr. Weiler's excellent cinematographs of pupillary reactions. He also spoke of the Eglfing Asylum outside of Munich, and called attention to the system of exchange of assistants between Prof. Kraepelin's clinic and the Eglfing Asylum. He also commented on the sensible plan of allowing a clinic assistant a three months' respite from ward duty in order to carry on special research.

Dr. M. S. Gregory said he considered Kraepelin's clinic one of the best in Europe. In other parts of Germany he did not think the institutions for the insane were as good as those in our own country, so far as the care of the patients was concerned. In scientific work, however, there was no doubt that both in Munich and in some institutions in other parts of Germany they were far in advance of this country.

Dr. Kirby, in closing, said he did not think the docility of the patients in Kraepelin's clinic could be altogether explained on the grounds advanced by Dr. Bailey; they certainly would not hold good in the case of the female patients.

Periscope

Archiv für Psychiatrie und Nervenkrankheiten

(Vol. 41, No. 2.)

15. Atypical Alcohol Psychoses. F. CHOTZEN.
16. Psychical Disturbances in Multiple Sclerosis. RAECKE.
17. Inanition in the Course of Mental Diseases and Their Cause. G. DREYFUS.
18. Unilateral Disturbances in Genuine Epilepsy. EMIL REDLICH.
19. Pathological Anatomical Alterations of the Brain in Leprosy, Leprosy Bacilli in the Gasserian Ganglion and the Anatomy and Pathology of Nerve Cells of the Brain in General. HUGO STAHLBERG.
20. Amyotrophic Lateral Sclerosis Combined with Multiple Brain Cysticerci. E. MEYER.
21. Agrammatism and Disturbances of Internal Speech. KARL HEILBRONNER.
22. A Contribution to the Opium-Bromide Treatment of Epilepsy following Flechsig (Zichen's Modification). P. SCHIRBACH.
23. The Korsakow's Symptom Complex in Brain Syphilis. L. ROEMHELD.
24. An Hitherto Apparently Undescribed Malformation of the Spinal Cord. A. WESTPHAL.
25. Syphilitic Disturbances of Sensibility on the Trunk. KNAPP.
26. Description of a Dermograph with a Report of Results of an Investigation Made with It on School Children. P. PRENGOWSKI.

15. *Atypical Alcohol Psychoses.*—Chotzen, after a detailed analysis of psychoses associated with alcoholic excess, together with the report of a large number of illustrative cases, reaches in part the following conclusions: In the present state of our knowledge of psychoses differential diagnosis is often difficult to make. To attribute chronic paranoid psychoses to alcohol is difficult because they cannot be distinguished from the psychoses of abstainers. The same is true of various conditions of alcohol hallucinosis. In Kraepelin's description of the hallucinatory conditions in drinkers various disease processes may be included whose clinical identity is not assured. The so-called Korsakow's symptom complex is frequently not typical, but from the outset difficult of interpretation. To distinguish also the psychoses occurring in acute and chronic infectious diseases from those occurring in chronic alcoholism is frequently a matter of much difficulty. Many details of similarities and dissimilarities are discussed at length in this paper to which space does not permit us to allude.

16. *Psychical Disturbances in Multiple Sclerosis.*—On the basis of three carefully studied cases Raecke draws attention to the often discussed psychical disorders observed in multiple sclerosis. He gives the opinions of various writers on the subject and adds comparatively little to the conclusions already reached by other writers. The possible association of multiple sclerosis with dementia paralytica is discussed and in general regarded as too unusual to merit consideration in spite of the fact that the mental disturbances often simulate those of general paralysis. Usually grandiose ideas in sclerosis develop late

in the course of the disease, leaving ultimately a condition of mental enfeeblement. In the special cases studied it was noteworthy that the patients maintained for a long time interest in their surroundings; that they showed relatively little dulness, and preserved their capacity for orientation. Finally it was observed that the dementia occurred only after long persistence of the signs of somatic disease.

17. *Inanition in Mental Diseases.*—Dreyfus writes an exhaustive article on the causes of inanition in the course of mental diseases, and brings to his aid in this research the histories of sixteen cases. The paper is much too detailed for intelligent review in a brief abstract.

18. *Unilateral Disturbances in Genuine Epilepsy.*—Redlich discusses in detail the hemiplegic conditions which come on in the course of genuine epilepsy in certain instances. As a result of this careful clinical investigation the general conclusion is reached that the hemiparetic symptoms under discussion most frequently are manifest only after the attacks, and in other cases are more conspicuous after attacks than at other periods. From this it is apparent that these conditions are to be ascribed in part at least to exhaustion. This, however, is not a wholly satisfactory explanation, since one hemisphere is evidently involved more than the other, and the suggestion is made that there is presumably an anatomical lesion as the basis of the condition. An elaborate discussion of the theories and opinions of other investigators is included in the article. Redlich is in general of the opinion that hemiplegic signs point to a certain local alteration in the brain or to an accentuation of diffuse alterations in definite areas. The question of differential diagnosis between hysteria and epilepsy also receives brief mention.

19. *Anatomical Alterations of the Brain in Leprosy.*—Continued article.

20. *Amyotrophic Lateral Sclerosis with Multiple Brain Cysticerci.*—Meyer reports a case chiefly interesting on the pathological side of amyotrophic lateral sclerosis combined with multiple brain cysts. The histological elements of the case are discussed in detail, and the paper is of value as a curious combination of disease processes. The writer regards the special peculiarity of his case to lie in the fact that there was found an adventitial infiltration of plasma cells and lymphocytes in cord, oblongata and pons which were regarded as the indication of a chronic inflammatory process.

21. *Agrammatism and Disturbances of Internal Speech.*—This paper is an exhaustive study of a case of aphasia with special relation to the so-called motor type. Certain of the conclusions which the writer reaches are as follows: Agrammatism may occur as the consequence of an otherwise slight motor speech disturbance; it may remain stationary for years; it is not associated with mental weakness; it is a primary defect and not secondary to the difficulty in motor speech. Considerable degrees of agrammatism may be associated with slight or even no difficulties in understanding parts of sentences and consecutive speech. Several other deductions are also drawn from this study.

22. *Opium-Bromide Treatment of Epilepsy.*—Schirbach after clinical trials of the opium-bromide treatment of epilepsy concludes that the method may bring useful results in carefully selected cases in which bromide alone is inefficacious. A further use of the method is altogether justified, and the dangers may be reduced to a minimum by

treatment in a hospital under suitable hydrotherapeutic and dietetic rules.

23. *The Korsakow Symptom Complex in Cerebral Syphilis.*—In this case of unquestioned syphilitic character all the symptoms of the Korsakow syndrome were present. Three possible explanations of this relation are given: First, that the mental symptoms may have been due to a diffuse disease of the vessel walls; second, that a possible gumma of the left hemisphere may, through increased intracranial pressure, have produced the psychical disturbance, and, finally, that the symptoms may have been produced through a syphilitic intoxication.

24. *Malformation of the Spinal Cord.*—Westphal publishes with many illustrations a report of a unique case of malformation of the spinal cord, in which a doubling took place at its lower end, associated with certain pathological alterations which finally led to the death of the patient. The illustrations demonstrate well the exceptional conditions which were found.

25. *Syphilitic Disturbances of Sensibility.*—On the basis of two cases Knapp draws attention to certain unusual distributions of skin sensibility in syphilis, which he regards as peculiar to that disease. He discusses at length the anatomical relations of the branches of the intercostal nerves.

26. *Investigations with a Dermograph.*—Prengowski has undertaken a study of the so-called dermographic phenomenon by means of a special instrument. His investigation was made in school children and he has carefully summarized the dermographic reaction, its intensity, its method of production, the time of its appearance after irritation, and other details of interest. E. W. TAYLOR (Boston).

Neurologisches Centralblatt

(Vol. 25, May 16, 1906, No. 10.)

1. Toxic Polyneuritis in a Case of Phthisis. W. SALOMONSON.
2. Hysteria in Animals. J. MAINZES.
3. Isolated Traumatic Paralysis of the Subcapularis and of the Musculo-Cutaneous Nerves. F. TIRCHLER.

1. *Phthisical Polyneuritis.*—The author describes two cases of phthisis, in the course of which multiple neuritis appeared. He does not believe that this was due to the disease, but believes that it was the result of the ingestion of creosote.

2. *Hysteria in Animals.*—The author comments upon the rarity of these observations, and describes three cases occurring in dogs. The symptomatology is open to doubt.

3. *Isolated Paralysis of the Subcapularis and Musculo-cutaneous Nerves.*—In the first instance the paralysis was brought on by a forward stretching of the arm, the lesion occurring between the beginning of the plexus and the entrance of the nerve into the supraspinatus muscle. Only fourteen similar cases are recorded. The musculocutaneous paralysis was also traumatic in origin, and only eleven similar cases are known.

(Vol. 25, No. 11, June 1, 1906.)

1. Concerning Hyperesthesia of the Peripheral Visual Fields. A. PICK.
2. The Bulbar Syndrome: Dissociation of Sensation with Cerebellar Ataxic Disturbances. S. E. HENSCHEN.
3. Sexual Anomalies in Animals. S. LOMER.

1. *Hypercesthesia of the Visual Fields.*—Cases of hypesthesia of the visual fields are more or less common, but the opposite condition has not been described. Pick describes a woman in a functional mental state who, when sewing for example, if anyone would pass by would be compelled against her will to look in that direction. Any visual irritation of the peripheral fields would bring on these movements of the eyeballs. The movements were either lateral or downwards and never upward in direction. The cause is difficult to ascertain, but Pick is inclined to believe it cerebral.

2. *A Bulbar Syndrome.*—Henschner, following the report of Babinski, Nageotte and Rossolimo, reports a very interesting clinical case of what appears to be a unilateral specific lesion of the bulb, giving the symptoms of involvement of the sensory fifth, sixth and ninth, and of involvement of pain and temperature sense of the right limbs. There were also present bulbar symptoms plus ataxia. In another clinical case, as a result of contracoup of the skull, there resulted an abducens paralysis, plus pain and temperature disturbances and ataxia of one lower limb.

(Vol. 25, June 16, 1906, No. 12.)

1. The Descending Connections of the Thalamus. V. v. Bechterew.
2. The Course of the Central Fibers in the Medulla. S. Sergi.

1. *The Thalamus and Its Descending Connections.*—Bechterew calls attention to the fact that in 1898 he described a tract of fibers coming from the optic thalamus which connected with the red nucleus, and thought that these fibers were a central connection with the so-called v. Monakow's bundle in the extra-pyramidal tracts. He calls attention to the work of Ernst, done in his laboratory, in which in experimental injury to the optic thalamus there were found two distinct sets of fibers coming from the thalamus and connecting with the nucleus articularis and the nucleus of the formatioreticularis respectively. Both of these tracts Bechterew had also described previously. In the experiments of Ernst no degeneration was found traceable to Monakow's bundle, but a tract was found which connected with a red nucleus of the same side. It can also be assumed that there is a connection between the thalamus and the opposite red nucleus, in opposition to Probst. Degeneration was also traced by Ernst which terminated in the anterior corpora quadrigemina.

2. *The Central Tract of the Hypoglossus.*—In a case of porencephalus with right-sided spasticity and atrophy, at necropsy was found an atrophy of the left lower central gyri with thinning of the upper posterior gyrus and of the supramarginal gyrus. Accompanying the usual degeneration of the pyramidal tracts there was found atrophy of the distal portions of the hypoglossal nucleus. The cell degenerations were not limited to particular groups. The afferent fibers of the same side were thinner and seemed underdeveloped. The nuclei of the hypoglossus in the proximal portions were not affected, and its corresponding afferent fibers seemed also normal, but the fibræ rectæ of the raphé on the opposite side were atrophic. The author concludes that the cortico bulbar hypoglossus fibers in man follow the pyramidal tracts to the bulb, where they are to be found in the fibræ rectæ of the raphé after which they cross over by means of the dorsal fibræ efferentes to the hypoglossus nucleus.

Graphic Kinesthetic Hallucinations.—Margulies returns to a description of these sensory phenomena now occupying so important a field in psychiatry, particularly in the study of dementia præcox and of hysteria. He gives in full detail the history of a hysterical psychosis in an eighteen-year-old student, in which motor graphic hallucination played an important genetic part. They entered consciousness with great suddenness, were perceived to be foreign and of external origin. The patient had no doubts about their reality, and believed them to be the word of God. The hallucinations in many instances were analogous to the "hearing one's thoughts spoken out loud" type of hallucinations, at times they were written in the consciousness of the patient, where they had an imperative character. In following the commands the patient sprang into the water and was saved from drowning with difficulty. The psychosis persisted seven days and full insight into the whole history was gained.

Neurasthenia and Sea Climate.—The author is in favor of a sea climate in the treatment of neurasthenics, especially if accompanied by the accessories of sanitarium regulations.

(Vol. 25, No. 13, July 1, 1906.)

1. Tumor in the Substance of the Motor Zone (Arm Area). The Differential Diagnosis of Cortical and Sub-Cortical Lesions. C. T. Van Valkenburg.

1. *Cortical and Sub-Cortical Lesions.*—Valkenburg records a case of a sub-cortical sarcoma of the motor area which gave the usual symptoms, and he attempts to differentiate cortical from sub-cortical tumors for the study of his case and others. The important differential diagnosis seems to be in the study of the type of Jacksonian convulsions. In cortical cases the convulsion always starts in the same part of the same limb, whereas in sub-cortical cases, although the convulsion is limited to the involved limb, the movements may start in different muscle groups. This is really an important differential point, and one not often recognized.

(Vol. 25, No. 14, July 16, 1906.)

1. The Prognosis of Tetany in the Mature Individual. L. V. Frankl-Hockwart.
2. Graphic-Kinesthetic Hallucinations. A. Margulies.
3. The Treatment of Neurasthenia by Sea Climate. Dr. Ide.

1. *Tetany.*—Continued article.

(Vol. 25, No. 15, August 1, 1906.)

1. Sensory Disturbances in Incipient Progressive Paralysis. J. Peltz.
2. The Prognosis of Tetany. L. V. Frankl-Hockwart.

1. *Sensory Changse in Paresis.*—Peltz, in a study of sensory changes in paresis, found that there was a diminution of the pain sensation of the skin—a hypalgesia or analgesia—of the whole body, with the exception of an area around the neck and of an area in the buttock and the upper posterior portion of the thigh. He also found an increase of tactile sensation in the buttock and the inferior parts of the back.

2. *Prognosis of Tetany.*—Frankl-Hockwart, in an extensive study of a large number of cases of tetany, comes to the conclusion that cases of tetany recurring in epidermic-endemic form in workmen, and in the period

of maternity are not as hopeful of recovery as previously thought. The paper should be carefully read.

(Vol. 25, No. 16, August 16, 1906.)

1. Epilepsy with Unilateral Appearances. Dr. Boetz.
2. A Case of Acute Loss of Consciousness of Alcoholic Origin. Dr. Juliusberger.
3. Acute Encephalitis and Apoplectic Lesion of the Cerebellum. F. Witte.
4. The Etiology of Progressive Spinal Muscular Atrophy. V. Vitek.

1. *Unilateral Epilepsy.*—Bratz records a case of genuine epilepsy with unilateral manifestations. He places the lesion in Ammon's horn.

2. *Acute Disturbance Due to Alcohol.*—A unique case of dissociation due to alcohol giving the picture of a phase of dementia præcox as elucidated by Stransky.

3. *Acute Encephalitis of the Cerebellum.*—On the seat of an old hemorrhage due to an arteriosclerosis an acute encephalitic process developed. The author considers this due to the pneumococcus. The lesion caused a stagnation of the blood supply, which gave the coccus an opportunity to cause the inflammation.

4. *Progressive Spinal Muscular Atrophy.*—Vitek records a case of an old acute poliomyelitis which came on in infancy, and in which twenty years later there appeared the changes of a progressive degeneration of the anterior horn cells in other portions of the cord. (Vitek refers to the German authors, but fails to mention the excellent paper and complete review of this subject by Potts in the March number of the *University of Pennsylvania Medical Bulletin*, 1903).

(Vol. 25, No. 17, September 1, 1906.)

1. A Further Contribution to Autogenetic Regeneration of Nerve Fibers. E. Lugaro.
2. Organic Weights and Idiots. H. Vogt.
3. Bilateral Athetosis. S. Klempner.

1. *Autogenetic Nerve Regeneration.*—Lugaro in some further experimental work reasserts his previous conviction that no autogenetic regeneration can occur in peripheral nerves in which the associated parts of the spinal cord and ganglia have been removed. Raimann recently contested this statement, but there was no certainty in his work that the ganglia had been removed. In the recent work of Lugaro no precaution was spared, and in those cases in which both spinal cord and ganglia were removed there was complete degeneration of the peripheral fibers, both by the osmic acid and Cajal methods. The few remaining axis cylinders which can only be demonstrated by the Cajal method belong to the amyelogenetic fibers, and belong to the sympathetic system.

2. *Idiots' Organs.*—An interesting article showing that other organs than the brain are deficient in idiots.

3. *Bilateral Athetosis.*—Klempner attempts to differentiate under the title of double athetosis a form of movement different from that usually understood by that term. This is not original with him. By it he describes bilateral movements of varying intensity which are choreic in character and are characterized by remissions. Disturbances of intelligence, convulsions, paralyses and spasms are not found in this disease, or are very little in evidence. In these cases the eating

reflex of Oppenheim is found. This is obtained by touching the buccal surface of the lips or the tongue, chewing, sucking or swallowing movements resulting in pathology as given. It is rather doubtful if such a clinical classification can be made; at least the author has failed to establish his point. WEISENBURG (Philadelphia).

Centralblatt für Nervenheilkunde und Psychiatrie

(30, Jan. 1, 1907.)

1. *Contribution to Study of Chronic Alcoholic Hallucinosis.* F. CHOTZEN.

Chotzen agrees with Wernicke that hallucinosis alcoholica acuta may culminate in a chronic form. He describes one case. His patient, thirty-four years of age, free from neurotic and vasanic taint, indulged in alcoholic excesses, developed a typical toxic delirium, later neuritic signs occurred, and with the subsidence of the acute symptoms, chronic hallucinosis supervened. The patient presented no evidences of defective retention or memory, and no deterioration could be demonstrated. The diagnosis was carefully considered; Dementia Præcox, Korsakoff's Psychosis, Paranoia, and Kraepelin's Hallucinatorischen Schwachsinn der Trinker, were eliminated because the clinical picture showed no characteristic features of those diseases. The author claims that chronic alcoholic psychosis may be precipitated by the following conditions: (1) Direct injury to the brain through disturbed metabolism; the latter caused by alcohol; (2) Poisonous action of alcohol upon the other organs of the body; (3) Nutritional disturbances due to alcohol; (4) Auto-intoxication superimposed by alcohol; (5) Affection of brain tissue and cerebral arterio-sclerosis, especially in the senile and involution periods, brought about by alcohol; (6) Constitutional psychopathic state. The last condition could not be considered in the patient in question because his family history showed no neuropathic or psychopathic traits.

(30, Jan. 15, 1907.)

1. *Psychoanalytic Method of Freud. Sadger.*—Contains no new material.

(30, Feb. 1, 1907.)

1. *Organic Contraction in Progressive Paralysis.*—T. S. HERMANN.

The author briefly discusses the motor symptoms of general paralysis. He claims that flexor contractures associated with immobility of the spinal column are of a rare occurrence. This condition is usually developed in the last stage of paresis. The contractures are so marked that the gait becomes affected. Passive motion in contractures of long duration is impossible. In some cases decubitus may originate. Contractures may develop in upper and lower extremities; more frequently in the latter. He reports six classical pictures of general paralysis. Three of them showed contractures of the lower limbs and in the other three both upper and lower extremities were involved. All of them disclosed muscular atrophy and fixation of the spinal column. In regard to the pathological anatomy, the author agrees with Berger that the lesion is situated in the cells of the anterior horn. Brief reference is made to Bechterew and Hoche. According to the former the pia-mater of the spinal cord and cord substance are dis-

eased; but the latter maintains that the anterior roots are affected. The following are his conclusions: (1) Contractures in paresis are in the last stage of the disease process and are associated with profound dementia and paralysis. (2) The contractures are of a flexor nature and occur in the lower extremities—in 50 per cent. of his cases both upper and lower extremities were involved. (3) Muscular atrophy and wasting accompany contractures. (4) All his cases were attended with rigidity of the spinal column.

M. J. KARPAS (Ward's Island).

Journal de Psychologie Normale et Pathologique

(Vol. 3, No. 6, Nov.-Dec., 1906.)

1. Variations in the Structure of the Cerebrum. P. GIRARD.
2. Hysterical Laughter. J. INGEGNIEROS.

1. *Variations in the Structure of the Cerebrum.*—Anthropologists have long imagined that, as a result of some measurement, weight, or other mode of calculation, a mathematical index might be established to indicate the relative intellectual status of any one given individual. So far their hopes have not been realized. In the present essay Girard attacks the problem in a somewhat different way, a way which provoked much brilliant and heated discussion along about the middle of the last century. Granting that two factors, the one somatic, representing the organic mass, and the other intellectual or psychic, representing the coefficient of cephalization, determine the volume and weight of the encephalon, the problem is to solve the manner and extent to which one or the other of these factors influences the form and intimate structure of the encephalon, especially the cerebrum. After reviewing the history of the earlier attempts to answer this question and after detailing some of his own experimental work, the author presents certain general conclusions. He says that in chemical analysis, and not in mere morphological observation, must the proper answer to the question be sought. He compares the white matter with the gray, and in their relative and comparative chemical composition he discovers a more or less definite index of the individual's intellectual status. In its general features the problem is solved thus: Taking into account the superiority of the size of the animal, the superiority of the intelligence, using the word in its broadest and fullest physiological sense, implies a particular structure of the brain corresponding to a certain proportional development of the gray substance and of the white.

2. *Hysterical Laughter.*—There is very little literature upon the nervous and mental pathology and pathogenesis of laughter, though the studies are numerous that relate to its physiology and psychology. Ingegnieros quotes some definitions from these studies and then takes up the pathology of laughter, particularly hysterical laughter, illustrating by case reports. He says that in ordinary laughter three factors are to be distinguished, namely, the pantomimic element, the emotional element, and the intellectual element. The phenomenon is thus a complex one and these various elements may associate and dissociate among themselves in numerous ways. There exists, therefore, a pathology of laughter that is purely pantomimic, one that is purely emotional, and one that is purely intellectual. These the author elaborates, differentiates and studies in great detail. The pathological pantomimic

laughter is seen in some of the spasmodic manifestations of hemiplegia, lateral sclerosis, bulbar lesions, etc.; in the exhibitions of imitative laughter occurring in idiots, dements, etc., and in the classical laughter in ore stultorum; in the tics with the manifestation of rictus; and in the convulsive movements of the muscular groups normally concerned in laughter, as for instance, in hysteria. In pathological emotional laughter, we meet with dissociation between the emotional state and its pantomimic expression. For example, the laughter accompanies a painful feeling. Sometimes there is a disproportion between the emotional state and its pantomimic manifestation. This is seen in the intense and irresistible laughter accompanying a really inadequate motive (fool laughter). It is also observed in the emotion of pleasure, with corresponding pantomimic manifestation, revealed by certain born criminals when relating the details of their crimes. Among the cases of pathological intellectual laughter are to be included those in which the laughter is provoked by a morbid process of reasoning (delirious laughter), by morbid perceptions (hallucinatory laughter), by obsessions, by false representative processes, etc. The erotomaniac, the victim of hallucinations, and the reasoning fool who find pleasure, for instance, in their illogical ideas, are all illustrations of this third form of morbid laughter. After discussing thus the general subject of the pathology of laughter, the author devotes the rest of his long article to hysterical laughter, taking up in detail its classification, its origin, its differential diagnosis, its paroxysmal manifestations, and its treatment by hypnotic suggestion. As these are all portrayed from long case reports rather than discussed in a set scholastic manner, the author's article is not one easily abstracted, though it is highly interesting and instructive.

(Vol. 4, No. 1, Jan.-Feb., 1907.)

1. Disorders of Emotional Pantomime among the Insane. DROMARD.
2. Contribution to the Physiology of the After-sensations of Taste. POLIMANTI.
3. The "Call of God." BENEZECH.

1. *Disorders of Emotional Pantomime among the Insane.*—In a preceding paper (JOURNAL OF NERVOUS AND MENTAL DISEASE, March, 1907, p. 207) Dromard discussed the disorders of voluntary pantomime observed among the feeble-minded. The present paper is the second of the series and is a study of the disorders of involuntary or passive pantomime among the insane. Of these involuntary, passive, emotional pantomimic disorders, some exhibit themselves as real disturbances of adaptation and seem to take their origin from a perturbation of the ideo-affective associations; while others appear to be mere disturbances of function resulting from an alteration in the apparatus which is specially set apart for the organization of the emotional manifestations, namely, the thalamus and its dependencies. In the disorders of adaptation there is detected an incongruity between the manifestations of the emotion and the emotional quality of the situation. In the disturbances of function, the performance of the pantomimic movements themselves is profoundly affected, whether that be by failure of inhibition or by defective dynamogeny. Each of these three forms of dis-

turbed emotional pantomimia the author discusses in detail and illustrates by case reports. (1) Of the first, paramimia, he concludes as follows: In certain patients there is a dissociation between the ideational activity and the affective activity. The emotion is not in consonance with the idea. It happens, therefore, that the pantomimic manifestations do not accord strictly with the accompanying speech. Indeed, paramimia is not so much an indication of a discrepancy between the pantomimic expression and the thymopsychic activity of which it is the portrayal, as it is an evidence of a discrepancy between this thymopsychic or affective activity and the noöpsychic or intellectual activity which ought to be normally fully commensurate with it. This is equivalent to saying that the phenomena now under consideration are not due to a break in the ideo-pantomimic connections, but to a break in the ideo-affective associations. And, if we admit that there is an indissolubility between the emotional life and the pantomimic activity, we will recognize the fact that here the pantomimic manifestations preserve their normal attributes, qualities, or fundamental mental relationships. This psychological observation proves that paramimia, properly speaking, is not a psycho-pantomimic disorder. Many writers give a broader definition to the term paramimia and make it include a number of disorders which Dromard speaks of under the respective heads of spasmodic pantomimia and dissociated pantomimia. Dromard believes that it is of special practical value not to classify these disorders according to their mere external characteristics or objective traits when one is studying them from the standpoint of psychology. (2) The disorders of emotional pantomime from failure of inhibition have their origin in a disturbance of the ideo-affective associations resulting from the thalamus being directly excited by an irritative lesion or, better still, from the loss of its cortical connections through a destructive lesion of the corona radiata, the cerebrum being thus prevented from exercising its proper inhibitory control. The motor discharges are therefore somewhat automatic in character and assume the form of a spasmodic pantomimic manifestation, which is always involuntary if not always unconscious. This spasmodic pantomimia is observed in patients who possess old organic lesions (hemorrhage or softening). It is not rare where the lesion is diffuse or disseminated. It has been studied in cases of cerebral syphilis and general paralysis. It is frequent in senile dementia. In the psychoneuroses, notably hysteria and epilepsy, it occurs: sometimes as remarkable exhibitions of automatic laughing and weeping. It is not uncommon in the psychoses, as for instance, catatonia and dementia præcox. (3) In contradistinction to the preceding type of emotional pantomimia is that in which there is a defective dynamogeny by reason of the thalamus not exercising over the lower executive centers the coordinating authority that it should, and in this way throwing the neuro-muscular apparatus and all its dependencies into a condition of nullity and clumsiness. In general these disorders are revealed in a dissociation and discordance of the pantomimic movements. So important are these the author proposes to devote a separate paper to their discussion.

2. *Physiology of the After-Sensations of Taste.*—This is a physiological study wherein Polimanti criticizes some earlier theories concocted to explain the modifications in the taste of certain substances when other substances are applied to the tongue. He concludes that the after-sensations of taste thus engendered are due to a modification,

physico-chemical in nature, of the membrane which incloses the gustatory papillæ.

3. *The "Call of God."*—Benezech reports the case of a scientific investigator who had a distinct auditory hallucination in which he clearly heard the voice of God calling to him to go out and labor in the missionary field. He gave up his scientific work and obeyed the call. The case is reported as a sort of sequel to Binet-Sangle's study of the prophet Samuel, who was looked upon as a cerebral degenerate.

METTLER (Chicago).

Miscellany

CASES SIMULATING CEREBELLAR DISEASE. A. A. Eshner (Journal A. M. A., March 23, 1907).

The author reports two cases suggestive of cerebellar disease. In one there was a reeling gait, unilateral weakness, impaired hearing and tinnitus, but the lack of objective symptoms and the fact that the disorders followed rather closely a mental shock, suggested hysteria, and treatment directed on this theory effected a speedy cure. The other case commenced with headache, vomiting, vertigo and ataxia, followed later by left hemiparesis and increase of reflexes, drowsiness, etc. There were no mental symptoms or ophthalmic or other cranial nerve symptoms, and the course of the disorder was afebrile. There was no specific history, but the patient gradually improved under treatment with iodids and mercury. The drowsiness, which seemed hardly to be accounted for by the bromids given for the intense headache, suggested meningeal or arterial disease.

THE PHYSIOLOGIC CONCEPTION OF DISEASE IN NEUROLOGY. L. Harrison Mettler (Journal A. M. A., February 23, 1907).

The author considers that the anatomic or organic conception of disease, the accounting for disorders by the structural changes found, though still dominant in clinical medicine, is passing, and that the idea that disease is an abnormal physiologic process is coming more to the front. In practical scientific medicine it is the abnormal physiology, the disturbed functions, that is the desired goal of study. This is the physiologic conception of disease, and while not opposed to the anatomic, on which it in part rests, it is a higher and more accurate conception than the latter. It has, nevertheless, been slow in acceptance, and while recognized by the most advanced leaders of medical thought, it is still inadequately presented in the teaching of the day. It is in neurology, he thinks, that the physiologic principle is the interpretation of disease, and its symptomatology is more conspicuous than in any other department of medicine. The neurone theory, which Mettler considers as essentially valid in spite of the modifications necessitated by recent discoveries, has given us one unit and that a physiologic one. In the nervous system it has given us a physiologic conception of its diseases, which renders the usual anatomic classifications of the textbooks confusing to the average student and practitioner. He gives a summarized explanation of his own tentative physiologic classification of nervous diseases, into those of the neuronic or functionating tissues and those of the supporting or nutritional ones, the former being characterized by degenerative, the latter by inflammatory processes,

and these again being subdivided. This classification was published several months before the appearance of Grasset's more radical and elaborate presentation of a physiologic classification, but Mettler thinks that perhaps at the present time, when the pathology and neurologic status of some diseases are still in dispute, a less radical change from the older classifications may have its advantages.

MULTIPLE NEURITIS SIMULATING PROGRESSIVE MUSCULAR ATROPHY. J. Grinker (Journal A. M. A., March 9, 1907).

Three cases, one of alcoholic polyneuritis, one of lead paralysis and one of progressive muscular atrophy, illustrating the futility of attempting to differentiate these conditions by objective symptoms alone, without study of the etiology and course of the disease are reported by the writer, who holds, nevertheless, to the advisability of clinically separating the cornual from the peripheral lower neurone disorders. His conclusions are given as follows: 1. Clinically, multiple neuritis may simulate a spinal atrophy as regards distribution of parlysis, absence of sensory symptoms and protracted course. 2. Progressive spinal muscular atrophy may resemble neuritis in the presence of pain, remission of symptoms and subacute course. 3. Etiology and course are still the best guides in the clinical diagnosis of the various muscle atrophies.

SURGERY OF THE SPINAL CORD. J. B. Murphy (Journal A. M. A., Mar. 2, 1907).

The surgical diseases and lesions of the spinal cord, contusions, concussions, punctured wounds, hemorrhage, fractures and dislocations, gunshot wounds, spina bifida, syringomyelia etc., and their management, are reviewed by the author. He holds that when the axons and ganglion cells that make up the substance of the cord above the cauda are destroyed, regeneration is impossible, and, therefore, in gunshot or stab wounds with immediate paralysis, operation is useless as regards hope of repair, except in the caudal zone, where the possibilities are the same as in the case of wounds of peripheral nerves. Hemorrhage, concussion and contusion without laceration may give rise to the same immediate symptoms as division, and a positive differential diagnosis is impracticable. The time and order of appearance of symptoms may be the only guides; there is no direct relation between the severity of the trauma and the degree of injury to the cord. Absence of immediate paralytic symptoms is not a guide—they may appear after days or weeks. Early spinal puncture may relieve paralysis due to hemorrhage which in some cases may be as complete as that due to division of the cord. Most patients with incomplete paralysis following fractures recover without operation, and when the displacement is not great the physician is justified in assuming that the cord is not suffering continuous compression and in refraining from operation. The special indications as regards operation in the various conditions above enumerated are pointed out and the technic of laminectomy is described. The danger of delay in conditions calling for operation, especially in non-malignant tumor, tuberculoma, etc., is emphasized. Late operations after necrosis in the cord has taken place are worthless. Surgery of the spinal cord, like surgery elsewhere, must be timely, *i.e.*, before the pathologic condition has passed the possibility of repair.

Book Reviews

A Treatise on Diagnostic Methods of Examination. By Prof. Dr. H. Sahli, of Bern. Edited, with additions, by Francis P. Kinnicutt, M.D., Professor of Clinical Medicine, Columbia University, N. Y.; and Nath'l Bowditch Potter, M.D., Visiting Physician to the City Hospital and to the French Hospital. W. B. Saunders & Company, 1905, Philadelphia and London.

Sahli's work on diagnosis has been a German classic for years and in its new fourth edition in the original it represents the best work of its kind in any language. The English translation from this last edition is a veritable storehouse of material and will be appreciated by the American physician.

It is preeminently a clinical work, maintaining at the same time its qualities as a technical manual. This quality of combining laboratory and clinical methods of investigation is that which gives the stamp of preeminence to the volume under consideration.

New features have been added, including a chapter on the clinical investigation of blood pressure by Theodore C. Janeway and newer chemical and physico-chemical studies on the urine included. The publishers deserve special praise for their part in making this a high class work.

Brown.

News and Notes

An International Congress on Psychiatry, Neurology, Psyschology and the Nursing of Lunatics will be held at Amsterdam, Holland, September 2 to 7, 1907, under the auspices of the Netherlands Psychiatry and Neurology Society. The congress will be organized along the lines of previous congresses at Brussels and Paris, with the important addition of a section for psychology introduced in recognition of the influence of that science upon modern conceptions and methods of treatment of nervous and mental diseases. The work of the congress will be divided into two parts: the General Sessions, at which the larger questions of general bearing will be taken up, and the Meetings of Sections for discussion of special questions. There are four sections: (1) Psychiatry and Neurology, (2) Psychology and Psycho-physics, (3) Nursing of Lunatics and (4) Exhibition. The section on nursing of lunatics will be of the same practical character which made it so valuable a feature of the congresses of Antwerp and Milan. French, German and English will be the languages used.

Every American whose interests lie in the field of psychiatry, neurology, psychology and the care of lunatics is invited to become a member of this congress. The only necessary formality is registration with the General Secretary, Dr. G. A. M. van Wayenburg, Prinsengracht 717, Amsterdam, together with the payment of the congress fee of $5.00. Contributions on special subjects will be welcomed, and those intending to make them are requested to send in synopsis before

May 1. The Secretary will see to the printing and translation of all articles, and will distribute copies among the members of the congress.

A more detailed program will be issued shortly, and any questions as to the organization and work of the congress will be gladly answered by the Secretary.

The Pathological Society of Philadelphia, which is one of the oldest, if not the oldest society, of its kind on this continent, will celebrate its semi-Centennial in May, 1907. Instituted at a time when pathology scarcely had a foothold in this country, it has kept pace with the tremendous development of that science, and has had a share, not only in giving Philadelphia its eminence as a medical center, but also in fostering the scientific spirit in America.

The celebration, which may rightly be considered an event of national importance, will extend over two days, Friday, May 10, and Saturday, May 11. On the first day addresses will be delivered by Dr. Frederick G. Novy, of Ann Arbor, Mich., on "The Role of Protozoa in Pathology;" by Dr. Simon Flexner, of the Rockefeller Institute, New York, on "The Newer Pathology," and by Dr. A. E. Taylor, of the University of California, on "The Dynamic Point of View in Pathology."

In the afternoon, at four o'clock, a commemorative meeting will be held in the Pennsylvania Hospital, where the first meetings of the Society, in 1857, took place. At this meeting, Dr. William Osler, Regius Professor of Medicine, Oxford University, will deliver an address on "Pathology and Practice."

At a dinner in the evening, prominent men from all parts of the country will respond to toasts.

An exhibition meeting of interest to pathologists, clinicians, and surgeons will be held on Saturday, May 11.

The date of the celebration will enable those to be present who have been in attendance upon the Congress in Washington, and those who are coming East a little in advance of the meeting of the American Medical Association.

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Original Articles

AMBULATORY AUTOMATISM.*

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"By the term ambulatory automatism is understood a pathological syndrome appearing in the form of intermittent attacks during which the patient, carried away by an irresistible impulse, leaves his home and makes an excursion or journey justified by no reasonable motive. The attack ended, the subject unexpectedly finds himself on an unknown road or in a strange town. Swearing by all the gods never again to quit his penates, he returns home but sooner or later a new attack provokes a new escapade." This explanatory definition of Pitres[1] is fairly good. We might add that while the patient may not act exactly like himself during his erratic trip, usually there is nothing obviously pathological in his conduct; and that on returning to normal consciousness, no knowledge remains of what transpired during the ambulatory period.

In its widest sense the disturbance occurs as a manifestation of quite diverse diseases. It has been observed as a post-traumatic state, in the disturbed consciousness of alcoholism, as a post-epileptic phenomenon or epileptic equivalent and as one of the voicings of hysteria. Some cases, I believe, are scarcely to be included in any of these categories, but seem to present the syndrome as a more or less isolated, or particularly striking manifestation of psychic instability or of that condition of vague definition called degeneracy.

Ambulatory automatism is not to be confused with the con-

*President's address before the thirty-third annual meeting of the American Neurological Association, held at Washington, May 7, 1907.

scious or semi-conscious flights of the insane, such as general paretics,[2] senile or precocious dements,[3] paranoiacs, melancholiacs, etc.

Then certain neuropaths are simply inveterate wanderers;[4] victims of an almost constant ambulatory or travelling obsession. Indeed, many persons have the ambulatory instinct or habit. The persistent pioneer of early days ever on the move ahead of civilization; the wandering "journeyman" who inconstantly works at his trade and never for long in the same town; the restless traveller for pleasure or health; the professional tramp, are familiar examples. Not to be definitely separated from these is a class of unstable, more or less defective individuals who mean to be stable and stationary but are unable to carry out their good intentions. Such a one, apparently settled for good, yields to a sudden impulse and starts for elsewhere. The impulse is based on fear, anger, weariness of occupation or environment, desire to see another locality or other persons; in short, on any of the ordinary motives of travel or removal but on a motive utterly inadequate in degree to move a normal person. Oftentimes such a patient is himself scarcely able to recognize the motive back of the impulse and is apt to attribute his running away to "just a sudden notion." This vagrancy of defectives often begins in childhood.[5]

Between all these groups of conscious errants and the ambulatory automaton there is some sort of relationship. The same individual may be first a conscious and then an unconscious wanderer. Such a disposition or habit of mind plus misty consciousness or secondary consciousness is quite apt to result in ambulatory automatism. Naturally, a recently acquired but intense mental impression or desire may play the same rôle as the crystalized habit.

As every neurologist has observed, some persons are seized at irregular intervals with a feeling of unrest, of "nameless longing," of discontent, and I believe this may be the determining element of an automatic flight. As Kraepelin,[6] Aschaffenburg[7] and Gaupp[8] have shown, such periodic fluctuations of spirit may reach a marked pathological degree, and these authors have correlated them with epilepsy and the inebriety of the periodic drinker or dipsomaniac. The question of this relationship I shall touch later, but there can be no doubt of the intimate relation of such transient affective states to ambulatory automatism.

A consideration of my own and of published cases has impressed me with the frequency of another element in the determination of these so-called fugues or flights. This is the inherent tendency of the patient to run away from trouble. Some persons are natural fighters, others are inclined to "grin and bear it," still others suffer in dumb though sullen submission, while a certain proportion of us prefer neither to combat nor to submit; we run away, consciously or unconsciously.

Tissié[9] lays great stress upon dreams as an exciting agent of the impulsion to start and Raymond[10] calls attention to the same thing but I think the importance of this influence has been exaggerated. It seems more reasonable to suppose that the subject dreams of running away or of visiting a certain place because the idea has been in his mind, than it is to assume that the dream originated the conception. In other words, both dream and fugue are the result of the frame of mind.

To the study of this, as of many other neurological questions, Charcot seems to have given the first effective impulse, although Hughlings Jackson and others had previously given it careful attention. In 1888 Charcot[11] published a typical case and the following year[12] further discussed the same patient. This case was reported as an epileptic one and clearly has had undue influence on subsequent writers, many later cases having been called epileptic, which were really hysterical or degenerative simply because the writers blindly followed Charcot's lead. In later years his case was restudied by Sous[13] who threw grave doubts upon its epileptic nature, adducing evidence tending to show that it was hysterical.[14]

In this country the subject has received little systematic attention. Of the forty-one bibliographic references in the critical review of René Semelaigne[15] twenty-seven are French, six Italian, five English, one German and two American[16]. Th following cases, therefore, seem to merit brief report and comment.

Case I.—On a Monday afternoon, July 27, 1896, a young woman brought her husband to the Chicago Policlinic with the statement that he had suddenly become deaf and dumb. After a little cross-examination it was learned that on the previous morning before breakfast they had had a difference and she, obviously the dominant one, had soundly slapped his face or boxed his ears. He took what she gave and a few minutes later went out for a

morning paper. She did not see him again until after ten o'clock that evening, when he returned without power of speech or hearing. His statement, made in writing when first seen and later verbally, was to the effect that not seeing a newsboy he had stopped for a single glass of beer and then started down the street to buy a paper. With one exception he remembered nothing from this time until he found himself at about ten o'clock in the evening in the business part of town, not knowing just where he was nor how he got there. He recalled having seen a street car pass a crossing and having noted that he heard no gong. He also remembered remarking that the streets seemed unusually quiet. When he regained consciousness he was weary, footsore and hungry. Stopping the first passerby he attempted to ask his way, and found that he could neither speak nor hear. In a small book he wrote the question, "How can I get to the Rush Street bridge?" knowing that once there he could easily find his way home. The gentleman pointed the way and although the patient was within a few blocks of his home he did not find the place readily, but passed the house and had to come back to it.

Speech and hearing aside, results of examination were quite negative, except that the tongue, especially the anterior half, seemed to be rather anesthetic. Nothing in the history indicated any form of epilepsy. I may add that the wife was agitated by the liveliest concern. Fear that her assault was responsible for an awful calamity evidently possessed her. These facts, together with the general demeanor of the patient, especially his lack of great concern over his condition, pointed to hysterical deaf-mutism and hysterical ambulatory automatism.

In writing, I told the patient that we would bring back his speech first and attend to the hearing later. Forthwith I passed a very strong faradic current transversely through the neck, informing him that in a moment he would be able to say *ah*. In a few seconds a feeble "ah" was heard, and we went through the vowels.

After about three treatments given within a week, speech was normal and three more restored the hearing, when the couple promptly disappeared. The result of treatment verified the diagnosis as far as the deaf-mutism was concerned, and under the circumstances I think it is only fair to suppose that the automatism was of the same nature. True, simulation cannot be excluded

with absolute certainty but I was able to satisfy myself that the case was one of disease and not of deviltry.

This, then, was a frankly hysterical case, relatively simple and of short duration. Because of its simplicity it serves well to typify one sort of ambulatory automatism. It is to be regarded as an example of vigil ambulism or hysterical somnambulism, Charcot's so-called second state. Given an inpressionable, unstable individual and a violent emotional shock, an hysterical attack is nothing out of the ordinary. Hysterical deafness from a blow on the ear is no great curiosity. Hysterical mutism from mental perturbation is nothing new. That this patient should at once have passed into a state of somnambulism instead of having a hysterical spasm may be regarded as a matter of disposition or of the personal equation. A young man of about the same age whom I saw some years ago, when castigated by his mistress had a fit of violent tremor or general clonus with extreme tachypnea. In the case under consideration I think we must assume that there was within his consciousness, and based upon his certain timidity, a wish to get away or to go somewhere for relief, possibly to go off until his spouse should be sorry for her treatment. This mental element it was which made the case ambulatory or wandering rather than convulsive, cataleptic or lamentative.

This impulsion to ambulation must pertain to every case, whatever its nature. Of the origin or nature of the impulsion we sometimes know but little and in the epileptic cases, I believe we know nothing at all of it. Doubtless something approaching reason or desire is always present, but a clearly defined *raison d' être* is seldom obtainable. In most instances the nearest approach to reasoning seems to be a process of auto-suggestion. Obviously, however, there can be no such thing as auto-suggestion *ab initio*. There must be some process of reason, some mental conclusion or some desire; at least some inclination or whim to be gratified, and each of these psychic states or processes must originally spring from something outside of the ego. It must go back to something in the shape of experience.

In the following case evidence of this inherent or pre-existing impulse to abandon his penates is easily traced.

CASE II.—A real estate clerk, forty-five years old, referred to me by Dr. Webster of Evanston, was first seen October 1, 1902.

What he complained of was severe recurrent pain about the right ear and over the right side of the face.

At nine years of age he had had scarlet fever complicated by double suppurative otitis and by "dropsy." For seven years thereafter aural discharge had been frequent and until the present time the patient had suffered with recurrent earache. Concerning the recent attacks of pain I could get no satisfactory history. His statements were too vague to allow of definite conclusions, but from his description, his manner and the almost negative result of examination I concluded that it was an hysterical pain or, at least, an ordinary earache enormously exaggerated by hysteria. He stated that the pain had been very severe, enough to "drive him crazy" for seven or eight weeks until, a week before his visit to me, he had been out all night in a rainstorm when the pain had suddenly stopped for some days. In an attempt to get the details of this somewhat peculiar happening, I elicited the following bit of history. On a Monday morning, after doing some work about the house (in Evanston, twelve miles north of Chicago) he had gone upstairs to change his "garden suit" for his office clothes but soon came down in the old clothes and went out of the back door without speaking to anyone. The last thing he remembered was starting upstairs. When consciousness returned it was night, he was sitting on the ground under a hedge, wet to the skin, cold, dead tired and footsore. He got to his feet and started to investigate his whereabouts but fell into a ditch and concluded to wait for daylight. After about two hours he made his way to a farmhouse and learned that it was Thursday morning and that he was 110 miles south of Chicago. Of what had transpired in the interval he knew absolutely nothing.

With this phase of the case I did nothing for several days. A few applications of strong faradism to the right ear and surrounding parts rapidly removed all pain and tenderness. Having by that time gained the patient's confidence and believing that his unconscious wandering was of hysterical origin, I hypnotized him and had no difficulty whatever in getting from him the following statement, which he repeated a few weeks later before the Chicago Neurological Society. During the recital he frequently stopped but promptly resumed when told to go on. Several times I interrupted with questions. Consequently, the written statement is

more consecutive than was his oral one but it is practically in his own language.

"I walked on Asbury Avenue and then across the prairie to Western Avenue, walked on Western Avenue to the Chicago River, where the car starts. Took the street car to 63d Street, on 63d Street car to Madison Avenue and then the surburban car to Whiting, Indiana. Walked a little way on Illinois Central Railway tracks to a railway crossing; when a freight train stopped I got on and it took me to Niles, Michigan. There a brakeman found me and put me off. Walked to South Bend, Indiana, four-five-six hours 'till it was getting dark. Went to sleep on the river bank. Wakened up at daylight, went to eat in a little restaurant with a sign "Open all night," then walked on past the big wagon works. Asked a man which way to go to get to Illinois. He told me to keep on south and then west. Walked on all day to another railroad and walked, and walked, and walked, going west. I see a freight train stop at a crossing and get into an empty car. I ride on train through a number of towns and after a time I see that we are coming to a larger town. As the train passes the station I see that it is Kankakee. We go through Kankakee across the river where the train stops and the watchman puts me off and drives me out of the freight yard. I am lost and don't know where I am going. I am walking, walking, always walking; it gets daylight. After a long time I meet a man and ask him where I am and he says twenty-five miles south of Kankakee. Forgot to ask him which way to go and so keep on. Went to a farmhouse for something to eat and the lady said she had only bread, and I said I would be very glad to get it. Walk, and walk, and walk. It got dark and I lay down along the road; feet tired, hurt awful bad, raining now, hard, I am getting wet, I must get up, I am so tired." (Began to whimper and stopped.)

I asked him if that was all and he said "Yes, that is all."

Evidently, at this point he awakened to find himself under the hedge. The location of the towns mentioned, the railway lines and the distances harmonize with the patient's tale.

He denied the occurrence of anything similar in his past but later I learned from others some facts in his history that certainly are important. About twenty-one years ago, before his marriage, having taken part in some rather questionable business transactions, he found it expedient to go west. Here, instead of set-

tling down, he led a sort of nomadic existence, travelling on a pony over a large part of Colorado and New Mexico. The whole occurrence evidences a tendency to abscond or an inherent instability which voiced itself as ambulatory unrest. What seems to have been his first fugue (or unconscious flight) occurred soon after his marriage twenty years ago. One Sunday, on his way home from church, he stopped for a shave and nothing was seen or heard of him for a week or two, when he returned and asserted that he didn't know where he had been. Again, fourteen or fifteen years ago, on the occasion of some domestic disagreement, he left home and was gone for a week, but this is said to have been a conscious absenting of himself. Nevertheless, it shows a willingness to escape trouble by flight.

Furthermore, I have learned that for some time prior to his visit to me he had been involved in financial trouble; trouble connected with alleged irregularities on his part. Five months after my examination he one day failed to keep a business appointment and nothing was seen or heard of him for several days, when his wife received by mail a power of attorney. The document had been executed in Chicago, the envelope was postmarked Denver and the address was poorly written; not in the usual hand of the patient. Since then nothing has been heard of him. In addition to the foregoing it must be said that from childhood on this patient had shown evidence of imperfections of makeup. Probably some would call him a degenerate. At any rate, he was to a limited extent a defective. As a school boy he did not take kindly to educational methods; showed an inapitude for study, an aptitude to play truant. He was unreliable in word and deed. In later years the same traits persisted. One occupation after another was tried and abandoned. Good advice and assistance were alike unavailing and his early infractions finally culminated in the first running away to Colorado already mentioned.

The relation of the face pain to his fugue is of some interest. In the literature I have found a good many cases in which before their flight the patients complained of more or less intense headache. In some instances this headache was not especially severe, probably only a part of the preautomatic nervousness and distress. In other cases it was intense and, I believe, a determining factor of the attack. For pain to occasion a hysterical outbreak is commonplace. As already indicated, whether this out-

break shall be convulsive, delirious or ambulatory will depend upon the personal equation or upon casualties. Here, as elsewhere, the pathological may be not much more than an exaggeration of the physiological. How many quite normal individuals when in distress of body or spirit must pace up and down or tramp restlessly from one place to another? How many others when unwell or worried instinctively seek change of scene, make a shorter or longer journey, for some ostensible reason or another?

CASE III.—Is quite similar to the foregoing one but the motive or driving influence initiating the deambulation is decidedly different. A. C., colored, laborer, forty-three years old, came to the dispensary of the Northwestern University Medical School, January 3, 1907. He has been married sixteen years, has four healthy children and his habits are good.

Twenty-five years ago he suffered a fracture of two ribs, twenty years ago had a venereal sore with suppurating bubo, at about the same time malaria, and eighteen years ago, influenza. In 1902 his home was washed away by a freshet and he lost all of his worldly goods. About a month later his leg was injured in the rolling mill where he worked as a skilled operator, and he was laid up for a month. Only a week after his return to work the mill shut down and he was thrown out of employment. He was now in debt, instead of having money ahead, everything seemed to go wrong, and he has not been so prosperous since. The reverses have greatly worried and distressed him. Two years ago in a railway accident, his head was badly bruised, some teeth knocked loose and he thinks he sustained "internal injuries." He was unconscious for about an hour. One year ago he was sandbagged and beaten into unconsciousness. In the history there is nothing to indicate either epilepsy or hysteria except that once he came home with ten dollars, gave them all to one of the children and then laughed and "carried on" like a child. Results of physical examination were negative.

His first fugue occurred about two weeks after the accident of two years ago. Since that time he has had fifteen or twenty. Ordinarily an attack begins with worry about the circumstances of his family and his poor earning power. This brings on headache and backache, he becomes restless, depressed, soon leaves the house and remains away for from one to five days. He has always returned of his own accord and when asked where he has

been responds "looking for work." Generally he has no idea that he has been gone for more than a few hours. Sometimes he can vaguely recall an incident or two of his wanderings. The rest is a blank. On his return he is restless, often somewhat dazed or confused and always wants to start right off again. Even when he has a job it is the same. He wants to hunt a better one; says he must have more money.

His wife has learned never to ask him for money and has forbidden the children to ask him for anything, because if he cannot gratify them he at once begins to fret and soon disappears. Evidently the occasion of his last wandering was his inability to buy Christmas presents for his family.

For purposes of diagnosis I tried hypnotism. Satisfactory hypnosis having been attained in the third sitting I obtained from him the following statement of his last trip.

"I left home Christmas morning and went to the West side where I had been working, to finish up the work, a temporary job. After I got through there I started out to hunt work and went first to the Boston Store. This was Christmas night. There I saw a man named Liller, foreman of the digging gang. The store was about to build an addition and they were excavating. He said he could not put me to work then but he might be able to the next morning. I then started home but came back and thought I would wait around until midnight, when there would be a change of shift. At midnight he could not put me to work so I again started home but came back and waited around until eight o'clock in the morning, the next change of shift. As I was then unable to go on I went over to the West side to look for work. The first person I remember seeing was an acquaintance named Campbell whom I met on the street. He asked me where I was going and I answered that I was looking for work. He gave me some lunch. After a while I met my cousin and went home with him. I said I was sleepy and he said "Well, you can sleep here," and his wife fixed up a bed for me. This was in the evening and I slept all that night and the next day until evening. Then I got up and went back to the Boston Store, but found no work. I waited around there all night and the next morning went over to the North side where I had been told they unloaded a lot of coal cars at a factory. To get there I crossed the Robey Street bridge and then walked up the Northwestern railroad tracks. This was in

the morning. I waited there a while and then went to sleep. I don't know how long I was there. When I wakened the men were eating lunch and asked me if I was hungry. I said I didn't care for anything to eat. They told me I had slept ten or twelve hours. I asked them about the cars to be unloaded and where I would find the foreman and what his name was. I finally learned the name of the foreman, which was Thompson, but could not find him and after a while started back home. I came down on the West side and on out to 18th Street, where I stopped and rested a while on the street. I then started east on 18th Street where I met a man with whom I had formerly worked. I do not know his name. He asked me what I was doing over there and I told him I was looking for work. He said "there is no work over here." He asked me what I was doing with the shovel and the rubber boots. I had borrowed these the morning I left home from a friend who lives near the corner of 20th and State Streets and had had them with me all the time. This acquaintance remarked that I looked tired and advised me to go home. This was at about two o'clock in the morning. I have seen this acquaintance once since and he told me that it was about that time. I continued east on 18th Street to State Street and then south on State to 35th and went home. I rang the bell and my wife came to the door. I gave her the boots, told her to take care of them and that I would be back in a few minutes. She asked me where I was going and I told her I was going over to see Al. Hamill and see if he could not get me a job at scrubbing. She made me come into the house and told me that I should not go out. The boy got up and made a fire and my wife warmed some water. I washed and went to bed."

Ten days later he was hypnotized before the class and repeated the account. It varied a little from the first recital but not essentially. On March 26, I again put him to sleep and received substantially the same statement.

About February first he secured employment as a railway porter and I did not see him for a month. He had had an accident, sustaining a fracture of the left wrist and a dislocation of the right elbow. Again I hypnotized him and received the following account of the trip before the last.

"Next to the last time I went away from home was long about the last of November. I left home in the morning to go to work

at the dock of the Anchor Line. I went to the warehouse on Kinzie Street and waited there for orders until nearly night. Then I got word to come back at seven o'clock in the morning, when a boat would be in to be unloaded. I started for home and got as far as 18th Street, when someone said the orders were to report at six o'clock the next morning instead of at seven as I had supposed, so I went back to see whether this was true. When I got back I found that one of the boys named Lee Hill had made a fire and some of the others were standing around it. They told me to wait and see the steward. I fell asleep and slept until about eleven o'clock. By that time the vessel was in and I saw the mate and said I would go home. I walked south to Polk Street and then went over to State to take a car for home, but in some way I got a car going north instead of south. When I discovered that, I thought I might as well go back to the warehouse, which I did and sat there by the fire until seven o'clock, when I went to work. After about an hour I didn't feel very well and so I quit work. I must have worked an hour as I received an hour's pay. I then started for home; walked to 18th Street and west on 18th Street to the railway tracks in the 18th Street yard. There I met a porter named Hainey and talked to him. He said he was going to stay in the car that day and all night as he had had a poor trip and staying in the car would save expenses. I lay down there, went to sleep and slept until nearly ten o'clock that night. When I woke up I said "Lou, why didn't you call me?" He said he thought I needed the rest, and besides, having me there was company for him and so he thought he would let me sleep. I then went home and my wife said, 'where have you been?' I told her I had been to work. She said 'why didn't you eat your lunch?' It seems I had not eaten my lunch which I still had in the pail."

Although hypnosis was used primarily as a diagnostic aid, I also have tried to influence the patient by suggestion, apparently with good results. He declares that he no longer is nervous and that the head distress which had driven him into his "spells" has entirely disappeared. He also says that his head is clearer, the sense of confusion gone and that he can better understand and remember the printed instructions to railway employés. He has received no medicine.

Scattered through the literature are plenty of cases similar to these three. Raymond[17] reports one of them. The patient was a

man thirty years old, a resident of Nancy in eastern France, of neuropathic family and himself a neurotic. At seventeen years he joined an expedition to South America, was wounded but returned to duty as soon as well and soon thereafter went to Africa. Some years later, after the death of his wife, he was with difficulty dissuaded from going off to Africa again. His fugue followed a period of continuous overwork and strain causing a number of neurasthenic symptoms and great increase of emotivity. One evening after several drinks with some friends in a café, he felt a severe pain in the head and started for home. The next thing he remembered was finding himself lying in the snow on the bank of a stream near a city. It was night and he was sore and weary. A street car took him to a railway station where he learned that he was in Brussels and the date eight days later than his last evening in Nancy.

This patient had a strong aversion to hypnotism and it was found impossible to hypnotize him, but by degrees he succeeded in recalling the principal events of his fugue. Prior to the flight he had been subjected to great emotional disturbances such as would not only tend to induce a hysterical attack but would also suggest the advisability of flight. Indeed, he had been threatened with an exposure to the police. All of these facts and the entire absence of any evidence of epilepsy, easily place the case in the hysterical category.

Proust[18] relates the case of a young lawyer in Paris, "an inveterate hysteric," who, after an altercation with his father-in-law left home and on coming to himself three weeks later was stupefied to learn that he had gone travelling through the department of Haute-Marne, had got into debt and had been convicted of swindling. In hypnosis he was able to recount all of his doings during the three weeks.

In this case, as in my Case II, there was evidence of previous instability and lack of ethical sense. Indeed, there had been infraction of the legal code. The extent to which this nervous and moral unreliability runs through the histories of these patients is very striking. Early in a study of the cases the questions are thrust upon one: To what extent are these flights voluntary? To what extent, if at all, are they unconscious? To what extent is the assertion of ignorance on the part of the subject simply a falsehood? I believe that there is an imperceptible gradation to

be traced from perfectly conscious, voluntary, rational (if unwise) flights with perfect memory for all their events, to perfectly involuntary, unconscious[19] flights with complete amnesia. Further, as already stated, the same patient may have conscious as well as automatic flights. He may begin as a voluntary traveller or fugitive and end in being an involuntary one.

Of all the published cases of hysterical ambulatory automatism, one of those recorded by Tissie[20] and by Pitres[21] of Bordeaux is probably the most remarkable. The history reads like a fantastic tale from fiction. The patient's first trip or attack occurred at the age of twelve, when he left his native town and walked to another twenty miles distant. Here his elder brother found him going about with a peddler. Touching him on the shoulder the brother asked "what are you doing here?" The boy seemed to waken as from a dream and was astonished to learn where he was and what had transpired. A month later he found himself in a town forty miles away. Some time after this he was sent out with 100 francs to pay a bill. The next day he came to himself in a railway train with a ticket for Paris in his pocket. In that city he was given fifteen days in prison as a vagrant and then, as his family declined to send money, he walked back to Bordeaux, two-thirds of the way across France. From this time on his wanderings were frequent and various. All the principal cities of France, Algiers, the Rhine country from Dusseldorf to Darmstadt and Frankfort, then Wurzburg, Nuremberg, Lintz and Vienna were included in his itineraries. Later he went over much the same route and later still visited Prague, Leipzig, Berlin, Posen, Varsovie and Moscow. Conducted by the Russian police to the frontier he wandered to Constantinople, then to Vienna, where a comrade put the idea of Switzerland into his head. He tramped by a round about way through southern Germany and visited the Swiss cities. During this time he had enlisted and twice deserted, but at Basle his impulsion sent him back to France and he surrendered to the police at Delle. After serving out his punishment at hard labor in the military camps of Africa he returned to Bordeaux, settled down to work, after some months believed himself cured and became engaged to be married. On the wedding eve he disappeared and came to, three months later, at Verdun, without knowing how he got there or what had transpired in the interval. Soon after this he came under medical

treatment and his escapades became less frequent and less extensive but they did not cease.

This case was at first supposed to be one of epileptic equivalent of the ambulatory type, like the ones described by Falret[22], Charcot[23] and others, but Tissié and Pitres plainly showed it to be one of hysterical equivalent or prolonged hysterical somnambulism. Examination revealed complete hemianalgesia and concentric contraction of the visual fields with some dyschromatopsia. More conclusive still, it was found that in the hypnotic state the patient could recount, what was otherwise unknown to him, the various incidents of his several flights. In 1894 I saw this patient in the service of Ballet at St. Antoine, and when hypnotized the minute exactness with which he related details of some of his long tramps of years before was truly surprising.

But a study of this man's numerous wanderings shows that they were by no means always automatic. Many a time he moved on like any other tramp just because the spirit moved him. Sometimes a fugue started unconsciously but when he came to himself, instead of going home he stayed where he was or wandered further. This indifference to or love of absence is noticeable in a number of other cases and, I think, is of assistance in determining their nature.

Among the earliest hysterical cases reported are those of Jules Voisin[24] and his pupil Saint-Aubin,[25] (eight cases) since which time many have been published.[26]

My fourth case is a more difficult affair than the preceding ones. It illustrates not only the severity which the syndrome may attain but the psychologic complexity of some cases: a complexity of etiology and of manifestation. Although the observation is far from complete, I think it presents evidence that some cases are neither epileptic nor hysterical.

CASE IV.—The patient, a man twenty-two years old, was seen July 5, 1902, through the courtesy of Dr. J. H. Hoelscher. The expressed wish of himself, his young wife and his friends was that I should facilitate his commitment to a state institution for the insane. The history obtained was, in brief, as follows:

Both parents, now dead, were nervous and both were, so the patient stated, inebriates. A brother and sister had died of nephritis following scarlet fever. The mother's only sister was very nervous. The patient had begun at an early age to mastur-

bate, smoke cigarettes and indulge in alcoholics—all excessively. As a child he had received no systematic training, his habits had been irregular and without due restraint. He ran about much as he pleased but says he was bright in school.

His first escapade in the way of fugue or flight occurred at the age of eighteen when he was attending a college in Indiana although, naturally, he had played truant in the ordinary way a great many times. He was walking through the campus when he saw passing the grounds a livery carriage on its customary trip to the railway station. At once he was seized with the idea of going somewhere. He called to the driver, was driven to the station and took the next train going west. As he had no ticket the conductor asked him where he was going, so as to collect cash fare. Up to that moment a definite destination had not occurred to him, but at the question Kansas City came to his mind and so he said "Kansas City." On the advice of the conductor he paid his fare to Chicago and thinks that there he purchased a ticket for Kansas City. Arrived at the latter place he realized that he had no business there, found his funds exhausted and wired for money. One of the professors went for him and he returned to the college. That this journey began as a mere whim and that his destination was determined by a stray thought are clear in his memory. I believe this to be important as illuminating the nature of the trouble. Most of the occurrences during this first flight are also remembered. Evidently the escapade was more the freak of an ill-balanced boy than a manifestation of disease. Certainly anything like epilepsy was out of the question.

From this time his fugues followed each other in rapid succession. Some were partially conscious. Some were begun consciously but ended unconsciously and the tendency was for them to become more frequent, more prolonged and more purely automatic. For instance, at about six o'clock one afternoon he found himself sitting in a restaurant, evidently having just finished dinner. From the general appearance of the place he concluded he was in the Bismarck restaurant of Chicago and asked the waiter something about trains on a certain railway to the suburb where he lived. As the waiter knew neither suburb nor road, he concluded that he was not in Chicago. Going into the street he bought a paper and learned that he was in Kansas City. In his pocket he found sleeping car receipts showing that he had gone

from Chicago to Omaha, from Omaha to Salt Lake City and thence to Kanses City, a total distance of nearly 3,000 miles. On several occasions he returned to Chicago with a number of partly used railway tickets, indicating that he had started for a distant city and had broken the journey at some intermediate point to go off in another direction, only again to change his destination midway of the trip. One morning he telephoned a friend to come to him at a certain hotel. The friend found him dirty, unkempt and quite used up. He was just back from a fugue and said "Lord only knows where I've been." He had partly used tickets for Denver, Cheyenne (Wyoming), Salt Lake City and Galveston, Texas.

The impulse to start generally came suddenly; and in spite of his disgust with himself after the termination of a flight, when the impulse came he followed it. As before noted, ordinarily he was perfectly conscious of the impulse. He said "something pushes me." On one occasion when he had reached home tired, hungry and penitent after an absence of some days, his wife went to a nearby shop for refreshments, only to find on her return that he had disappeared. Another time while sitting apparently contentedly at home he made some flimsy excuse and stepped out. Being fearful that it was the beginning of an escapade, the wife telephoned to her brother and they hurried to a certain railway station. Something the patient had said within a day or two led the wife to expect that he would leave by that road. A few minutes before the departure of a through train for the far west, the patient appeared, with a new travelling bag wherein was newly purchased clothing, and went aboard the train. He had a preoccupied air, recognized neither wife nor brother-in-law, and when they attempted to persuade him to leave the train was irritable and rebellious. Finally, they got him out of the car and into a carriage where his wife broke down and sobbed. Her grief seemed to waken him, he suddenly became like himself, tenderly inquired what was the matter and apparently knew nothing of what had gone before.

Pursuant to his wish, he was committed to an insane asylum but, naturally, soon tired of it and made his escape. This was a conscious flight. He was arrested as a possible car thief but quickly explained who he was and whence he had escaped. He was

returned to the asylum but procured his release by process of law and I have lost sight of him.

In this case I had no opportunity to attempt hypnosis but I doubt if his fugues were hysterical. On the other hand, I believe they were not epileptic, though I have no doubt some authors would have classed them as such. In the spring of 1901, a year before I saw him and three years after his flight, he had a "fainting spell." Since then he had had one every few weeks or months. From his description it was impossible to determine their nature but he was positive that they bore no relation to the fugues. There were no hysterical stigmata.

In addition to what he himself told me, I learned that he was unstable, unreasonable, extravagant, impulsive and foolish in many ways. More than this, I was informed that he had indulged in many dishonesties, some petty, others of graver magnitude. Not only would he raise money for his fugues by deception and illegal means, sometimes with considerable cunning, but at other times in a perfectly conscious and deliberate way would perpetrate swindling operations. In short, what this man did during his automatic periods was not very different from what he did in the, for him, normal state.

I must admit that I have no cogent reasons for not calling this an hysterical case. Quite possibly the whole trouble may be hysteria developed on a basis of degeneracy. Certainly in many respects it is similar to my Case II, to the celebrated case of Tissié and to many others surely hysterical. But the very complexity of the case puts a doubt into my mind. The gradual passage of perfectly conscious voluntary truancy into automatic fugues is scarcely characteristic of hysterical attacks. Sometimes it seemed a toss up whether this patient would spend a sum for one of his fugues or for some other form of debauch. It was not only in the way of journeys that he yielded to sudden impulse and gratified it by hook or by crook. Once he was taken with the idea of giving his wife a handsome ring. Instantly he proceeded to swindle a jeweller out of one. On a Saturday, he missed a train which was to bring him home for Sunday. He was furious and immediately spent his last dollar to charter an engine. On another occasion he started to row a boat a certain distance up a stream but encountered shallows and other difficulties. He became quite beside himself, swore he would get there if it killed him and finally did accomplish the job, hav-

ing lost half of his clothes, ruined what he had left and exhausted himself to the point of illness. At times such as these he was not unconscious at all but "besides himself" to the degree of absolute indifference to surroundings and consequences. Afterwards, too, memory for the details was hazy. This same odd mixture of the conscious with the unconscious, of known whim with indefinite impulsion and slightly hazy memory with total amnesia is seen in many of the recorded cases.

Until more definite tests are applied to individual patients, until the cases are more carefully studied and perhaps until we know more of so-called secondary states of consciousness, no final classification of ambulatory automatism can be made. But arbitrarily to force a case like the preceding one into the rubric of either epilepsy or hysteria seems to be acting prematurely. Such are the cases I prefer to call degenerative. Degenerative they certainly are. A more definite, a more desirable designation must be for the future. We know so little of the nature of these secondary states, so little of consciousness and so little of memory that it is presumptious to say that all automatic wanderings must be traumatic, toxic, epileptic or hysterical. Numbers of writers have described the patients, called by Tissié[27] *captivés,* who are consciously in the grip of an imperative impulse; some have adopted the term *détérminisme ambulatoire* of Duponchel,[28] which indicates the same thing; many, as I have said, have written of patients partially conscious and with only partial amnesia and have classified them in various ways, but all seem to be agreed that as soon as a flight is purely automatic with complete amnesia it must be epileptic or hysterical if it be not traumatic or toxic and the patient be not the victim of a well defined psychosis. To this I am not prepared to agree. The more the cases are studied, not as examples of ambulatory automatism but as individuals, the more resemblance one sees in many of them to other defective, unstable or degenerate folk. Particularly suggestive is their similarity to the peripatetic myth makers so well described by Dupré,[29] Kraepelin[30] and others. For such a case of morbid personality to become one of ambulatory automatism nothing is needed but the addition of some amnesia or periodic alterations of consciousness. Who shall say that such secondary consciousness or amnesia must be either epileptic or hysterical? The difficulty of definitely placing each case is well illustrated by the following.

CASE V.—L. C., a clerk, twenty-eight years old, of good family history but himself always nervous, married two years, was referred to me June 17, 1904, by Dr. Quales. At thirteen he is said to have had a sunstroke and has had a good deal of headache ever since. During the last year he had had half a dozen attacks of intermittent pain in the right lumbar region and right flank, each attack lasting a couple of days. They were very annoying but never kept him from work. He worried considerably about them. Four months before he had begun to have "neuralgic" pains all over the body, worst in the neck. His physician made a diagnosis of muscular rheumatism. He had had none of these for six weeks.

Until about a year before he had been a man of exemplary habits and a consistant church member. At that time he began to drink some and to gamble. Soon he was in debt, tried to recoup his losses and lost more. Likewise he drank more but never heavily; never enough to interfere with his work and never so that his wife noticed it. Naturally, he lied to his young wife, deceived his parents and his pastor. The whole thing worried him greatly and constantly. Three or four weeks prior to my examination he had begun to have severe headaches. These also worried him and he decided to consult a well known physician.

On Sunday evening, June 5, he had a bad headache and after retiring was very nervous and could not sleep. On Monday he received his pay, about $55.00, quit work earlier than usual, took several drinks and went down town to consult the physician he had determined upon, but too late to see him. His memory is misty for events after the start for down town. He does not remember where he got off the street car but does recall passing a certain populous corner. When he came to himself he was sitting in a railway train which was standing at a station. In his hand was a railway ticket and upon it he read "Suspension Bridge to New York." He thought "What do I want in New York? I'll get off here." He did so and learned that he was at Niagara Falls, (over 500 miles from Chicago). For a time he walked around "trying to collect his thoughts" and then went to a hotel. He had only $5.00 in his pockets. The fare from Chicago to New York is about $25.00. He does not know whether it was Tuesday or Wednesday but thinks it was Tuesday. He disavows having had any intention or idea of running away but when he realized where

he was, he was afraid to go home, afraid something would happen and frightened by the severe headache he was having. He feared he would lose his mind or do something rash. For about two days he wandered about the place in a somewhat dazed condition. On Thursday he wrote for his wife. He thought of sending a telegram and then it "skipped his mind." After writing he seems to have lost track of events again. By Sunday, the 12th, he was out of money and about to apply to the police when his wife arrived. When he saw her he burst into tears and clung to her. He wept frequently through the day. In appearance he was pale, haggard and unkempt. He was quiet, talked little and slowly, walked slowly, and appeared to be ill. The following day they left for home, reaching Chicago the day after, June 14, three days before I saw him. After arrival at home he was entirely rational but slept poorly, felt tired and weak and complained of headache.

Results of examination were very meagre. The patient seemed listless and was not communicative but answered all questions promptly and to the point. I saw him first in the morning when the pulse was 108 and the temperature 100. At five-thirty in the afternoon after three doses of phenacetin, 7½ grains each, pulse was 108, temperature 99.4. Pulse and temperature remained about the same until June 22 when he went to the country. On his return July 2, the temperature was normal and remained so but the pulse continued high.

The most careful inquiry failed to elicit any evidence of epilepsy except that on several occasions shortly before his fugue his wife noticed that he was absent-minded. The most flagrant instance was that he took the baby carriage out of the house and soon afterward asked her where he could find it.

I saw this patient again March 24, 1907. He has had no further attacks of ambulatory automatism, has worked hard and steadily in the same position, his habits have been correct and he has saved money. No treatment has been used. When he is very tired he is apt to be quiet and occasionally his wife notices that he is absent-minded or forgetful. As nearly as I can ascertain he has exhibited absolutely nothing which could be construed as indicating any form of epilepsy. Nor has he shown signs of hysteria. Except for the one year of backsliding he has always been an exemplary man and since his escapade has been as steady as a clock. For sixteen years he has been with the same firm and they never

have complained of him as an employé. To call him a degenerate is impossible. Never having been really intoxicated his fugue scarcely could have been alcoholic. For the few days that he was under observation, conditions seemed to me to be unpropitious for hypnotism and later I had no opportunity.

There is the same difficulty in placing the case of Bregman.[31] His patient was a lad of fourteen in whom the wander instinct seems to have been almost congenital. At four years of age he showed a strong predilection for hiding himself and his fugues began at seven years. The first time he left home was to visit the grave of a recently deceased brother of whom he had been extremely fond. From that time his flights were numerous and of gradually increasing length. Many of them simply those of a juvenile tramp; others were partly automatic. At least there were lacunæ in his memory of them and these lacunæ he tried to fill by fantastic inventions. The boy was not an epileptic, apparently not hysterical, nor was he an imbecile. To the ordinary tests he was normal morally and mentally. If to his inherent topographical instability, a little more change of consciousness had been added, he would have been a perfect automatic wanderer. The question waiting for an answer is, to what transitory alterations of consciousness short of a known psychosis, is such a subject liable.

As an example of the germ from which the degenerative form of ambulatory automatism may spring I may cite the case of a man now thirty-two years of age, son of a nervous mother but level-headed father. As a schoolboy he was most unsatisfactory, could not apply himself, learned little and played a good deal. Later he was sent to business college with small profit. He was tried in every department of his father's manufacturing establishment, was always inefficient and stuck to nothing. At twenty-five he married, soon disagreed with his wife and her people and within five months ran away, because he feared bodily violence at the hands of his brother-in-law. For three or four days he wandered about the streets without going to bed and when found could not give a complete account of where he had been and what he had done. Then began a series of longer or shorter absences. Sometimes they seemed to happen in the most casual way. He would go out for the evening, carelessly stay too late, be ashamed to go home, make a night of it, be more ashamed in the morning and remain away, generally wandering aimlessly about. Sometimes he seemed

to go away in obedience to an impulse or impulsion and later a desire for alcoholics seemed to have an influence, not only in starting him, but also in keeping him away and in contributing to the amnesia. Various positions were procured for him; he could hold none of them. Finally, he was sent to a city 500 miles away to do clerical work. One night after working over hours he was suddenly seized with an idea that he would go home. On the instant he started, walked half of the way and for the other half stole his way on freight trains. The principal events of this journey he remembered but the details were decidedly misty.

It is of interest to know that since a course of treatment for alcoholism four years ago and the definite settlement of his conjugal troubles, this man has not had a fugue and has done tolerably well in a small business way. Had the element of fear not been eliminated, had not life been made easy for him and his environment attractive he was in a fair way to become worse. And he needed only the addition of more obscured consciousness to have presented typical ambulatory automatism. Just what it is that obscures the consciousness in such cases and induces the amnesia: just what the nature of this obscuration or "disintegration" is, I cannot surmise. But I cannot admit that it is epileptic and I believe that it is not always hysterical.

After noting the frequence with which attacks of ambulatory automatism are preceded by a desire or longing or fear tending to start the patient on a trip, it is not surprising to learn that many cases have been reported by military men.[32] Especially is the trouble frequent in countries of compulsory military service. Indeed, many of the German and French cases reported by civilians relate to desertion or absence without leave. The military cases do not differ from others except in their special forensic bearing. In the reports there is the same confusion as to nature and, consequently, the same disagreement as to classification found in other publications. But, to my mind, the pregnant facts are the great relative frequency of fugues in the army and the circumstance that what the young soldier perpetrates as an unconscious or automatic fugue is exactly what he would have liked to do consciously. This point I shall mention again.

The relation of fugues to alcoholism is interesting but still needs much elucidation. One thing is clear. A distinction must be made between fugues in an alcoholic and alcoholic fugues. An

alcoholic may be a degenerate, a hysteric or an epileptic and may have any of the various kinds of flights pertaining to these subjects. A good example is the case of Souques[33] which was one of conscious deambulation due to an overwhelming impulsion; what Régis has called dromomania. Each flight followed a drinking bout but started the first thing in the morning when the patient was not intoxicated. There was simply the impulsion to tramp straight away without a conscious object. The flights lasted twenty-four to forty hours and the patient had normal remembrance of all that transpired. The attack terminated in a nervous attack of trembling and weeping. Fully conscious of all he was doing and clearly realizing the unreasonableness of his actions, this patient cannot be said to have been a victim of automatism, but in my opinion such a case does not differ radically from those in which consciousness and memory are partially obscured, nor these latter from some of those with perfect automatism and amnesia. What seems to have been an alcoholic flight with peculiar amnesia, in a *déséquilibré,* was that of a patient seen in 1902.[34]

Case VI.—At about noon a man, apparently forty-years of age, accosted a policeman, saying that he could not remember his name nor where he belonged. The officer brought him to the Passavant Hospital. On admission he was not intoxicated, no evidence of trauma was found and results of physical examination were negative. He talked fluently, correctly and rationally but knew neither his name nor residence, whence he had come nor his destination nor names of relatives. He was quiet and entirely amenable, perfectly clear as to surroundings but distressed by his loss of memory. Appetite and sleep were good. By evening he began to have a misty recollection of having driven about in a cab with a couple of women, of having visited a theatre and several resorts and having had a number of drinks. It appeared to him that the cab man had put him out. He recalled having awakened or come to on the street at dawn and having walked about until he spoke to the policeman. The next morning he remembered that he had come to Chicago from New York and had gone to a hotel near the station, but of the start and the journey he knew nothing. Later he recalled that he had been attending the yacht races in New York and had drank much champagne, but he could not remember the names of his friends. Soon he began to recall scenes of his childhood and soon after this recalled his sister's

married name and also that of his mother who had married a second time. About a day later he joyfully told the interne that his name was Reese Williamson and that he had friends in Chicago. By degrees he recalled the names of these friends but when they were communicatd with, they disavowed knowledge of any such person. After the patient had been in the hospital about a week he asked the interne again to mention to some acquaintance certain doings in which they had participated. The interne returned with the message that the occurrences were correctly stated but that they related to a certain Mr. A. B. Whereupon the patient suddenly sat up in bed and exclaimed "Why I am A. B.," and this proved to be the case. Other events came back to him rapidly but the thing he could not remember, or said he could not, though he was in the hospital a month, was that he was then a deserter from the United States Army.

In this case, as in many others, investigation showed that the patient had been for many years a man of bad habits, reliable only in his unreliability, swayed by every desire or whim, a moral coward, given to evasion and duplicity. Finally, as a last resort, his friends had got him into the Army, from which he had deserted. He asserted that he never had had anything like the fugue which brought him to the hospital, but this statement I had no opportunity to confirm. An item of interest is that he had been in the habit of assuming the name Reese Williamson when (voluntarily) going out on disreputable escapades.

One of Heilbronner's[35] cases is quite similar to this except that his patient had repeated attacks. He had been a bad boy, twice expelled from school and frequently out all night. During his military service he had deserted without apparent cause. He never started on a flight unless he had been drinking and always drank excessively during the attack.

What the relative frequency of the hysterical, epileptic and degenerative cases may be I would not attempt to estimate. That many are hysterical is clear. When one comes to select the epileptic cases from the literature he is at once confronted by the problem of deciding what the criteria of epilepsy really are. To acquire for one's self a fairly definite working opinion is not very easy; to formulate in set terms what is and is not epilepsy is exceedingly difficult; to harmonize the opinions of only the most distinguished writers is impossible.[36] For some a so-call-

ed, psychic equivalent must be marked by some sort of a fit, great or small. Christian's dictum was "There is no epilepsy if there be not this sudden, complete, absolute loss of consciousness.[37] For the diagnosis of any epileptic disturbance, Ræcke[38] demands two data: 1. Clinically, the mental disorder must bear the stamp of epilepsy. 2. The existence of genuine epilepsy must be established.

Binswanger[39] in speaking of abortive attacks says that the diagnosis is certain only when undoubted epileptic (*i. e.*, convulsive) siezures are observed in addition to the anomalous attacks and that the same holds good for the marked psychic forms. In another place[40] he protests against the tendency of some writers (Lombroso) to attribute to epilepsy, even when other indications of the disease are lacking, all such sudden and violent outbursts as epileptics are prone to exhibit.

On the other hand, writers who recognize the psychic equivalent as itself being an epileptic fit, naturally are willing to make this diagnosis when all the ordinary signs of epilepsy are wanting. They even dispense with loss of consciousness and amnesia. For instance, in the diagnosis of epileptic insanity Samt[41] simply eliminated the ordinary signs of epilepsy and Kraepelin follows him when he says "The diagnosis of epilepsy can just as clearly rest upon a well-marked dreamy state (Dämmerzustand) as upon a typical convulsive seizure."[42]

Following the lead of Kraepelin, Aschaffenburg,[43] after dwelling upon the moodiness of epileptics proceeds to elaborate the thesis that the moodiness is of itself an epileptic equivalent, even when the patient shows no other signs of the disease. A full discussion of the question would take too much space, but I beg to remark that because a prisoner (his patients were criminals) has unaccountable and indescribable periods of depression with a feeling of apprehension, or unrest, or longing, or homesickness, or internal irritability, one is scarcely justified in making a diagnosis of epilepsy. As regards epileptic fugues this author further states that he has seen all stages of transition from the purely automatic attack with complete amnesia to a simple purposeless wandering about, with complete consciousness and unimpaired memory of the entire time. He even puts into the epileptic class one patient who regularly went home to sleep every night after tramping about all day.

The extent to which the epileptic conception of imperative deambulation is sometimes, I might almost say habitually, stretched is well exemplified in a paper by Donah[44] who reports three cases. First, he not only contends that loss of consciousness is no necessary part of an epileptic seizure, but makes the same contention even when a so-called attack extends over months and the patient during that time lives a fairly normal life. Nor is his faith in the epileptic nature of the fugue shaken by the fact that there is no amnesia whatsoever of the fugue period.

His first patient, a man thirty-eight years old, had had a fall upon the head at the age of seven. Ever after, *at the same time of the year,* he had become possessed by an imperious inner longing (Zwang) to wander or travel. At first his wanderings lasted only a few days, later weeks and finally up to five months. Before one of these long trips he begged, borrowed and stole, until he got a goodly sum together. Having thus deliberately prepared for a journey, he left on horseback, sold the horse, travelled by train to Budapest, Vienna, Hamburg and there took ship for New York. Finally, he tired of America and returned to Hungary. He came to this country because a scamp of a chance travelling companion persuaded him into it. He remembered perfectly all the details of the entire trip and expressed great penitence for his misdeeds. To call such an escapade epilepsy, it seems to me, is to burlesque pathology. We might almost as well say that the ordinary vagabond tramp who winters in the poorhouse and starts out in the spring, automatically abstracts the hen from the roost and epileptically slips on into the next township. I may add that the patient was an irresponsible loafer and drinker who abused his wife; that during the examination he burst into a fit of sobbing and the only "seizure" he ever had was one his wife observed at night, when he threw himself about in bed and scratched his face and chest.

In some respects Donath's second case resembles my second; in other respects my fourth. The patient was a man aged forty-nine who, as a fourteen year old boy had wandered all about Hungary, though he easily could have had a steady position. The pathological flights he had had for three years; at first only once a year for two or three days, later two or three in a year lasting a week or ten days. It is to be noted that at first there always was an ascertained exciting cause; that is, some cause for run-

ning away such as fraud, theft, gambling debts. Several times he left because of excruciating headache and said himself that the headache compelled him to go. During his observation in the hospital he had one of these headaches and remarked that if he were outside he certainly would start on one of his journeys. Fancy an epileptic saying "If I were outside now I'd have a fit!" Frequently for twenty-four hours before running away he had tinnitus or a sense of roaring in the head. Donath considered this to be an epileptic aura. But a twenty-four hour aura seems to me altogether extraordinary. Further, this patient, by good means or bad, always provided himself with funds before leaving, and on his last escapade was constantly writing to his wife for money. I confess my inabilities to conceive of an epileptic making elaborate preparations and when he is ready having his seizure.

Donath's third case was a young man of nineteen, who seems to have been a rather ordinary, unstable, moderate defective. He was dishonest, untruthful and lacking in sense of shame. Caught stealing he strenuously denied it and afterward confessed. There was no amnesia, no loss of consciousness and never a convulsion. Unless one assumes, as the author apparently does, that a fugue, even though voluntary and conscious, is itself evidence of epilepsy, I can see no reason whatsoever for invoking this disease to explain such cases. The author's definition of epilepsy, supposed to cover attacks of any form or origin, is surely broad enough and loose enough. He says it is "a pathological excitation (Erregung) of the cerebral cortex which suddenly appears, periodically returns, typically runs its course and quickly subsides." Verily, to borrow an expression of Binswanger, this is shoving the boundaries of epilepsy into the dim distance, but even this definition could scarcely cover several days' preparation, a twenty-four hour aura and a fit lasting five months. In none of Donath's cases was hypnotism tried.

To be sure, the cases and the position of Donath are rather extreme but not very exceptionally so. So good a man as Schultze[45] in reporting three cases seems to assume that in the absence of a definite psychosis, ambulatory automatism means epilepsy. His cases are very similar to those of Donath. There is the same inherent instability, the same emotional variability, the same lack of ethical sense, the history showing the same ingrained tendency to run away, to travel or wander. Especially do they show be-

fore and during the attacks an extreme suggestibility as to when, where and how to go. His cases likewise show the same lack of evidence of epilepsy, aside from the fugues themselves, and many positive signs pointing to hysteria or a similar state. There was the same conscious impulse to go and generally memory of the trip.

In a later paper Schultze[46] takes a less positive stand on the epileptic question. Obviously, he has been influenced by the paper of Heilbronner, presently to be mentioned, but he still contends that the cases of the former paper were epileptic. In this second contribution he reports nine cases of various kinds but says he has never seen an hysterical one. It is rather interesting to note that while he is quite ready to make a diagnosis of epileptic fugue in the absence of characteristic fits, he is unable to make a diagnosis of hysteria without the classical stigmata. He never tried hypnotism. One of his cases, as he admits, is very difficult to classify. Like many others it indicates to me that a new class or new classes of automatic wandering will have to be recognized and defined.

In a quite recent paper Donath[47] reports three more cases and reiterates his former opinions but it is rather obvious that he, too, has had his confidence somewhat shaken by Heilbronner's paper. Of these three cases not one is certainly epileptic, though the first may have been, and the third one the author himself admits was not epileptic.

Sous[48] reports one personal case and nine from the literature. His thesis is strongly tinctured with the influence of Legrand du Saulle and Charcot and consequently hysteria is not seriously considered. He says that hysterical ambulatory attacks are generally preceded and followed by violent hysterical convulsions and that in the absence of such demonstrations, the diagnosis of a hysterical fugue cannot be made. Now we know better. Nor, without more evidence than he has given us, can I accept as epileptic the celebrated case of Legrand du Saulle.[49] When this patient came to, he was aboard ship just entering a harbor. On inquiry he learned that the city was Bombay. He had gone from Paris to Havre and thence to Bombay without in the least attracting attention and could remember nothing of the trip. That an epileptic fugue could be so tranquil and so prolonged may not be im-

possible, but it needs something more convincing than assumption or assertion to establish its epileptic character.

It seems strange that Charcot and his pupils did not hit upon hysteria as an explanation for some fugues, for they were very near it many times. This is notably true of the clear-headed Garnier[50] who recognized his patient as hysterical and explained a momentary memory of the fugue events by supposing a momentary and casual lapse by the patient into his secondary consciousness; but apparently the author never thought of trying to induce this secondary state by hypnosis and thus to obtain details of doings in the automatic period.[51]

No less interesting than this unusual oversight of the Paris school, is the manner of the discovery of hysterical ambulatory automatism. The remarkable patient of Tissié had been observed for years and carefully studied in hospitals. The diagnosis always was epileptic automatism and as such the case had been lectured upon in clinics. Tissié himself had studied and treated him for months when, apparently more haphazard than with definite design, he hypnotized him and learned that in hypnosis the subject could tell the story of his unconscious flights.

But long since the time of Tissié, as I have tried to emphasize, many writers, good ones too, have gone on in the epileptic rut for no obvious reason. The case of Féré[52] is strongly suspicious of hysteria. Binswanger[53] in reporting two cases hardly requires of himself in the way of diagnostic criteria what he demands of others. He remarks, too, that the patients act "exactly like one hypnotized," actuated by a "strong psychic impulsion." The analogy certainly is more indicative of hysteria than of epilepsy. The cases of Gerstacker[54] and Burgl[55] are not at all convincing. The latter considers a dreamy state (Dämmerzustand) itself as conclusive of epilepsy. This would exclude the possibility of hysterical cases. Of those who have taken a broader view Heilbronner[56] is particularly to be mentioned. His painstaking paper is too long to be abstracted but I may state that he reports thirteen cases and comes to the conclusion that the epileptic ones are relatively infrequent. He has tabulated forty-five cases, twenty-eight of which were considered by the reporters to have been epileptic. But Heilbronner shows that only fourten of the twenty-eight presented any symptoms of epilepsy and of these fourteen, many showed positive signs of hysteria. Quite properly he cautions against

attributing to epilepsy every abnormal manifestation in an epileptic. One of his own patients, a lad of sixteen, was probably an epileptic but there was nothing to indicate that his escapades were caused by epilepsy. The same may be said of some of the cases of Schultze. Colman[57] reports a case supposed to be due to epilepsy. Though the patient never had shown signs of the disease, there was epilepsy in his family. The fugue itself was almost exactly like that of my second case. In the absence of evidence why assume that it was epileptic rather than hysterical or of some other origin?

In an excellent paper containing a fine psychologic analysis Woltär[58] reports a case which formerly would surely have been considered as epileptic and which would now be so regarded by many had not the patient been hypnotized. In the history there was nothing to indicate hysteria and on examination no stigmata were found and yet in hypnosis the patient was able to recount all the details of the amnesic fugue period. This case is further of special interest because the patient was under observation for several days of the secondary consciousness period without this fact being suspected. It was thought that this period had passed before his admission to hospital, so natural was the patient's demeanor.

I do not wish to be understood as doubting the existence of epileptic wandering. Unequivocal cases are sufficiently numerous in the literature. But I do insist that this diagnosis has been made too often. Study of reported cases and observation of my own has impressed upon me the frequency of several traits militating against epilepsy.

As I have already noted, the great majority of well-marked cases have been conscious of a strong impulse to start off; not only conscious but so acutely so that the feeling equaled an emotional strain not to be controlled: an inward pushing or longing not to be overcome. This has absolutely no analogy in any form of epilepsy. More than this, yielding to the impulse often carries with it a certain gratification, like satisfying an appetite, akin to the relief afforded by a hysterical explosion after repression and something like the solace experienced by the confirmed *tiqueur* when, after a period of suppression, he "lets go." Needless to say, this is utterly foreign to epilepsy.

Related to this impulsion must be the longing of many young

military recruits to go home, to see the loved ones or to free themselves from army restraints. But that this inward pressure induces an automatic flight is decidedly against its epileptic nature. An epileptic does not have procursive epilepsy, *petit nal*, epileptic mania or epileptic automatism in accord with his previous disposition or desires but according to the unaccountable dictates of his unaccountable disease. The mere fact of the relative frequence of ambulatory automatism during the period of compulsory military service speaks against its epileptic nature.

Several times I have alluded to the patient's preparation for a fugue. Supposing that such a wandering, once begun, were an epileptic equivalent, the procedure might be likened to a convulsive epileptic removing his clothing, inserting a gag, wrapping himself in a feather bed and then having his fit.

Quite noteworthy is the fact that the patients are not more concerned about themselves. Ordinarily if a man found himself lying in the snow, like Raymond's patient, or sitting under a hedge 125 miles from home, like my second case, or in Moscow like the case of Tissié, he would be seriously alarmed. A great many of these patients have no fright at the time and no great worry about it afterward. More than that, many of them instead of hurrying home, stay where they find themselves or voluntarily wander farther. Schultze has noted this point and attributes it to the low grade of mentality of the patients. His explanation I believe to be good for some of the degenerate cases but the fact is also entirely in accord with the traits of hysteria; it cannot be harmonized with epilepsy.

What appears to me extraordinary is that in a number of cases reported as epileptic, the patient has expressed repentance for his flight and even has promised not to do it again.

Early in my investigations I was struck by the enormous preponderance of men. In the very considerable number of reported cases, I have found only two women. Their fugues were hysterical and not long continued. But one other writer (Heilbronner), so far as I know, has particularly noted this point. It seems to me to be strong evidence, not proof, against the frequency of epileptic fugues. An epileptic fit is not adjusted to the personal disposition, temperament and inclinations of the patient nor to his surroundings; a hysterical attack frequently, nay generally, is. The erratic doings of degenerates or unstable individuals are

more or less in accordance with custom and environment. The incidence of epilepsy is about equal in the two sexes and the fits of women do not differ from those of men. Then why no ambulatory automatism in women? On the other hand, it is quite natural that a hysterical woman should not go off on a long and complicated journey but, for her attack, should have one of the "regular" manifestations of the disease.

In any given case epilepsy could not be excluded because bromide gives no relief but it is a striking fact that instances of ambulatory automatism surely stopped by this drug are scarcely to be found. If the disorder were frequently epileptic, instances of marked relief should not be rare.

Finally, the frequence with which automatism fugues are started by casual conditions is distinctly against their epileptic nature. That a man should have an epileptic seizure when he gets into debt, or quarrels with his wife; when he is threatened with arrest or tires of his job; when he has a bad headache or wants to get more money, might occur, but as a coincidence only; not as a regular thing.

Turning now to some of the recognized traits of epilepsy we cannot evade the almost invariable rule of sudden onset, disordered (not necessarily violent) action, short duration, loss of or greatly impaired consciousness and amnesia of the fit period. Even the so-called dreamy states of epilepsy comply with these conditions. The actions of epilepsy are without motive. Rarely are they purposive and when so they are of short duration. To all of these rules there are exceptions but I am unable to reconcile our knowledge of epilepsy with attacks covering a period of many days or weeks, during which the patient leads a lucid, not obviously unreasonable life with no signs of a seizure. And the more the cases of extended fugue are studied the more inconsistencies with epilepsy are found. Spratling[59] has published a conclusive case lasting twenty-eight days but during this time the patient had several ordinary epileptic seizures and the whole period of automatism seems to have been made up of a number of shorter periods succeeding each other.

The evidence seems to show conclusively that attacks of psychic epilepsy may occur without the accompaniment of a big or little fit. Especially is this true of a single attack. If, on the other hand, the patient with psychic epilepsy be observed for any con-

siderable period, additional evidence of the disease is pretty sure to show itself. In not one of forty-five cases of epileptic transitory disorder of consciousness observed by Siemerling[60] did he fail to detect other epileptic or epileptoid seizures.

ADDENDUM.

CASE VI. Since the foregoing was sent to the printer I have seen another case which again shows a divergence from what might be called the classical type of ambulatory automatism.

The patient was a gentleman fifty-nine years old, of a nervous family and himself somewhat eccentric and quite impressionable. Until a recent date his habits had been very good. About twenty years ago he had what was called nervous prostration for about a year, during which time he had many inconstant physical discomforts, insomnia and sundry phobias. He suffered greatly from a sense of chill along the back whenever he lay down to sleep so that he had a horror of going to bed. Finally he made a complete recovery.

For a number of months before the fugue presently to be mentioned, the patient had been struggling with grave business troubles and had got into the habit of taking more alcohol than was his wont, sometimes to excess. Finally, after much hard work, he believed that he had arranged a deal which would relieve him of all trouble and embarrassment, when one morning he learned that he had been tricked and that he was financially ruined. He was absolutely crushed; saw no hope and felt that the only escape was suicide.

Leaving the office where he had received the aforementioned information, he started out with no definite object but an ill-defined purpose to end his troubles by putting himself out of the way. Finding that he had but sixty cents in his pocket he thought he would go to the bank for money but this idea then passed out of his mind and the next thing he remembers was meeting an acquaintance in a distant part of the city, far from his home and almost equally far from the business district. The friend remarked that he was "off his beaten track," to which he replied that his factory was not far away. The next thing he recalls was passing a certain well-known building on the out-skirts of the city, at least twelve or fourteen miles from where he had spoken to his acquaintance. At this time it was growing dark, he was

footsore and weary and he has an impression that he would have taken a car for home had he had any money. But he found that he had spent the sixty cents with which he had started and so continued walking. He next remembers walking through a suburb and finally seeing a larger house than the others where there was a light. He went to this building, recognized it as a hotel, and walked in. In the clerk of the hotel he recognized a former employée and found that it was about twelve-thirty A.M.

He was put to bed but did not sleep well and remained in bed or about the hotel all of the following day. That night he slept somewhat better and by the next morning felt more like himself and telephoned for a friend who at once went for him.

It is probable that he had walked the entire time from about ten o'clock in the morning until after midnight, as the feet of his socks were entirely worn away and he was completely exhausted. Physically the patient is unusually strong and active for his age and a great walker. Frequently he walks ten miles for pleasure.

During the day spent at the hotel there was not only great physical fatigue but also what we may suppose was great mental exhaustion. Lassitude was very pronounced and though the patient was quite conscious and perfectly rational, both present and past events were hazy. When his friend appeared the patient broke down and sobbed freely. After return to his home nothing abnormal was noticed except that he seemed very quiet and not disposed to meet people. I saw him four days after his return, just one week from the beginning of his fugue, and it was then evident that he was worried about the occurrence, that he did not like to talk about it and that he feared it meant a return of the nervous breakdown of twenty years ago.

Examination revealed none of the so-called stigmata of hysteria, there was nothing either in the history or examination to indicate epilepsy, and the physical examination revealed nothing except a slightly hypertrophied heart and a suspicion of sugar in the urine. Blood pressure taken in the recumbent position with a Stanton instrument was 130. Two days after my first visit I made an attempt to hypnotize the patient and again on the following day but did not succeed, though he became slightly drowsy. He then left the city.

It seems perfectly clear that this could not have been an epileptic case and I see no sufficient reason for calling it hysteri-

cal. Neither is it reasonable to suppose that it was alcoholic as the patient had not sufficient funds to procure intoxication. It was simply a case of disturbed consciousness due to severe mental shock in an overwrought, nervously exhausted and naturally somewhat unstable person. The additional element necessary for the production of ambulatory automatism was his feeling that there was no practical way out of his difficulties, and he did in his disturbed consciousness what many a person does during perfect consciousness; namely, wander about in a rather aimless way preparatory to a projected more or less indefinitely determined upon suicidal attempt.[61]

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[11]Leçons du Mardi, Vol. I., p. 113.

[12]Ibid. Vol. II., p. 303.

[13]G. Sous. De l'automatisme comitial ambulatoire. Thèse de Paris, 1890.

[14]Undoubtedly Charcot was influenced by the previous graphic writings of Legrand du Saulle. Etudes medico-légales sur les épileptiques, 1877.

[15]Annales Médico-psychologiques, 1894, p. 71.

[16]I have not made a painstaking search of the American literature. Doubtless many cases have been observed and a considerable number published under various titles. Punton (Kansas City Medical Index, May 1893) reports as "hysterical aphonia" a case quite like my case I, except of longer duration. But it was mutism and not aphonia. And when my paper was all but finished I received a reprint of Dr. Angell's paper, "A Case of Double Consciousness," etc., (Journal of Abnormal Psychology, Oct., 1906.) McCarthy (Jour. of Nervous and Mental Disease, 1900, p. 143), has also published a case, but the flights were so short as scarcely to fall within the category of those under consideration.

[17]Leçons sur les maladies du système nerveux, 1894-5, p. 529.

[18]Bulletin Médical, 1890, p. 107.

[19]This word is here used loosely as meaning another or greatly altered consciousness. Of course a person who walks about and does things, must have some degree and sort of consciousness.

[20]Tissié. Les alienés voyageurs. Thèse de Bordeaux, 1887. Ibid. Les Rêves, Paris, 1890.

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[40]P. 273.

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TWO CASES OF DISLOCATION OF THE EYE-BALL THROUGH THE PALPEBRAL FISSURE.

By Beverley R. Tucker, M.D.,

CLINICAL ASSISTANT AT THE PHILADELPHIA ORTHOPEDIC HOSPITAL AND INFIRMARY FOR NERVOUS DISEASES.

The first case is one of cerebral gumma with exophthalmus so great as to cause a complete dislocation of the eye-ball.

This case is reported because of the many extraordinary symptoms which made the diagnosis difficult, among which was the extreme exophthalmus; and because of the patient's satisfactory recovery. The following notes are abstracted partly from the records of the Pennsylvania Hospital from the service of Dr. Morris J. Lewis and mainly from the records of the Orthopedic Hospital and Infirmary for Nervous Diseases also in the service of Dr. Lewis, to whose kindness I am indebted for the privilege of reporting the case.

The patient, S. L., age forty years, white, married, occupation housework, gives the following history:

Family History.—Father paralyzed at the age of sixty years and later died of pneumonia. Mother paralyzed at the age of forty-five years, and later died of diabetes. Balance of family history negative.

Past History.—She had the usual exanthemata as a child. In 1901 had rheumatism and intermittently since. In 1903 had a discharge from her left ear, the ear has discharged at times since and she has been somewhat deaf. In 1903 also, she had what she calls "water pox," probably poison ivy, and says her sister's children had it at the same time. A year later she had some "throat trouble."

She has been married 20 years, having no children born but one self-induced abortion. She will not admit specific infection.

On November 12, 1903, she came to Dr. J. K. Mitchell's clinic at the Orthopedic Hospital with a left facial palsy beginning six days before. Ten days previous she had caught cold after being overheated. The palsy got worse for three days, and then began to improve. At the clinic an effort to protrude the tongue was ineffectual and the tongue inclined to the left in the mouth. Food was collected on the right side of the mouth. She was treated and greatly improved.

In the year 1903 she states that she had a forward and backward tremor of the head, which soon cleared up, but she has had a recurrence since.

In April, 1905, she had what she calls a "stroke," involving the face only. The right side she states was first paralyzed and then the left.

July 12, 1905, she was admitted to the service of Dr. Lewis at the Pennsylvania Hospital. She came to the hospital in the ambulance complaining of pain in the back of the neck and inability to walk because of weakness. Her eyes were noticed to be prominent, and she stated that they had been somewhat so all of her life. She described attacks resembling petit mal, which came on suddenly with no aura. She uttered a cry, became set and rigid, her head twitched and she thinks she lost sight during attack, which would last only a few moments. She did not remember occurrences during this time, and would come out of them crying. Did not fall nor bite tongue. Had not had one of these attacks for over a year.

Nine weeks before admission into the Pennsylvania Hospital, she had pain starting at the crown of head and passing down neck. Was sewing at a machine when this pain first came on. These seizures of pain came with increasing frequency, since which her eyes have been more prominent and her deafness increased.

She has also had four or five chills during the nine weeks before admission.

The tongue protruded straight. Heart, lungs and abdomen practically negative.

The patient was nervous.

The case was diagnosed at this time as "Exophthalmus of unknown origin and brain tumor" followed by an interrogation mark.

While in the hospital her eyes became very prominent and on one occasion her left eye protruded to such an extent that it became dislocated from the orbit through the palpebral fissure. This occurred while a nurse was looking at her. The lids contracted behind the eye-ball.

Dr. Harlan was sent for and replaced the ball and stitched the lids together over it. After some weeks the exophthalmus was less and the stitches were removed. Never again was the exophthalmus so great as to cause this startling phenomenon. The attack came on so soon after the admission, that no ophthalmoscopic examination was made at the time.

On August 5, 1905, she was discharged from the hospital very much improved. She was in the hospital a little over three weeks. The treatment consisted mainly of sodium iodide and rest in bed.

Ten days after her discharge she was readmitted. The pain in her head had returned and her nervous symptoms had increased. Her neck was stiff, and her head drawn back. Her eyes were thought to be somewhat less prominent.

She was found to have anesthesia to touch, heat and cold on the right side of the face from a horizontal line through the center of the nose nearly to the vertex.

Her jaws opened with difficulty and the muscles of mastication seemed rigid. Her tongue protruded with difficulty and turned slightly to the right. Her knee jerks were plus. Her ear discharged—she fainted once while in the hospital.

She improved greatly in a few weeks. When discharged she had less anesthesia and less protrusion of the eyes. The rigidity of the neck disappeared. The treatment was strychnine in moderate doses and potassium iodide in large doses. Diagnosis same as former admission.

About two months later, on November 1, 1905, she came to the clinic of Dr. Lewis, at the Orthopedic Hospital and Infirmary for Nervous Diseases, complaining of severe pain in the jaw. She says the jaw appears to dislocate at times. Her mouth opens poorly and she frequently bites her tongue while talking. Has difficulty in chewing and swallowing and complains of general depression and weakness.

She is poorly nourished and has a cyanotic, asymmetrical face. Her eyes are very markedly prominent especially the left. She can close the lids over them upon attempt.

Her thyroid is moderately enlarged. Her pulse weak and 102 beats to the minute. Heart action regular. Temperature 101 2-5 deg. F. Respiration 22.

Knee jerks: Right exaggerated and left more so. There is no clonus. Dynamometer R. 20., L. 15.

With each wink of the eye there is a simultaneous fleeting, muscular contraction of the right angle of the mouth and right side of the chin.

The mouth is somewhat drawn to the right.

The cervical and upper six dorsal vertebræ are tender and painful. Pressure on the side of neck causes cyanosis and weakness.

She was admitted the next day, November 2, 1905, to the wards, and put to bed. A soft fluctuating swelling was found upon the scalp in the upper left occipital region. This was tender to pressure. It was opened two days later and found to contain pus. It healed promptly and temperature became normal. Pressure over the atlas and axis causes intense agony and the head is thrown back because of retraction of the neck, she is very nervous. Knee jerks found to vary in intensity but always present.

There is some tenderness over the liver. Diffuse enlargements are found on the upper right tibia, on the middle of the left tibia and on the inner one-third of the left clavicle Bending the head back and forth causes tremor and pain.

There is "pins and needles" sensation on the upper lip.

Over an area from a line horizontal through the center of the nose to the vertex on the left side all sensation is diminished, but not absent, below it is hyperesthetic or normal.

On the right side of the face, and on both sides of the upper lip are hyperesthesia and hyperalgesia. There is a reflex movement of the muscles of both sides of the face upon stroking the upper lip.

There are spots of loss of heat and cold sense on both sides of the face. The jaw jerk is absent.

The next day, November 3, 1905, improvement was marked. The patient was brighter and more cheerful. Movement of the facial muscles was freer. Discharge from the ear ceased. Areas of disordered sensation have disappeared.

November 4 to December 13, 1905, she improved somewhat.

December 13, 1905. While sitting in a chair she fell forward in an apparent faint. She was perfectly unconscious for a few moments and recovered suddenly and cried. She had no bad after-effects from this attack. She said that she had had these "spells" before.

December 22, 1905. She went home looking well and like another woman. She had fattened 16 pounds, and her eyes were much less prominent. Her treatment in the hospital consisted mainly of rest, potassium iodide in increasing doses up to 200 grains daily, and small doses of Fowler's solution. She is to come to clinic.

The following special examinations were made while she was in the hospital.

Dr. Freeman examined her ears and found pus in the left middle ear and believed the ethmoid also involved. A few days later, November 7, he found the aural canal practically closed. On December 7, he found the canal more open, and the membranes dry and posterior superior wall in the proper position.

Dr. de Schweinitz made the following eye examination.

Vision. O. D. and O. S. 6—6. Edges of disk normal, no vessel change. Hyperopia of one D. No spots in fundi nor swelling of disks. Bilateral exophthalmus, no diplopia. Abduction causes nystagmoid movements in each outward effort. Convergence is normal. No von Graefe nor Dalrymple sign, no Möbius sign. No dislocation of the lids.

Her blood and urine were examined several times without finding any pathogenic change except that the hemoglobin on November 6 was 55 per cent.

She came to the clinic from time to time and showed steady improvement. On December 5, 1906, she was seen at her home by me. Her ears have not discharged for months. Her eyes are still less prominent. There are no nystagmoid move-

ments. Convergence is good. There is no disturbance of sensation of any sort. Her thyroid is full but not noticeably enlarged. Muscle movements are better on the whole right side of the face. Knee jerks are slightly diminished, but present.

She states for the first time that during 1903 or 1904 after she had had the left facial palsy, she fell on the ice and struck the back of her head hard enough to make her vomit.

The case presents many features of diagnostic interest. Three years ago she undoubtedly had a 7th nerve peripheral palsy which has gotten completely well. The forward and backward tremor of the head, which occurred also during this year (1903) and which returned for a while a year or so later may have been a precursor of her more serious troubles of 1905.

The so-called stroke she describes in April, 1905, as paralyzing first the right and then the left side of the face, and only the face, we can only ascribe to cerebral syphilis. She never admitted this infection, but the gummatous nodules and the effect of antisyphilitic treatment proved it. Her hypalgesia and hyperesthetic areas were peculiar and can only be explained by the multiple specific lesions.

The condition of her eyes would lead one naturally to think of exophthalmic goiter, but she hardly had enough other symptoms of Graves' disease to justify the diagnosis.

Her unconscious attacks were not epilepsy as there was no froth at the mouth, no tongue biting, no true movements, and no history of their occurrence previous or later. They somewhat resembled hysteria and if they were hysterical this condition was superadded and could not account for her other symptoms. There were reasons for not thinking these attacks simple fainting spells, as for instance, her rigidity. Atypical Jacksonian epilepsy must be considered, which in the writer's opinion, they probably were.

The scalp abscess was probably an infection through a hair follicle from her discharging ear. This abscess could not however account for the intense pain in the back of the neck, for this occurred before the abscess and continued after it had healed.

The great pain, the increased knee jerks and the retraction of the neck and stiff muscles, lead us rather to the conclusion of a specific meningitis.

There are indeed many points of interest in this case, the discussion of which would add too greatly to the length of this paper.

In conclusion it is fair to state that the woman is apparently well, performing her daily duties with comfort.

The second case is one of exophthalmic goiter in which

the eye-balls were dislocated through the palpebral fissure.

This case occurred in the service of Dr. Morris J. Lewis at the Orthopedic Hospital and Infirmary for Nervous Diseases, Philadelphia. I am indebted to Dr. Lewis for permission to report the case.

The patient, Miss E. C. B., aged twenty-six years, came to Dr. Lewis's clinic April 25, 1906.

Family history. Father alive, but "nervous." Mother alive but in delicate health. One brother well, and one has "spasms." Five sisters well though one had chorea as a child.

Past illness. The patient had the usual exanthemata. Had typhoid when ten years of age, and again when seventeen years of age. She had convulsions once when a child, lasting three days. Menses never troublesome. When nineteen years of age she noticed that her eyes were becoming prominent. Her ears discharged at this time also. About six months after this she noticed some enlargement of the neck. She has had difficulty in respiration, but less trouble now.

At present her pulse is 96. Her neck just below the chin is 12¾ inches, and at the greatest part 14¼ inches. She states that her neck was larger and that she was given Fowler's solution two years ago and again six months ago, which seemed to reduce its circumference. Her thyroid is markedly enlarged. She states that at times the exophthalmus has been so great that each eye-ball has protruded from its socket about six times, sometimes without known external cause and sometimes when wiping them with a handkerchief, etc. She reduced them to their proper position herself. Dr. Lewis at clinic touched gently the lower lid of the left eye, and to the surprise of all present the globe protruded beyond the palpebral fissure, which contracted behind it. Dr. Langdon immediately replaced the eyeball by manipulation, and makes the following report as to her eye conditions.

"Marked exophthalmus and eyes equally prominent. Palpebral fissures wide. Motions full and convergence good. Pupils equal and normal in reaction. Media clear and fundi normal. Von Graefe's sign present at intervals. Hyperopia O. D. 2 D. O. S. 3½ D.

She was admitted to the hospital April 29, 1906. Auscultation of her heart showed accentuation of the 2nd pulmonary sound, and the 1st pulmonary booming in character, otherwise negative. Her pulse while in bed ranged from 60 to 80 beats per minute. Her urine was normal. May 3, 1906, her hemoglobin was 90 per cent., red cells 4,940,000, white cells 9,000. June 6, 1906, she became hoarse, then almost lost her voice, also her ears were discharging. Dr. Freeman was called, he found the right ear drum almost gone and the left perforated. She had an acute trachitis and her right vocal cord was

inflamed. Dyspnea was marked. Turpentine stupes to throat and inhalation of tr. benzoin co., menthol and creosote frequently administered, relieved her. She was quite ill for some ten days. Her temperature ranged from 98 to 100 deg. only. After this time her improvement was steady, and she was discharged August 25, 1906. Her thyroid had become smaller and her neck measured May 20, 1906, in its greatest circumference 13¼ inches. About this time a mass of granulations was removed from her right ear and her hearing improved.

When she left her eyes were much less prominent and they never became dislocated again. She gained 13 lbs. in weight and her nervous system was much quieted.

August 29, 1906. She returned to the clinic still improving. She was given potassium iodide gr. ten. She is to be glassed at the University of Pennsylvania.

December 1, 1906. She writes that she has been working since October 5. Her neck measures in its greatest circumference 14 inches. Her eyes have been getting better all the time, and she concludes by saying "Everyone that has known my condition for the last eight years is astonished at my improvement."

Society Proceedings

THE BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY.

Nov. 15, 1906.

The President, Dr. Tuttle, in the Chair.

A CASE OF CHLORAL DELIRIUM.

By Dr. Mitchell.

The resemblance to alcoholic delirium was emphasized. A shoemaker of sixty-two began to take chloral sixteen years ago for insomnia; and it became his habit to take gr. 15-30 for many years every night. Also he used alcohol moderately up to ten years ago.

Four years ago his family first noticed he was more restless and he took more chloral. At night he fancied he heard horns and bands of music, and later voices. He would be irritable and unreasonable in reaction to hallucinations, which would appear irregularly for a time, and then he would seem as well as usual for some months, only to have a recurrence. Three years ago he took a large dose of chloral, with suicidal intent. Afterwards he increased his daily average allowance to 75 gr.

Nine months ago he formed delusions explanatory of his hallucinations; his neighbors were conspiring against him, and he bought a revolver to protect himself against incendiaries. He lost weight, became tremulous, and had a delirious attack lasting weeks; he saw strange animals and men, and heard abusive epithets; he was restless and disorderly. This acute attack subsided rapidly, but he soon resumed chloral with a recurrence of delirium; and he was committed to the Danvers' Hospital.

He was poorly nourished and his face cyanotic. His gait was feeble and unsteady; pronounced Romberg; fine fibrillary tremor of the facial muscles; tremulous and husky voice; general tremulous incoördination; reflexes generally lively; pupils reacted normally; and considerable blunting of tactile sensibilities existed.

Mentally he was disoriented; his attention could not be held in conversation; restless and disorderly, moving his bed about, throwing his bedding around, trying to climb out of the window and keeping constantly in motion. He had visual and auditory hallucinations; he was apprehensive.

In a week he had improved and was able to sleep without drugs. At the end of four months he was practically well and had gained in weight so that he left the hospital, able to sleep better than for years and without drugs.

Dr. Dewey said he saw recently a case of what he considered delirium due to the salicylates. A man who formerly used liquor quite freely, but who had drank none for several months, was taken suddenly with a severe pain in his hip, but without fever. His physician, thinking that the pain was rheumatic, gave him large and frequent doses of salicylates which relieved the pain. On the fourth day he had ringing in his ears and became delirious. He saw beautiful scenery on the ceiling. Later he saw

long needles in the bed clothes and heard his wife taking medicine in her mouth, the glass syringe striking her teeth.

When seen this condition had lasted three days. He then recognized that the first hallucinations were not real, but believed that his wife had been taking medicine and that the physician who had just examined him had put some medicine on his chest with his stethoscope. He was well oriented.

His physician reported that in three days he was practically well.

LATE EPILEPSY IN A WOMAN OVER SIXTY YEARS OF AGE.

By Dr. Southard.

The subject showed a general arteriosclerosis. The vessels at the base of the brain showed a trivial degree of diffuse thickening. The tissues in general showed involution changes.

The reader called attention to the fact that although numerous sections from various parts of the nervous system were examined, a progressive Marchi reaction could be demonstrated in one focus alone; viz., about a small cyst of softening of the left hemisphere of the cerebellum. Minute dissection of the remainder of the nervous system revealed no other focal lesion. Microscopically no characteristic lesions were found, except focal perivascular gliosis in the interior of the spinal cord and in various parts of the cortex. None of these perivascular glioses appeared to be accompanied by a Marchi reaction. There was a characteristic pigmentation of the cells of the fusiform layer in numerous regions.

The case must be counted one of late epilepsy developing upon an organic basis.

TUMOR OF THE RIGHT FRONTAL LOBE.

By Dr. H. W. Miller.

The specimens presented show a tumor in right frontal region which is of interest from the size of the growth, the duration, and the complex of symptoms manifested at various times during the course.

The clinical history in brief is as follows: A male, sixty-four years of age, upon admission to the Taunton Insane Hospital in January, 1894; excessive alcoholism; onset with insomnia, pain in the head, irritability, some memory disorder, and convulsions (nature and number not ascertained) for three months before admission.

At the time of admission physically well nourished, but pale, with motor symptoms suggestive of a peripheral sensory motor neuritis. Pupils unequal, right dilated, left contracted; sluggish reaction to light and accommodation; knee jerks absent; edema of the lower extremities; tremor of tongue, lips and facial muscles. He had at no time convulsions in the hospital, nor was there any vomiting until his final illness.

In three months he recovered from the motor disturbance so that he was given parole and did outside work. He frequently complained of frontal headache, a feeling of dizziness, and pain in his legs, which he spoke of as rheumatic.

At first he was mentally much confused, disoriented, elaborated very poorly; in brief, was in a somnolent, semi-stuporous condition. These symptoms subsided with the physical improvement so that five months after admission it was stated that he showed only a mild degree of general intellectual impairment. His condition remained stable till June, 1896, when he was discharged as much improved.

One year later he was recommitted. In the interim he had indulged freely in alcoholic drinks; had a few (?) convulsive attacks. Shortly before his return he became despondent, restless; complained of a feeling as if the upper part of his skull was falling off; often held on to his head; said that he was dizzy; and he screamed frequently at night. Physically at this time he showed some Romberg, equal but sluggish pupils, absent knee jerks, and by no means persistent headache.

The more acute mental symptoms rapidly passed off, and following that time he showed only slight irritability, temporary attacks of depression, restlessness, rare hallucinatory episodes, and a distinct moral deterioration of the nature of sexual perversion.

The above in brief characterizes his condition up to the present year. On April 6, 1906, twelve years after his first admission, he had a fainting spell with vomiting; became bewildered and stupid, passed into a comatose condition, and died five days later. The fatal termination was apparently due to the rupture of some of the vessels belonging to the new growth.

The tumor was located in the right frontal lobe, commencing anteriorly 1 cm. from the tip, 6 mm. from the medial surface. Its greatest diameter was in a position 5 cm. from the tip where it measures 5½ cm. From this location backwards it decreased in size. The posterior extremity was found 11 cm. from the tip of the frontal. This extremity involved the anterior third and the anterior second of the anterior capsule and a small portion of the inner part of the lenticular nucleus. The lower, inner involved the gray cortex.

The degenerative changes are well shown in the specimens. The protrusion into the longitudinal sinus left a depression on the inner side of the left frontal lobe into which the ends of two fingers could easily sink. The septum lucidum was pushed to the left, its walls thickened, and the cavity of the fifth ventricle almost obliterated. The optic commissure was not interfered with; the optic nerves of normal size, and the right had a few bands of adhesions to the tumor mass; the right olfactory atrophied.

Histologically the growth was a glioma, very vascular, with a predominence of fibrils, but also with many neuroglia cells. Remnants of nerve cells and nerve fibers were found well within the tumor mass.

A CASE OF FRONTAL TUMOR.

By Dr. Walton.

The tumor so far simulated cerebellar tumor that operation in the latter region was concurred in by most of the neurologists who saw the case. The patient was a young woman, referred into the wards of the Massachusetts General Hospital by Dr. Taylor, and seen while there by Drs. Putnam, Paul and Waterman, as well as by Dr. Walton.

The early complaints were pain over the right eye and down the right side of the face and neck, loss of vision and vomiting, commencing somewhat over a year ago. During the past two months there had been severe and constant pain in the occipital region, and back of the neck, with drawing of the head backward, and to the left shoulder, the pain extending to the arm with subjective numbness. There had been hysterical attacks with screaming. There was unsteadiness on standing with possibly a tendency toward the right. There was double optic neuritis with marked projection of the disc on the left. The right pupil was larger than the left, both reacting to light. The patient answered questions in a fretful

manner, indicating a change of disposition, which should perhaps have suggested frontal tumor, but which is by no means pathognomonic of such tumor. There was no asynergia, but decided diadikokanesia in the left arm in which she was unable to make rolling movements; for example, with the facility shown on the right.

Primary operation in the sub-occipital region by Dr. Beach showed tense and bulging dura. The patient died before the secondary operation could be undertaken.

Autopsy by Dr. Wright showed rounded tumor-mass projecting into the cavity of both lateral ventricles on each side of the septum lucidum and just anterior to each caudate nucleus. These masses appeared continuous with one another beneath the septum lucidum and just anterior to the pillars of the fornix. The right frontal lobe was largely occupied by dark, soft, spongy, finely reticulated tissue extensively infiltrated with clear fluid (glioma).

Dr. H. C. Baldwin asked the reader if there had been any complaint of pain or rigidity in the back of the neck.

Dr. Baldwin stated that rigidity of the muscles of the neck was mentioned as one of the symptoms of tumor in the frontal lobes of the brain. No case of tumor of the frontal lobe with this symptom had ever come under his observation; but during his service in the Massachusetts General Hospital this last summer a patient was admitted to the surgical wards suffering from a bullet wound which involved both frontal lobes. The patient made a perfect recovery and had no symptoms except pain and rigidity of the muscles of the neck.

THE RELATION OF PSYCHOGENIC DISORDERS TO DETERIORATION.

By Dr. Adolf Meyer.

After a brief statement of the position taken in his "Fundamental Conceptions of Dementia Præcox" offered as part of a discussion at the meeting of the British Medical Association at Toronto, August, 1906, Dr. Meyer reported two cases which showed the importance of a constructive conception of the disease for the purpose of an adequate sizing up and adequate therapeutic measures. The first patient had three periods of classical hysteria preceding the outbreak of the final broader psychosis, which at first could hardly be distinguished from a hysterical disturbance, but in the course of a number of aggravations and relapses, took the shape of an outspoken catatonic deterioration. In this psychosis the same conditions which had before fed the hysterical episodes reappeared with the characteristic reaction type of a deterioration process, and as relatively easily traceable unbalanced and unbalancing substitutive reactions. The patient is now in a state of catatonic dementia. Non-recognition of these factors appears to have been a serious element of almost every aggravation in the process. The therapeutic efforts all tended to submerge the psychogenic issues in more or less drastic somatic measures. The perfectly glaring lack of penetration into the mental difficulties of the patient led to a series of disastrous blunders. A readjustment of the management of the patient with due attention to the psychogenic constellation has brought about a certain practical change, though, of course, no radical alteration in the undoubted defect which has now existed for a number of years.

The second patient is one in whom a certain constitutional peculiarity

and several short episodes of hysterical emotional outbreaks preceded a typical fantastic catatonic development remarkably clearly traceable to definite material of experience. A comparison of the stereotyped mode of presentation by the routinist with that obtained in the full history and observation of the case, formed the foundation of a discussion of the therapeutic needs of such situations. The main purpose of the communication was to show the lines along which to utilize the reconstructive conceptions of disease. The excellent helps furnished by Kraepelin's prognostic nosology should not be taken for more than a place where we can take a new breath and from which to go forth for more fundamental work. The exclusion of deterioration from practical nosological entities such as psychasthenia and hysterical states has a certain justification, but as the studies of Janet show, the hysterical and psychasthenic mechanism may continue to form an essential part of super-added unfavorable constellations, and although from a prognostic point of view these psychasthenic and hysterical factors may then be subordinated, from a therapeutic point of view they may be the very points which must be kept in evidence in any plan of arresting the disorder and in providing the patient with material for a sound reconstruction. Grasset's recent sketch in the *Revue de Psychiatrie*, and the many valuable contributions to psychotherapy by members of the Boston Society of Neurology and Psychiatry, tend greatly to systematize what undoubtedly every physician must have tried in such cases unless he was wholly under the dogma of prematurely rigid disease-conceptions. The aim of modern psychopathology is not merely the dogmatic diagnosis, but such a knowledge of the facts and their working together that they can be used for therapeutic and prophylactic work.

Dr. Knapp said that Dr. Meyer's paper has emphasized one point which is perhaps a common truism in all neurological work, but which should be the rule in every department of clinical medicine. The prognosis and the treatment must vary with the individual and not be dependent solely upon the diagnostic label we put upon the individual's condition. Even when we recognize that the patient is the victim of an incurable disease we should not sit down hopelessly and do nothing. We all recognize, for example, when we make the diagnosis of tabes, that in some cases the prognosis is comparatively benign and the patient may go on for years with little discomfort, and that our treatment is absolutely different according as the patient is ataxic, or suffers pain. In dealing with the few diseases of the brain which are miscalled "mental diseases"—although every disease of the brain presents "mental" symptoms—we must not be too prone to make a diagnosis of a given disease from the existence of certain symptoms such as negativism, katatonia or mental retardation. In these, as in other brain diseases, we must recognize the fact, which is generally recognized in regard to such symptoms as aphasia or paralysis, that a lesion in a given locality always gives rise to the same symptoms no matter what the pathological process may be. This law is too often ignored by the alienist, although it has been emphasized by Wernicke, whose treatise on psychiatry, however, was based upon the solid foundation acquired in the preparation of his masterly treatise upon brain diseases. We are told to beware of the man of one book. If the alienists were less disposed to swear to the words of their single master, Kraepelin, they would perhaps recognize more frequently that his nosology was not final. What we lack, however, are more definite clinical methods in the

study of brain disease. If we had them we could determine more accurately whether we were dealing with compulsion, stupor and apathy or whether the morbid process had gone on to complete cell destruction and to incurable dementia. If we had these methods the difference to which Dr. Stedman has alluded would disappear. Dr. Knapp said he could not, however, so readily give up the elder belief that "dementia" is a secondary state of an incurable nature. He believed that the so-called "primary" cases go through a phase of stuporous confusion first, from which they may recover, but, if not, that they finally pass into a state of incurable dementia. In some cases it is possible clinically to distinguish between these two states.

Dr. Folsom said with regard to psycho-therapeutics there was more done fifty years ago than until quite recently. Isaac Ray and Dr. Bell were masters in it. Prof. Tyler was appointed to the Medical School nearly fifty years ago, and some of us can remember his careful analysis of symptoms and what was mis-called "moral treatment." It is a pleasure to see psycho-therapeutics coming in vogue again after Virchow's cellular pathology has had its swing. It is a singular fact that Tyler's course of instruction was entirely omitted in the history of the Harvard Medical School recently published.

Dr. E. E. Southard raised objections to the use of the term *psychogenic* on general grounds. He maintained that the term might lead us too far when used in connection with various acute psychoses. Whereas it must be conceded that the histopathologists have scarcely made out a convincing brief for dementia præcox as related with structural alterations, nevertheless it is equally going too far in the opposite direction to insist that these cases have a psychical basis. This question should be held open until more facts are produced. It seemed equally apposite to maintain that various psychoses are patrogenic or metrogenic or hetairogenic as to say that they are psychogenic.

Moreover, philosophically speaking, it might be alleged that a strictly theoretical use of the term psychogenic would commit one to the interaction hypothesis. With respect to the interaction hypothesis, we are still in the position of waiting for more facts. In brief, though it seems absurd to speak of the anatomical origin of many mental diseases, it is equally true that the burden of proof rests with one who insists on the psychical origin of various mental diseases.

Dr. Walton said the work of Kraepelin, which has brought order out of chaos, should not be judged by so unimportant a question as whether the choice of the term dementia præcox is a happy one. Dr. Meyer's suggestions are most timely that the task is not completed when the diagnosis is made. The one characteristic underlying the "psychogenic" disorders is the obsessive temperament, of which Dr. Meyer's cases give evidence in the doubts, fears, scruples and anxieties antedating alienation. That the mental balance of the obsessive may be to a greater or less extent restored by training there is no question, and even in such cases as deserve the title and the prognosis "dementia," the collapse may be at least postponed by efforts in this direction.

Dr. Cotton said as he understood Dr. Meyer's interesting and instructive paper, he hardly saw where he repudiates Kraepelin's work or discredits what Kraepelin has done for psychiatry, and he should like to be clear upon that point. It seemed to him that our zeal in working for diagnoses under this classification has not been misdirected, but that the

very act of probing deeply into the details of our cases, in order to establish a diagnosis, has made Dr. Meyer's paper a possibility. It has certainly given us a clearer idea in regard to symptomatology and has brought us to a point where causal factors can, or should be recognized. And that having reached this point we should not be satisfied merely with making diagnosis, but should now be able to go further and derive some benefit from our work, and utilize the facts obtained by following Kraepelin's methods.

In closing the discussion Dr. Meyer explained first in what sense he used the conception of "psychogenic" factors. In this respect the contrast of psychogenous disorders to general paralysis proves most instructive. The main factors of the disturbances in the cases of "dementia præcox" belong to the sphere of mental reactions, or better, of reactions which could not be thought of as other than "mental." An attempt to express them in terms of nerve physiology would eliminate many fundamental facts which we only know in terms of psychological experiences. In general paralysis the development of the disease depends on the intercommunication of syphilitic infection. In psychogenic disorders we deal with the inability of the individual to adjust himself to certain vital constellations, and certain uncorrected and inadequate mental factors remain the directing element of dynamic processes. The transformation of the symptomatology of general paralysis and also the disappearance of the text-book description of raving mania since the diminution of drastic measures and kindred modes of dealing with patients, shows, of course, the importance of mental constellations in any disease, even those not plainly precipitated by ill-digested and upsetting mental experiences or mental reactions. But certainly the radical management of psychogenic disorders could not be thought of without due attention to their psychic mechanism which in individuals with inadequate defenses can become part of a process of deterioration. In this respect Kraepelin has given us excellent help towards arriving at a position of order replacing the former bewildering confusion. But, after the preliminary survey with his principles, we must venture into the fundamental work of a *reconstructive* study, but not without a warning against losing one's self in pondering. Nissl's arraignment of psychologizing interpretations would only be justified if imaginative construction took the place of rigid observation of fact; what the speaker advocated is the orderly use of the facts at hand and the throwing off of the dogmatic routine which seems to make of a diagnosis a protective against the reproach of medical impotence in difficult but not necessarily fundamentally ill-fated cases, rather than a natural and helpful sign-post in the material for the orthopedics of habits and physical and mental life of the patient. In this respect the moral treatment of the excellent physicians of the past had decided shortcomings because it worked with a conventional moralizing psychology, whereas to-day we have an adequate knowledge of the rôle of substitutive reactions and automatisms and a more adequate sizing up not only of the odds, but also of the constructive possibilities required for adequate reaction. In all this, we could not value enough the great help derived from the studies of Janet and others, and the growing tendency to peal out dynamic principles which cannot fail to become of importance in every nosological and therapeutic activity.

That psychogenic factors are to be considered in a disease or are possibly at the bottom of it, does, of course, not make the disease any

less real and serious; but the recognition of the factors is one of the essential avenues for help and will alone free the patient from the sad faith in routine bred in many hospitals. It is certainly absolutely essential for non-institutional care of mental disorders.

NEW YORK NEUROLOGICAL SOCIETY.

Nov. 28, 1906.

The President, DR. JOSEPH FRAENKEL, in the Chair.

THE MECHANICAL TREATMENT OF NERVOUS DISEASES.

By Dr. H. S. Frenkel, of Heiden, Switzerland.

By the mechanical treatment of nervous diseases was understood a treatment which did not deal with drugs nor physical means, such as hydrotherapy, electricity, etc., but one wherein the therapeutic factors depended upon the functions of the muscles themselves, and might consist in active or passive movements, or in changes of nutrition and assimilation, as was supposed to be produced by massage of the muscles. The mechanical treatment was not much appreciated by the scientific world until the results of the re-educational treatment suddenly suggested the possibility of improvement or cure in organic disease. One of the most important diseases in which the mechanical treatment had been tried, following the good results of the educational treatment of locomotor ataxia, was hemiplegia. To improve the power of standing and walking in the hemiplegics, Dr. Frenkel laid a great deal of stress on exercising the healthy side in order to make it compensate in a certain degree the function of the paralyzed side. The system of "Bahnender and Mermender Therapy" had been recommended also in the chronic spastic spinal cord diseases, for instance, lateral sclerosis and multiple sclerosis, but Dr. Frenkel had never seen any improvement in these cases; on the contrary, he had observed a more rapid course of the disease. A special point of interest was that those cases of multiple sclerosis with intentional tremor of the hands, and unaccompanied by muscular weakness, could be improved by exercises graduated similarly to those of tabetic ataxia of the upper extremities.

ORTHOPEDIC TREATMENT IN NERVOUS DISEASES.

By Dr. Frenkel.

One of the diseases in which one saw excellent results from orthopedic procedures was a spastic paraplegia due to spondylitis. Dr. Frenkel had himself seen some cases of total paraplegia with all the signs of spasticity disappear completely by extension continued for a long time. In a special form of pseudo-muscular hypertrophy in which the atrophy of the soleus muscle was combined with a slow retraction of the tendon, he could improve the act of walking by an orthopedic apparatus which fixed the foot at a right angle, so that in walking the toes did not touch the ground first. He cautioned against ever allowing tenotomy in organic nervous diseases where there was muscular weakness. In poliomyelitis anterior improvement would not follow any exercise, but good orthopedic measures could greatly benefit the patient. In multiple neuritis in the acute

stage, mechanical treatment should be absolutely avoided. In chronic alcoholic multiple neuritis convalescence in many cases would be shortened by the use of moderate massage and passive movements. In paralysis agitans, where the other muscles were in a good condition, by carefully suggested active innervation one could make these patients walk normally again for a time.

From what he had said before, one could conclude that the optimistic idea which prevailed at least in Europe among the practicing physicians and also amongst the neurologists who did not occupy themselves with this question, was not well founded, in so far as the degenerative motor lesions of the brain and spinal cord were concerned. There was danger that neurology, through this optimism, would be set aside from the ways that were successfully pursued by internal medicine, and which might bring us to the knowledge of the course of disease, and thus to a causal instead of symptomatic treatment. He meant by this the biological methods of investigation which were so closely connected with bacteriological and chemical methods, such as the investigation of organic poisons and their antidotes. There was a field for experimental investigation in the central nervous system. If the nature of the different forms of degeneration of the nerves, of sclerosis, etc., could be recognized as being produced by certain poisons, it would not be utopian to say that antitoxins would be found. In short, neurology should not content itself with dazzling installations of mechanical and electrical appliances for symptomatic treatment, but it should take an important part in the research of the causes of the diseases with which it had to deal. If neurology did not apply itself more to the casual researches than it had hitherto, it was certain that internal medicine would replace it in this most important part. For this reason, he was proud to say that the newly built nervous clinic in Berlin, with which he was connected had gotten recently the most perfect installations for chemical, experimental bacteriological and biological investigations.

Dr. M. Allen Starr said he was very much interested in Dr. Frenkel's paper, particularly as he had covered such a large ground and had pointed out to them lines of therapeutics in a great many different directions. Personally, he was familiar with Dr. Frenkel's written works upon tabes and its treatment, and had been wonderfully impressed and surprised at the success he had had in the treatment of several of his own private patients that he had sent to him on the other side. Dr. Starr supposed that a good deal of this mechanical treatment was limited to the treatment of tabes dorsalis. It had not occurred to him that there were so many various types of nervous disease which were open to this method of education of the muscles. For this reason, he said he was impressed with what had been read, which opened up a very hopeful view. It seemed to him that if this mechanical treatment, when carried out in tabes, could be applied to other diseases, it was our duty to apply it. Dr. Starr said that after a careful study of the method, he would adopt it. He believed that particularly in hemiplegia, decided benefit would accrue to the patients from proper training of the muscles. He was impressed with the statements made regarding paralysis agitans, because he was convinced from his own experience in the treatment of that disease that benefit could be had from the proper education of the muscles. He had had several cases of paralysis agitans where material benefit followed massage given by Swedish masseurs who had educated the muscles. He

was convinced that the method had a very wide application. He believed they were much indebted to Dr. Frenkel for bringing this subject before them, and showing the wide application of the method.

Dr. B. Sachs said he was indeed very much interested in Dr. Frenkel's conservative views, and he thought it was safe to say that his opinion regarding the limited application of the mechanical treatment in organic nervous diseases were shared by the larger number of American neurologists, and perhaps by the orthopedic surgeons as well. But there were one or two points in which his conservatism exceeded the views held by some here. If he understood Dr. Frenkel correctly, he stated that he did not think the mechanical treatment aided very materially in those conditions in which the limbs were paralyzed from organic lesions. Dr. Sachs was not disposed to agree with him on this point. There was one period, for instance, in the treatment of hemiplegias, in which he thought the spastic contractions might be benefited. In his own experience with patients recovering from apoplexy and hemiplegia, if the patient possesses a certain amount of motion, if you ask him to try and move the limb more and more, you would find that by such encouragement, movements will be made by the muscles of the affected side, and the patients would ultimately succeed better than if this effort was not directed to those parts. While it was difficult to force a path through sclerotic tissues he believed motor impulses could be forced through, if the tissues were not wholly destroyed. Following these apoplectic seizures, he believed more could be done than Dr. Frenkel implied, although in the main he was disposed to agree with him. There was another form of organic disease successfully treated in this country, and Dr. Sachs' experience with it dated back to an early association with Dr. Gibney, and that was in post-hemiplegic athetosis and in post-hemiplegic contractures; here, the application of suitable splints to the paralyzed limbs for weeks and months would result in some relaxation of the contractures, and this relaxation offered some possibility of education by the method advised by Dr. Frenkel. Personally, he was thoroughly convinced of the possibility of improvement by this mechanical restraint, and he adopted it as a routine measure in the hospital in most cases of hemiplegic contractures.

Dr. Virgil P. Gibney apologized for not having posted himself on the treatment of tabes, as practiced by Dr. Frenkel; his only apology was that he regarded the disease itself, from his own standpoint, as a *noli me tangere*. He had never seen anything in orthopedics which would help in tabes except after resolution of the disease itself. He believed there were a great many convalescents improved by mechanical appliances. He had been assured by neurological friends associated with him that the muscular power could be restored by this method. He was not quite sure whether he understood Dr. Frenkel correctly when he said that in the contractures of poliomyelitis division of the tendons resulted frequently in loss of power. He was inclined to believe that he misunderstood the doctor. If there was anything of which Dr. Gibney was firmly convinced in his mind it was that lengthening the tendons in poliomyelitis by tenotomy did not impair their function. This was almost self-evident, after long observation of these cases. He knew that this impression prevailed among the laity, and also among certain physicians. He believed that often the neurologists would be very glad to have the tendons divided in order that the foot might be brought to a right angle with the leg, and so get a useful limb. Such a division does bring about

the desired result, and in the same way does division of the muscles about the hip and leg. Dr. Gibney said that he could imagine that a very free division of the sartorius, the adductors and other muscles about the hip, might result in a dangling leg, thus making the patient worse. But when one found the muscles shortened, making it almost impossible for the patient to stand erect because of an extreme lordosis, he sometimes questioned whether it would not be better for the patient to have a dangling limb rather than a hideous deformity. He had failed to see good in these cases result from massage or manual traction.

Regarding atrophy of muscles caused by steel bands or other restraining appliances, he said he had seen many cases where illy-constructed appliances to palsied limbs had caused atrophy of the muscles; yet it was hardly fair to call such apparatus orthopedic appliances; they were the appliances of the shops. The ordinary orthopedic surgeon applied an apparatus for the relief of a dangle limb, the result of a poliomyelitis, aimed to get the points of support away from the muscles. The orthopedic surgeon's idea was to limit the motion in such joints as the hip, knee and ankle; it was believed that such a strain on the joints, by distortion of the limbs, and by hideous positions on the floor or on the bed, retarded repair and frequently rendered the patient more helpless. Thus the hip muscles stretched over night, while the patient slept with the limbs hyperextended, so that the heels touched the buttocks. The ligaments of the knee were likewise strained, and what little power remaind in those muscles was exhausted. Direct relief is not claimed for such an apparatus; it does not *per se*, develop the muscles, but it does enable the patient to assume the upright position, thus improving the circulation in the limbs, and preventing those fibres not wholly destroyed by the disease from overstrain. It also helps the masseuse and medical gymnasts to carry on their work better, and the neurologist to better reorganize power in muscles when relieved of tension.

Dr. George W. Jacoby said he was very much interested in the final remarks of Dr. Frenkel that neurologists would lose their occupation unless they gave up the systematic treatment of disease and paid more attention to experimental, bacteriological, and biological investigations, asking them to first take up the causal relationship in nervous diseases. Much attention has been given and is being bestowed the world over upon the etiology of nervous diseases; bacteriology and biology are special subjects for which the neurologist will find little time, considering the demands already made upon him. Neurology is by no means barren of practical results, and he had no fear that it would be absorbed by general medicine. Dr. Jacoby thought that Dr. Frenkel's attitude towards the mechanical treatment of nervous diseases had perhaps been too skeptical, except in his attitude toward the treatment of ataxia in tabes, about which he is very enthusiastic, and with cause.

In the mechanical treatment of nervous diseases, it seemed to Dr. Jacoby that two distinct lines could be drawn on the treatment of affections of the peripheral neurone; *i. e.*, in affections of the peripheral nerves, of the muscles, and of the anterior horns we were able to accomplish most by mechanical means. If we waited until the acute process had passed, we were able to make the muscular fibers yet existing do double work, making them take the place of the complete muscles in certain groups, and thus causing marked functional improvement in certain patients. He also believed that in the early stages of poliomyelitis,

by placing the limbs in a certain position, so as to cause a relaxation of the antagonists, we could prevent the contraction of these antagonists, a condition which is the cause of so much trouble.

When, on the other hand, we were dealing with disease of the motor tracts, we confronted an entirely different question. Here, excepting in those muscles which were bilaterally innervated, we could do nothing. If they were bilaterally innervated, nature could be aided and the opposite centres could be made to do the work of the two.

In the treatment of tabic ataxia the question too was entirely different; here it was not a question of the treatment of motor impulses, but a question of treatment of sensory defects. So long as the limb has a minimum of sensibility, Dr. Frenkel can re-educate the muscles, by means of this remaining amount of sensibility, and he could by this means convey to the central organs the knowledge of the position of the limbs. He made the minimum sensibility do the work of the entire amount that previously existed. This whole question Dr. Jacoby said was a very interesting one.

DISTURBANCES OF VISION IN A HYSTERICAL PATIENT, PRODUCED BY EXAGGERATION OF BINOCULAR ASSOCIATION.

By Prof. Pierre Janet, of Paris.

Dr. E. Gruening said that it gave him great pleasure to open a discussion on Dr. Janet's paper, because we should then have an opportunity to hear him speak again in closing the discussion. He said he was especially indebted to the Neurological Society for the invitation to be present to hear the classic and plastic recital of this remarkable case of hysteria. The eye is a favorite arena of functional disturbances due to hysteria, whether they present an exaggeration, or what Dr. Janet calls a deficit of function. Every oculist is more or less familiar with various phases of hysterical manifestations in the organ of sight. Thus we may observe complete blindness of one or both eyes, without anatomical substratum, diplopia, homonymous or heteronymous, due to fleeting paralysis or contraction of the one or the other ocular muscle, narrowing of the visual field to such a degree that only central vision remains, and the patient may read the finest print, yet be unable to find his way about. The manifestations are of so protean a character, and so impossible of explanation on physiological grounds that psychology has been called into service to supply working hypotheses. In hysteria, as in a fairy-tale, everything is possible, and the "nil admirari" on the part of the physician is nowhere more applicable than in its manifestations. Dr. Gruening said it would have been interesting to have had a chart of the visual field of that particular patient. Dr. Janet had not mentioned whether or not the field had been taken. The theory that the patient did not see with her good eye, because she hungered for binocular vision, and for this reason could not make use of her monocular sight was interesting, but not borne out in practice. People who lose one eye, have, as a rule, no difficulty in adjusting the remaining eye to the demands of their occupations. This, of course, does not apply to a case of hysteria, because almost anything can happen in this fourth dimension of medicine.

Dr. Gruening, in concluding his remarks, said he wished to state that to hear Dr. Janet present a case was a rare literary treat.

Dr. Pearce Bailey said that the history of the case reported by Prof.

Janet illustrated well the new lines of work being done abroad and in this country. Namely, the case of a woman with atrophy of the optic nerve of one side, the other side being normal who nevertheless was unable to see. Sight was restored after a careful analysis of all the psychological conditions which could account for its loss of vision, and applying appropriate means to counteract them. We are much indebted to Dr. Janet for analyzing functional cases hitherto unobserved and unstudied, showing the necessity for a careful analysis and study of every psychic symptom, and after a complete study of the case, applying the correct psychic remedy. The case reported by Dr. Janet was a good example of his work, and Dr. Bailey emphasized strongly the value of this work, which he had done for years in the Salpêtrière.

Dr. B. Onuf related a case which, although not of the same character as the one reported by Dr. Janet, seemed to belong to the same class. It illustrated how in a hysterical patient a group of physiological functions normally under conscious control may by habit hypertrophy detach itself from conscious guidance as to almost form an automatism of its own.

The case was one of a girl, with hysterical stigmata, such as contraction of the visual fields and analgesias and thermo-hypesthesias of a regional character, who on the basis of a slight refractional error (viz., a myopic astigmatism of one-half diopter of both eyes, combined in one eye with a myopia of one-quarter diopter), developed a spasm of the apparatus of fixation, *i. e.*, convergent squint, contraction of the pupils and spasm of accommodation. When seen, she had, in addition to this, a marked blepharospasm. The latter was regarded as grafted on the spasm of the apparatus of fixation through the habitual contraction of the pupils, which formed part of the spasm. This habitual smallness of the pupils made the retinas over-sensitive to light. When in the course of the disease the wearing of glasses, correcting the refractional error, had led to a subsidence of the spasm of fixation, and with it to a dilatation of the pupils, the increased influx of light thus resulting gave rise to an exaggerated form of the reaction normally following such increased exposure of the retina to light; namely, a blepharospasm.

Dr. Adolf Meyer said that the two guests of the night, who came from abroad, had brought reports in a field of neurology which, fifteen years ago, would have received but little thought and probably would have met with little hope; namely, the field of the functional consideration of neurology. So much had to be and could be done in the anatomical sphere and in localization that naturally study went in that direction. While it was not so very long ago that it was stated that neurology had about reached its limits, Dr. Meyer thought that they were beginning to see a widening horizon, even though there were limitations to the anatomical consideration. We must learn to study functions, as a condition for further function. Both Dr. Frenkel and Prof. Janet had shown clearly along what principles these studies must be made and how they were to be applied. This did not only pertain to the fields they had touched on; it held as well in the domain of psychiatry. Dr. Meyer commented on the brilliant presentation of Dr. Janet on how necessary it was to enter patiently upon the details of the individual's reactions, in order to reach the disturbing factors, and what is needed to build with, and how we should not mind merging in the use of hypotheses, etc.

JOINT MEETING

OF THE

NEW YORK NEUROLOGICAL SOCIETY

AND THE

PHILADELPHIA NEUROLOGICAL SOCIETY.

Held in Philadelphia, Nov. 24, 1906.

The President of the Philadelphia Neurological Society, DR. D. J. McCARTHY, in the Chair.

(*Continued from page* 330.)

PSYCHASTHENIC ATTACKS SIMULATING EPILEPSY.

By William G. Spiller, M.D.

In the *Journal für Psychologie und Neurologie,* Vol. 6, 1905-1906, Oppenheim discusses peculiar attacks under the title of Psychasthenic Convulsions. Although he has spoken briefly of this condition previously, he gives in this recent paper a full presentation of his views. Convulsions may occur in certain forms of neurasthenia, in cases which are not hysteria nor epilepsy nor organic. The first contribution to this subject was made by Westphal in 1872 (*Archiv für Psychiatrie,* Vol. 3), in his paper on agoraphobia, and according to this author the occurrence of convulsions with agoraphobia is not uncommon, and they may be seen as frequent signs of various psychopathic and neuropathic conditions. Oppenheim refers to the fact that Westphal's views have not received general acceptance.

As Oppenheim presents the subject, the individuals are intensely neurotic or psychopathic from birth, and show the first sysptoms of this diathesis in childhood. The neurasthenia is of the grave type which has been regarded by French writers, especially Janet and Raymond, on account of mental abnormalities as psychasthenia. The tics, states of anxiety, phobias, obsessions and vasomotor disturbances predominate. On such a foundation, with, however, some immediate cause, such as emotional disturbance, mental or physical overwork, alcoholic indulgence, especially by one unaccustomed to it, sleeplessness or a period of anxiety, the attack develops.

This may be only deep unconsciousness with involuntary defecation and micturition, or there may be also convulsions, biting of the tongue and rigidity of the pupils.

Usually only a few of these attacks occur, interspersed with periods of vertigo, anxiety, etc., and the tendency may disappear under proper hygienic treatment.

These attacks are not hysterical, every hysterical stigma is wanting, and the attacks themselves are not hysterical in character. Oppenheim dismisses the question of any resemblance to hysteria in a few lines. The differentiation from epilepsy is more difficult.

(1) The attack in itself cannot be distinguished from that occurring in epilepsy. The patient is not an epileptic, he has not had such attacks in childhood or early youth, he is always neurasthenic or psychasthenic, and always periods of anxiety, phobias, tics or vasomotor disturbances have preceded the convulsions.

(2) A special cause for the convulsive attack is always necessary, such as overexertion, mental or physical, anxiety, vertigo, etc.

(3) The condition is merely an episode in the course of the psychasthenia, the attacks are few, or there may be only one during the life of the individual.

(4) The attack may resemble fully the epileptic, but on the other hand there may be variations, thus profound unconsciousness may occur without convulsions, or the convulsions may be limited to a few muscles, or they may persist after consciousness has returned. The condition may resemble petit mal.

(5) Intelligence and memory do not become impaired even though the attacks may be numerous.

(6) The treatment should be mental, bromides are of little value.

Acquired neurasthenia probably never causes these convulsions. Oppenheim prefers the name of "psychasthenic convulsions," even though convulsions are not always present; "psychasthenic attacks" he regards as too comprehensive.

Mistakes of diagnosis may be made easily, indeed, Oppenheim himself has made them, as in one of his cases an organic condition was present, and Dr. Spiller is inclined to think that the danger of mistake is especially great as regards the dreamy state of epilepsy described by Hughlings Jackson under the name of uncinate group of fits. Dr. Spiller said he had not been able to find any reply to Oppenheim's views, and as yet they seem to have received little attention. The subject is, however, one of importance, because of the resemblance of these conditions to epilepsy, The word epilepsy conveys much dread to the patient and his relatives, but far more important is the fact that not only the diagnosis, but also the treatment and prognosis of the psychasthenic attacks are essentially different.

Dr. Spiller reported two cases.

Case I.—C., twenty-one years old, consulted him about five or six years ago, at which time the following notes were made:

A maternal uncle died in an insane ayslum. No convulsions had occurred in the family of either parent. The mother of the patient is very irritable, easily excited, and somewhat quarrelsome.

The patient has had five brothers, but no sisters. His father has shown great artistic talent. The first son is a sculptor and is irritable. The second son does not appear to be neurotic. The third son was very eccentric. He tried to commit suicide two or three times while at home, and finally succeeded. He at times became very much depressed and occasionally when he had these attacks he would wander away and stay away over night. Once he was absent two days. Before he wandered away his expression would become peculiar so that his relatives would know he was about to leave home. In one attack he went a distance of several hundred miles.

The fourth son seems to be normal. The fifth son is the patient. The sixth son is sixteen years old and is afraid to go into the dark.

C., the patient, has never had convulsions. When he was seventeen years old he had his first visual hallucination. He was in church. He heard the minister begin his sermon and then as he looked across the church he noticed that a certain man was looking at him. At first he liked the face, and something in it reminded him of a boy of whom he had been very fond, and whom he had not seen for about two years.

This boy had taught him masturbation. In this first attack he did not know what occurred about him, he got up and came out of the church after the service was over, and the money he had intended to put in the plate he had still in his hand. Whether this was unconsciousness or not is uncertain. He did not speak during the attack. After this first attack he began to hate and fear the face, and always had a warning before seeing it, "a sort of spasm would go through his whole body," or if he were holding a book his hand would tremble violently, and then if he looked across the church the "face" would be looking at him. It was always the same face and had always the same sneering expression. Except on one occasion the man was always in the same part of the church. He was not motionless, but was not seen by the patient to walk out of the church except on one occasion, when he followed the patient. During the first year the man was observed always in the same church, then he was seen in another church, and later was seen repeatedly on the street. The whole figure of the man was visible, but the patient spoke of the hallucination as "the face."

If the patient fixed his eyes upon the wall he could bring the figure of the man before him, but it did not seem real to him, and did not "satisfy him," as he expressed it.

The attacks occurred every Sunday during the first year, but not so frequently during the second year, and during the third year not more than four times. The patient believes the face is real. The attacks have been frequent proportionately as masturbation has been frequent.

He can always tell the day in advance that he will see the face. His eyes seem to be out of focus, he cannot keep them focused upon the model he is copying, and if he tries to do so he gets a bad headache, then becomes sleepy for the rest of the day. The aura usually occurs about four P. M. He has always had a bad taste in his mouth during the aura, "like cheese after you have eaten it the night before" or a "musty taste." If he falls asleep after the aura he has very vivid dreams, on one occasion he saw a comet coming toward him and exploding, and on another he saw clearly the face of his brother, who a few days later committed suicide. He has had the aura without seeing the face the following day, but has never seen the face without having had the warning the day before.

Within about fifteen minutes after the visual hallucination he gets sleepy and stupid, and does not know what is going on about him. The vision is followed by fullness of the head and palpitation of the heart. He has formed the habit of taking a back pew in church so he can hide behind the people and from the vision, and support himself against the wall. Only on one occasion he saw two children with the man. Each time he has had the vision his "eyes have gotten out of focus" and objects seem to move to and fro. He then has had a sick feeling and had to sit down. He has the same taste in his mouth during the hallucination that he has during the aura. Immediately after seeing the man his "ears ring like bells and insects all singing together." He has had occasionally aural hallucinations without other disturbances; *i. e.,* he has heard his mother calling him or bells ringing.

The young man is very intelligent. He has never wet his clothing during an attack nor cried out, nor bitten his tongue. When he feels he is about to have the vision and resists it, his face flushes, he gets cold and hot alternately, and all objects appear queer.

Dr. Spiller said he had seen this patient again within the past few

months. He is in excellent health. The visual hallucinations lasted one or two years after he was first seen, and then ceased entirely. They gave place to a difficulty in swallowing. The man believed he could not swallow and was depriving himself of food. An examination showed nothing abnormal to explain the dysphagia. This condition lasted about a year and a half and then disappeared.

Interesting in this report are: The neurotic family history; the attacks of wandering in one brother; the aura always preceding by one day the visual hallucination and associated with a bad taste in the mouth, suggesting Hughling Jackson's uncinate group of fits, and associated also with ocular disturbances and followed by drowsiness; the occurrence of the hallucination at first always in one place, a church, and therefore in a crowd, but later in other places and on the street; the resemblance of the face seen to that of a boy who had taught him masturbation; the frequency of the attacks proportionate to the frequency of masturbation; the bad taste in his mouth and sick sensation during the attack; the possible unconsciousness in only one attack; the drowsiness, fullness of head and palpitation of the heart following an attack, and the absence of all convulsions.

Case II.—A. B., thirty-six years of age, consulted Dr. Spiller Sept. 10, 1906. He was a patient of Dr. Radcliffe Cheston, and at the head of a large business. He comes of a neurotic stock. His father had nervous prostration, and now is tormented by unreasonable doubts as to the manner in which he conducts his business.

The patient is a hard worker. Some years ago he undertook to study a profession, but as he was engaged all day he was obliged to study at night. After passing his examinations he "went all to pieces," as he expressed it. He would sign a letter and after a few minutes would tear the envelope open to see whether he had signed the paper. On one occasion he visited his sister, and wandered about the house in a dazed condition. He was very irritable. Two or three years ago he went away for complete rest for fourteen weeks, and since then he says he has "felt his nerves" more. He had difficulty in fixing his mind on his work, and has frequently repeated his actions in order to be sure he had done his work properly, and often would read a paper without being able to fix his attention upon it. He took exercise in the same energetic manner in which he carried on his business.

He is the father of three healthy children, two of whom are "high-strung." He gives no history of sexual irregularity. He had never had any attacks of any kind before July 4, 1906. In the early part of the summer of 1906 he went to Europe, and took an automobile trip from Paris lasting two days. He travelled about two hundred miles each day. The weather was not very warm, but he was much tired by the trip. After the second day's journey, in the evening while talking to some one he fell and was said to be unconscious. His face was a little flushed, and his eyes had a vacant look. He was "perfectly limp" about ten minutes, then he became rigid in his feet, and had involuntary movements of the upper limbs of a purposive character. He was put to bed and had what he called a chill; he shook all over, his teeth chattered, he became red in the face and cold in the feet, but the thermometer showed no rise of temperature.

After this first attack he had weak spells in which he would sit with a vacant expression. These attacks occurred once or twice a day and

nearly every day. He was not unconscious in these attacks, although he seemed to be "wandering in mind."

The second severe attack occurred July 13, and in this he fainted. Other attacks occurred July 17 and 21. In the latter he had a vacant expression, became limp, then got up and tried to walk and fell after he had gone upstairs. He then became unconscious and was rigid in his entire body, but had no convulsive movements. After this attack was over he got up and went to bed, and had a chill and headache in bed.

Attacks occurred on July 24, 26 and 27. After the first two or three attacks he had a warning in a general weakness and sensation as if he were "charged with electricity." His memory began to be impaired. The attacks continued every few days until he got on a steamer, Aug. 29, on his return journey. He had two attacks on board the steamer. In all, he had twenty-five major attacks. The last attack was on Sept. 3. In one attack, when walking alone he fell and became covered with mud.

Before taking the steamer he had an attack in which he was unconscious one hour, and in this his body was more rigid than in the other attacks. In two attacks the face twitched, but convulsive movements were confined to the face.

He had taken a drink of whiskey or beer daily, occasionally some claret, and had smoked about twenty cigarettes daily. He had also been much worried about his wife's condition, as she had been in a hospital three times.

Before leaving America he had been having headache since April, 1906, more on the right side and in the parietal region or over the mastoid process, but this pain disappeared. There was no mastoid disease.

This was the history as obtained from the patient and his wife at the first interview. A further study of the patient by himself revealed some interesting facts. He said that sometimes when he was supposed to be unconscious he was not so, and the so-called rigidity was often purely voluntary on his part and was the result of an attempt to get the numbness out of his forearms and ankles. While he was on the ocean on his way to Europe he kept control of himself, but when he reached Paris he did not care and gave way to his feelings. He did not care whether he fainted or not, as it was a temporary relief to do so, and if he felt like sitting still and looking at a spot upon the wall he did not make a mental effort to avoid it. He would become weak after dinner and he began to dread this period, and if he could have been fooled regarding the time of day he believed he would not have fainted. He does not know why he fell.

The man presented no hysterical stigmata nor signs of organic disease. The treatment was psychotherapy.

He was seen again on Oct. 19, 1906, by Dr. Spiller. He had been weak twice, and had had constant slight headache. On one occasion he felt an attack was coming on, his wife urged him to resist it, but he pleaded with her to be allowed to fall, pushed her from him and said she must let him fall. His attacks have usually occurred when his wife was present. At this time he was again on a vacation with no business to occupy his mind, and he felt that he need not control himself. He said that if he were asked any time in the day whether he had headache he would reply yes, and yet usually he was not aware that he had headache. It was not real pain that he experienced, but as he expressed it, it was "consciousness that he had a head." He said if he had known that falling would have injured him he would never have fallen.

Dr. Morton Prince said that he thought pathologically these cases were to be regarded as types of hysteria, though the statement needed some explanation. We must make a distinction between a clinical and a pathological classification of disease. As he viewed the matter, hysteria from a nosological point of view is a clinical conception. That is to say, in practice it is customary to classify cases as hysteria purely on grounds of clinical symptom-complexes. That being the case it is purely arbitrary how extensive we shall make our symptom-complex.

On the other hand, we can make our classification on psychopathological grounds; in which case we might include under hysteria cases which clinically would exhibit diverse clinical manifestations. The question is therefore comparable to that of clinical and pathological diphtheria. We may limit clinical diphtheria entirely to membranous inflammation, but pathological diphtheria would include all cases in which the specific bacillus was found, irrespective of the clinical manifestations.

In an analogous fashion, if we can find a psycho-pathological basis for hysteria, we might include many cases in such a pathological conception which clinically appeared to have little resemblance.

Now recent researches seem to have shown that hysteria can be referred to a psycho-pathological basis, which is functional dissociation on the one hand and motor, sensory or ideational automatisms on the other. For instance, anesthesia, amnesia and paralysis are due to dissociation, while contractures, tics, hallucinations, hystero-epileptic attacks to automatisms. Either dissociation or automatisms may predominate and undoubtedly our knowledge of these alterations needs to be further worked out, and we are only on the threshold of a complete knowledge of them. But a classification based upon pathology must give us a far better conception of the disease than a mere clinical differentiation. The only question is whether our pathological conception is correct.

It is for the above reason that Sidis drops the word hysteria entirely and uses such words as psycho-pathic dissociation, psycho-pathic automatisms, etc.

Dr. Prince realized that such cases as Dr. Spiller reported are not those which ordinarily are clinically classified as hysteria, but he believed that fundamentally it would be found, if they were subjected to a searching analysis, that psycho-pathic dissociation and automatism was the underlying pathology; therefore he called them hysteria.

He also thought that Janet was too precise in making such a sharp difference between hysteria and psychasthenia. That writer's conception of hysteria necessitates a dissociation of consciousness producing a subconsciousness of which the subject is not *aware*. The question then of awareness of the obsessing ideas with him is a test of dissociation, but plainly we may have dissociations of which the subject is perfectly aware, as for example, dreams, hynotic states, automatic writing and speech, etc., etc. The obsessions of psychasthenia differ only from those of Janet's hysterics in that the subject is aware of the former and not of the latter. He did not believe this was a sound distinction, although he would not class all so-called psychasthenics as hysterics.

Dr. Spiller's first case, that of C., reminded the speaker of the case of M——ll, which he had reported with Dr. Sidis. The main difference being that the automatism in Dr. Spiller's case was purely visual, while in the M——ll case it was both visual and motor. The vision which M——ll experienced was that of his employer, whom he always saw in a dream at

the beginning of his attack. Dr. Prince thought that if Dr. Spiller's case C. had been hypnotized, as was M——ll, we would have found various sub-conscious phenomena which would have thrown light upon the attacks and would have placed it in the pathological category of hysteria, if we accept the above pathology of hysteria. He admitted that there were cases which are difficult with our present knowledge to bring within the above pathological conception of hysteria, and it may be that as Dr. Spiller claimed, we shall have to make a third division to be called strictly psycho-epilepsy. We should then have epilepsy, hysterical-epilepsy and psycho-epilepsy. The clinical problem would then remain, into which division to place any given case. This perhaps seemed to be taking a back track, but he should want to see the pathology of hysteria thoroughly worked out before making a third group of distinct pathology.

To illustrate his views, Dr. Prince described a couple of cases which he had seen. The first case was that of a woman who, a short time ago, had appeared at the clinic of the Boston City Hospital, complaining of epileptiform spasms, from which she had suffered for the past six months. The spasms occurred daily, sometimes several attacks taking place during a day. While the patient was being examined an attack occurred, apparently so far as one could see without ostensible cause, though, as it afterwards appeared, an unsuspected emotional cause was really present. The attack as witnessed was of the following character:

The patient remarked that an attack was coming on. She was standing at the time and described, on request, her sensation as they developed. There was first a sort of aura consisting of a feeling of being "gripped" in the region of the sternum. At the same time she experienced a strong beating sensation as of the heart in the same region; then she began to feel faint and was obliged to sit down. At this moment there developed a feeling in the lower abdomen as if it was being strongly drawn upwards; then followed a succession of spasmodic movements of the abdominal muscles, while at the same time there were clonic spasms of the diaphragm causing a series of inspiratory movements. Finally the muscles of the larynx and neck were thrown into strong clonic spasms. At this point she made an effort to speak, but though she moved her lips and tongue no sound was produced. From later investigations it appeared probable that this aphonia was due to tonic spasms of the laryngeal apparatus. (She knew what she was trying to say; namely, to describe her sensations.) The attack then passed off, leaving her for a few moments weak and with a severe headache. The whole attack from the beginning of the aura to the end of the spasms lasted perhaps two minutes, while the weakness and headache lasted a few minutes more.

The attack had all the appearance of Jacksonian epilepsy, for which for the moment it was mistaken. It was soon learned, however, that the attack could be brought on by a blow upon any part of the body; by this method, for the purposes of study, the attacks were brought on as often as desired. Further study and analysis revealed the fact that the attacks originated in an emotional shock and fright which she had received some six months previously and developed out of a delirious condition into which she fell as a result of the fright, and in which she imagined that she had epilepsy, or the same disease from which her mother suffered. This fear still persisted and brought on the attacks. It was very easy by suggestions to completely cure her.

The second case to which he referred was one which he had already

reported with Dr. Sidis. The subject, a young man, for five years had suffered from a series of attacks extending over a whole week, and which when superficially observed appeared to be Jacksonian epilepsy. During this week a condition representing status epilepticus developed. Through hypnosis it had been possible to show that the convulsions were due to subsconscious ideas originating in a fright which he had received five years before. The memory of the circumstances attending this fright had recurred periodically, but sub-consciously ever since. A cure was also brought about in this case.

Since this discussion, Dr. Prince has seen a case which in 1894 had been diagnosed by himself and one of his colleagues as petit mal, but which now in the light of a more searching analysis is plainly seen to be one of some form of psycho-epilepsy, and is made up of functional dissociation and automatism, call it by what name you will.

These cases illustrated the principle on which in his opinion such cases should be pathologically classified as hysteria, though clinically the manifestations might be very different from those which we are accustomed to group under this name.

Dr. L. F. Barker said that the subject of psychasthenia and its relation to other psychoneuroses was interesting them in Baltimore, and he, together with Dr. H. M. Thomas, the neurologist to the Johns Hopkins Hospital, had been studying a number of cases recently in the light of the more recent literature. They had come to the conclusion that a sharp differential diagnosis was often difficult, and that the boundaries of the individual psychoneuroses are as yet not very well defined. He was glad to hear Dr. Spiller's interpretation of the cases he had studied. The loss of consciousness in the attacks would seem to separate the cases from typical psychasthenia, at any rate if we accept Janet's definition of the syndrome. According to this French investigator the relation of psychasthenia to epilepsy is a very close one. He believes that in both diseases there are remarkable oscillations of the psychologic tension. He attributes both diseases to lowering of the psychologic tension. In epilepsy, however, the lowering is great and sudden and results in complete loss of consciousness for a brief time, after which the tension again rises, he thinks, so much that the patient feels very well, and does not even complain of the sensations of incompleteness which are so characteristic in psychasthenia. In some of the so-called epileptic deliria the true psychasthenic states are more nearly approached. Indeed, psychasthenia, according to Janet, may be regarded as a very attenuated chronic epilepsy in which the lowering of psychologic tension is continuous, and yet is never so great as to lead to complete loss of consciousness. If these conceptions are right, the cases described by Oppenheim, and to-night by Dr. Spiller, would have to be grouped as cases of psychasthenia with epilepsy. It is obvious that the place of such cases in nosology will depend entirely upon the definitions we give to the terms psychasthenia and epilepsy. The case Dr. Prince describes would, he thought, undoubtedly fall among true hysterical cases rather than in the psychasthenic group.

It seemed to him that the psychasthenic state may occur, perhaps, in a whole group of diseases of different etiology, just as the old typhoid state is now known to be a condition which may be manifest in infectious diseases of different origin. We must study our cases at present very objectively; later when we have collected sufficient data, a more specific differentiation will be possible.

Dr. C. K. Russell, of Montreal, Canada, said that one of Dr. Spiller's cases reminded him very much of a case observed at Queen's Square. It was a girl of about twenty-two, who had been in the hospital fourteen months previously with a condition very similar to what Dr. Spiller described. She had attacks starting with great fear, and thought she saw girls working in a room. She had never seen the girls or room before. In later attacks she saw rooms with golden pillars, then she saw some animals; but her attacks were all of the same nature and over a certain period were very similar. She was looked upon as having a hysterical condition. She left the hospital, but was kept under observation. She came back when he was there last year, and with these same attacks. Although her eyes were examined regularly there was nothing ever found until she had been in the hospital two or three days when she developed optic neuritis, which increased rapidly, and when they were discussing operation she suddenly died. At autopsy there was found a large tumor invading the temporo-sphenoidal lobe.

Dr. Dana said he was much interested in the description of these seizures because he had been studying and trying to classify them himself for some time. A year ago he had read a paper before the Academy of Medicine upon a condition which he called "para-epilepsy," in which he described a number of forms of sensory seizures which he thought could not be classed with hysteria or epilepsy and had ventured to suggest that they should be grouped together as a special periodical neurosis. The paper was only provisional and had never been published. There was such a difference of views as to what was psychasthenia and what was hysteria that it seemed difficult to him to come to conclusions about the nature of these attacks unless we agreed as to what we mean by the terms. There was a group of morbid mental conditions which were characterized by morbid fears, doubting mania, abulia, fixed ideas. These had been classed under the different names of degenerative insanity, neurasthenic insanity, psychasthenias, etc. He did not think the word psychasthenia exactly included this type now. But he thought all neurologists recognized the type as a special and easily recognized one. The sufferers from this condition which he had called "phrenasthenia" were subject to periodical seizures of various kinds. Some of them like that which Dr. Spiller described, though he had seen extremely few of them—only two. In most of the cases the seizures were slight in character and might be called the *petit mal* of phrenasthenia. In many of these patients their trouble began with an attack. For example, a patient he saw recently, after studying hard for two years, was sitting one evening smoking a strong cigar, when he suddenly felt a tremendous sensation of the nature of a general paresthesia come over him as if it were a *douche*. It began in the head, passed down like an aura from head to feet. He fell back and went into a condition of partial unconsciousness, with shakings and tremblings. In the course of an hour or so revived. After that he had a number of attacks coming several times a week in greater or less violence. His fear of the return of these seizures gave him agoraphobia. Many morbid fears are largely based on the fear that the patient will have one of his seizures. This class of attacks should be put with the seizures of psychasthenics.

(*To be continued.*)

Periscope

Brain

(Vol. 29, No. 114, 1906.)

1. An Address on Mendelian Heredity and Its Application to Man. W. Bateson.
2. A Contribution to the Study of Amaurotic Family Idiocy. F. J. Poynton, J. H. Parsons and Gordon Holmes.
3. A Case of Orbital Encephalocele, with Unique Malformations of the Brain and Eye. J. H. Parsons and George Coats.
4. A Study of the Various Changes which Occur in the Tissues in Acute Diphtheritic Toxemia, More Especially in Reference to Acute Cardiac Failure. Leonard S. Dudgeon.

1. *Mendelian Heredity in Man.*—Bateson presents a brief sketch of the Mendelian hypothesis of heredity in its applications to some of the diseases of mankind, notably color blindness, congenital cataract, etc. He urges the study of hereditary diseases in the light of the Mendelian hypothesis. The paper does not permit of an abstract.

2. *Amaurotic Family Idiocy.*—The authors, from an examination of three cases of this affection, show that: (1) There is strong evidence that it is a primary disease of the nervous elements. These are affected not only throughout the whole nervous system, but also in the dorsal root ganglia and in the retina, and so universally that not a single normal cell can be found. The changes which have been described, and all the evidence that could be obtained on the mode of their development, prove that the disease is not due to a primary affection of the neuroglia, but that the proliferation of the latter has only followed the degeneration of the nervous elements. Further, there is no evidence to suggest that the changes of the nervous elements resulted from any vascular condition, there was no visible disease of the vessels and no signs of either old or recent inflammation. (2) Though they employed different methods for the demonstration and estimation of pathological changes in the different parts of the neurone, it can be asserted that the nerve cells are relatively more affected than the fibres. In many systems, as in the direct cerebellar tracts, the dorsal and ventral spinal roots, and the optic system, there may be no visible change in the fibres though the cells from which they spring are severely altered. From this fact it may be concluded that the affection is a primary cell disease, and that the alterations visible in nerve fibres are only secondary to this. (3) The condition of the nerve cells as demonstrated by the elective stains (Nissl's, Bielschowsky's), for their component parts, shows that the interfibrillar protoplasm is very much more severely affected than are the neurofibrils, and suggests that the primary change is disease of the interfibrillar protoplasm, and that to this the alterations in the neurofibrils are secondary.

Certain conclusions can also be drawn on the etiology of the disease from its morbid anatomy in conjunction with its clinical symptomatology: (1) It is not due to arrested development. If it were so, the clinical symptoms of the disease would be probably evident from birth which has not been so in any of the cases observed; and there is no reason why a mere

arrest of development should produce a progressive and invariably fatal disease. There is also little anatomical evidence of mal-development, and the appearance on which Sachs lays so much emphasis can be explained by the wasting the cerebral convolutions must undergo as a result of the disease. Further, the most easily-obtained evidence of the completed development of the nervous system, myelinisation of the brain, proves that the final development of the different parts of the brain is completed at different periods in a fairly long space of time, and is not completed till a few months after birth. But the examination of these brains does not indicate greater abnormalities in the regions which develop late than in those where development is completed early in intra-uterine life; *e. g.*, the visual cortex, which is myelinated early, is quite as severely affected as the prefrontal region where the myelinated fibres appear late. If, however, the disease dates from the earlier months of extra-uterine life, the development of the fibres which myelinate late may be checked owing to deficiency of trophic influence from the diseased cells. (2) The negative results of the bacteriological examination and the entire absence of such reactions in the vascular and lymphatic systems as frequently accompany bacteriological infections suggest that the disease is not due to bacterial toxins. The clinical facts also argue against this theory; its family nature, its occurrence only in the Jewish race all militate against this view. (3) The final conclusion possible, which is supported by the microscopical investigation, is that the disease is due to some inherent biochemical property of the cells, as a result of which it undergoes certain changes which result in its degeneration, and, consecutively, in degeneration of the parts of the neurone (neurofibrils, axis-cylinders and myelin sheaths), the normal existence of which is dependent upon it. The cell changes have not the characters of a simple atrophy; in fact they seem to be due to an excessive growth of the protoplasm which later undergoes degenerative changes. This fact is not in favor of Sachs' hypothesis that the pathology of the disease can be described by the term abiotrophy (a term suggested by Gowers to represent an inherent defective vitality in the cell), or of Schaffer's suggestion that it may be explained by Edinger's "Ersatz-theorie," which assumes that elements are inherently feeble and undergo degeneration when exposed to the strain of life to which they are not normally resistant.

3. *Orbital Encephalocele.*—The authors present a detailed report of an extremely rare congenital abnormality of the eye, and an apparently unique malformation of the brain. Three congenital abnormalities of the globe were present: (1) Coloboma of the choroid, (2) Coloboma of the optic nerve entrance: the ectasia had taken place to the inner side of the intervaginal space, and the latter has not been displaced with the nerve, but has retained its normal position, (3) A central or macular coloboma. The globe abnormalities were probably due in large part to the orbital encephalocele. An isolated mass of brain substance had probably been snared off at an early period of foetal life and had interfered with the proper budding of the primary optic vesicle. It seemed probable to the writers that this alone would not explain the anomaly. Since there was no defect below, either in the nerve or in the retina, it seemed evident that a secondary optic vesicle was formed and closed in a similar manner, and that the defects in the globe were caused by the pressure of the brain

masses on the growing secondary optic vesicle. The macular coloboma remains inexplicable.

4. *Changes Due to Diphtheria Toxemia.*—Dudgeon contributes a lengthy, well-illustrated and detailed morphological study of the tissue changes in acute diphtheritic toxemia, paying particular attention to the changes thought to be responsible for acute cardiac failure. Sixteen cases served as a basis of the examinations in conjunction with extensive experimental work. The changes found in the tissues coincide with those with which pathologists are already familiar. He concludes, however, (a) That the most important lesion in the acute cases is a fatty change of the heart muscle and diaphragm, which is due to a direct action of the toxins on those tissues, (b) That similar fatty changes may be found in certain of the important viscera, especially the adrenal gland and liver, (c) That the expression "cardiac paralysis" in acute diphtheritic toxemia should be abolished and replaced by "acute cardiac failure," (d) That the changes found in the nervous system are secondary factors and not the primary cause of cardiac failure, (e) That the antitoxin, if given in sufficient quantity and within the first forty-eight hours, may prevent or considerably diminish the possibility of death from cardiac failure.

JELLIFFE.

Miscellany

FRACTURES OF BASE OF SKULL. (Annal. of Surg., December, 1906.)—Drs. L. R. G. Crandon and Louis T. Wilson analyze 530 cases of fracture of the base of the skull. The mortality was 44 per cent. Alcoholic intoxication was present in 32 per cent., of whom 38 per cent. died. Many of these developed delirium tremens, which could not be wholly differentiated from symptoms of laceration or meningitis. Forty-four per cent. were unconscious at first examination, and fully half the conscious ones were dazed or stupid. Fifty-eight per cent. of the unconscious died, against 33 per cent. of the conscious. Early delirium was usually due to alcoholism, but when late, infection was the cause. Hemorrhage from at least one cranial orifice was present in 74 per cent., the order of frequency in this series being the ear 281, nose 168, subconjunctival 53, two ears 47, mouth 44. In practically all cases of aural hemorrhage the drum was ruptured. Where pharyngeal bleeding occurs the mortality is very high. Respiration as a symptom is unimportant except in primary shock and toward a fatal termination, and the quality of the pulse is of greater significance than its rate. In 117 cases presenting stertorous breathing 106 died. A subnormal temperature seems to indicate shock or alcoholism. Nearly all those with high initial temperature died. The escape of cerebrospinal fluid was noted in only 27 cases of this series. The most striking result of the study of pupillary change was that in 142 cases without reaction 131 died. Of these 530 cases 59 were operated, with a mortality of 53 per cent. The writers conclude that only those cases should be trephined where either hemorrhage seems the most important part of the clinical picture or the course is considered one of continuous and progressive intracranial compression. Patients with non-reacting pupils, or unconscious with stertorous respiration are nearly always bleeding deep in the brain substance, and are hopeless. It is urged that in suspected basal fracture that rest in bed for three full weeks be enjoined. COWLES (New York).

CEREBROSPINAL MENINGITIS. By J. S. Billings, Jr. (Journal A. M. A., June 2).

Dr. Billings gives a history of the epidemic of cerebrospinal meningitis in New York City in 1904-05, with the methods employed to meet it. It

began in the early part of 1904, and continued through 1905. During 1904 there was a total of 1,083 deaths and a death rate of 4.6; during 1905, 1,511 deaths and a death rate of 6.3. Fifty-five per cent. of the cases were males and 45 per cent. females. The cases occurred mostly in children, only 19 per cent. being adults and only 1 per cent. over 50 years of age. Italians seemed particularly susceptible and negroes the least. The great majority of the patients were evidently of the poorer class, 76 per cent. residents of tenement houses and often living under insanitary conditions. In only 6 per cent. had there been any direct exposure to other cases of the disease and in only a small number was there evidence of direct transmission. In the majority the attack began without antecedents; only 6 per cent. were in bad health. The onset was sudden in all but 5 per cent. Stiffness of the neck was the most common symptom and closely following it came vomiting, headache and convulsions. Eruption was present in 30 per cent. of the cases; it was petechial in 19 per cent., and herpetic in 11 per cent. Nasal discharge was noted in only 13 per cent. Of complications, those of the eye were most frequent, next came muscular paralyses, then otitis. In 33 per cent. of the cases, lumbar puncture was performed to confirm the diagnosis and the meningococcus was found in 82 per cent. In the remainder the diagnosis was by clinical signs only. Death usually was due to coma and exhaustion. About 7 per cent. of the patients died on the first day, less than 34 per cent. during the first five days and 39 per cent. after ten days. Recovery was complete in 84 per cent. of the recoveries. Diphtheria antitoxin was employed in 313 cases, with a mortality of 69 per cent. Large doses were no more effective than small ones. While its harmlessness was demonstrated, no credit is given it as a curative agent. In conclusion, Billings says that little light has been thrown on the mode of transmission of the disease, nor has any effectual treatment been discovered. It is to the laboratory that we must look for further light. One imortant fact brought out is that, in all probability, the disease is much the most infectious during the first two weeks. The Department of Health acted on this knowledge, enforcing quarantine, etc., during the first two weeks and insuring disinfection of rooms and bedding. As a possible consequence of this the number of deaths reported for the first six weeks of 1906 have been 102, as compared with 170 for the same period in 1905.

Uniocular Optic Neuritis and Retinitis. By A. A. Hubbell (Journal A. M. A., July 7).

The author remarks that the ophthalmologic text-books are comparatively silent on the subject of unilateral inflammation of the optic nerve and retina. After a number of references to the literature, which, he says, is also comparatively scanty, he reports and analyzes eighteen cases of his own observation within the past fifteen years. It is difficult, he finds, to fix any definite etiologic relations of the conditions. One-sided nephritis existed in one case; there was more or less arteriosclerosis in five patients, two of whom also had albuminuria; valvular disease of the heart, together with previous cerebral hemorrhage, was found in one patient, while the remainder were classed as healthy and without cerebral, vascular or kidney disease. In a quarter century's experience in ophthalmologic practice, he has never seen a case of optic neuritis that was not uniocular except when there was syphilis or brain tumor. He has never seen a case of neuroretinitis or retinitis that was not uniocular, except when there was double orbital cellulitis, Bright's disease, diabetes mellitus or syphilis. He does not claim, however, that this must have been the experience also of others. The treatment he has employed has been almost invariably the administration of iodid of potassium, which he thinks has been of benefit in some cases. In others it seemed ineffective. Hubbell considers the condition more common than is indicated in ophthalmologic literature.

Book Reviews

THE DISSOCIATION OF A PERSONALITY. By MORTON PRINCE. London, 1906.

Miss Beauchamp is a person in whom several personalities have become developed; these follow in regular succession, often changing from hour to hour. The self with which she was born may change to any one of the other selves, each of which has a different character. Two of the selves have no knowledge of each other or the third; when one of these is replaced by another there is a blank in memory; when it reappears it finds itself of what it has said or done just before. Miss Beauchamp is an actual case of Stevenson's Jekyll and Hyde.

Many cases of such multiple personality have already been reported in considerable detail. Dr. Prince makes an exhaustive study of this one. It would be hopeless to present in brief form the mass of details that he has collected. Portions of the book make very interesting reading, although there is a lack of systematic presentation making it very often confusing.

SCRIPTURE.

THE MANAGEMENT OF A NERVE PATIENT. By Alfred T. Schofield, M.D. P. Blakiston Sons & Co., Philadelphia, Pa.

The book represents the author's ideas of psychic treatment of functional nervous disorders and goes over the ground of ethics to be practiced between the consultant and the consulting.

The idea is strongly brought out that the fascination quackery has for people is mainly by the means of suggestion, and that the only way to overcome the evil is that physicians learn the value of suggestion and use it, thus obviating the necessity of irregular practitioners.

The essential traits in the physician are shown to be sympathy, patience, perseverance and attention to details. The author is resourceful in suggestions regarding the management of a case.

He takes as a premise that matter is governed by mind, and the latter not always being in the same vigor there are consequent deviations in the organic processes, the first aim is to get the patient's confidence, change the outlook on life and influence the unconscious mind so as to energise it for good; suggestions to be made indirectly to the unconscious mind by environment and treatment and changing the course of thought.

The value of hypnotism is doubted, emphasis is laid on a change of environment during sickness and afterward, and that the recovered health must never be used above its limit.

The general ideas are broad in scope, there is perhaps too much entreaty for the ideas advanced, instead of a bold slash at the principles involved.

It is not a book so much for the specialist as it is for the newly-graduated student.

S. D. LUDLUM.

JAHRESBERICHT UEBER DIE LEISTUNGEN UND FORTSCHRITTE AUF DEM GEBIETE DER NEUROLOGIE UND PSYCHIATRIE. Herausgegeben von Dr. E. Flatau und Dr. S. Bendix; Redegiert von Prof. Dr. E. Mendel und Privatdozent Dr. L. Jacobsohn. IX. Jahrgang. Bericht über das Jahr, 1905. S. Karger, Berlin.

The ninth volume of this masterly review has appeared. It is arranged in the same complete and thorough manner as its predecessors. It is unqualifiedly the most valuable general reference book that we possess, and no active worker in neurology or psychiatry can afford to be without it.

JELLIFFE.

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Original Articles

UNILATERAL PARALYSIS AGITANS OCCURRING AFTER HEMIPLEGIA.

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Unilateral paralysis agitans, so-called, is not an uncommon condition. Two distinct forms can be recognized: that in which the more obvious symptoms of the disease appear first on one side, or are at least more pronounced upon one side, and that in which the paralysis agitans has developed equally, but subsequently has, in consequence of hemiplegia, ceased, either temporarily or permanently, upon the paralyzed side. I have been unable to discover the record of any case in which, some years after an attack of hemiplegia, paralysis agitans has developed and remained restricted to the healthy side. The following instance of this condition, therefore, seems worth reporting.

Mrs. D., now 76 years of age, had, at the age of 69, a stroke of apoplexy, causing paralysis of the right side. Speech was affected; the mouth was drawn to the right, and the leg and arm were paralyzed, but apparently incompletely. The speech soon became normal again, and there was a considerable return of power in the right arm and leg. At the age of 73—four years after her attack—she began to notice shaking in the left arm and hand, which has continued to the present time, apparently increasing very moderately in severity.

Owing to the somewhat imperfect memory of the patient, the family history is untrustworthy, and the early previous history, possibly for the same reason, appears to be unimportant. The present condition is as follows:

She is very stout; the legs are edematous; the peripheral arteries are in a state of advanced sclerosis; there is a loud systolic murmur, heard best at the aortic cartilage, and transmitted clearly into the vessels of the neck. Speech is apparently normal,

although the patient claims that she cannot speak as well as she did a year or two ago. The pupils are equal, and react to light, but she states that her eyesight has failed recently. The expression is mask-like. In the left hand there is a persistent tremor. At the wrist this consists in movements of extension and flexion; the thumb is in a position of apposition, and, with the fingers, exhibits the characteristic pillroller's tremor. When the patient attempts a voluntary movement the tremor stops completely, and may remain absent for as long as two minutes, but if the movement continues for a longer time the tremor reappears, at first faintly, but soon becoming more violent than during rest. There is no tremor whatever of the right arm or hand during either rest or movement. There is moderate impairment of power in both arms and hands, apparently about equal in degree, but the movements of the right arm appear to be more limited than those of the left. The patient states that within a year the left arm was considerably stronger than the right. There is a tremor in both feet at the ankle, resembling the oscillations of clonus. This tremor disappears if the feet are forcibly flexed dorsally. The tendon reflexes of the arms and legs are absent. Distracting the patient's attention by asking her to count or look at the ceiling does not cause them to appear, and Jendrassik's method cannot be tried. The Babinski reflex is not present on either side. Walking is impossible, probably in part due to weakness, to the severe edema of both legs, and to the excessive weight. While sitting in her chair there is no sign of either propulsion or retropulsion. There is distinct rigidity to passive movement of the left arm.

To summarize the case briefly: Seven years ago a woman of 69 developed right hemiplegia, from which she made a partial recovery. Four years later she first noticed tremor in the arm and hand of the opposite side, which has continued until the present time. There is now the typical symptom-complex of paralysis agitans in the left arm, with paresis of the left arm, and tremors in both feet. In short, there is an incomplete Parkinsonian hemiplegia, and some traces of an old organic hemiplegia.

The first observation of a case of unilateral paralysis agitans is credited to Marshall Hall, but as the tremor in his case (a young man) developed only upon voluntary motion, then becoming violent, and as there were also curious movements of the eyes, and disturbances of speech, it is fair, I think, to conclude that his patient was suffering from multiple sclerosis. Later observers, however, have generally united in believing that paralysis agitans,

at least in its incipiency, is nearly always unilateral, although they all state that in the course of a few years it becomes generalized. (Brissaud, Eulenberg, Erb, Berbez, Wollenberg, Williamson, and Gowers.) Fuerstner is the only author who states positively that the disease may be limited to one side throughout its entire course. Dutil records a case of 2 years' duration, atypical in some respects, in which the tremor had remained limited to the left side. The other symptoms were general in character. Berbez, in 28 cases examined in Charcot's clinic, found 3 cases in which the tremor was unilateral, and 7 in which it was more pronounced on one side than on the other. The three unilateral cases were in the early stage, and there was no reason to believe that extension might not ultimately take place.

Regarding the arrest of the tremor by hemiplegia, there seems to be very little definite information in the literature. Eulenberg and Lamy, both of whom have written comprehensive articles on the subject for systemic works, have failed to mention it at all. On the other hand, Gowers, Wollenberg, and Williamson speak of it as a well-known fact. Gowers states that, if the paralysis is not complete and permanent, the tremor may return, and Williamson states that the arrest of the tremor after cerebral hemiplegia is usually transient. Williamson makes the assertion that there are two cases on record in which the arrest of the tremor was permanent. Examination of the literature cited in the monographs of Wollenberg and Williamson has failed to show upon which actually recorded cases their statements were based.

The practical value of observations of unilateral Parkinson's disease, and the effects of hemiplegia upon the tremor, consists of its aid in the determination of the localization of the lesion. This localization has been made in every part of the motor tract, from the cerebral cortex to the muscular fibers.

The results of histological examination and investigations have been contradictory. Changes have been found in the spinal cord similar to those found in the cords of old persons suffering from general arteriosclerosis, but these changes are by no means constant, observations by competent neuro-pathologists having failed to reveal any changes at all. (Fuerstner.) No characteristic changes whatever have hitherto been found in the cerebral cortex, although the majority of neurologists now believe that the lesion is situated there. A few, basing their opinion upon some of the

symptoms, and the fact that occasionally tumors of the peduncle have caused symptoms resembling paralysis agitans, believe that it is in the basal ganglia. Blocq and Marinesco believe that the lesion affects chiefly the motor tract in the lower portions of the brain.

It seems hardly worth while to repeat the various arguments in favor of the cerebral localization. That based upon the unilateral occurrence of the disease is not entirely satisfactory because, in disseminated sclerosis, even when the cord is chiefly involved, the symptoms may be preponderant upon one side, and the fact that paralysis agitans almost invariably invades the other side, would be rather in favor of a general brain disease, or at least a disease affecting the brain bilaterally, even if unequally, than a disease affecting one hemisphere. It is easy to understand how diffuse lesions of the pons or medulla might, under exceptional circumstances, produce tremor only upon one side of the body. In practically all recorded cases there have always been symptoms that show that the opposite side was sharing in the disease. Neither does it seem to me that the cessation of the tremor, after hemiplegia, necessarily indicates a cortical situation. It merely suggests strongly that the cause of the tremor affects the central motor neurones, not the peripheral neurones. And this view is supported by the frequent reappearance of the tremor with the restoration of power, which means a recovery of the central motor neurones. It is of somewhat more significance that the moderate injury of these central neurones may serve to check the subsequent development of the tremor in the injured side.

There is no question that, in a part that has been subject to a tremor, it can be more readily produced than in a part in which tremor has never occurred. In the same way, the tremor of paralysis agitans, if inhibited for a time by a sudden injury to the central motor mechanism, may be apparently easily re-established; but if the central motor mechanism has never learned to produce a tremor, then it is unable to acquire it after a moderate degree of injury.

Of course, in the present case, although the time that has elapsed since the onset of the tremor in the left arm is rather longer than usually occurs without the development of a tremor in the right arm, no definite statements can be made as yet. It

is still possible that bilateral tremor will occur, just as, seemingly, it has already occurred in the feet; but even if it should subsequently occur, it will be justifiable to ascribe the long delay to the hemiplegia.

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A STUDY OF REFLEXES OF THE LOWER EXTREMITIES IN SIXTY CASES OF PARESIS, WITH A SPECIAL REFERENCE TO THE PARADOXICAL REFLEX.*

By Alfred Gordon, M.D.,

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A systematic study of cutaneous and deep reflexes in paretic dementia has been made by very few observers, and that of Babinski's sign particularly only by a few isolated writers. The consensus of opinion is that the "phénomène des orteils" in paresis is an exception. Very recently Robert and Fournial (*Revue Neurologique*, No. 21, 1906) studied 66 cases and found Babinski's sign present only in one case. The reason of this rarity it is apparently difficult to explain, as on one hand involvement of the motor tract in paresis is the rule, and on the other Babinski's reflex is unquestionably a sign of an involvement of the motor pathway in its central or terminal ends.

An ensemble study of all the reflexes well known to be the expression of disease of the motor tract appeared to me to be of some scientific interest.

As I have shown from my first clinical studies (Journ. of Nerv. and Ment. Dis., July, 1906), and subsequently from the anatomo-clinical observations that the paradoxical reflex is "a sign of irritation or early stage of a lesion of the motor tract," it occurred to me that a study of this sign in a disease in which Babinski against all expectations is absent, may be of some pathogenetic value.

Anglade in 1898 (*Arch. de Neurologie*), Wyruboff in 1900 (*Revue Neurol.*), have shown on a very large number of pathological specimens that the participation of the cord in paresis is a constant fact, and while the posterior columns are very markedly affected, the pyramidal tracts are to a lesser degree, but nevertheless invariably involved. Wyruboff's study shows that all paretic cases from the anatomo-clinical standpoint can be divided into three groups.

(1) First concerns cases characterized by markedly ex-

*Read at the meeting of the American Neurological Association, May 7, 8 and 9, 1907.

aggerated knee-jerks and anatomically by a degeneration of the posterior columns in the thoracic region and a recent degeneration of the pyramidal bundles.

(2) In the second group the knee-jerks are abolished, which corresponds to a considerable degeneration of the posterior columns in the lumbar regions and some degeneration of the pyramidal tracts.

(3) In the third group there is a marked degeneration of the posterior columns through the entire cord and again some changes in the pyramidal bundles.

Klippel, who made extensive studies of paresis, is also of the opinion that a degeneration of the motor neurone in general and of its portion in the cord particularly is a constant phenomenon in paretic dementia.

The conclusion at which one can arrive from these studies is that while the motor pathway is always involved, nevertheless it is only to a very slight degree in the majority of cases. As Babinski's sign is usually the clinical expression of a well-defined degenerative lesion of the motor tract, the very moderate degree of pyramidal involvement will perhaps satisfactorily explain the rarity of the "phénomène des orteils" in paresis.

On the other hand the study of the paradoxical reflex in organic nervous diseases suggests very strongly that it is the expression of a slight or early involvement of the motor tract. This is proven beyond any doubt, I believe, by the anatomical cases reported: one by Dercum (JOURN. NERV. AND MENT. DIS., Sept., 1906, p. 593), and the other by myself (*Revue Neurol.*, No. 22, 1906).

The presence of this reflex in a large number of paretic cases and the comparatively slight involvement of the pyramidal tracts are therefore highly interesting and logically must be considered more than a simple coincidence. In the study of 60 well developed cases of paresis I found the paradoxical reflex present in 42 instances: in 35 cases on both sides and in 7 only on one side.

The reflex was present in 39 cases together with increased knee-jerks and in 3 cases with absent knee-jerks. The latter fact is quite remarkable for this reason, that in all my previous studies the paradoxical reflex was always found to be asso-

ciated with exaggerated patellar tendon reflex.

This anomaly is only apparent. The pathological studies of the above writers show that even in cases of paresis of tabetic type with loss of knee-jerks, alongside of total degeneration of the posterior columns, there is invariably also some involvement of the pyramidal fibers.

In two cases with increased knee-jerks the paradoxical reflex was absent. In one case with normal knee-jerks the paradoxical was also absent.

In the seven cases with one-sided paradoxical reflex, the latter corresponded to an exaggerated knee-jerk which was present on the same side and absent on the other.

Babinski's sign was present only in 3 cases on both sides and one side in 4 cases. It coincided with spasticity and markedly exaggerated knee-jerks.

The antagonistic relation of Babinski's and paradoxical reflexes observed by me in my previous studies is fully corroborated also in my present investigation. In all cases except two where Babinski was present, the paradoxical was absent, and vice versa.

Oppenheim's reflex deserves special mention. Some neurologists expressed the opinion at the time of my first communication that the paradoxical reflex is perhaps a modification of Oppenheim's. In my second series of organic cases reported to the Philadelphia Neurological Society in December, 1905 (JOURN. OF NERV. AND MENT. DIS., July, 1906), Oppenheim's reflex was absent in 41 instances out of 58; only in 5 cases it was present together with the Babinski and paradoxical reflexes. In the two pathological cases mentioned above the paradoxical reflex was conspicuous, all others, except increased knee-jerks, were absent, and operative procedures were decided upon because of the exclusive presence of the paradoxical reflex. One case was seen by Keen, Dercum and myself, the other by Mills, Dercum, Da Costa and myself. In my present study of paresis I laid special stress upon the relation of the paradoxical reflex to Oppenheim's, and the result was this, that while the former was present in 42 cases, Oppenheim's only in 8 cases, in 4 of which it was bilateral and in 4 unilateral.

Ankle-clonus was present only in 11 cases, in most of which it was slight, and only in one case pronounced.

CONCLUSIONS

The present study appears to me to be of certain importance for the following reasons:

The old triad of reflex phenomena, viz., exaggerated knee-jerks, ankle-clonus and Babinski's sign, which we are accustomed to find in hemiplegia or in any other organic disease with a distinct degenerative lesion in the motor pathway, is here markedly dissociated. While the first is present in the majority of my cases, the other two are rare. Of the two more recently described reflexes, Oppenheim's is also rare, while the paradoxical is frequent.

In view of the fact that the pathological studies of competent observers show a constant presence of motor fibers' lesion, and that the latter is in the majority of cases only very slight,— the rarity of Babinski's and Oppenheim's reflex, also of ankle-clonus, and the frequency of the paradoxical phenomenon acquire I believe for this reason a valuable clinical significance.

These observations were made in the presence of Drs. H. M. Stewart and B. Robinson, internes at the Philadelphia Hospital. I am indebted to Dr. Hawke for the courtesy extended to me.

SENSORY AND MOTOR DISTURBANCES IN PARTS ABOVE THE DISTRIBUTION INVOLVED BY DEFINITE ORGANIC LESIONS OF THE SPINAL CORD.*

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From the Department of Neurology and Laboratory of Neuropathology of the University of Pennsylvania.

The object of this paper is to call attention, first, to the gradual extension of the areas of disturbed sensation above the peripheral distribution related to definite lesions of the spinal cord, and secondly, to the marked increase in the tendon and skin reflexes in the same parts, and in parts above. These facts have not, heretofore, been noted.

These observations are based upon three cases, one with necropsy. In all, the lesions were of long standing, being 35, 29 and 4 years in duration, and the sensory phenomena were carefully studied and noted at varying intervals. The lesions were of traumatic origin, and the symptoms were of such clearness that it was possible in each case to make a clear-cut diagnosis in definite localities of the spinal cord.

The cases are as follows:

Case 1. Morgan. Was admitted to the nervous wards of the Philadelphia Hospital June 13, 1899, where he still is. His past and family history are of no importance, has never used alcohol or tobacco; and has never had syphilis.

When 27 years of age he was struck across the lumbar spine by a piece of iron and rendered unconscious. When he recovered, he was unable to use his lower limbs, and has never had more than slight use of them since. He has never had any bladder or rectal disturbances, or bed sores. Considerable pain was present in his lower limbs for some years after the injury, and girdle sense has always been present.

Notes by Dr. Van Epps made in January, 1900, state that the patient had from January 1 to 14 herpetic eruptions accompanied by much pain in the left side of the body extending from mid to mid line laterally and from costal margin to iliac crest posteriorly, and the pubic line anteriorly. The vesicles gradually disappeared but the pain continued for some time.

Examination on November, 1900, 28 years after the injury,

*Read at the meeting of the American Neurological Association, May 7, 8 and 9, 1907.

by me, denoted almost total loss of power in the lower limbs. They were considerably wasted and spastic, more so the right. The cremasteric, patellar and plantar reflexes were present and prompter than normal; ankle clonus was present on each side, but the Babinski reflex could not be obtained.

Sensation for all forms was absent to a line corresponding

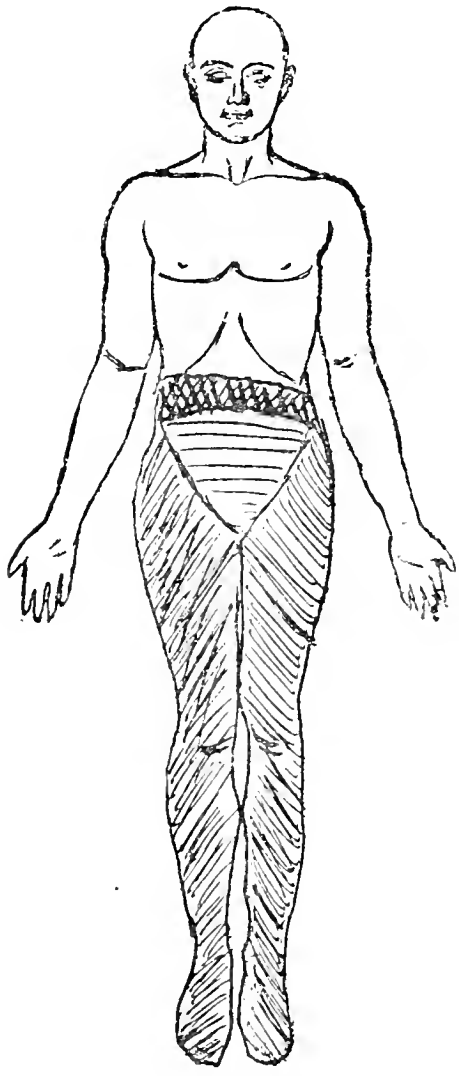

Fig. 1.

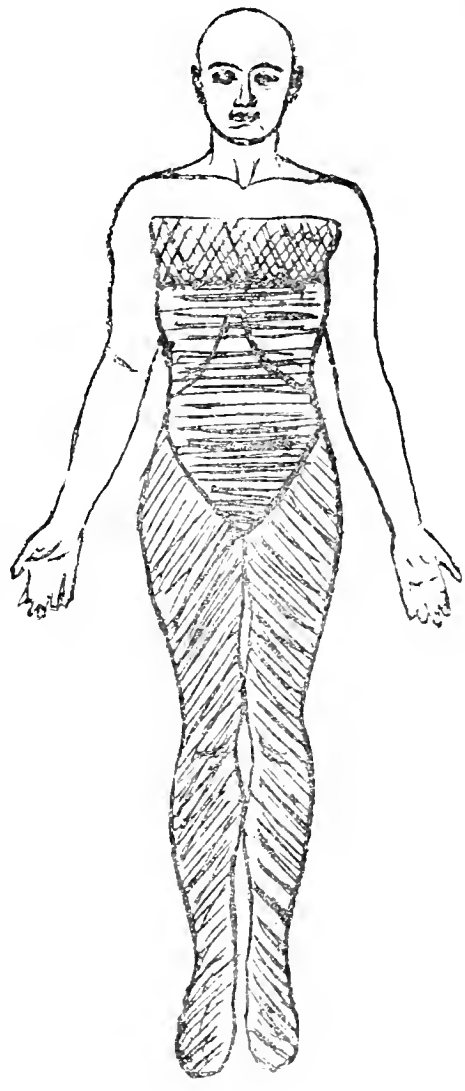

Fig. 2.

Fig. 1. The shaded areas represent disturbed sensation in 1900.
Fig. 2. The shaded areas represent disturbed sensation in 1907.

with the umbilicus. Above this point pin prick was appreciated, but touch and temperature sensations were not normally recognized until two inches above the umbilical line; just above this line there was a zone of hyperesthesia and the patient complained here of a girdle sense.

Examination by me on February, 1907, seven years after the last examination and thirty-five years after the injury,

showed practically the same condition as to his motor power in the lower limbs. He could only slightly move the toes and knees. The cremasteric and patellar reflexes were still increased, but ankle clonus could not be obtained; the Achilles jerks were present. Plantar irritation produced flexion of all the toes. The bladder and rectum were normal.

The state of the sensation altered considerably. In my first examination of this year, he was unable to recognize touch, pain and temperature sensations over the lower limbs and abdomen up to the line of the nipples, and sensation did not really become normally appreciated until about the first intercostal space. Subsequent examinations have confirmed this, with the exception that pin prick was occasionally interpreted correctly below the nipple, but never more than two inches above the line of the umbilicus. (Fig. 2.) Bone sensation as tested by the tuning fork was diminished in the lower ribs, but was gradually better appreciated as the upper ribs were approached, and was normal about the second rib.

Power in the upper limbs was normal, and there was no apparent atrophy present. In the finger to nose test some ataxia was noticeable, but this was not marked. There was some hypotonia in the elbow joints. The sense of position was about normal, but on one occasion when absolute relaxation was obtained, it was demonstrated to some extent in the fingers, wrist and elbow joints. The biceps, triceps and wrist reflexes were prompter than normal, more so in the right side. On tapping with the percussion hammer over the chest and shoulder prompt contraction could be obtained in the corresponding muscles, so that practically reflexes could be obtained wherever desired.

X-ray examination made by Dr. Leonard D. Frescoln showed an inflammatory thickening (probably periostitis) around 10th to 12th thoracic and 1st lumbar vertebræ.

Summary. A man of 27 years, as a result of a severe blow to the 10th, 11th and 12th thoracic and first lumbar vertebræ sustained complete paralysis of the lower limbs. There must have been an almost complete transverse myelitis of the 9th, 10th, 11th and 12th thoracic spinal segments. An examination made by me 28 years after the injury denoted a spastic paralysis of the lower limbs with increased tendon reflexes, and disturbed sensation up to a line 2 inches above the umbilicus. The bladder and rectal functions were at no time involved. Examination made also by me 7 years later and 35 years after the injury showed the same condition as to motor power in his lower limbs, but sensation was disturbed to the first intercostal space. The tendon and skin reflexes in the upper limbs and in the reflex arcs of the chest and shoulder muscles were markedly exaggerated.

Case 2. Egan. Was admitted to the nervous wards of the Philadelphia General Hospital, May 11, 1892. His family and past history were of no importance and there was no history of alcoholism or syphilis. In 1884, at the age of 35, the patient lost his balance while on a running freight car, and fell to the track, striking his lumbar spine. He then lost

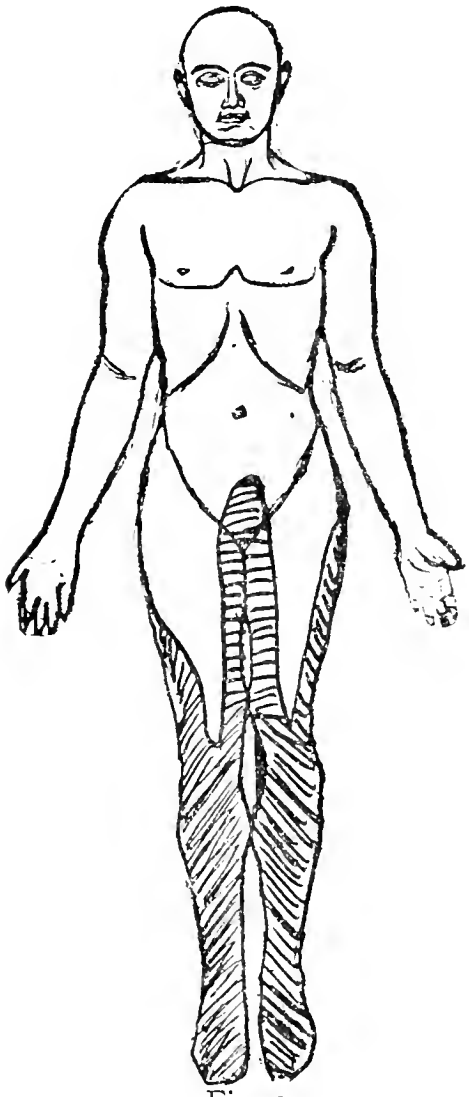

Fig. 3.

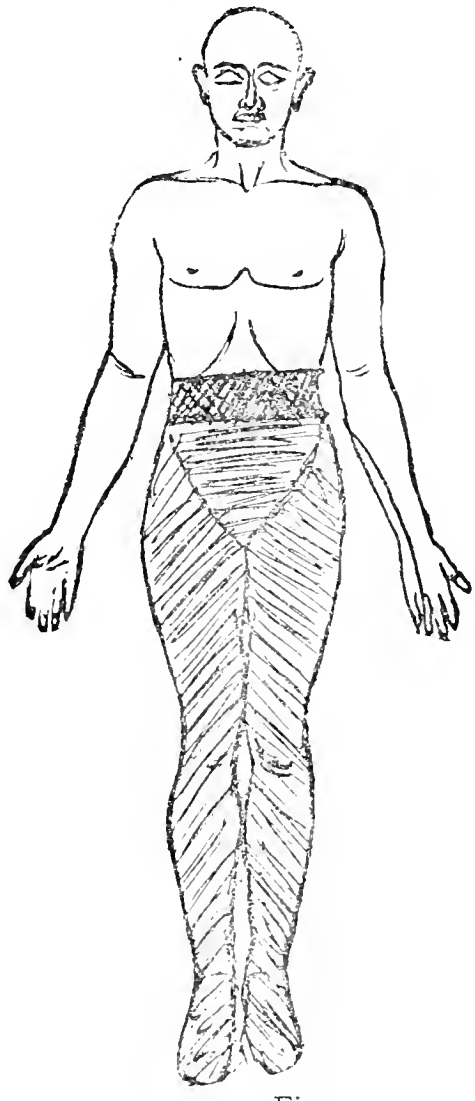

Fig. 4.

Fig. 3. The shaded areas represent disturbed sensation in 1899.
Fig. 4. The shaded areas represent disturbed sensation in 1907.

almost all power in his lower limbs and control of his bladder and rectum.

Notes made in June, 1899, 15 years after the injury, by Dr. G. E. Pfahler, state that the patient had loss of power in the lower limbs with the exception of some ability in flexing the right thigh on the abdomen. Bladder and rectal functions were completely lost. Sensation was lost as indicated in chart No. 3.

Examination by me in February of this year (1907), 23 years after the injury and eight years since the last examination, showed almost the same condition as regards the motor power of the lower limbs. The only movement possible was that of slight flexion of the right thigh on the abdomen. The toes were extremely hyperflexed and cyanotic. All of the tendon reflexes in the lower limbs were lost. Plantar irritation produced no movement of the toes. Bladder, rectal and sexual functions were totally lost.

Sensation for all forms was totally lost in the lower limbs and buttocks, with exception of a small area just in the inner surface of both thighs, and over the lower parts of the abdomen, to an irregular line about two inches below the umbilicus. From this line to a line drawn about 1 to 1½ inches above the umbilicus, sensation became gradually better appreciated, being normal at the above point. (Chart No. 4.)

Power and movement was normal in the upper limbs. The biceps and triceps tendon reflexes were very prompt, more so on the left side. On tapping with a percussion hammer over the chest and shoulders prompt contraction could be obtained in the corresponding reflex arcs. The abdominal reflexes were lost on both sides, as were also the cremasteric. In the finger to nose test, some ataxia was present, more so in the left limb. Sensation was normal in the upper limbs.

The X-ray examination denoted caries of the body of the third lumbar vertebra, especially towards the left side.

Summary. A man of 35, as a result of a fall, became completely paralyzed in the lower limbs with total loss of bladder and rectal functions. There must have been a complete transverse myelitis of all portions of the cord below the 2nd and 3rd lumbar segments. An examination made 15 years after the injury denoted almost a complete flaccid paralysis of the lower limbs with the exception of a slight ability to flex the right thigh on the abdomen. The bladder and rectal functions as well as all of the tendon reflexes were totally lost. Sensation was disturbed over both lower limbs, buttocks and perineum with the exception of a tringular area over the front of the thighs. An examination made 8 years later and 23 years after the injury showed the same condition as to the motor power in the lower limbs, but sensation is now disturbed to a line about 1½ inches above the umbilicus. The tendon and skin reflexes in the upper limbs and over the chest and shoulder are markedly increased. There is some ataxia in the upper limbs.

The third case, a brief history of which is only given, does not illustrate the sensory and motor changes which are the subject of this paper, but is used because it was a case of traumatic myelitis in which a careful microscopic examination

was made, and in which the pathological findings are illustrative of the changes which probably have occurred in the two similar clinical cases here recorded.

Case 3. This case was recorded by Dr. Spiller and myself in the *Review of Neurology and Psychiatry,* October, 1904. A young man, because of an injury to the back, sustained complete paralysis of both lower limbs and of the bladder and rectum. All of the tendon reflexes of the lower limbs were lost. When examined by me one year after the injury, sensation was lost to a line corresponding about 2 inches below the umbilicus, and further examination made 3 years later and 4 years after the injury denoted absence of all sensation to a line drawn about 1 inch below the umbilicus.

A careful microscopic examination of sections taken from the upper part of each segment of the spinal cord from the 10th thoracic to the upper part of the cervical enlargement showed that as high as the upper part of the 10th thoracic segment, the cord was completely destroyed. Many normal fibers entered the cord in the posterior roots of the 9th thoracic segment.

Examination of the sections above the 9th thoracic segment stained by the Weigert hematoxylin method showed that the posterior root entrance zones, and the columns of Burdach and Goll were much better and more deeply stained than the other portions of the cord. A marked thickening was also found in the walls of the arteries, besides the usual ascending secondary degenerations.

We have here, therefore, two cases of definite lesions of the spinal cord; one involving the 9th to the 12th thoracic segments; and in the other the spinal cord below the 2nd or 3d lumbar segments. In both the lesions were of long duration, being 35 and 23 years, and the sensory symptoms were studied and noted at intervals of 7 and 8 years respectively. The areas of sensory disturbances increased in the period of 7 years in the first case from a line drawn 2 inches above the umbilicus to the 1st intercostal space, and in the second case from irregular areas over the thighs to a line drawn about 1½ inches above the umbilicus. In these areas of increased sensory phenomena, sensation was lost absolutely only about 2 or 3 inches, and above these parts there was a gradual shading off of disturbed sensation. The important point is that the areas of disturbed sensation have markedly increased above the limits previously found.

What are the causes of this increase in the sensory disturbances? The first answer which naturally presents itself, is an extension of the myelitic process, and secondly, the possible influence of the secondary ascending degenerations.

Let us consider first the possibilities of the occurrence of an ascending myelitis. It is well known that whenever the spinal cord is injured, even in a restricted portion, that the parts both above and below the point of injury may have small areas of hemorrhage or of softening. In fact the whole extent of the spinal cord may be so involved. In most instances after the subsidence of the acute process, these areas of softening and myelitis disappear and no trace of them is to be found, and clinically no symptoms appear. It is more than probable, however, that they are replaced by neurogliar tissue and we may have here the origin of a future sclerotic process.

The spinal cord receives its blood supply from the anterior and posterior spinal arteries, both of which arise from the vertebral, and from the intercostal branches. These intercostals have ascending and descending branches, which anastomose freely with one another and with the anterior and posterior spinal arteries, making an arterial network about the spinal cord, from which numerous small branches penetrate the surface. The gray matter receives its blood supply principally from the anterior spinal arteries, and it is to be noted that this supply is largely from different branches than that of the white matter. It is also to be remembered that the arteries which enter the cord are terminal. Considering this, it can be readily understood how a destruction of a number of spinal segments would cause interference with the blood supply, first of the whole cord, and after the acute symptoms have subsided, especially of the contiguous parts. This arterial obliterative process in common with all degenerations has a tendency to increase, and if the patient lives long enough, would involve considerable portions of the spinal cord.

In the case with necropsy (Case 3) the arterial degeneration is evident. Sections of all parts of the cord show marked endarteritis and this seems to be much more apparent in the lower parts of the cord near the lesion, and especially so in the periphery and in the lateral columns. The vessels in the gray matter seem less sclerosed, the reason for this greater endar-

teritis in the white matter of the cord being the more ready involvement of the arterial supply. This has only recently been called attention to by Allen.[1]

It is probable that a similar arteritic process is present in the clinical cases here recorded, but of much greater intensity as the lesions are of 35 and 23 years' duration as compared with 4 years in the case with pathological findings. This endarteritis causes impairment in the nutrition of the parts; this resulting in a gradual necrobiosis and impairment of function. In this is to be found the probable explanation of the increase in the sensory disturbances.

What influence, if any, do the secondary degenerations have? This question has long been in dispute, from the time that Charcot first explained the occurrence of the contractures in hemiplegia as the result of secondary degeneration of the motor columns. Take, for instance, in the case with necropsy. Here as a result of a myelitis in the 9th thoracic segment, marked secondary degenerations are to be found in the cervical cord. In studying such sections stained by the Weigert hematoxylin method it seems as if these extensive degenerations must have some influence, if not upon the neighboring fibers, at least upon the fibers which mingle with them.

Let us try to analyze this. In a lesion of one internal capsule interrupting the motor columns, secondary degeneration results throughout the whole extent of the pyramidal tract. Such degeneration will, at first, only be apparent by the Marchi and later by the Weigert hematoxylin method.

The question is whether this has any influence upon the structures which are associated with it. We assume that there is a connection between the cells of the anterior horns of the spinal cord and the motor columns, and if this is true, as we have every reason to believe, a degeneration of the motor columns should have influence upon these cells, and yet pathologically this has never been satisfactorily demonstrated. That is no argument, however, that this does not actually occur. Clinical evidence shows sufficiently that in hemiplegia there are atrophy and vasomotor disturbance; these have never been satisfactorily explained, but are probably due to a loss of

[1] A. R. Allen, University of Pennsylvania Medical Bulletin, 1905.

tone of the whole motor system of which the motor nuclei in the anterior horns of the cord play a prominent part.

When a paralysis occurs, as for instance, because of a capsular lesion, how is loss of power produced? There is as a result of this lesion, immediate disturbance of physiological relation between the cortical motor cells, the pyramidal tracts, the cells of the anterior horns, the anterior spinal roots, the peripheral nerves, and the muscles, tendons and fascia these supply. More than this, there must be some disturbance of the normal relation with the sensory arc.

This disturbance of physiological relation, or "diaschisis," as Von Monakow has termed it, is, of course, immediate, and will become permanent in varying degree; this depending upon the extent of the original lesion. And how is this disturbance of relation manifested? Clinically by loss of power and pathologically by degeneration of fibers. This degeneration, which is called secondary, is progressive as shown by the reaction to the Marchi and Weigert stains. So far our microscopic methods have failed to demonstrate any changes in the cells of the anterior horns, and in the nerves and muscles. This, however, is mainly because of the recuperative power possessed by the motor cells on account of the innervation received from other sources, and the independent trophic action which they possess. We see then that what is termed secondary degeneration is really nothing more than the physiological or mechanical death of a part.

What action, if any, then, has this secondary degeneration or physiological death of a part? Evidently none, for whatever has produced this, has at the same time produced loss of function in the related parts. Secondary degeneration, however, does produce from the very nature of its slow death, some physiological disturbance. Take for instance the motor columns. Even though there is a complete destruction of these as a result of a capsular lesion, other motor fibers, the so-called extra-pyramidal bundles, join the motor columns in their course downwards. Whether these bundles intermingle freely with the motor columns in the pons and medulla, or whether they are independent in their course, has not been fully determined; but it is known that they ultimately join the motor columns in the spinal cord and probably mingle

with them. We can readily imagine that the degenerated or dead fibers must have some detrimental action upon the healthy fibers.

This can be better demonstrated in the so-called ascending or sensory degenerations. Take, for instance, in Case 3, in which because of a lesion in the 9th thoracic segment, the secondary degenerations are marked. As we go higher and higher, newer fibers enter by means of the posterior roots, and join either the posterior columns or the columns of Gowers and the direct cerebellar tracts. These newer fibers intermingle, or at least, accompany the degenerated fibers.

This influence is probably the result of a vascular change. The blood vessels that supply or are in association with a degenerating or degenerated tract must share somewhat in the sclerotic process, and it is conceivable that as a result of this obliterative arteritis, some degeneration is produced in the contiguous or intermingling healthy fibers.

In the case with necropsy a careful examination of sections of the spinal cord above the lesion showed that the fibers in the posterior columns and in the entrance root zones were much better or more deeply stained than the fibers in the other parts. The pallor in the centripetal columns can be explained as the result of secondary degenerations, but this does not explain the apparent degeneration in the motor columns. Retrograde atrophy of the pyramidal tracts has been described, and it occurs in the motor columns above destructive lesions of the spinal cord. This is as we should expect, for when physiological relations are disturbed, as they would be in such case, there must be some disturbance of function in the whole motor system as has been indicated above. This disturbance is manifested pathologically by a downward degeneration of the motor columns below the point of lesion, but is not apparent in most instances in the parts above, at least, by our pathological methods. This is because these fibers still retain their trophic cortical innervation. Reasoning from this then, retrograde atrophy must occur in every case, in the motor as well as the sensory tracts.

The increase in the sensory phenomena in the parts above the peripheral distribution related to definite lesions of the cord, has an important practical bearing. Most of our obser-

vations upon sensory areas are based on definite lesions of the spinal cord, in which no attention has been paid to a possible increase of the sensory phenomena, as is pointed out in this paper. Manifestly then such observations in which the lesions are of long duration cannot be as accurate as those which are the result of lesions of a shorter time. In this is to be found one explanation of the great variance in the sensory diagrams.

The second part of this paper is to again call attention to the increase of reflexes in the parts above definite lesions of the spinal cord. This clinical fact was first called attention to by me in 1904. ("Clinical Report of Three Cases of Injury to the Lower Spinal Cord and Cauda Equina."[2]) In this paper I called attention to the increase of patellar jerks in a lesion of the cauda equina. As this subject is an important one and as no other writer has contributed to it, some of the remarks then made pertaining to the subject will be quoted.

"I have been unable to find any reference to this condition in the literature at my command, except in Thorburn's[3] contribution to the surgery of the spinal cord. This author mentions the following cases with increase of the patellar reflexes, but pays no special attention to this subject. One case I have found also reported by Franz Volhard.[4]

"First case, cited by Thorburn from Kirchoff; backward crushing of the first lumbar vertebra causing a degeneration of the fourth and fifth sacral segments, the only symptoms being paralysis of the bladder and rectum and increased patellar reflexes. Sexual and sensory changes are not mentioned.

"Second case: a partial compression of the cauda equina about the level of the last lumbar vertebra, causing severe neuralgic pains in the sciatic and pudic distributions, a weakness of some of the muscles of the lower limbs, but no complete paralysis and no anesthesia of the limbs. The bladder and rectal functions were paralyzed. The patellar reflexes were slightly exaggerated, but there was no ankle clonus.

"The third case is cited by Thorburn from Oppenheim, and was one of fracture of the first lumbar vertebra, causing a my-

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[2]The American Journal of the Medical Sciences, May, 1904.
[3]Philadelphia, Blakiston & Son, 1899.
[4]Deutsche med. Wochen., 1902, No. 33.

elitis of the conus medullaris. There was a slight weakness of the calf muscles, otherwise no loss of power or atrophy. Anesthesia was limited to the peroneal region and buttocks. There was paralysis of bladder and rectum. Here also the knee-jerks were exaggerated, but there was no ankle clonus.

"The fourth case, by Volhard, of a tumor of the cauda equina, as proved by necropsy, with motor and sensory symptoms, absent Achilles jerks, and weakness of the plantar reflexes, but both the patellar reflexes and the cremasteric reflexes were exaggerated.

"In all four of these cases, therefore, the exaggeration of the patellar reflexes was probably caused by lesions below the reflex arc."

In the two clinical cases now recorded, the tendon reflexes in the upper limbs, that is, the biceps and triceps, were very prompt and the skin reflexes over the shoulders and chest were exceedingly exaggerated. Tapping over any portion of the chest, shoulders or back produced prompt and marked contraction in the corresponding reflex arcs. How is this to be explained? Quoting again:

"It is difficult to find a satisfactory explanation for such a phenomenon as this, but evidently the reflex arcs are in some way thrown into a state of excitation in these cases. No writer has made this a subject of careful study, and it seems therefore important to emphasize the fact that a reflex may be increased by a lesion in the spinal cord below the portion in which the reflex arc is represented. Considerable evidence is offered that in the nervous system there are both depressomotor and excitomotor fibers for the different reflexes, these having their origin in the brain; and it may be that other excitomotor fibers arise. We know that spinal roots on entering the spinal cord give off descending branches that pass downward in the posterior columns; the function of these fibers is entirely unknown, but we must assume that in some way they affect the function of the lower segments. It is probable that in a similar manner the lower spinal segments exert some influence over higher segments, and there is no doubt whatever that in the antero-lateral columns degeneration of short fibers occurs upward. It is presumable that these fibers exert some control over higher segments than those in which they

arise, and in this way possibly cause an exaggeration of tendon reflexes."

It is also probable, as has been shown in the present paper, that the disturbance in the arterial supply in the parts above definite lesions of the cord, produces a disturbance in the functions in the motor columns, this being further influenced by the retrograde atrophy. As has been shown by Rothmann, the only constant symptom of a lesion of the motor column is an exaggeration of the reflexes. Here, then, is to be found the explanation of the reflexes in the parts above lesions of the spinal cord.

The following conclusions can be drawn from the study of this paper:

First: An injury to the spinal cord will cause at first interference with the blood supply of the whole cord. If, as a result of such an injury, there should be produced a limited lesion of the spinal cord, there will be interference with the blood supply of the contiguous areas. This interference will be progressive, thus causing arterial obliteration and necrosis in the contiguous parts.

Second: This progressive degeneration will cause disturbance of function, it being manifested clinically by a gradual extension of the areas of disturbed sensation, and by increase in the tendon and skin reflexes.

Third: A lesion in any portion of the spinal cord will cause disturbance of physiological relations in the associated parts. This disturbance is greatest directly after the injury, and becomes less in the course of time.

Fourth: Secondary degenerations "per se" do not produce any direct symptoms, for whatever has produced secondary degenerations, has at the same time caused loss of function in the related parts. Secondary degenerations, however, cause some physiological disturbance. Every degenerating or degenerated tract has healthy fibers from other sources mingling with it. The arterial degeneration present in the involved tract will cause degeneration in these healthy fibers, and of the immediate fibers surrounding the degenerating tract.

KORSAKOFF'S PSYCHOSIS SUPERIMPOSED UPON MELANCHOLIA.*

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In April 1906 issue of the Journal of Nervous and Mental Disease, there appears an article by Dr. Wm. A. White, in which he sets forth the theory that there is no reason for believing that a patient already suffering from one form of mental disease may not develop another during the course of the primary disorder, or as he expressed it at one point in his article: "That because a person has manic-depressive insanity is no reason he should be immune from the ordinary diseases that affect the brain and impair the mind. He further lays particular stress upon the fact that the clinical picture of a given psychosis may be greatly modified by episodic intoxication and infection processes, resulting in what would be confusing and anomalous symptoms did we not recognize the possibility of such a combination. To his very able and clear exposition of this theory I can add nothing, and would refer you to his original communication, but this belief appeals to me as a very sound and tenable one, and I wish to present for consideration the report of a case which seems to have very clearly shown the co-existence of the characteristic symptoms of melancholia of involution and Korsakoff's psychosis.

As a matter of fact, the question has been raised as to whether the group of symptoms described as constituting Korsakoff's psychosis is merely a syndrome, or a definite clinical entity; but the considerable majority of investigators have accepted the latter view.

The following is a report of the case in question:

A. C., female, white, single, aet, 44, American, occupation none. One of four children, the other three being quite normal. She comes from a very refined, and intellectual family. No unfavorable hereditary history. The patient has, however, since childhood shown signs of constitutional degeneracy. At school she was one-sided and erratic in her accomplishments. Notably brilliant in certain subjects, she was greatly deficient in others, particularly the exact sciences. As she grew into young womanhood, she manifested many very noticeable peculiarities of conduct.

*Read before the Brooklyn Society for Neurology, Oct. 25, 1906.

She was seclusive, could never be particularly interested in anything, lacked initiative, and failed to show the "get up and hustle" of the average American woman of her intellectual attainments. Though not of robust health, she had never had any serious or prolonged illness previous to the present one. She never used drugs or alcohol in any form. Signs of the menopause presented some months previous to the onset of her illness, and she has not menstruated since December 1905.

She has masturbated for many years, and at intervals greatly to excess. This latter was the case during the autumn of 1905.

In the spring of 1905 she began to manifest undue emotional depression, which increased, and according to the account of her family physician, gradually evolved into the clinical picture of melancholia—painful emotional depression, ideas of self-unworthiness, and then distinct delusions of self-accusation.

She was sent away to the country for a time, and was then taken back home for a few months, remaining there until December last. It is to be carefully noted that the family physician, whom I consider quite competent to judge, states positively that up to this time she had not presented the amnesia and disturbance of orientation that subsequently became so marked for a time.

In December there came on suddenly an attack of severe pain in the head, face, and arms, lasting about 48 hours. It was looked upon at the time as neuralgia, and there were no physical sequelæ after the abatement of the pain to indicate that it was anything else. This, however, I do not consider as absolutely proven. Mentally, she did show a distinct change for the worse, and as her brother expressed it, "completely went to pieces." He then sent her to the sanitarium, (Dec. 21,) which however, is not an institution for the insane, and hence the reports that I have been able to get as to her mental condition while there are far from being as minute as I would wish. However, they indicate that she was much depressed emotionally, had marked self-accusatory delusions, thought her soul eternally lost, etc. At times became exceedingly agitated, and begged persistently for poison, "because she could not and would not live any longer." Once she tried to put her head in the fire, and at another time talked about jumping out of the window. They were uncertain as to the condition of her memory, but stated that orientation was impaired. There were no local evidences of neuritis. Her deep reflexes were increased, and her pupils dilated. Pulse rate tended to be high, particularly in her periods of excitement, when it reached 105-120. Her family physician states that normally her pulse rate is 75-80.

She was admitted to the Long Island Home, Feb. 13, 1906.

She was that morning brought from the other sanitarium to New York on a private car, and from thence to Amityville by special train, accompanied by her brother and a nurse.

The initial examination showed that she was in a rather poor physical condition, badly nourished, very weak, so that she walked unsteadily, skin was muddy, tongue was coated, pulse 120, temperature normal, pupils normal, patellar reflexes greatly exaggerated, superficial reflexes normal, no disturbance of sensation demonstrable. Her facies was dull and indicative of mental torpor.

She understood what was said to her, and her answers as a rule were relevant, though occasionally she interjected a question or remark whose relation to the subject under discussion was not apparent. There was very little mental activity present, and it was frequently necessary to urge her in order to secure a reply to questions. There really seemed to be a question as to whether apprehension was entirely clear, for she was unable to clearly grasp the full meaning of any other than short questions.

She possessed no true insight, though she said that she had been ill, and that her physician had ordered her to bed.

It was in the field of memory and orientation that the most startling revelations were made. She had but the most vague and indefinite recollection of having been on the train with her brother that morning, and was absolutely unable to give any details of the trip here, though she did know that she had been in New York City that morning. She at first said that she came to the Home that morning in company with her brother and sister (it was really her brother and a nurse), but a short while afterward she declared that she was still at the other sanitarium (which I will designate as X), that she had been here 5 or 6 weeks, and had never been on Long Island. She said she went to X in June, but did not know of what year—thought it might be 1904. At first she said she did not know the present month, but finally said it was June, though she still adhered to her statement that she came here (to X) in June, and that she had been here five or six weeks. She was unable to see the inconsistency in this. She was unable to tell what she had had at the previous meal, and had no recollection of certain statements that she had made to her nurse that morning. Fifteen minutes after this visit she had but the most vague and indefinite recollection of ever having seen the examiner before, and had forgotten all about the visit.

Feb. 14, 1906.

Passed a sleepless though not very restless night. Talked a great deal about suicide, and her wish to commit the act. Very difficult to induce her to take sufficient food. There is very much more mental activity to-day, so that she answered my questions readily, and spontaneously entered into conversation.

Her whole thought seems dominated by the idea that her family have deserted and repudiated her and left her to her own resources. She relates quite in detail, repeating the exact words used, a conversation supposed to have occurred between her and

her brother when he visited her here (at X) three or four days ago. He told her that he had discovered certain things, and that now he is done with her, that she could provide for herself in the future, that he would no longer pay her board here, would no longer furnish her with clothing, etc., because she had grossly deceived him. As a matter of fact, no such conversation ever did occur, nor did she see her brother at all on the date she states. She becomes reticent when questioned as to the details of why he had spoken to her so, but admits that it was because of her wrong-doings, "about which she does not care to pray."

In all of this, to which she constantly reverts, and assures me over and over again is true, she gives no evidence of acute emotional depression, but on the contrary, there seems to be rather a lack of emotional feeling of any kind, so that her emotions seem to remain at an uniform level, with neither tears nor smiles.

The same disturbance of orientation and memory continues. She has been here five or six weeks, came Dec. 21, 1905, and it is now June 8, 1906. Is unable to see the inconsistency. She has no recollection of my visit and examination of yesterday. She thinks she has seen me several times, but don't know just when. Could not tell what she had had for breakfast. Only after very pointed suggestions from me did she recall having been on the train at all recently, entirely denying it at first. She still insists that she is at X, and will not accept my statements to the contrary, and has forgotten that I explained to her yesterday her present location and how she got here. Her appreciation of the passage of time is much impaired, and she says it has been three or four days since she saw her brother who was here with her yesterday.

She is ready to bridge over the past with ready explanations, though she does not glaringly fabricate spontaneously. However, she does make certain highly improbable statements about occurrences at X, such as that one of the buildings there fell down, that another one burned etc. (I have subsequently learned that these statements are without foundation in fact).

Given two numbers to remember, she was unable to repeat them ten minutes later. This amnesia occurs in the presence of clear apprehension.

Pulse remains very rapid.

Feb. 16, 1906.

She was very depressed and agitated yesterday evening because her family had deserted her, etc. Her family has suffered great wrongs and calamities on her account, her brother has lost all of his property and his son died from grief, all caused by her wickedness. She wished she was dead, felt that she ought to be in hell, and begged the nurse for chloroform. This morning, however, she is fairly cheerful and ready to smile on occasion, though

she still holds the same delusions. No evidence of acute mental pain arising therefrom.

Her mental processes are active, apprehension is unimpaired, and her answers to direct questions are usually relevant, though a peculiar condition is to be noted, when in the midst of a sentence she breaks off into another on an entirely irrelevant line.

Hallucinations of hearing are present.

When questioned she says her brother did not bring her here to this place, but that she cannot remember the name of the place he did take her to. "He took me to the place where the fire occurred. They are all Presbyterians there, and the fire destroyed that beautiful stone porte cochere."

Q. "What place is this?

A. "You said it was some place on Long Island."

Q. "Would you have known that had I not told you so?"

A. "No."

Q. "Do you remember being on the train with your brother on the trip here?"

A. "No. My brother has never been here. Just before the fire he went to New York with me."

Q. "How long ago was that?"

A. "Six or seven weeks."

She says she does not remember anything about how she got here.

Q. "Where were you before you came here?"

A. "I don't know that either. Was it not at Dr. Weis's that the sanitarium broke down? I give it up. The large things I remember perfectly, but the little things I forget."

Q. "When did you go to X?"

A. "Ah, that's the place. I went there in December."

Q. "Of what year?"

A. "Of this year."

Q. "Well what year is this?"

A. "1907, is it not?"

Q. "What month is this?"

A. "February."

Q. "How long have you known me?"

A. "Three or four days."

She cannot remember the number that I gave her yesterday to repeat to me this morning.

February 18, 1906.

She has been exceedingly anxious and depressed during the last two days in response to her depressing delusions. She also believes that her friends are seeking her, and that she must go away with them. To this end she fights and struggles with her nurse to the last ounce of her strength. She says she hears her brother's voice in the next room.

Last night she was much distressed because the ship was sinking and she could not escape. Ten minutes later she wanted to borrow fifty cents with which to get away from here. Apprehension and comprehension of what was said to her were perfectly clear throughout this.

Amnesia as previously noted.

February 24, 1906.

Continues very anxious and restless much of the time, but particularly at night. Many self-accusations. Says she has been here seven or eight weeks. That she has been two weeks in the room to which she was removed four or five days ago. Gave the correct date yesterday, and said that she was at Amityville.

February 27, 1906.

She remembers that I yesterday told her that my name was Stevens, but I could not convince her that such is true. Yesterday she told me that she was very sorry for Dr. Stevens, whom she had caused so much trouble. She would not believe that I was Dr. Stevens. She forgets her nurse's name from day to day.

Q. "How long have you been here?"

A. "I do not know. I thought this was the end of March, but the nurse tells me that it is the end of February."

Q. "How many weeks have you been here?"

A. "I don't know. I haven't the least idea, and cannot reckon it. I was at two or three other places before I came here, and I don't know anything about how I got here."

Q. "Well, approximately how long do you think you have been here?"

A. "I should say about five weeks." (2 weeks).

Q. "Do you remember anything about a trip on the train with your brother?"

A. "I simply remember starting from X with my brother, a nurse and Dr. Shyro. I don't remember having seen them since. I don't think my brother did come here with me. I don't remember. I am confused."

Q. Can you tell me the year and month?"

A. "1905, I guess. The nurse said it was the end of February. I supposed it was later."

She does not know the name of the institution, but says it is in Amityville. She has been told the name of the institution many times. She makes many mistakes in relating what she had for the previous meal, and altogether her memory for passing events is very defective.

She is quite depressed, despondent, and self-accusatory, and wishes to kill herself.

She has improved very much physically, sleeps fairly well, and appetite is good. Pulse remains very rapid, much of the time as

high as 130. Patellar reflexes remain greatly exaggerated. Cutaneous sensibility normal.

March 15, 1906.

Recently painful emotional depression has been more prominent, and much of the time she is in an exceedingly agitated state, begging most persistently for poison, and trying to injure herself by every means in her power. This is because she thinks she has been so wicked and has caused her family so much trouble. She thinks she is going to be turned out in the cold without any clothing, or that she is to be deserted by every one and left here alone. Memory and orientation practically unchanged. Pulse is not quite so rapid—about 100. This pulse has throughout been unaccompanied by any elevation of temperature, and is not due to agitation, for it continues so in her periods of quiet. Hallucinations of hearing active. Improved physically; sleeping 5 to 8 hours nightly without hypnotics.

April 8, 1906.

Continues in the same agitated and distressed condition previously noted. Hallucinations of hearing still present, but less active.

Since the last note a very marked improvement has occurred in the field of memory and orientation, so that now no glaring defects of either would be noted on a superficial examination. Some defect does, however yet remain, so that she cannot give an accurate account of what she had for the previous meal, and makes mistakes with reference to the time elapsing between events. She does not know definitely how long she has been here, usually saying six or seven weeks. While she always knows the year, and generally gives the month correctly, yet she is not perfectly certain about this or what time in the month it is, so that it would be very easy to deceive her in this regard.

Is in fairly good physical condition.

April 17, 1906.

To-day the clinical picture is clearly and purely that of melancholia of involution. She is greatly depressed with the most painful delusions of self-accusations. She becomes very agitated in reaction to these at times, and is always very anxious for death, for she knows she can never be forgiven for her sins. Her depression and distress have been more acute during the last few weeks than at any time since her admission.

Her memory for both old and recent events, and orientation are now normal.

Hallucinations of hearing have been absent for two or three weeks. She now has a fairly good disease insight into what her condition has been during the past few months, realizes that she has had hallucinations of hearing, that her memory has been im-

paired, and speaks of the condition of confusion that she was in. Patellar reflexes remain very much exaggerated. Pulse 100.

From this time until about the middle of June her condition showed little change. She then developed a line of nihilistic delusions in connection with her ideas of self-accusation, and has held them with little variation until the present, (Sept. 20).

There is no such place as New York City, and never was. There is not nor ever was any America, or world. She is on one little piece of land, and this is crumbling away into nothingness, with every thing that is upon it. She sees imaginary persons, wagons and horses going about, but these are imaginary only, and soon vanish. All these things, her former belief in the existence of her father, mother, of the world, God, etc., really existed only in her imagination, and now since she has had her eyes opened, she sees it all in its true light. The people about her are spirits, reembodied for the moment, but when they leave her they return to the beautiful spirit world, and her constant cry is that she may be allowed to return to that beautiful land with us. She declares that she has committed sins which she knows have eternally shut her out from that paradise, but she pleads that she may be forgiven, and feels that she is now suffering the most awful torture in being thus excluded. On this account she is greatly agitated and distressed all the time. Is perfectly oriented, and memory for passing events is very good indeed.

She has not menstruated since last December.

I cannot pass it by without briefly directing attention to that very interesting condition present in connection with her nihilistic delusions, viz., a feeling that a change has taken place in herself, the changed way the past seems to her in retrospect, and a feeling that her surroundings are strange, unnatural, and different from what they once were. This symptom, usually spoken of as the "feeling of unreality," has attracted some attention recently, articles on the subject having been published by various writers. Packard[1] has reviewed some of this literature and defines the condition, that is, the sensation, as one arising from "a disorder of apperception, which in turn is due to an association difficulty of some kind." The associational defect—a disharmonious and morbid interaction of the higher associational processes—would seem to be the main cause in this patient. Grossly, I think we must consider it a disturbance of consciousness.

This is not the place to enter into a long discussion of this symptom, however, so I will simply detail that part of an examination made Oct. 13, 1906, which brings out this symptom. More than that would be to diverge from the main object of this paper, which is to show the presence at the same time of two forms of mental disease in the same patient.

[1]Packard. Jour. of Abnormal Psychology, June, 1906.

"Paradise is all around, but we can not see it with human eyes, and I am afraid I will never see again except with human eyes. My mind has been made a blank so that I can only see dirty city streets. I don't see what brought me here. It was all a horrible dream. (She hears music down stairs). Instead of the beautiful music of paradise I have my head all full of the most horrible stuff. I don't understand it at all. I don't know what the spirits do. All I see now is steam and smoke." (She does not mean this literally I am sure.) "I have nothing but visions of rows and blocks of stupid houses. I know there is some land and some water, but I know there is not the world I thought there was. There is something called the sun."

Q. "Why do you not believe in the existence of these things?"

A. "Why? Because I am not a fool. It is because I have had my eyes opened.—I was made to dream there was a city, world, etc., after I lost my place in Paradise, and now I know from what I see outside and what they say that it isn't so."

Q. "Now what is it you have seen and heard that makes you believe or feel as you do?"

A. "Lots of things. How things grow and how they don't grow, but oh Doctor, you know all about it. I am no fool. I only wish now that spirits could die. Miss M., told me I have paid dearly for my folly."

Q. "What did she mean by that?"

A. "For having had evil thoughts before I came here."

Q. "What change have you noticed in the way things grow etc?"

A. "Well, one tree will have cherries one time, and then have pears, and you know that can't be."

Q. "Where did you see such a thing?"

A. "Why, over by the cottage there is a tree which had a few cherries on it, and then pears." (As a matter of fact, there is a pear tree and a cherry tree at the place she mentions).

"I have seen trees move their positions. When I came here those clumps of green trees were close together, and now they are far apart."

Q. "Do you feel that you are physically different?"

A. "No, but I have not thought much about it. I know that I still have a headache once in a while, that my knee still gives me some trouble."

Q. "Do you feel that any change has occurred in you since ten years ago?"

A. "I was not alive ten years ago. Up until about a year ago I was a spirit."

Q. "Did you, or could you suffer pain then?"

A. "No. That is only the imagination of the last ten months. Every one that has been here has simply been a good spirit play-

ing a part. I cannot understand how I got here. By that I mean I cannot understand how I ever lost my place in Paradise."

To sum up briefly, then, what have we? First, a defective constitutional basis, with psychic stigmata of degeneracy. Then at the age of 44, or the involutional period, there developed a mental disorder which has continued already for more than eighteen months. This psychic disorder was characterized first by painful emotional depression, delusions of self-accusation with a strong religious coloring, and later, suicidal tendencies. Seven or eight months after the onset of this condition occurs the episode of headache and pains in the arms of 48 hours' duration, followed by an accentuation of her symtoms. If not immediately, at any rate within the next six weeks, there is superimposed a condition of disorientation for time and place, an extreme amnesia for passing events, great disturbance of the time element in memory, fabrications, dreamy delusions, and hallucinations of hearing, underlying which is the original state of emotional depression, with certain modifications, and self-accusatory delusions. This amnesia, disorientation, etc., continues for some six or seven weeks, and then rather rapidly disappears, her memory becomes excellent, fabrications cease, orientation becomes perfect, and hallucinations disappear, leaving her in the original state of severe emotional depression, painful self-accusatory delusions, despondency and utter hopelessness, and a wish for death, and still later, nihilistic delusions.

Now this is not the clinical picture of melancholia of involution, pure and uncomplicated. Such it evidently was during the first five or six months of its existence, and undoubtedly is now. Such a delirium does not occur in that disease. Undoubtedly we do see cases of melancholia where the patient, because of his intense mental agony, is so dominated by his damning delusions and is so agitated in his reaction thereto, that he may fail to take material note of extraneous happenings transpiring during this agitation, and at which times it is impossible to get him to co-operate with the examiner and tell of those things which he really does know and remember; but such was not the case with this patient. At the times my examinations were conducted she was quiet and composed, and frequently not particularly depressed. Her apprehension and comprehension for the moment were clear, as it had

been at the time of the occurrence of those matters with reference to which her memory was tested. To-day, when her memory is as accurate as is that of an average normal individual, she is even more depressed and distressed than she was at the time this amnesia was noted. Further, to-day in her most agitated states, she does not present any such disturbance.

What I have said in reference to the connection between agitation and memorial co-operation may also be applied to the state of orientation. Disorders of orientation do occur in melancholia, without question, but in such cases of melancholia there would seem to be present a much more marked degree of clouding of consciousness to be associated with such a degree of disorientation as was present in this case.

The special significance that I would be disposed to attribute to the hallucinations occurring in this case is due to the fact that they seem to have set in with the disorientation and amnesia, and to have disappeared with the same. Aside from this coincidence, they would have attracted no special attention, since hallucinosis is a comparatively common symptom of melancholia.

The emotional attitude of this patient, during the stage under consideration, was not uniformly that usually characteristic of melancholia. A part of each day, and particularly at night, she was much distressed and showed an acute emotional reaction to her delusions, while at other times she seemed almost devoid of mental pain and talked readily about her self-accusatory delusions without any appearance of mental suffering therefrom. In fact, at times her attitude and conduct were such as to strongly suggest a state of advanced emotional deterioration. This and the variable nature of her reaction to her delusions, constituted her departure, in an emotional way, from the usual picture of melancholia. She does not show this emotional dullness now, but is always keenly depressed.

I think we may say very positively that she did present definite fabrications, which is another symptom not characteristic of melancholia.

Such are the features that raise the question as to whether or not it is an uncomplicated case of melancholia, and would seem to me to justify a negative answer, and a belief that here we have a case primarily one of melancholia, upon which has been engrafted

a secondary symptom complex, viz., Korsakoff's syndrome or psychosis.

The amnesia and its peculiar features—exceeding defect of memory for events of a few minutes or hours before, in the presence of practically clear apprehension, and the great disturbance of the time element—, the disorientation, the fabrications, all of which were so very marked, go to make up quite clearly the clinical picture of Korsakoff's psychosis. Then, too, the *episodic* nature of the appearance and disappearance of these symptoms would speak for their consideration as a superimposed condition, since we have the patient in a given condition previous to their onset, and in a similar condition after their disappearance.

Furthermore, I believe that we may look upon the very rapid pulse rate as a point in favor of the belief in the presence of Korsakoff's disease, and that it is an expression of the toxemia present with a special reaction upon the cardiac nerve supply, or of a neuritis affecting the pneumogastric. Mills, Lloyd, Sharkey, and others report cases of alcoholic multiple neuritis with marked tachycardia, sudden death from heart failure, etc. Tachycardia was a very prominent symptom in an unquestionable case of Korsakoff's that we had recently under treatment. It began at the initiation of the disease and continued, though diminished, for three months after the subsidence of all active symptoms, both mental and physical. In A. C. it was present on admission, and not until five months afterwards did her pulse rate drop down to its normal average of 80.

Of course the weak point in my claim that Korsakoff's psychosis was co-existent here, is the fact that we have no evidence of the occurrence of a definite neuritic process, unless such was the nature of the pains in the head and arms. When I saw her there were no sequellæ other than the rapid pulse and a certain degree of muscular weakness, that would warrant the assumption that a neuritis had occurred. We are told, however, by those who have made a particular study of Korsakoff's psychosis, that the occurrence of a neuritis, per se, is not necessary, for the toxines may effect the cortex cerebri alone, so that the only symptoms produced are in the psychic field; and a number of such cases have been reported. In others, the neuritic symptoms have been so slight as to escape attention.

Certainly we had here presented the Korsakoff's syndrome.

Society Proceedings

JOINT MEETING

OF THE

NEW YORK NEUROLOGICAL SOCIETY

AND THE

PHILADELPHIA NEUROLOGICAL SOCIETY.

Held in Philadelphia, Nov. 24, 1906.

The President of the Philadelphia Neurological Society, DR. D. J. MCCARTHY, in the Chair.

(*Continued from page* 419.)

DISCUSSION ON APHASIA, ESPECIALLY WITH REFERENCE TO THE VIEWS OF MARIE.

Dr. Charles K. Mills said that neurologists owe some gratitude to Pierre Marie for reviving interest in the study of aphasia and for showing that some of the classical views about cerebral disorders of speech are worthy of re-examination, if not of revision. Marie has already found some supporters for his view, and has awakened the attention or opposition of distinguished neurologists like Dejerine and von Monakow.

The task assigned to Dr. Mills was simply that of opening a discussion which it is hoped will bring out new data and a full expression of opinion from the members of our two societies. After a glance at the views of Marie and the manner in which these have been opposed by Dejerine, he would content himself with a rather dogmatic presentation of personal views.

The most important of Marie's assertions are: that auditory, visual and motor speech centers do not exist; that aphasic phenomena are not due to interference with auditory, visual, or other images, but to intellectual deficit, this deficit causing difficulty in the comprehension of speech and interfering with numerous didactic processes; and that Broca's convolution takes no part in the function of speech and its lesions in the production of aphasia. Considering how universal has been the opposite of this last opinion about Broca's convolution, this is perhaps the most startling of his assertions. Marie admits that Wernicke's aphasia, Broca's aphasia, and anarthria are clinical facts, but explains these facts, especially as regards Broca's aphasia, very differently from his predecessors,—asserting that Broca's aphasia is nothing but Wernicke's aphasia complicated with anarthria, or anarthria complicated with Wernicke's aphasia. Anarthria, as he understands the term, is due to lesion of the lenticula and its environment. According to Marie, the regions, lesions of which produce aphasia, are all included in Wernicke's zone, which he defines as composed of the supramarginal (inferior parietal) and inframarginal gyres and the posterior extremities of the first two temporal convolutions,—in other words that cortical region composed of the gyral masses which curve around the extremities of the Sylvian and the parallel fissures.

In his replies to Marie, Dejerine maintains the classical or at least the usually accepted views regarding aphasia, holding with reference to sensory aphasia that the long accepted theory of centers for sensorial images cannot be successfully attacked, and that the diminution of intelligence sometimes exhibited by aphasics is dependent upon disruption or disturbance of the cerebral mechanism of speech rather than the aphasia upon the intellectual loss or deficit. He stoutly maintains that Broca's convolution plays an important part as a speech centre, explaining the cases in which it is involved and aphasia does not result by the compensatory action of the opposite hemisphere, and cases of Broca's aphasia without lesion of Broca's convolution by the fact that the motor speech zone includes other parts, as the anterior insula and the foot of the second frontal gyre. He adheres to his theory of sub-cortical motor aphasia due to lesion of the fibers entering and leaving Broca's convolution. He holds that the existence of the real or the apparent sensory aphasia in motor aphasics is usually only temporary, and in any case is not to be explained in the way that Marie suggests. Anarthria or dysarthria, according to Dejerine, is due to lesion of the motor projection fibers and is different from cortical or subcortical motor aphasia. He contends also that the cases with numerous or extensive lesions described by Marie are cases of total aphasia, the lesions involving all or a large part of the speech zone; also that Marie fails to recognize cases in which Wernicke's zone and the lenticula are not involved and yet the Broca syndrome is present. Dejerine believes that a purely unilateral lesion of the lenticula does not produce anarthria.

Dr. Mills' views regarding the questions in dispute between Marie and Dejerine are largely those of the latter, differing in some particulars which will appear in the course of the discussion. He summarized these views as follows:

1. The centers concerned with speech may in a general way be designated as sensory and motor.

2. Under sensory should be included lower and higher sensory centers, the lower being places of storage for the auditory and visual memories of words, letters and all else which take part in language on its recipient side. The higher sensory areas have sometimes been termed the concept areas; they are regions in which names become associated with the ideas of objects named, and in which probably other somewhat evolved and complex sensory processes take place.

3. The lower and higher sensory areas for speech and their associated structures are all included in the posterior association area of Flechsig, the concrete concept or concrete memory field of the writer. They do not belong to the cerebral primordial or projection fields, but have been evolved out of these,—a fact important to remember when considering the question of intellectual deficit in aphasics.

4. For the purposes of this discussion all the sensory centers for speech,—auditory and visual,—may be regarded as included in Wernicke's zone as defined by Marie, with the addition of some portion of the third temporal convolution. It needs only to be remembered when speaking of the supramarginal convolution as a part of Wernicke's zone that only the caudal or posterior half of this convolution is to be taken into account.

5. The cortical motor center for speech is situated in the insula and in the hinder part of the left third frontal convolution.

6. In the lower third of the precentral convolution are situated the cortical motor centers, concerned with phonation, articulation, and facial expression,—in short with all movements which take part in spoken language. These motor centers taken together constitute an utterance or executive speech center, and are of necessity connected with Broca's convolution.

7. The motor center for speech is not connected by projection fibers directly with the bulbar nuclei, but indirectly through the motor cortex. Whether the cortical speech centers proper are connected directly with the lenticula or other basal centers not bulbar, is not yet positively determined, but probably the cortical executive center is connected with the lenticula and thalamus.

8. The lenticula has motor functions, and like the precentral cortex is subdivided into centers for movements of different parts of the body, as for the leg, arm, and face, necessarily therefore having within its borders motor centers of some sort concerned with articulate speech. The part played by the lenticula in motor speech, however, is different from that which is performed either by the motor center included in the insula and Broca's convolution, or in the cortical center for movements of the face and tongue.

With regard to the cerebral zone of speech, and also other regions of the brain, we are not much beyond the threshold of our knowledge of the subdivision of function.

In some quarters a tendency to rebel against the extreme differentiation of the brain into areas and subareas and centers is exhibited, but close attention to the facts, particularly those which are being obtained through clinico-pathological observation, would seem to show that this differentiation is even much greater than has been supposed by the most ardent believer in localization.

Sensory aphasia, according to Marie, is the only real aphasia. He would have us believe that all our slowly and patiently acquired views with regard to memorial images, auditory, visual and for all the senses, so far at least as the discussion of aphasia and the cerebral phenomena of speech are concerned, should be cast aside; that in Wernicke's zone, as already defined, are not the long recognized auditory and visual centers and their elaborate connections, but that this zone is an intellectual area or center and that the aphasia of Wernicke is due, not to disturbance of auditory and visual processes, lower and higher, variously combined, but to intellectual disintegration.

The centers, visual and auditory, concerned with speech are, as Dr. Mills has already stated, part of the great concrete memory field, or posterior association area of Flechsig. It must be admitted that this is a region concerned with thinking, with concrete ideas, but these very forms of mental activity have for their bases sensorial images and their correlation with each other and with other cerebral processes. The posterior association area is a psychic zone, higher in grade than the fields of projection and lower than the prefrontal psychic zone concerned with abstract conception and the highest intellectual functions. The intellectual deficit described by Marie and observed by all students of aphasia, is conditioned by destruction and disturbance of the sensorial cerebral centers, and of the structures connecting these with other parts of the brain. When Marie admits, as he does, that Wernicke's aphasia is a clinical type with

the characteristics described by Wernicke and others, he largely admits all that is asserted by those who hold to the more commonly accepted views regarding auditory and visual aphasias. One may, if he so choose, regard the two sets of convolutions which curve around the extremities of the Sylvian and of the parallel fissure, as an audito-visual "intellectual" center—a center composed of auditory and visual subcenters with correlating intrinsic and extrinsic structures.

One might ask Marie in this connection some questions of such simplicity as to scarcely appear to have scientific value. What for example does he regard as the basis of the intellectual activity of his psychic center? Is he going back to the ancient mythical and metaphysical ideas of some spirit dwelling in this or that part of the brain? Is intelligence with him some indefinite essence separated into different parts, each dwelling in some particular region of the cerebrum? If he recognizes higher and lower grades of intelligence or intellectuality, does he regard them as dependent upon some subdivided entity which he calls the intellect, or does he believe that they are in some way correlated to sensation and motion and to physiological processes of perception and apperception which have their anatomical substrata in different regions of the brain? In brief, what is his exact definition of an intellectual center, disturbance or disintegration of which gives rise for instance to the intellectual deficiency which is present in his aphasia?

It seemed to Dr. Mills that Dejerine has the best of the argument as regards the question of intellectual deficit depending upon interference with sensorial processes, and not these processes upon intellectual impairment. It must be said, however, that Dejerine, in common with others from which list Dr. Mills could not exclude himself, may have spoken at times without due consideration of aphasics having no impairment of intelligence, just as at other times too great stress may have been laid upon intellectual impairment. If the cases are closely studied, it is true, as Marie asserts, that such impairment of intelligence will be found in varying degree.

Too much emphasis cannot be placed in this connection upon the individual capacities, natural and acquired. Aphasic patients like those seen at the Bicêtre, the Salpêtrière, and at the Philadelphia General Hospital, are most frequently individuals of no great natural endowment and sometimes of little or no education. Lesions which interfere with the cerebral zone of speech either on its sensory or motor side will cause in such persons an impairment of ability to carry out mental processes to a greater degree than in those of a higher order of intelligence. Dr. Mills had again and again noted striking differences in the ability of aphasics to understand and carry out directions or to act for themselves along lines requiring intelligence, such patients presumably from their symptoms suffering from lesions of the brain of similar extent and location.

In discussing the part played by sensorial images, attention should be more strongly fixed upon the fact that it is not alone destruction or disturbance of sensory centers concerned with interior speech that causes aphasic phenomena and interference with intellectual processes, but that these sensory centers are of various grades of simplicity and complexity according as they administer to lower or higher sensory concepts, and also that the disruption or disturbance of the associations between sensory centers for the different senses, and for lower and higher conceptual pro-

cesses are the agents in causing aphasia as much as the assaults which are made upon isolated sensorial centers. Moreover, the breaking of association between the audito-visual portion of the cerebral zone of speech with all its intricate associations and correlations and the motor portion of the speech zone and the projection system add both to the aphasia regarded simply as a speech disorder, and as due to deranged or diminished intelligence, but that the loss of interference with memorial images is the main cause of the aphasia, is fundamental.

The references of Dejerine to insanity are interesting in the support which they give to his views. He refers for instance to the fact that hallucinations among the insane support the idea of sensorial images. He does not perhaps lay sufficient stress upon the fact that such hallucinations and illusions are more dependent upon the disruption of associations between centers than they are upon the interference or destruction of the centers themselves. The writer gave some attention to this subject in a paper recently presented to the section on psychology of the British Medical Association at the meeting held in Toronto, September, 1906. As shown in that paper the study of the hallucinations of the insane is in a large and proper sense a study of an interesting phase in cerebral localization, a view which has been supported in the past by many alienists and neurologists. Dejerine's reference to general paretics is one which must appeal to every alienist. How often do we observe cases of general paresis with more or less advanced dementia still showing no true aphasia, either sensory or motor, notwithstanding the fact that because of the nature and site of some of the lesions, anarthric or dysarthric affections of speech may be present?

The symptomatology of disease of Wernicke's zone will differ according to the size and extension of the lesion. If the lesion be, as it usually is, of considerable extent, the main symptoms will be word deafness (sometimes nearly complete, although more often partial), word dumbness more or less pronounced, dyslexia, paralexia or alexia, slowness or difficulty in word speaking (paraphasia), repetition of the same words and phrases, and at times abnormally rapid paraphasic speech or logorrhea. The sensory aphasic, whatever may be the limitations or the extent of the lesion producing his symptoms, if such lesion is confined to Wernicke's zone, is not deprived of the power of speech, like the motor aphasic suffering from a lesion of the cortex or subcortex, or cortex and subcortex of the insula and Broca's convolution.

Dr. Mills did not give much space to the consideration of the clinico-pathological evidence favoring the existence of centers for word seeing, word hearing and other forms of memorial imagery, and also of tracts in the audito-visual zone connecting these centers with each other and with other parts of the brain. Such evidence is to be found in the well-known works and articles on aphasia, and to a certain extent it has been marshalled by Dejerine in his part of the interesting discussion now under way. Dr. Mills had from time to time recorded cases with such symptoms as word blindness, word deafness, word dumbness, object blindness, alexia, paralexia, paraphasia and the like; and believed that most of these will at least stand the test involved in referring them to lesions of a true audito-visual zone.

It needs to be said that word deafness and word blindness are rarely monosymptomatic, although Marie admits that the latter may be: and

that cerebral lesions are only in very rare instances either purely cortical or purely subcortical.

Probably one of Dr. Mills' own cases, now well known in the literature of the subject, approached as nearly as any recorded case to an illustration of word deafness due to an isolated and limited lesion.

This case was first recorded by him in 1891. The two cerebral hemispheres are still in his possession and have been brought here this evening. The patient, fifteen years before her death, had an apoplectic attack, previous to this time not having had any known affection of hearing, sight, or speech. As the result of this attack she became word deaf, and had a marked form of paraphasia, although she continued to read, and to some extent to write. She was described, however, as having mixed up her words in writing. She could hear and appreciate musical and ordinary sounds, like a bell, a knock, or the tick of a clock. Six years before her death, she had a second apoplectic attack and soon after became totally deaf, or nearly so, as to sounds as well as to words. She was also, as a result of this second seizure, partially hemiplegic on the left side. At the necropsy in this case, even before the membranes were removed, it was noticed that the first temporal convolution was smaller and thinner than usual, and that at the posterior extremity of this convolution about opposite the upward turn of the posterior extremity of the Sylvian fissure, was a depression which included also a part of the adjoining second temporal convolution, the depression being about seven-eighths of an inch in diameter. This depression, as was proved subsequently, was the result of an old embolic or hemorrhagic cyst. On the right side was an old and very extensive hemorrhagic cyst which had completely destroyed the first, and almost completely the second, temporal gyre, the island of Reil, the retroinsular gyres, the lower extremities of the central gyres, and to a large extent, but exactly how much was not determined, the ganglia and capsules. The first temporal, retroinsular, and subfrontal (Broca's convolution) gyres were greatly wasted.

We can have a motor aphasia which is distinct in its symptomatology from a sensory or sensorimotor aphasia or from a dysarthria or anarthria, the dysarthria and anarthria being used in the commonly accepted sense of impossibility or difficulty in articulating, enunciating and vocalizing words or expressions. Motor aphasia due to lesion of the cortex and its immediate subcortex may be complete or partial, according to the degree of destruction of the cortex and subcortex. In complete motor aphasia the patient is unable to speak at all, although as is well known, in cases almost complete he sometimes retains a single or a few recurring utterances. If the case is one of absolutely pure motor aphasia, the patient may have no paresis of the organs concerned with speech. He is unable to recall words which describe objects or which are used in the formation of phrases or sentences. Apparently he cannot arrange language for utterance. One of his difficulties might be expressed as Broadbent so long ago expressed it, as "inability to propositionize"; and indeed this fault or loss has been expressed in various ways, none of them entirely satisfactory. The patient is word dumb, but more than this. It is said that the psychomotor memories concerned with language are lost, but this does not make the matter much clearer. Again, it is sometimes said that the motor aphasic cannot build words, phrases, or sentences. Not a few cases of motor aphasia as seen in practice are partial at first and become

complete as in a case of advancing tumor. Sometimes the motor aphasia is complete or nearly so for a time, but disappears and reappears, these varying conditions of motor speech disturbance being due to variations in the condition of the lesion and in the physiological activities of the parts associated with the centers affected by it.

The motor aphasic may exhibit some dysarthria or anarthria, this being especially noticeable with regard to certain sounds as those made by dentals and labials. It may be a question whether the dysarthria or anarthria of a motor aphasic is due to the influence of the lesion on the true motor speech center, or on adjoining or connected parts, as the centers at the foot of the precentral convolution or perhaps those in the lenticula.

Motor aphasics may have trouble both in reading and in comprehending spoken words, but these defects are not the same in degree and probably not the same in kind as in the case of sensory aphasics. Troubles of this kind present early in a case of motor aphasia due to a severe lesion may disappear entirely and not be a part of the residual syndrome. They are doubtless due either to the effects produced by the edema and other temporary states or to the diaschysis of von Monakow—that is, to interference with the physiological actions of the parts with which the center attacked is anatomically and physiologically connected. Von Monakow, in expounding diaschysis diagrammatically, indicates a lesion placed in some portion of the cortex. Coming to and going from this place of lesion are association fibers to other portions of the cortex, and also commissural connection through the callosum to the other hemisphere, and presumably, from the other hemisphere through the callosum; also fibers going from the cortex to the bulbar nuclei, etc. A certain physiological interchange of function takes place between the different but correlated areas, so that destruction of one area necessarily causes a change in the physiological state of other centers or areas with which it is correlated.

Von Monakow calls attention to the importance of giving more heed to negative cases; also to the fact that much still remains to be learned with regard to many phases of the subject of aphasia, Marie for this reason deserving the thanks of neurologists for the interest which he has excited in the subject.*

The difficulty in understanding spoken language sometimes exhibited by motor aphasics is so little marked as to need considerable study for its detection. The amount of this difficulty is conditioned to a certain extent by the original intelligence, education and training of the aphasic.

In a correct sense the cerebral zone of speech is a single great mechanism, but it has many parts. If the machinery in one part is interfered with, the effects of such interference may extend to all parts.

One case recorded by Dr. Mills has helped to convince him of the part played by Broca's convolution and the insula in the function of speech. This patient was long an inmate of the insane department of the Philadelphia General Hospital. Dr. Mills saw him first about nine years after an apoplectic attack which left him aphasic. His language was very restricted. He could speak only a very few words or phrases, or one or two very short sentences. Word dumbness was a marked feature of the case, although he learned by training to name objects like a watch, a pencil or

*This reference to von Monakow was introduced since the discussion at the meeting of the two societies, his paper having appeared about that time.

a knife, with some facility. The expressions he made use of were such as "very pretty," "thank you," etc. He had a curious recurring utterance which he made use of when he attempted to read aloud. It was, "England, oh, my soul, England, oh, my soul!" He would read, apparently understanding what he was reading, and then make use of this expression in a loud and rather oratorical tone. He was apparently able to read, although with difficulty. He was tested as regards this matter, for instance, by asking him to select words from different parts of the page which he was reading, and which he would do correctly, although taking some time. What he said was said with distinctness and clearness. No paralysis of the muscles of articulation, enunciation or phonation was present. He had no anarthria or dysarthria in the usual sense in which these words are employed, but was a true motor aphasic. He could understand what was said to him. The patient was able to recognize objects by touch, hearing, taste and smell, but as a rule was not able to name the object. The case was clearly one of marked, although not absolutely complete, motor aphasia. The patient could write many single words or short combinations of words correctly, holding his pen or pencil with ease and firmness.

In some of the cases in which motor aphasia has been present, the convolution of Broca apparently remaining intact, lesions have been present in both the external capsule and the anterior part of the internal capsule, parts which are closely related to the insula and the convolution of Broca.

In the cases cited by Marie in which so-called motor aphasia was present, and in which the lenticular nucleus was implicated in a destructive lesion, parts neighboring on this ganglion were also nearly always involved. These parts included the insula and the external capsule. The insula and its subjacent cortex, by many as by the writer, have been included with Broca's convolution and its subcortex in the so-called motor center for speech; and the probabilities are that in the large lesions to which Marie refers the subcortex of at least part of the motor speech center is involved.

The fact that atrophy or involution of Broca's convolution sometimes takes place after destructive lesion involving Wernicke's zone on one or both sides is an argument in favor of the view that Broca's convolution is an integral part of the zone of speech. In the personal case of sensory aphasia to which Dr. Mills had referred, that of a patient who had suffered from word deafness and paraphasia because of a lesion limited to a small area at the junction of the hinder portions of the first and second temporal convolutions, and who afterwards became totally deaf from a destructive lesion attacking the temporal and central convolutions and other parts of the opposite side, the post-mortem examination showed that Broca's convolution among other parts was greatly atrophied. The attack, causing the word deafness, it will be remembered, occurred fifteen years before death, and that which brought about complete cerebral deafness, nine years before death.

While sensorial and motor aphasia are separate and independent clinical syndromes, many of the cases of aphasia observed in practice are, because of the nature and especially because of the extent of the lesions causing them, sensorimotor, or as they are sometimes called, mixed or total aphasias. The lesions in these cases often involve, as is well known, such

parts as the superior temporal convolution, the insula, Broca's convolution and its subcortex, the central and the supramarginal convolutions, the lenticula and the external and internal capsules. They are instances of widespread destruction of all parts irrigated by the middle cerebral artery. In other instances the cerebral regions supplied by this artery are involved in varying degree, giving a more or less confusing smyptomatology made up of a combination of sensory and motor speech phenomena with other symptoms.

Marie's views would seem to be largely built up upon the record of cases of these so-called total aphasias.

What is necessary in this discussion is in the first place to marshal the symptomatology of cases in which the lesions have been confined to the audito-visual (Wernicke's) zone; secondly, that of cases with lesions limited to Broca's convolution or the insula and its immediate subcortex; and thirdly, that of cases in which the lesions were confined to the lenticula. Cases of each of these descriptions are on record and give a particular symptomatology.

Much stress is laid by Dejerine upon the distinction between cortical and subcortical motor aphasia. Such a distinction can no doubt at times be made; at least isolated cases of subcortical motor aphasia have been recorded, their symptomatology differing somewhat from that of the cortical forms of Broca's aphasia. For reasons, however, which must be very clear to every practical neuropathologist the lesions causing the aphasia, in fact massive lesions of all sorts, are very rarely purely cortical. The vessel which closes or which is broken has its branches both in gray and white matter, and hence the softened area or the hemorrhagic cyst usually involves both. It is probable, as Dejerine says, that a subcortical motor aphasia, if of pure type, will leave certain powers or capacities of the patient,—as that of writing,—intact, or but little affected, but such a case will be of rare occurrence. The usual symptom picture, whether the lesion be in Broca's convolution or the insula or in both, or in whatever part of the cerebral zone of speech, is one that is due to a lesion which is jointly cortical and subcortical. Dr. Mills excluded here those cases of lesion of the internal capsule and corona radiata, sometimes observed, in which no portion of the gray matter, either cortical or central, is diseased; but even in capsular lesions and in lesions of the corona outside of the usually accepted cerebral zone of speech the gray matter is often involved.

Motor aphasia, according to Marie, is simply Wernicke's aphasia plus anarthria. Dr. Mills did not see how this definition is to be accepted unless we give a new meaning to anarthria, which indeed Marie seems to have done. The generally accepted definition of anarthria is that it is a defect or difficulty in speech, especially connected with articulation. Even if we expand this definition as Marie has suggested, so as to include defects of the expiratory, phonatory and articulatory mechanism, it is still clear that such an anarthria does not constitute motor aphasia in the accepted sense. Many of the motor aphasics whom Dr. Mills had studied have presented no anarthria or dysarthria. Some partial motor aphasics are dysarthrics; some monoplegics or hemiplegics have anarthria or dysarthria which is regarded by careless observers as motor aphasia, but anarthria and motor aphasia are quite distinct, although the two may be combined in the same case. Add to the well recognized phenomena of sensory

aphasia such as partial word deafness, paraphasia, etc., an anarthric defect of speech, and the resultant will not be a true motor aphasia. Defective vocalization and articulation will be added to the sensorial phenomena, and yet the patient may be able to speak, although in a stumbling manner.

To what is anarthria or dysarthria due when present from a cerebral lesion,—from a lesion above the bulb? In some cases it is undoubtedly due, as is now well known, to lesions usually bilateral of the internal capsule; in others to lesions of the subcortex of the facial and orolingual region, and in others to lesions of the orolingual cortex itself.

Dejerine believes that anarthria producing the pseudo-bulbar syndrome never occurs from a unilateral lesion, either cortical or capsular, the cases observed by him having always been instances of bilateral lesions. He believes that the centers which come into play in the mechanism of speech have in reality a bilateral cortical representation.

The question of the correctness of Marie's views with regard to the part played by lesions of the lenticula in the production of motor aphasia necessarily involves the discussion of the entire question of the functions of this body. Dr. Mills had the opportunity of making personal observations on several cases with necropsies in which destructive disease of the lenticula was present. The lesions, however, were rarely strictly confined to the lenticula. In the laboratory of neuropathology of the University of Pennsylvania, Dr. William G. Spiller has the specimens from some of these cases, with others collected by himself. One or two of these specimens have been brought here for the inspection of those taking part in this discussion. Later it is the intention of Dr. Spiller and the writer to take up the question of the functions of the lenticula largely from the point of view of personal clinicopathological studies.

Probably the work of Mingazzini on the symptomatology of lesions of the lenticular nucleus is the most valuable contribution to our knowledge of this subject. As the result of carefully made personal observations and of a study of cases recorded by others he concludes that a focal lesion, even if of small size, which involves only the lenticular nucleus never fails to manifest itself with motor disturbances, these motor symptoms showing themselves as dissociated or generalized paralysis or paresis to which are sometimes added irritative symptoms.

Mingazzini discusses the speech disorders which he, in common with Nothnagel and others who preceded him, believe result from destructive lesions of the lenticula. These are particularly described as dysarthrias, and Mingazzini holds that they would appear to result only from lesions of the left lenticula. He gives cases with necropsies demonstrating the fact that lesions of the right lenticula do not, while those of the left if peculiarly situated do, cause dysarthric affections.

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Dr. James Hendrie Lloyd said that Marie's paper on aphasia is more important for what it indicates than for what it really says. As a pronouncement on the subject of aphasia it is noteworthy chiefly for attempting to tear down all the work that has been done by others, and to erect instead an edifice which is largely theoretical and purely Marie's. Viewed in this light it does not seem to be deserving of all the attention it is receiving; and Dr. Lloyd failed to see that it marks an epoch.

But from the standpoint of what it indicates—as showing, for instance, a tendency—Marie's paper is of some significance. It is a protest, or a reaction against the extreme academic school which has long been paramount. This academic, or diagrammatic school has elaborated this subject of aphasia beyond all reason. A recent author gives twenty-eight varieties of aphasia. There is hardly a monograph in which this excessive subdivision is not more or less in evidence. We see the nicest analyses made, and the whole subject partitioned out, each variety being assigned to a limited area of the brain cortex. Dr. Lloyd did not hesitate to say that these fine-spun schemes are not so much in accord with bedside observation as they are mere evidences of a burning of the midnight oil. We even hear it said that there are special and distinct centers for the grammatical parts of speech, as nouns and names, but for his part Dr. Lloyd had never been able to subscribe to those extreme views, and he believed he was within bounds when he said that it is impossible at the bedside to verify all this theoretical psychology.

His own doubts on this subject have been growing greater and graver for some years. Bedside observations, very faithfully made and in large hospital clinics with ample material, have not enabled him to make many of the fine distinctions which are so popular. In most cases that he saw the phenomena are mixed. Thus with motor aphasia there is some auditory aphasia and word blindness, especially in bad cases, and in the early stages even of mild cases. There is also, as Marie points out, some failure of intelligence, or mental confusion, so that it is most difficult to say just how much is aphasia properly so-called, and how much is due to mental defect. He had been in the habit for some years at Blockley of calling these cases *sensori-motor aphasia,* as indicating that both the sensory and motor elements of speech are involved. He knew that the criticism is brought against these views that those who hold them are lacking in the power of precise observation. But that criticism is the worst kind of a begging of the question. Precise observation is not a monopoly, even of those who can discern twenty-eight varieties of aphasia.

In a recent case at Blockley there was complete motor aphasia, with some degree of word deafness and word blindness; also very evident mental dullness; but the brain cortex on gross inspection, after hardening in formaline, was not involved, and the lesion was confined to the internal capsule and lenticular region. Dr. Lloyd has now two cases of right-sided hemiplegia under his care in the Methodist Episcopal Hospital in neither of which is there the slightest evidence of aphasia of any kind. He did not agree with Marie that the cortex must necessarily be involved in order that there should be aphasia proper, for he had certainly seen genuine aphasia in cases of purely lenticular lesions. He said this, however, with due appreciation of the fact that we have no right on mere naked eye inspection to say that the cortex is not involved when there are large underlying capsular lesions. Even a microscopic examination might not suffice to determine whether the brain cells in life had been able to

functionate in cases in which the circulation had been so seriously obstructed as by large underlying hemorrhages or softenings.

This led him to say a word about the difficulty of interpreting pathological changes. We have in many of these cases great ripping lesions, tearing up large areas of the brain substance. Even in one or two of Marie's pictures it looks as though large parts of the side of the brain had been torn out. In such cases precise conclusions are often unwarranted. The damage even in small lesions is often much more extensive than it seems to the naked eye. He had seen a minute but fatal hemorrhage in the spinal cord which seemed to be confined to the anterior parts of the posterior columns, but under the microscope the damage was seen to be much more extensive, acting in fact like a total transverse lesion. So in the brain, a seemingly small lesion may be responsible for much greater and more widespread damage than it is credited with. Observation in some of these cases can hardly be exact; and as some of them are reported and described no proper attempt is made to delimit the lesion. We are totally deprived of all opportunity to experiment in order to prove anything in aphasia, for of course the anthropoid apes are of no value for such a purpose, and we cannot experiment on the human subject under ether. Dr. Lloyd often feels like protesting against the dogmatic statements that are based on improperly observed pathological changes. Will the time ever come when some enthusiast will have the temerity to excise carefully the so-called auditory speech center or the angular gyrus in a normal human brain? He should not like to be either the excisor or the excisee.

Marie asserts that in every aphasic there is trouble to comprehend spoken language, but that the tests ordinarily used are not adapted to bring this defect out. Dr. Lloyd thinks this is a criticism that should be taken to heart and put to the test by some of the advocates of pure motor aphasia. It will doubtless be found in many cases if the demands made of the patients are a little complicated (not merely an order to "put out your tongue," or "hold up your hand") that Marie is right in this respect. He knew that he had often found it to be so.

Another most important point is Marie's statement that in every case there is some loss of intelligence. It is surprising how commonly it is assumed that intelligence is intact in aphasia; that there is nothing the matter with the mind but a speech defect; although it should be evident that because of this very speech defect the difficulty of determining the integrity of the mental processes is almost unsurmountable. Dr. Lloyd would not tarry here to attempt to discuss the vast question of the dependence of thought upon the faculty of language; it is enough to point out that this dependence is very close, and that those persons who calmly assume that in aphasia there is no mental defect, take much more for granted than either the facts or the science warrants. Upon this important subject Marie's criticism ought to do some good, and should invite to a less superficial view of these cases. If the attempt is made to induce an aphasic to use his mind for abstract thinking, it is usually soon evident that he is out of his depth. He can "put out his tongue," and "hold up his left hand," but he cannot sustain any complex train of thought, and the mere attempt soon worries and exhausts him. This raises the important medico-legal question of the will-making power of aphasics, as in the celebrated Parrish case.

The emotional life is usually well preserved in aphasics, as Marie says,

and this gives a superficial appearance of preserved intelligence. But the emotions are easily excited, even in the weak-minded, and are a very poor gauge of the extent of the intelligence.

Marie asserts that Broca's motor aphasia and Wernicke's sensory aphasia are very analogous, with the capital exception that in Broca's aphasia the patient cannot talk. This inability to talk is merely an anarthria, which is a sort of pseudo-bulbar palsy due to a lesion of the internal capsule. His scheme is a very simple one. The seat of all true aphasia is in a comparatively limited area of the brain cortex, and includes the supramarginal convolution, the angular gyrus, and the posterior ends of the two upper temporal gyres. When the capsule is involved, there is added an anarthria. Broca's classic convolution is thrown out entirely, as having nothing to do with aphasia. As has been already said, Dr. Lloyd thinks this cheme of Marie's is entirely too theoretical and dogmatic, and is open to the same criticism that can be brought against that of other schemers. But in some details, to which Dr. Lloyd had already referred, he believed Marie's criticism is based on just grounds, especially his claim that the superfine analysis of aphasia into many varieties is largely academic. Yet he would not go quite so far as Marie. In a large and general way he believed aphasia can be subdivided into a few types. In one type the motor defects predominate, and this can be called the motor type. In another, the sensory element is more conspicuous, and this can be called the sensory type. But he believed it is a mistake to go much beyond this simple plan in our present knowledge of aphasia. As for the localization of these defects in the brain cortex, he believed that the zone of speech is much more extended than the area which Marie has mapped out.

Dr. F. X. Dercum briefly stated Marie's position with regard to aphasia; namely, that aphasia is an intellectual deficit; that it is a unit; that it is not made up of sensory aphasia on the one hand and motor aphasia on the other, but that by lesion of the zone of Wernicke there is established an intellectual deficit for the comprehension of spoken language; that in so-called sensory aphasia the lesion involves the zone of Wernicke, that in so-called motor aphasia there is, in addition, an involvement of the region of the lenticular zone. Lesion of the lenticular zone gives rise to anarthria. Therefore in so-called motor aphasia, we have merely ordinary Wernicke aphasia plus anarthria. Dr. Dercum then detailed the results of his studies of fourteen cases of aphasia according to the method of Marie, for the determination of the presence of intellectual deficit. Some aphasics cannot comprehend a single word. More frequently they comprehend things that are relatively simple. As a rule they can execute simple instructions, but not complicated ones. Some aphasics have great trouble in executing a single act; others are embarrassed by a direction to perform two consecutive acts, and others fail when three or four are attempted. In all of his fourteen aphasics, Dr. Dercum found an unquestionable deficit. Four were unable to carry out any instructions, no matter how simple; two invariably failed when an instruction containing two factors was given; one was able to perform one instruction fairly well, but usually failed when the instruction contained two factors. Four were able to execute instructions containing two factors, but always failed when they contained three. Three could execute instructions containing three factors, but usually failed when this number was exceeded.

Similar facts were elicited when the attempt was made to have the

patient carry out *written* instructions. Seven of the fourteen could read; that is, they could read single words, written or printed. Thus one patient could read the word "hand" and would correctly indicate the object upon his own person by raising and exhibiting his own hand. He could read the word "head" and indicated this portion of the body. A short sentence embodying these two objects; namely, "hand" and "head," was now placed in writing before him, thus, "Put your hand on your head." He failed absolutely to comply. He evidently could not comprehend the sentence and was as helpless in the presence of a written instruction as he had been in the presence of the verbal instruction. Other interesting illustrations were given.

Dr. Dercum contended that the intellectual deficit maintained by Marie must be unhesitatingly admitted. He pointed out that this deficit is special in character. It differs, of course, from the deficit seen in arrested development on the one hand and in dementia on the other. The deficit is lacunar, involving one function or a closely related group of functions.

Dr. Dercum was unable to classify his aphasics into motor and sensory. He failed also when he attempted to arrange them into groups according to the presence or absence of anarthria. The motor speech difficulty appeared in each case as something added to the essential symptoms of the aphasia; an anarthria added to the failure to comprehend words or sentences.

Dr. Dercum's clinical studies were confirmatory of the results obtained by Marie. Further, Marie's contention that motor phenomena may be produced by involvement of the longitudinal bundle and by the isthmus which connects the zone of Wernicke with the region of the basal ganglia, bears a strong probability of truth, but it is only the confirmation of pathological observations which will definitely determine the question.

Regarding the third left frontal convolution, the occurrence of isolated lesions of this region in right-handed persons without producing aphasia must be extremely significant. Certainly such instances entitle us to the legitimate doubt as to the real function of the third left frontal.

Dr. E. D. Fisher said that what we understood by an intellectual defect must be in regard to speech or in regard to writing. He had a patient who illustrates this. He attends to his business regularly. He is a hemiplegic and not an aphasic. He could not calculate in figures and yet his mind as a mind was as active as it ever was in business relations. He thinks there are many aphasics who are not intellectually aphasic in any way.

Dr. Starr said he had read these papers by Marie very carfully and given them a great deal of thought. As a lecturer on aphasia it was very interesting to draw diagrams, as Dr. Dercum said, and show what we thought ought to be the beautiful varieties of this disease, but, unfortunately, if you had to show the patients whom you thought illustrated the lecture it was sometimes hard to bring harmony between theory and fact. It had often been very difficult to find a case which corresponded exactly to the distinctions which were laid down in regard to motor and sensory aphasia. He believed Marie was right in calling attention to the fact that the majority of aphasics are doubly aphasic, that there was a sensory and a motor element combined in the majority of aphasics. He thought, on the other hand, that Marie had taken a decided step backward in trying to enforce this idea as applicable to every aphasic. He thought everyone who had studied

a large number of aphasics could put his finger here and there on a case which was decidedly and distinctly motor, or entirely sensory, and if cases of this kind existed, it overthrew Marie's contention absolutely and brought us back to Dejerine's point. It seemed they were both right in part; that as Marie said aphasics were often both motor and sensory, but that Dejerine was also right in that there are cases of very limited lesion with purely motor or purely sensory symptoms. He had seen examples of both kinds. Exner was the first to figure it out in his wonderful diagrams 'way back in 1881, limited lesions causing pure types of aphasia, in Wernicke's region sensory aphasia and in Broca's motor aphasia. We have been acting upon this localization in brain surgery in the removal of tumors and abscesses from the different speech areas of the brain with success, and he therefore was not prepared to give up the idea of separate aphasic areas.

Dr. Joseph Fraenkel said that there are undoubtedly cases of pure sensory and pure motor aphasia occurring. It is true, of course, that such pure types are rare. Some years ago Dr. Onuf and he analyzed the clinical and autopsy findings of all cases of motor aphasia thoroughly reported in the literature. Out of a total of about 110 cases they found only about nine cases in which the speech disturbance was the result of a strictly localized lesion in Broca's center. Out of this number, only three showed the aphasic disturbances to be permanent. In the other cases there was a more or less marked recovery of the speech faculty in spite of the permanence of the lesion. Most of the other cases showed widely distributd lesions, so that it is impossible to conceive that these lesions should give rise to a clearly defined clinical picture.

Periscope

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(Band 28, Heft 2-4.)

5. Osteoacusia and Its Relations to the Sensation of Vibration. NEUTRA.
6. Contribution to the Study of Spondylose Rhizomelique. MINGAZZINI.
7. Acute Multiple Gangrene of the Skin, with Investigations Upon the Necroses Produced by Pure Hydrochloric Acid. ZIELER.
8. Contribution to the Knowledge of Congenital Muscular Defects. STECHE.
9. A Case of Defect in the Muscles of the Shoulder-Girdle and Their Compensation. CAPELLE.
10. Communications Concerning Hysteria. 1. Hysterical Mutismus in Combination with Hysterical Asthma After an Accident. STINTZING.
11. Hysterical Somnolence with Choreatic Movements. GROBER.
12. Complete Cutaneous and Sensorial Anesthesia in a Case of Traumatic Hysteria. SEIFERT.
13. Contribution to the Origin of Brain Pressure by Brain Tumors, and Other Diseases of the Brain, and a Peculiar Form of Swelling of the Brain that May Be Observed in These Conditions. REICHARDT.

5. *Osteoacusia.*—Neutra, after a very exhaustive discussion of bone conduction, reaches a series of conclusions of which the following is an abstract. Hearing of a tuning-fork placed upon any peripheral portion of the body depends upon bone or cartilage conduction. This behaves in all respects as the conduction of vibrations through the cranial bones. If the external ear is closed during this conduction, the sound's intensity, and usually also its duration, are increased. This may possibly be due to psychical as well as physical causes. Osteoacusia is most distinct in those cases in which the bones are subcutaneous. It is affected by various diseases of the skeleton, and is not affected by organic sensory disturbances. If the tuning-fork is placed upon the spinal column it is found that kyphosis increases the duration of the sound, lordosis diminishes it. Osteoacusis is entirely independent of the sensation of vibration. Slight changes of the latter cannot be determined. The bones play only the part of reflectors, the sensation being perceived in the pressure nerves of the soft parts. In addition to the bones, firm connective tissue, and even contracted muscles may act as reflectors. Therefore, the sense of vibration must be regarded as a modified pressure sense. For these tests tuning-forks having from 100 to 200 oscillations per second are the best. The absence of either osteoacusia or the sensation of vibration upon a place where they are normally present is, with certain precautions, a definite sign of either hysteria or simulation.

6. *Spondylose Rhizomelique.*—A man of twenty-nine, began, at the age of 19, to have pain in the lumbosacral region, and in the legs. Later, during his military service, he had pain in the left thigh which terminated in ankylosis at the hip. From this, however, he made a partial recovery. Subsequently when examined by Mingazzini the patient was found to have marked limitation of movements in the knee and hip joint, which disappeared

under spinal anesthesia, and were evidently due to pain. There was also pain in the muscles of the neck. The spinal column gradually became more and more deformed until the upper portion was at right angles to the lower portion. Radiographs of the hip showed alterations in the bones. Mingazzini regards the deformity of the spinal column as due directly to the alteration in the anterior vertebral articulations, and not to the effect of gravity.

7. *Skin Necroses and Hydrochloric Acid.*—A girl of twenty-two had for about half a year suffered from a curious eruption of the skin. First there was a hyperemic area upon which welts appeared with white, shining centres, and a circular erythema. In the course of a week either a superficial scale separated, leaving normal skin beneath, or there was a very superficial area of gangrene. There were no general symptoms. Later some small blisters were occasionally seen around the eruption, and still later larger blisters appeared. The mucous membranes were not involved, and keloids never formed. There were no disturbances of sensation. The histological examination of one of the lesions showed hyperemia and edema of the cutaneous and papillary bodies, with slight round-cell invasion around the capillary bodies. As the lesions resembled very closely those produced by dropping raw HCl upon the skin, such artificial lesions were examined microscopically, and indicated a distinct necrosis proceeding from the surface, entirely different from that of the hysterical lesions.

8. *Muscular Defects.*—The first patient, a boy of twenty, had complete defect of the left serratus. There was high position and hypoplasia of the left shoulder blade. The second case, a boy of eight years, lacked on the left side the pectoral muscles, and the latissimus dorsi, and there was imperfect development and weakness of the serratus. Anomalies of the skeleton consisted of shortening of the clavicle and rudimentary development of the left hand. A fold of skin extended between the thorax and the upper arm. There were also some developmental disturbances of the skin of the left breast, the mammary gland on that side being entirely absent, and the skin abnormally thin. The third case, a man of twenty-six, had on the left side defect of a portion of the pectoralis major, and of the whole of the pectoralis minor. There were some defects in the ribs, deformity of the left hand, and a fold of skin between the left arm and the chest. The skin on the left side was thin, and the mammary gland was absent. The fourth case, a man of twenty-four had almost complete loss of the left trapezius muscle, without any other conditions. The fifth case, a man of twenty-two, had absence of the deltoid, but the disturbance in function in this case was exceedingly slight. The sixth case, a man of fifty-two, had congenital absence of several muscles in the ball of the thumb on both sides. There were practically no functional disturbances, and no congenital anomalies otherwise. Steche calls attention to a combination of a group of conditions associated with defect of the pectoral muscles, which includes, in addition to disturbances of the skeleton, the skin and mammary gland. The loss is always unilateral, and is never hereditary. He collects cases recorded in the literature, and tabulates them, and explains the inability of determining any cause, and in conclusion reports a case in which the mother of a patient suffering from muscular dystrophy, presented this uniateral defect in the pectoral muscles, which the autopsy indicated, was congenital.

9. *Shoulder Girdle Defects.*—Capelle describes a case with congenital

absence of the latissimus dorsi on the left side. He discusses very fully the functional disability that results from this, and also the method by which it is in part compensated. In addition to the latissimus, examination also showed absence of the pectoralis minor, and of the lower portion of the serratus.

10. *Hysterical Mutismus.*—A man of twenty-nine suddenly experienced severe pain while at work. He subsequently became weak, had some vomiting and severe asthma, as a result of which he was unable to work. Later he became unable to speak, even to whisper, although his understanding of spoken speech was perfect. The movements of the vocal cords were not impaired. The reflexes were all increased, but not pathological. A diagnosis of hysteria was made, and the patient recovered rapidly under psychic treatment. The condition corresponds exactly to that described by Charcot.

11. *Hysterical Somnambulism and Chorea.*—A child of three and a half years of age had several attacks, probably hysterical in nature, and then became lethargic. The diagnosis was doubtful; meningitis, possibly tuberculous, was suspected, but never conclusively proven to be present. After three months of the lethargic state he recovered consciousness, but was dumb, and had a functional stiffness of the right wrist. Subsequently there were several brief attacks, typically hysterical in nature, followed by complete recovery.

12. *Traumatic Hysteria.*—A man of thirty-eight fell violently upon the ground, striking his head and left arm. Subsequently he developed a condition diagnosed as traumatic hysteria, in which he emaciated, became pale, was melancholic, had profuse sweating during investigation, and had numerous tender points. There were also some cutaneous anesthesia, but no disturbance of the special senses, or of motion. The reflexes were normal. Later he had an attack of confusion lasting two days, followed by total blindness in the left eye, unilateral loss of cutaneous sensation on the left side, and loss of hearing, taste and smell on the same side. There was concentric contraction in the visual field in the left eye, and the general condition grew worse. Still later, after a slight infection, he developed hysterical mutism. Another confusional attack was followed by partial anesthesia in the right hand. If, during this period, the right eye and ear were closed he went into a somnolent condition. Later he lost the hearing in both ears, was unable to speak for a prolonged period, and finally developed hysterical paresis of the left arm. During the course of the disease the patient repeatedly expectorated blood. Lasègue's test, substituting for visual control the sensation of the skin in order to obtain movement in the anesthetic hand, was not successful, but if the anesthetic hand was laid upon a sensitive portion of the skin, and either touched with the sensitive right hand, or first observed with the right eye, the fingers could be freely moved. Some other very interesting experiments are also described. These cases of extensive distribution of the hysterical symptoms are rare.

13. *Brain Tumor and Pressure.*—Reichardt calls attention to the importance of the position of the tumor for the production of intracranial pressure. This also occurs in simple hypertrophy of the brain. He reports a number of cases of brain tumor, and studies in each the cause of the pressure. In the first case a huge tumor, causing increase in the pressure purely by its bulk, was found. In the second case an extra-

cerebral tumor, weighing 162 gm. produced very little pressure because there was an atrophic area in the brain in which it was accommodated. In the third case a huge glioma of the left hemisphere had produced severe internal hydrocephalus. The brain, however, was distinctly atrophic, and this served to explain the very mild symptoms of intracranial pressure that were present during life. In the fourth case a small tumor containing about 100 cc. of clear viscid fluid and pressing upon the right frontal lobe, caused symptoms of severe intracranial pressure. In this cyst a small glioma was found. An histological examination of the brain revealed the presence of a productive gliosis. The patient was young, which probably accounts for the increased resisting power of the brain, and the symptoms of pressure. In the sixth case (cholesteatoma) were symptoms suggesting multiple sclerosis. The brain showed distinct indications of increased intracranial pressure due to a swelling, not the result of edema, but of a severe hyperemia, probably to be brought into relation to the action of the tumor. The seventh case was one of periodic dementia, there were evidences, at the autopsy, of severe intracranial pressure (evidently of acute origin) shortly before death. The brain was swollen, but no cause for this could be found. The ninth case was practically the same. Reichardt insists upon the necessity of a better comprehension of the term "swelling of the brain." There are varieties of this, and possibly they may serve to explain why sometimes a small tumor will cause a high grade of intracranial pressure and a large tumor will not do so. His studies have been based upon a comparison of the relation of the capacity of the skull to the weight of the brain, and he believes that such studies are important. He also believes that the choked disc must be regarded not as a indication of the size of the tumor, but of the intensity of the intracranial pressure, and—to a certain extent—of the resistance or reaction of the brain to the irritant.

(Band 28. Heft 5-6.)

15. Investigations Upon the Idiomuscular Hyper-Irritation (The Idio-Muscular Contraction of Schiffs). CURSCHMANN.
16. The Relations of Congenital Weakness of the Ectodermal Germinal Layer to the Development of Tabes Dorsalis. BITTORF.
17. The Syphilogenic Diseases of the Central Nervous System, and the Question of "Syphilis à virus nerveux," with Introductory Remarks by W. Erb. FISCHLER.
18. Brief Communication. Further Remarks on My Article, "Studies Upon Oppenheim's Feeding Reflex, and Some Other Reflexes." FÜRNROHR.

15. *Idiomuscular Hyper-Irritation.*—Curschmann, after a careful review of the literature of the idiomuscular contraction, proposed to himself a study of its nature and duration, and also of the wave-like contractions. At the same time he made a careful examination of the irritability of the muscle examined, and of the general muscular system, as well as the mechanical and electrical irritability of the motor nerve and the muscle. Furthermore he studied the relation of the tendon reflexes, the vasomotor irritability, and finally, the occurrence of the spontaneous movements of the muscles. These results are carefully tabulated, although the tables are not reproduced in extenso. Curschmann summarizes them

as follows. Idiomuscular super-irritability and Schiff's waves occur exclusively as a result of mechanical irritation, and pre-eminently in the presence of pathological emaciation. They may be absent under favorable circumstances in women, young children, and in uncomplicated senile emaciation. They do not, however, represent a symptom of wasting, but are the expression of a specific irritability of the muscle. This phenomenon is not the product of the degeneration and death of the muscle; at least, not in the functioning muscles of persons suffering from disease. It may be that deficient water in the tissue is of some significance. At any rate, the fundamental cause resides in the muscular tissue, and is probably due to the action of various toxic materials upon the contractile substance. Disturbances of the central or peripheral motor neurones has no influence upon the occurrence of the idiomuscular tumor formation. Indeed, the latter seems to require at least the essential features of coördination and central trophic influence. There is no relation between it and the tendon reflexes. The degree of idiomuscular contraction is directly proportional to the general muscular irritability. This is also true of Schiff's waves, and therefore, both are probably the expression of an alteration in the irritability. Almost invariably there is found in the more extreme cases of idiomuscular super-irritability a similar increase in the mechanical, and probably the electrical, irritability of the nerves. This must not be regarded as of etiological significance for the muscular phenomena, but probably as an expression of a simultaneous change in the irritability produced by the same poisons. Whether the idiomuscular super-irritability bears any relation to the involuntary fibrillary twitchings of the muscles, or involuntary movements as a whole of muscle groups, is doubtful. But it is certain that those diseases which produce secondary tetany also give rise to the idiomuscular super-irritability, Schiff's waves, and the most extreme type of super-irritability of the nerves. There is no relation, however, to vasomotor super-irritability.

16. *Tabes and the Ectoderm.*—Bittorf has made a careful study of the literature of tabes dorsalis, in order to determine whether there is not some congenital anomaly or vice of construction in the nervous system which is the basis of its development. He regards as sufficient proof for this the discovery that the patient has a neuropathic heredity, because this indicates a diminished resistance of the central nervous system. Among the most important factors in the ancestry are alcoholism, suicide, mental and nervous disease, and epilepsy. The statistics are obtained from sixteen men and fifteen women. Of these thirteen of the men and twelve of the women showed in their ancestry mental disease. This can be compared with five of eighteen men with neuropathic heredity, and two of fourteen women. He concludes with a study of the physical signs of degeneration in his cases. Eight men and seven women had less than five of these signs; eight men and eight women had more than five. Of the cases for comparison 25 per cent. had three of the signs and none had one.

17. *Latent Syphilis.*—The causation of tabes and paresis is still a subject for investigation. It is not clearly understood how the infantile forms occur, nor what is the nature of the congenital forms. Further, there are records of groups of persons infected with syphilis from the same source in whom a large percentage have developed one or the other of these diseases. These observations have given rise to the suggestion of a peculiar form of "syphilis à virus nerveux," or, to the hypothesis of

Hitzig that there is an additional infectious substance usually conveyed at the same time as the other infection. In an effort to answer these questions Fischler, upon Erb's suggestion, has undertaken an analysis of the cases found in the literature. The first group includes the cases of juvenile tabes and paralysis. It appears that the disease has not yet been recognized in the first years of life, and therefore, probably does not occur until later. Moreover, even in hereditary syphilis it may not occur until after puberty. From among these children it is possible to exclude the so-called accessory factors: Tobacco, alcohol, excesses, struggle for existence, etc.; indeed, in some it is expressly stated that, being sickly from birth, they were especially shielded. In the majority of cases congenital syphilis could be determined. In a few cases syphilis acquired in infancy, chiefly from the wet-nurse, appeared to be the cause. In some of the latter cases anti-syphilitic treatment was energetically employed, but nevertheless, paresis subsequently developed. The senses appeared to be about equally effected, a pronounced difference from the conditions occurring in adults. In many cases there is a hereditary neurotic tendency.

In the second group of cases is included a series of cases in which both husband and wife suffered from tabes, tabo-paresis, or from tabes or paralysis in combination with other syphilogenic disturbances. A study of the material indicates that syphilis is the chief cause, and that hereditary family influences have little to do with it. Nor does it appear that a similarity in the manner of life is of much influence. Another series of factors, such as the excessive use of tobacco, etc., can also be partially or completely excluded. Certain cases in which other persons than the husband and wife have been infected from the same source and developed the disease seem to render the etiological relation of syphilis more direct. Fischler admits the frequency with which paresis or tabes occurs in only one member, but also calls attention to the frequency with which syphilis has reached the non-communicable stage by the time marriage is undertaken.

The third group includes various combinations of tabes, paresis and the syphilogenic diseases of the central nervous system. The number of cases is large, and in the great majority it is possible to determine the existence of a distinct syphilitic infection. It is remarkable how frequently in these cases both parents, as well as the children, are affected, in nearly 50 per cent. These cases support the view that in certain cases of syphilis there is a marked tendency to the production of diseases of the central nervous system.

The fourth group includes an interesting series of cases in which several persons not in any way related were infected from the same source, the majority of them in some instances, and sometimes all, developing tabes, paresis or syphilis of the central nervous system. The number of these instances is necessarily small, in all probability chiefly because it is so difficult to determine a common source of infection in a number of unrelated people. If they are related, as in families, it appears that such groups may be frequently recognized. Fischler believes that everything in these studies speaks for the existence of a lues nervosa.

18. *Feeding Reflex.*—Fürnrohr calls attention to the fact that Goltz, in some experiments upon dogs, observed a reflex closely analogous to that of Oppenheim. SAILER (Philadelphia).

Centralblatt für Nervenheilkunde und Psychiatrie

(XXX., 1907, Feb. 15.)

1. Ideas of Reference. M. Rosenfield.
2. Number of Syphilitic Cases in Copenhagen and the Number of Paretic Dementias in Skt. Hans Hospital. P. Heiberg.
3. Trional Cure. Wolff.

1. *Ideas of Reference.*—The author reports three cases, the clinical picture of which, in addition to retardation, depression, and general inactivity, were dominated by delusions of persecution with ideas of reference. The patients did not react to hallucinations, showed no evidence of katatonic symptoms, expressed no hypochondriacal ideas, and intelligence was intact. The recoveries were rapid, complete, and without any defect in the psychical sphere. In regard to diagnosis Rosenfield does not regard these disease pictures as acute abortive paranoia, but classifies them, according to Kraepelin, as manic depressive insanity (depressed form).

2. *Syphilitic Cases in Kopenhagen and Paresis in Skt. Hans Hospital.*—Heiberg maintains that there is a causal relation between the total number of syphilitics of Kopenhagen and the paretics of Skt. Hans Hospital. Two and a half per cent. of all syphilitics in Kopenhagen develop paresis. He draws his deductions from the following table:

Number of Syphilitic Cases in Kopenhagen.		Death from Paresis in Skt. Hans Hospital (15 years later).	
Year.	No. of Syphilitics.	Year.	Number of deaths.
1864-1880	13,500	1879-1885	321
1881-1890	13,500	1896-1905	321
1891-1905	20,000	1906-1920	500 (to be expected.)

3. *Trional Cure.*—Dr. Wolff claims that trional shortens the duration of excitement in certain mental diseases. In his 12 cases (5 cases were reported in *Centralblatt,* 1901, and 7 cases are reported in the present number), 7 were manic depressive insanity, 4 belonged to the infective exhaustive groups, and 1 was an anxiety psychosis, trional cure was applied with success. His treatment is outlined in the following manner: For fourteen days patients received trional from 2 gm. to 3 gm. daily, later the drug was reduced to 0.5 or 1 gm. In most of his cases the drug was administered through a stomach tube. On the second week patients' sleep much improved, psychomotor unrest diminished in frequency and intensity, and excitement soon subsided. Trional was used also with advantage in allaying the excitement in two patients who suffered from dementia præcox, paranoid form and katatonia, respectively. In the trional cure no injurious or unpleasant after-effects were observed, except one case showed marked evidences of intoxication, but patient soon recovered. The author, however, fails to call attention to the fact that urine should be frequently examined for hematoporphyrin. It is to be remembered that "when trional is taken in full dose for several weeks, it produces very distinct alterations in the blood, which are manifested by hemotoporphyrinuria—a state in which the urine is dark red and almost blood. The drug should be stopped at once when the urine begins to be red and saline purgatives must be used freely (Hare)."

Morris J. Karpas (Ward's Island.)

Review of Neurology and Psychiatry

(Vol. V. No. 1.)

1. A Plea for the Study of the Intermedio-Lateral Cell-System of the Spinal Cord. A. BRUCE and J. H. H. PIRIE.
2. A Report of Two Cases of General Paresis with Focal Symptoms. A. HOCH.

1. *Intermedio-Lateral Tract.*—It is known that division of the connecting fine medullated fibres between the sympathetic and the spinal roots causes a *reaction a distance* in the cells of the intermedio-lateral tract; also that division of the cervical sympathetic produces similar cell-changes in the corresponding intermedio-lateral tract and not beyond it. The analogy of the relationships of the splanchnics to the cord suggests for them a similar origin. Bruce and Pirie, in their interesting article, report two cases, with autopsy, in which there was involvement of the sympathetic system and lesions of the intermedio-lateral tract adequate to have produced the symptoms in question. In one case loss of sweating in the lower extremities had developed during the course of an acute atrophic paralysis in an adult. The other case was one of acute exophthalmic goitre, in which death took place during an access of hyperthyroidism. In the first case, there was complete flaccid paralysis of the muscles of the abdomen and of both lower extremities. The case had a sudden onset, and during the four months of its duration there was an intermittent temperature of hectic type from 99°-101°, and later 101°-102°; once as high as 106°. With many of the accessions of temperature there was profuse perspiration above the level of the sixth intercostal nerve, while below a sinuous line two and a half inches above Poupart's ligament there was absolute loss of sweating except over a small area on the inner side of both thighs. Abdomen perspired in irregular patches. On autopsy of this case a congestive and hemorrhagic condition was found in the lumbar and sacral and lower dorsal segments, with partial thrombosis of the anterior spinal vein and in some of the arteries of the posterior roots and in a few of the lateral coronary branches passing towards the intermedio-lateral tract. Motor cells were degenerated, both of the anterior horn and the intermedio-lateral tract. The direct cerebellar tract and Clarke's column were only slightly affected. The writers conclude that there is a special area for sweat secretion in the lower limb, and suggest a special examination of this area, in suitable cases, in the intermedio-lateral tract.

In the case of exophthalmic goitre reported, the nerve-cell changes were limited to those cells contained in the intermedio-lateral tract. They were most pronounced in the 3 D. and 4 D. segments, less in the 2 D. and 5. D., and slight in the 1 D. and 6 D. segments.

The only deductions to be drawn are, apparently, that it is in the intermedio-lateral tract of the segments involved that we should look for the nervous lesion of this disease, inasmuch as it is these segments which give origin to the roots, stimulation of which produces retraction of the upper eye-lid, protrusion of the eye-ball, acceleration of the heart, and sweating and vaso-regulation of the upper part of the body. The writers are not prepared to say that this nervous lesion is the primary one in exophthalmic goitre.

2. *Two Cases of General Paresis.*—Dr. Hoch presents careful histories

and post-mortem findings showing, in the first case, intense processes, more marked on the left side, which had given rise to apoplectiform attacks with subsequent focal symptoms. The patient had exhibited sensory aphasia, explained by changes in the first temporal convolution; and a peculiar visual disorder due to changes in both calcarine fissures. The visual disorder was bilateral. The patient was not blind, but bumped into objects on either side. His attention on the right side could not be attracted by movements or objects, and on the left side only to about 50°, and this with difficulty and to a limited degree.

In the second case similar intense processes gave rise to convulsions, followed by transient paralysis in one leg and aphasia; later, twitchings and loss of power in right arm and again aphasia. The right arm presented at first ataxia; then, after a slight apoplectiform attack, athetosis. The latter ceased after renewed attacks and gave place to twitching, while the arm became more helpless and the reflexes of that side increased. The aphasia was characterized by an almost total inability to speak or write, but there was also marked dementia. On autopsy there was found a marked general paralytic process with greater involvement of the left side, and special involvement of the anterior central and first temporal convolutions and the angular gyrus. The motor tracts were degenerated, left more than right. The third frontal convolution was not involved in this case.

(Vol. V. No. 2.)

1. Circumscribed Hemorrhagic Cortical Encephalitis, with the Report of a Case in which the Lesion was Limited to the Motor Zone, the Chief Clinical Manifestations Being Jacksonian Epilepsy. C. K. Mills.
2. Eight Cases of Hereditary Spastic Paraplegia. E. Jones.
3. An Investigation Into the Arrangement of the Achromatic Substance of Nerve Cells, and of the Changes Which It Undergoes in Various Forms of Mental Diseases. W. M. Smith.

1. *Cortical Encephalitis.*—Mills reports a case clinically and pathologically. The patient was an old woman of eighty-three. Jacksonian attacks occurred over a period of about a week before death, and involved the left face and left hand. During attacks the left eye was closed, brow wrinkled, left nostril dilated and the platysma contracted; there was no frowning or movement of the jaws or eye-balls. The movement of the hand was confined to the deep extensors of the fingers and thumb and group of ulnar extensors. Between convulsions there was great weakness of the lower left face and left arm with wrist-drop. Upper arm and shoulder girdle movements were possible, but were also weak. Tongue protruded to the left. Left conjunctiva greatly inflamed. Sensation and reflexes normal. There was almost complete loss of convergence and of movement of eyes to left and of associated upward eye movements, the latter being more marked on right side. Necropsy showed an enlarged soft heart and patches of sclerosis, and ulceration of the aorta. There was a limited area of softening of the cortex cerebri anterior to the bottom of the central fissure due to hemorrhage and a hemorrhage of recent origin, in the outer portion of the right occipital lobe. Dr. Spiller's microscopical report was, briefly, that there were numerous hemorrhages within the cortex and a very few in the subjacent white matter. They were limited

in extent, the transition from the normal to the hemorrhagic cortex being quite sharp. The adjacent pia was hemorrhagic and contained numerous fatty granular cells and round cells. The affected cortex contained considerable black granular pigment. There was slight round cell infiltration about the small vessels of the peduncles.

2. *Hereditary Spastic Paraplegia.*—Jones' eight cases were of a single childship. Symptoms were the same in all the cases with slight variations. There were in each case spasticity of the lower limbs, talipes equino-varus and changes in the reflexes indicative of an organic affection in the pyramidal tracts. Paresis was slight. The inequality in degree of the cardinal signs was not proportional to the age of the patients. The arm jerks in all were increased. The jaw jerk was obtained only in the two worst cases. In no case were pseudo-bulbar symptoms present. All cases affected were boys, the ninth child of the family, a girl, escaping. No assignable cause was found. All cases showed first evidences when beginning to walk, between ages of one and two years. All the cases ran a similar progressive course, never reaching total incapacity. No cases were found in the ancestry going back 150 years.

3. *Nerve Cells in Mental Diseases.*—Two methods were employed; viz., Bethe's original and Lugaro's colloidal silver method. The former to show the achromatin arranged as fibrils streaming through the cell and indicating nerve-cell relationship and conduction; the latter to show the substance arranged as an interlacing meshwork in the cells and greater detail of structure. Ramon-y-Cajal's No. 3 method and the pyridin methods of Donaggio were also tried. Brain tissue from healthy animals were used for comparative purposes. After a review of some theories of others of the intracellular fibrillary arrangement the pericellular network, anastomosis, development of cells, etc., the author gives his own investigations on normal material from the ox, sheep, pig, and cat, and then states the pathological changes in the neurofibrillar elements of nerve-cells which he claims to have met with in thirty cases of insanity. The precentral convolution was always examined, and in many cases parts of the medulla, cord and cerebellum also. Several interesting drawings accompany the descriptions. Space will not permit report of the changes found except to say that the achromatic structure of the cells as described is profoundly affected in mental diseases, and that the changes differ in degree rather than in kind in the various conditions. Six cases of general paresis, six of chronic brain atrophy, two of epileptic dementia, three of chronic mania, one of diabetes mellitus with melancholia, and the others of dementia, were studied.

CHAS. E. ATWOOD (New York).

Miscellany

FAMILY CARE OF THE INSANE AND FEEBLE-MINDED. Alfred Petren (Hygiea, 1905).

This is a paper which describes the author's travels in Germany, undertaken for the purpose of determining the status of this subject in the latter country. As a purely personal narrative it loses some value from the fact that a number of German psychiatrists had already gone upon record on the subject of family care of mental maladies; so far at least as their personal experience is concerned. (Alt, Wahrendorff,

Nawratski, Falkenberg and others.) The author appears to have visited chiefly such medical centres as had been already made the subject of public description of family care, etc.; so that his narrative seems designed to do no more than convey the testimony of an eye-witness to his colleagues in Sweden.

The pamphlet as it stands is not adapted for reviewing. The style is not only cursory, but there is a total absence of schematic arrangement, statistics, summaries, etc. It might be classed as a piece of medical feuilleton, or as an ordinary medical letter of a traveler—a so-called Reisebericht or Reise-brief, which is a common feature in all medical journalism.

As for the subject-matter, it may be found in a more acceptable form in the original articles upon this subject, one of the latest and best being that of Alt: "Die familiäre Verplegung der Kranksinnigen in Deutschland," 1903.

DEGENERACY. P. C. Smith (Edinburgh Medical Journal. New Series, Vol. XXI, No. 2).

The writer treats the subject as an entity, investigating the mode of its transmission, and giving an account of its pathology, etiology, symptoms, complications, diagnosis, prophylaxis, and treatment. Degeneracy he defines as a state of imperfect development, originating probably in malnutrition on the part of an ancestor, or of the individual during the period of growth, affecting many or all of the bodily systems and functions, and always involving a dissolution of heredity. The symptoms, he states, consists of defects, structural or functional, present at birth or shown during development. The psychical symptoms he classifies into idiocy, imbecility, moral insanity, criminality (some forms), volitional insanity, sexual perversions, and "neurasthenia minor" (neurotions). Anatomical stigmata, he thinks, are not shown to so great an extent in infancy as later. In the slighter forms of degeneracy, the changes in the nervous and glandular systems are as yet unknown. There may be irritable weakness of the nervous system, defective metabolism, diminution of sexual power, hyperplasia or hypoplasia of muscles, bones, ligaments, blood vessels, skin or connective tissue, or one or more gross anatomical abnormalities; but the chemical and histological changes in the nervous and glandular systems are unknown. Degeneracy is found among all civilized nations, in all ranks of society, and in both town and country. Its causes at any one epoch should be looked for in the conditions of a generation or two previously. As regards neurasthenia, the writer holds that where there is much neurasthenia there is a good deal of degeneracy. Degenerates, he says, are more liable than sound persons to attacks of indigestion, to rickets, infantile scurvy, catarrhs, infections and relapses in infective diseases, to local syncope, local asphyxia, and local gangrene, and to the various neuroses and psychoses. They furnish also a disproportionally large number of cases of arteriosclerosis, Bright's disease, consumption, tabes, and perhaps heart disease. The great majority of the insane, he thinks, are degenerates. Tabes and may be other organic nervous diseases affect the neurotic by preference.

In the diagnosis between insanity and degeneracy, from the medico-legal point of view, lawyers might admit a condition of "diminished

responsibility" in those who, as the result of developmental defect, have stronger impulses and weaker resisting influences (instinct, ingrained habits, will) than normal persons.

The writer contends that degeneracy may be eradicated from the race chiefly by the regulation of marriages and by mental and physical education from birth; celibacy is inculcated; the monastery is suggested for some neurotics; and it is urged that the subject of degeneracy should be studied by schoolmasters, jurists and criminal lawyers.

C. E. ATWOOD.

CEREBRO-SPINAL FEVER. WM. OSLER (The Edinburgh Medical Journal, New Series, Vol. XXI, No. 3).

Osler points out several interesting features. Sporadic cases are always with us, but epidemics have occurred in periods of 10 or 15 years since the recognition of the disease in 1805. The first known outbreak began in America and prevailed also in some parts of Europe for eight or ten years. The second, also in America and Europe, occurred in 1837. The third in 1850, and lasted all through our Civil War. A fourth epidemic began in 1871, and a fifth in 1901. In New York in the past two years there have been nearly 4,000 cases with 3,000 deaths. At present the disease is causing alarm in Belfast and Glasgow. A second peculiarity of the disease is that epidemics occur in very widely separated areas, in which it prevails severely, but does not spread widely. It is never pandemic, like influenza. Some of our severest epidemics were in the mountains of West Virginia, and in the mining regions of Pennsylvania, and last year one of the worst epidemics on record was among the Silesian miners. Another peculiarity is that the mortality ranks very high, for an acute infection, perhaps next to the plague. Its mortality is from 50 to 75 per cent. Lastly, among the infections, it is the most virulent. Death has occurred from it within six or eight hours. Other cases may be exceedingly mild and transient.

The specific germ of the disease is the diplococcus intracellularis meningitidis. It is found in the exudate in the brain and cord, and in the secretion in the back part of the nose and throat. Osterman last year found it in the throats of 17 out of 24 persons attending upon patients, but who had not the disease. A certain type of meningitis, the posterior basic, is due to the same organism, so that in reality cerebro-spinal fever, while not occurring as an epidemic, does exist in this sporadic variety all over the country. There are also sporadic forms of pneumococcus mengitis which occur in house-epidemics.

Cerebro-spinal fever has probably the same low degree of contagiousness that we see in pneumonia. It is much more a spinal affection than any other form of meningitis. We have as special symptoms the stiffness of the neck, the muscular rigidity, and the cutaneous sensitiveness. All forms of meningitis the author considers fatal except the cerebro-spinal which gives us from 20 to 40 per cent. of recoveries. The skin eruptions vary in different epidemics. Arthritis may occur, or early deafness, dumbness or blindness. The meningococcus should be sought for, by lumbar puncture, early in the disease, as at the end of a week or ten days it may not be present. The disease does not often prevail beyond the winter season. Where the disease is prevalent the nose and throat of attendants and others near by should

be examined bacteriologically and carefully treated. The hot bath, frequent lumbar puncture, the serum of Wassermann and of Flexner are mentioned in treatment. C. E. ATWOOD.

ON INSANITY, WITH SPECIAL REFERENCE TO HEREDITY AND PROGNOSIS. A. R. Urquhart (The Edinburgh Medical Journal, N. S. Vol. XXI, No. 3). Lecture I—Prolegomena.

In this, his first Morison Lecture on the subject, Dr. Urquhart gives an interesting historical review of the methods of study and investigation of insanity from the time of Hippocrates down, and discusses some of the modern opinions. Acknowledging himself to have been a follower of Sankey, whose generalization that all insanities begin with melancholia and tend to pass through mania and dementia unless interrupted by recovery (or death), he now recognizes later influences and lauds especially the work of Bruce and Robertson; that Bruce has finally brought insanity into the category of other somatic diseases through his clinical studies into the toxic nature of insanity; and that Robertson's conclusions in reference to general paralysis, the failure of the organism to protect itself against bacterial invasion, he thinks may be extended to forms of ordinary insanity which hitherto have evaded the skill of the pathologist; and finally states that it is necessary to revise the opinions of yesterday and recognize that the physical conditions are the more important considerations which render insanity an affair of medicine. In compiling statistics, the neuropathic heredity should be more carefully studied. Want of mental balance, eccentricity, alcoholism, paralysis, should be especially inquired into. It is his custom, so far as possible, to construct graphic charts of each family under observation. From these it appears that the incidence bears heaviest upon the eldest members of the families in fraternity, and that there is a fairly constant diminution of frequency as the families increase in size.

C. E. ATWOOD.

PLASMA CELLS IN NORMAL GASSERIAN GANGLIA. E. Meyer (Anat. Anzeiger XXVIII, Bd. 3, 4).

This article as reviewed by Dr. Goldstein of Königsberg (Centralblatt f. Nervenheilkunde, January 1, 1907), says that Meyer was able to demonstrate plasma cells in all human Gasserian ganglia. The cells were situated within the capsule and they were either singly or in groups. His observations are of great interest inasmuch as plasma cells are usually found in pathological conditions of brain and spinal cord. MORRIS J. KARPAS (Ward's Island).

TUMOR OF THE HYPOPHYSIS WITHOUT AKROMEGALY. Kollarits (Deutsche Zeitschrift f. Nervenheilkunde, Band 28, Heft. 1.)

Kollarits tabulates a series of cases in which there was tumor of the hypophysis cerebri, without akromegaly. Among these he includes two cases of his own. Usually these tumors occur in young persons, but Kollarits is of the opinion that a careful consideration of the cases suggests that tumor of the hypophysis is merely one of the symptoms, and not the cause of akromegaly.

J. SAILER.

A New Algesimeter, with a Critical Description of the Previous Algesimetric Methods. Thumberg (Deutsche Zeitschrift. f. Nervenheilkunde, Band 28, Heft 1).

Thunberg describes his algesimeter, which consists essentially of a lever to one end of which a needle is attached, and to the other end a weighted screw, by which it can be brought into a state of equilibrium. The arm to which the needle is attached is divided into ten parts. The instrument is brought into a state of equilibrium and then weights hung upon the arm. The position and size of the weight determines the amount of pressure exerted. This pressure is increased until the patient experiences pain. The paper is concluded with a valuable critical description of the various instruments hitherto devised for the purpose of measuring sensation. J. Sailer.

The Symtomatology of Hemiplegia. Heilbronner (Deutsche Zeitschrift f. Nervenheilkunde, Band 28, Heft 1).

Upon the cadaver it may be observed that the outer contour of the thighs is more convex than during life. The same appearance may also be observed in the affected leg in cases of recent hemiplegia. A somewhat similar appearance may be observed in the calf and arm muscles. Heilbronner calls this condition "the broad leg." It is not present in sleep, nor in unconsciousness, nor in profound narcosis. It is present in severe acute polyneuritis, but was not found in tabes, in chorea, in Huntingdon's disease, or in hysterical flaccid hemiplegia. It is not certain that it will serve in the differential diagnosis of cerebral and spinal hypotonia. The broad leg is not dependent upon the disappearance of the patellar reflex, or even upon the return of voluntary movement; but if it persists the contractility of the quadriceps to percussion upon its tendon is usually lost. After some discussion of the reflexes and muscle tone Heilbronner reaches the conclusion that in the course of recovery from hemiplegia the following series of events occurs: First, return of active motion; second, return of the contour-preserving tone; third, the return of the reflex muscle tone. With reference to certain other clinical phenomena he states his belief that the superior restoration of the function of the leg is merely apparent. He also discusses the hemiplegic gait, and calls attention to the importance of educating hemiplegics how to walk properly. He describes a curious form of rhythmical movements of the arm during walking, which are entirely involuntary. They consist of a flexion and lifting of the forearm, and may occur when apparently all the symptoms of hemiplegia have disappeared. J. Sailer

The Status Hemiepilepticus Idiopathicus; Eight Clinical and Anatomical Observations. Müller (Deutsche Zeitschrift. f. Nervenheilkunde, Band 28, Heft 1).

Although it is generally supposed that partial or Jacksonian epilepsy is due to some focal lesion in the brain, a number of observations have been recorded in which such partial epilepsy has existed although examination of the brain has been entirely negative. Müller collects from the service of Nonne eight cases of partial epilepsy. The first, an alcoholic and luetic man of 26, who had had an injury to the head. There was left sided status epilepticus not relieved by trephining. The macroscopical examination of the brain was negative; apparently

a miscroscopical examination was not undertaken. The second case, a chronic alcoholic, died in left-sided status epilepticus. At that autopsy there were various lesions of the body, but the brain was macroscopically normal. The third case, a chronic epileptic thirty-eight years of age, had 306 attacks involving only the left side of the body. At the autopsy an area of softening was found on the basilar surface of the right frontal lobe. Elsewhere the brain appeared to be normal. The fourth case, a boy of nineteen, had symptoms of acute meningitis, then left-sided epileptic attacks and death. There was a diffuse chronic meningitis in the base of the brain, but no alteration in the substance of the brain. The fifth case, a woman of thirty-seven, had epileptic attacks limited to the left side; there was a history of injury to the brain. There was high fever, cyanosis, and loss of pupillary reaction. The ophthalmic examination indicated disturbance of the optic nerves. At the autopsy nothing abnormal was found either in the brain or body, and a microscopical examination of the former organ was negative also. The sixth case, a girl of eighteen, had albuminuria, and right-sided epileptic attacks. The brain was normal; the kidneys only slightly affected. There was hypoplasia of the aorta. The seventh case, a man of thirty-seven, in the course of diabetes mellitus, developed typical Jacksonian epilepsy. At the autopsy the brain was normal, but there were changes in some of the abdominal organs. The eight case, a boy of six, had general epileptic attacks at the age of four years. Two years later he suffered from epileptic attacks, limited at first to the right side, then to the left side of the body, profound coma, gnashing of the teeth, and trismus. Later he developed measles, and made a complete recovery. It seems wisest, in the absence of more definite knowledge, to regard these cases as atypical forms of general epilepsy. J. SAILER.

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Original Articles

THE CLASSIFICATION OF PSYCHO-NEUROTICS, AND THE OBSESSIONAL ELEMENT IN THEIR SYMPTOMS.*

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The present trend in psychiatrical circles seems in favor of simplifying the classification of the psychoses, of looking into their nature, and of grouping together cases representing variations of a common faulty tendency.

The object of this communication is to emphasize the influence of the obsessive tendency in the psychoses and to suggest grouping together those cases which present variations of this tendency.

Among the neuroses, psychoses, or psychoneuroses with marked obsessive element and amenable in greater or less degree to psychotherapeutics, may be mentioned *tic convulsif*, (or more appropriately, *tic obsessif*), habit chorea, hypochondria, neurasthenia (psychasthenia), hysteria minor, and manic-depressive tendencies in mild form; *folie du doute* may be mentioned to remind us that some of those conditions are already merged under more general diagnoses.

Certain cases offer sufficiently distinctive characteristics to warrant their separate classification. Analysis, however, shows that they are all variations of a common tendency, and experience reveals that many cases present, in various degrees, symptoms of all these classes. The latter cases it is not only useless but misleading to classify other than under some such general term as psychoneurosis, or still more appropriately, obsessive psychosis.

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All sufferers from these varied forms of mental disorders are ideo-obsessive, and it takes no great stretch of the imagination to trace in all these morbid manifestations the obsessive tendency, the modification of which by educational therapy is the important practical consideration.

The movements of *tic convulsif,* however automatic they may appear, originate in the compelling impulse, and further study of the case will disclose the ideo-obsessive constitution.

Folie du doute results from an obsession to satisfy the doubt. If the victim of chronic indecision doubts which of two tasks first to take up, it is because of an overscrupulous insistence that he take up the right one first. This habit of mind once fixed, the indecision extends to the most trivial questions, but the habit of mind is the same as if the question lay between two actions involving a principle of right or wrong, justice or injustice, advantage or disadvantage.

In this form of disorder, training, especially self-training, under competent supervision, is often effective, whether directed toward the important or the unimportant matters. In fact, such training, directed first against unimportant obsessions, may gradually extend to the more important. I have known more than one doubter who finally learned to say even in important matters, "Others make mistakes, why should not I? It is better that I decide this question wrong than that I allow my mind to become unbalanced by chronic indecision."

Hypochondria results from the obsession to be well, to feel comfortable, and to be safe. The hypochondriac is obsessed to have his every sensation correspond to an ideal firmly established in his mind, an obsession unshaken by argument, ridicule or reproach. The sexual hypochondriac is the victim of an obsession that his organs shall convey to him a certain sensation, and shall perform their functions in a certain way. If they fail to satisfy these requirements he is in distress. If the heart-beat of the hypochondriac varies from an ideal rhythm and character established in his mind as the result of an obsession, his alarm becomes acute, the blood rushes to his head, his mind becomes confused and he is unable to proceed with his duties. Such a patient, after systematic training, may so far alter his ideals as to free his mind from these fears and to comfort himself in these matters like a normal individual.

The *phobias,* closely allied to the hypochondriacal fears, result from an obsession to be always perfectly safe, as well as perfectly comfortable and perfectly well. More than one victim of this form of disorder has been restored to normal mental balance by persistent practice in relaxing mentally and physically, and learning to say to himself, "The worst that can happen to me is nothing compared to losing my mind. It is better that I should break a bone and be laid up for months than that I should be imprisoned for life by hypochondriacal compulsions."

Neurasthenia, a term including the most varied clinical pictures, is generally attributed to overwork or other stress of circumstance. In point of fact, the ranks of neurasthenia are recruited largely, if not entirely, from the ideo-obsessive. In the majority of cases it is not the overwork or the overstrain which has produced the breakdown, but the mental constitution of the individual which makes all work disastrous. The business or professional man who breaks down with neurasthenia will be found generally to have succumbed under a burden no greater than is carried successfully by competitors and associates, whose methods are more reasonable and whose mental poise is more equable. The embryo neurasthenic is opposed to leaving any detail of his business to his subordinates, carries his work into the night hours, takes no vacation, and will not sleep until all the tangles of his life, past, present and future are straightened. If advised to take a vacation before his breakdown, he declines to do so on the ground that he is too busy and that he does not enjoy himself anywhere except at his work. He presents *ill-directed mental activity* rather than the feebleness implied by the word asthenia. Nor has material gain been made by substituting phrenasthenia or psychasthenia. Asthenia is a misleading word to apply to one who is ready and willing to walk miles to satisfy a doubt, or to ascend a dozen flights of stairs to avoid an elevator. The symptoms precipitated by stress are daily matched by those developed in idleness. *Neurasthenics carry their stress with them.*

I have seen more than one individual of this sort completely alter his ideals, devote himself so assiduously to an avocation as to forget his business while in its pursuit, and to bring himself to say, "No matter if I do not enjoy the vacation, I shall enjoy my work the better for having stayed away from the office for

a definite period." One professional man learned to surrender the details and much of the responsibility of his work to subordinates by saying to himself, "I can better afford that they make mistakes, than that I break down and have to go to a sanitarium for an indefinite period, or perhaps abandon my work entirely."

The fatigue following simple excessive work without faulty mental tendency is physiological, not pathological, and in this event some such term as the "brain fag" of Tuke is preferable to either neurasthenia, phrenasthenia or psychasthenia.

While *manic-depressive* insanity in its extreme form is little amenable to treatment, a moderate tendency thereto may be lessened, and even rendered inert, by training and exercise in the direction of establishing a healthy emotional poise. It requires no great stretch of imagination to assume that the exhibition of manic-depressive tendency may result, in part at least, from an obsession to give way to and externalize the emotions. Here is an example of flight of ideas I recently took down.

"Are you blue?"

"Blue, true blue, red, white and blue, one country, one king, no, not one king, one president, we are going to have a new president—President Hearst, cursed, the worst." Who does not recognize the modest prototype of this elaborate rigmarole chasing itself through his brain as he walks the street in jaunty mood? And who has not moments in which his feelings approach those of the depressive form of this disorder. There can be no question that an inherent tendency to manic-depressive insanity may be lessened by persistent training from childhood in the direction of resisting the obsession to give way to and externalize the emotions, of preserving the emotional poise and of pursuing the even tenor of one's way. While marked cases of this disorder may well be separately grouped, there are many at present thus classified, which partake so far of the other faulty mental habits of the obsessive that it seems forcing a point to classify them more closely than under the term psychosis, or obsessive psychosis.

There is unnecessary insistence on the part of clinical observers to place every psychopathic individual in some one definite class, according to the prevailing mental faults.

May I cite the case of a woman of middle life whose parents

were long-lived but invalids; one uncle was an invalid, and one relative is in a insane hospital. Her own mental trouble dates back several years. After caring for her mother, and settling her estate, she became nervous, complained of inability to do things, preferring to remain unoccupied. She has been back and forth between a sanitarium and home, improving somewhat at the former, but at the latter unable to take care of herself, physically strong but uneasy and restless, fearing she would lose her mind, spending most of the time in a rocking-chair, practically never in definite employment, unable to decide upon or carry out any line of conduct. Her mental horizon is limited to insisting upon her weakness of body and going over arguments to fortify this idea. She expects to die every night and says they have broken her body at the hospital. Talks at night and says she should be allowed to do so as she is at the point of death. Questioned regarding her symptoms she states she is too tired to talk, but once started becomes voluble upon the subject of her sufferings.

This case partakes of the characteristics of so-called neurasthenia, manic-depressive and hypochondria. Is it necessary to place her definitely in either class?

I have an athletic friend, the picture of physical health, a man of exceptional ability, who bid fair at one time to abandon his profession on account of mental tribulations. He feared to travel alone and he feared to stay at home; he dreaded his work but feared to leave it off. He spent hours pondering and discussing the question whether he should go to a sanitarium, just what he would do when he was there, and what its effect would be upon him. He would conjure up and worry over every conceivable comment that this step would occasion. He dreaded to be away from home lest something happen to his family; he dreaded to go home lest his wife learn of his fears and not understand them. In his depressed mood his heart was the main object of his solicitude. I have examined this organ again and again as have various other physicians without being able to convince him of its absolute soundness.

After years of mental torment during which efforts were made on the part of several physicians (apparently at the time unsuccessful, but doubtless useful as sowing the seed) he broke down completely and agreed to go to a sanitarium. There he

came under the immediate care of a physician of peculiarly happy method, who devoted himself to training out his obsessions by a judicious combination of stimulation and neglect. At the end of three months he returned, laughing at his own fears. He resumed the same work under which he had previously broken down, and has continued it for months without exhaustion. He takes the chances others do and comports himself in every way like a normal individual.

Should this case be classed as hypochondria, neurasthenia or manic-depressive? It partakes of the characteristics of all, and so in many of these cases, however we may focus our attention upon one or another mental peculiarity, careful examination of the intellectual and emotional life history will develop a similar combination.

In the Neurological Department of the Massachusetts General Hospital the tendency has increased to class such cases under the psychoneuroses, at the expense particularly of neurasthenia. During the year 1906 the diagnosis psychoneurosis was made 91 times; the diagnosis neurasthenia was made only three times, whereas it was made during the same time in the medical department 127 times.

This change of classification does not signify a waning ability on the part of the neurologists to distinguish the different forms of psychoses; it results rather from a growing conviction that we were wasting time in trying to make these combination cases conform to a single type, time better employed in the attempt to modify by psychotherapy the faulty mental tendencies. To the comment that nothing has been gained by substituting one *omnium gatherum* for another, the obvious answer is that it is better to use the word psychoneurosis correctly, than the word neurasthenia incorrectly.

It is no more necessary or accurate to subdivide these complex cases according to their besetting faults than it would be to insist that the tortoise-shell cat shall be classified as either yellow, black or white, according to the prevalence of either of these colors in her coat.

In a certain number of cases, it is true, the symptoms are so well-defined that it is advisable to place them in a definite subclass. This applies to a certain extent to hypochondria, to a greater extent to hysteria, and to a still greater to manic-depres-

sive. We have come to recognize certain symptoms as distinctly hysterical, such for example are aphonia, anesthesia (particularly hemianesthesia), and the paralyses, hyperesthesia, the contractures and the convulsive attacks and the angio-neurotic edemas. Cases exhibiting such definite signs may well be classed as hysteria. Such cases, though doubtless presenting other signs of mental deviation, cannot be traced directly to the obsession, except in so far as the insistent desire from childhood to have one's way, and to be an object of sympathy (a desire which doubtless often aids in the development of the symptoms) may be regarded an obsession.

Babinski[1] has recently proposed to define hysteria as the neurosis constituted by all the disturbances which are susceptible of being cured by persuasion, direct or indirect. He proposes to call it pithiatism, from *peitho,* persuasion, and *iatos,* curable. He says hysteria is the only affection susceptible of being cured by psychotherapy. I should take issue with this conclusion and with this classification.

Many more cases are appropriately classed as manic-depressive. Doubtless, however, a large number of cases are at present so classed at the expense of straining a point. Take the following case.

Case. A bright young woman engaged in clerical work. Her father is in an insane hospital. She has been for some time tormented by fears, scruples and self-reproach. She has never recovered from the effect of a certain letter conveying a reproach upon a member of her family. She fears she has sinned irreparably. She not only regards it her duty to help support her father but contributes from her wages toward the support of another relative. After the death of this relative, who left enough funds for the support of her father, her mental state is not in the least relieved by the lessening of her burdens. She writes that she fears she exaggerated some statement in her former visit, but still feels that her sins are heavy. She is now cast down because she has not the proper degree of affection for her father, and says it were better she had never been born. During the past year she has been torn by doubts whether to marry. She has insistent ideas regarding air in the room. She is inclined to think others do not want her around. She is physically strong and can walk

[1]Tribune médicale, Sept. 22-29, 1906. Abstracted in Jour. Amer. Med. Ass'n, Feb. 2, 1907, Vol. XLVIII., No. 5.

miles. She performs her work with accuracy and despatch.

Whether this case be classed under manic-depressive, neurasthenia, or elsewhere (surely not as dementia præcox in view of her work), the underlying characteristic seems to me that she is constitutionally ideo-obsessive and that her case is another instance of obsessive psychosis.

Since writing the above I have received a communication from the patient stating that what I told her about putting herself through rigid mental training has been a great help to her. "My distress of mind is passed,—it all seems like a wretched nightmare, wherein I greatly exaggerated, and very unjustly blamed myself. Life once more looks worth living, and I would thank you for all the time you spent so carefully consulting with me."

It is, of course, too early to say whether this frame of mind will be lasting, or whether it is merely a phase of manic depressive tendency. Such cases can be multiplied indefinitely.

Conclusions.

Many psychoneurotics offer a combination of the symptoms classed under neurasthenia, hypochondria, *folie du doute, tic convulsif*, habit chorea, manic-depressive and hysteria minor. Many, if not most, of their morbid mental and physical tendencies may be traced to the obsession.

In these cases the treatment is more important than the exact classification: unless the symptoms of one or the other disorder are definitely preponderant it lends more to clearness to include the general term psycho-neurosis, or still better, obsessive psychosis, than to insist upon a more distinctive classification.

The result of simple fatigue, without obsessive or other morbid mental tendency, is physiological, not pathological. In such cases, therefore, the term "brain fag" of Tuke would answer every purpose and be less misleading than neurasthenia or even phrenasthenia or psychasthenia.

The term neurasthenia, though convenient, like "nervous prostration," for popular use, is inaccurate, misleading and unsatisfactory, and can be discarded so far as scientific records are concerned. Nor has any material gain been made by substituting psychasthenia or phrenasthenia. The majority of the cases thus classed partake so far of the various morbid mental states peculiar to the ideo-obsessive, that they are best included under the general designation, psychoneurosis or obsessive psychosis.

THE USE OF SOCIAL INTERCOURSE AS A THERAPEUTIC AGENT IN THE PSYCHONEUROSES, A CONTRIBUTION TO THE ART OF PSYCHOTHERAPY.*

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Abstract. Meaning of the terms Psychotherapy and Social Intercourse as used in this paper. Etiological factors open to treatment. Treatment directed to the change or removal of the known indirect and contributing causes as opposed to the treatment of the unknown direct or fundamental causes. Problem in Psychotherapy largely the former. Re-education and development of a personality. Use of selected individuals as the foundation of a new social life. Methods, difficulties and limitations. Results and conclusions.

In this paper I plan to outline briefly an experiment in psychotherapy based upon the realization that an individual whether normal or abnormal reacts favorably or the reverse to the various people and circumstances among which his lot happens to be cast. I wish to suggest further that in certain instances these factors can be to some degree influenced by forces external to them, and that this influence can be consciously exerted in that direction or in those directions which may seem to be most favorable to the individual. I hope likewise to be able to prove that an effort of this kind falls within the scope of a neurologist's therapeutic activity.

The object of this therapeutic experiment is a case of hysteria in a woman with the usual complicating factor of neurasthenia. Thus the attempt both in respect to the psychical disease and to the treatment made use of may well come within the meaning of the term psychotherapy.

Probably the most striking phenomenon in neurology to-day is the sudden interest manifested in the subject of psychotherapy. In the past three or four years the literature has been plentifully supplied with papers, monographs and books dealing with this subject. The better of them reflect the spirit of a widening grasp of the power inherent in the action of one mentality upon another. There runs through some of them a desire, scarcely more than indicated, directed towards the development of some

*Read at the thirty-third annual meeting of the American Neurological Association, May 7, 8 and 9, 1907.

tangible technique or method by which these forces subtle though they are, may be set most economically in action. The chief obstacle to a wider use of psychotherapy is found in the lack of an analysis of the force implied in the term, a means by which it can be applied and a measure of its utility. Freud's various papers dealing with psycho-analysis may be said to be the most definite attempt at a systematized technique that has as yet appeared. Its complexity and difficulty of application, to say nothing of the etiological factor which is a necessary complement of the method speak most surely against the hope of anything approaching a general use of this system. Perhaps in no better way can the truth be emphasized that psychotherapy is largely an individual therapeutic art than the utter failure of psycho-analysis to meet the average neurologist's requirements.

It is therefore evident that the most that can be done at present is to note such individual instances of the use of psychotherapy as they appear and to attempt to see in them such general principles as seem to be demonstrated. In such a manner very gradually some kind of a generalized technique may be built up out of which each individual may obtain the procedures or the suggestion of them which will be useful in his special need.

The term psychotherapeutics as used in this paper implies the use of all forces outside of drugs, manipulative and so-called physiological methods which can be directed toward curing or making better an individual the subject of functional disease of the nervous system. The definition is sufficiently broadly devised in order that the attempt to be outlined in this paper may be covered. It assumes likewise that the influence inherent in the personality of the therapeutist may be considered fairly a factor and that this does not necessarily detract from whatever therapeutic proof there may be in the efficacy of the experiment.

It is the author's belief that the neurological therapeutist in common with others of a less restricted field has the right to regard as possible curative agents any factor found in the social life of his patient or further any factors which he may choose to inject into that social life. By the term social life is naturally meant the sum of all the activities which arise from the relation of an individual with others of his kind. This relationship may be organized or unorganized, complicated or simple as the case may be.

It is apparent that an effective therapy in hysteria must be based upon a sound conception of what hysteria is and to what hysteria is due. These are fundamental necessities for the permanent solution of any problem in therapy. It is just as apparent that at the present moment we are in no position to satisfy either of these demands and we are consequently forced to admit that hysteria exists largely in the person of an individual who has what we are accustomed to regard as hysterical symptoms. In other words our therapy is compelled to busy itself not with the attempt to remove or touch the cause but to moderate the effects. That is, the indirect factors which are present in determining the persistence of hysterical phenomena are the only ones that are really open to treatment. Not only must this be admitted but it must be acknowledged that the genesis of the hysterical symptoms is not understood.

In spite of a vast amount of work of which special mention might be made of Freud, Janet, Sidis and Prince and others we are in no position to say where nor how the hysterical phenomena play their part, that is in a psychical sense. If for example we are prepared to regard them as products of sub-conscious activity we are met with a divergence of opinion not only on this score but likewise on the question of the existence of such a thing as the sub-conscious. Whichever way we turn we are face to face with the fact that hysteria offers no certain field for the exercise of a therapy aimed to remove or remedy first causes. On the other hand there is an increasing lot of data which has to do with the indirect or secondary causes. These influence the progress, the degree and the persistence of hysteria in a very definite and tangible way. It is these causes which appeal to me to be the legitimate object of psychotherapy and this paper is concerned with an account of how such causes were attacked in an individual instance of hysteria.

The Case. From a purely clinical point of view the case offers nothing of interest. A woman 35 years old coming from a marked neuropathic family has for the last seven or eight years been the subject of a group of symptoms which are commonly found in hysteria. In addition there exists side by side with them a group of symptoms depending upon an abnormal degree of fatigue and irritability. These point to neurasthenia. In other words this patient presents the not uncommon neurasthenia hys-

teria symptom-complex. It might be of interest to state that in addition to the purely mental symptoms of hysteria there were present objective indications of the disease such as sharply limited areas of hyperesthesia, hyperesthesia over the ovarian region, hemihypesthesia, increase in the deep reflexes, conjunctival anesthesia, etc. The data of the past history that the limits of this paper permit to be mentioned are, an unhappy marriage ending in divorce, the husband disappearing four years ago, two gynecological operations, one induced abortion on account of hyperemesis gravidorum. There were no other notable illnesses and no other etiological factors that could be definitely placed. The patient has one child now about fourteen years old. It might be mentioned here for the purpose of making clear certain points afterwards to be alluded to that Mrs. J. came to St. Louis from Massachusetts knowing no one and having so social opportunities. During this time she lived with a brother and son, keeping house for them. In this limited circle, with no amusement, no opportunity for social intercourse, no deviation from the household regime, three years were passed. She came under my care a completely hopeless and discouraged woman, one that had run to seed mentally, physically and it might be said morally.

The case from the very outset appealed to me as one that offered an unique opportunity for an experiment in psychotherapy in its widest application. Here was a woman physically normal, well educated, with a latent talent for minor literary activities, a sufficient idealist, gifted with imagination, no acquaintanceship, and a consistent and eager desire to get well. This latter tendency was based upon two factors which in cases of this kind are of importance. First an instinctive love for the physically healthy and normal, and a keen desire to lessen the burden resting upon the shoulders of her brother.

I wish to preface the account of the therapeutic attempt by the remark that no mystery was allowed to exist in this case at all. Every step taken was carefully explained to the patient and her co-operation demanded. The group of symptoms were taken as existing and no attempt was made to investigate them further than to be aware of their presence. No insistence was made concerning the importance of their disappearance in respect to the favorable progress of the case. I mean that their origin was left unexplained and the conditions which were present and which

seemed to act towards their continuance were attacked. As soon as a thorough understanding was reached a scheme of treatment was planned which was to last for a long time. I emphasized this because I believe that one of the causes of failure in psychotherapeutics lies in the fact that no definite and consistent plan is either considered or carefully followed. In some mysterious way the mind is supposed to be influenced by the persuasive or suggestive power of another mind and the result achieved is often as much of a mystery as the manner in which it was brought about. In fact to many of us psychotherapeutics is a sort of negative process, the chief distinction about it being that drugs are not given. It is this careful planning that marks, I believe, the first step towards the building up of a technique.

Briefly, the therapy was planned to meet the following fundamental needs:

1. Need of a social existence in which the patient might feel a constantly growing activity and importance.

2. The necessity for the development of some serious business in life in which the patient could regard herself as fulfilling some purpose and further as giving her the chance to feel herself gain in mastering over whatever this employment might be.

3. The need for the creation and the development of an ideal of living apart from the mental and physical sufferings to which this woman had been so long accustomed. The mechanism by which the first was accomplished was in outline as follows:

Certain friends and patients of mine and some who were neither, were considered solely from the point of view of their possible personal influence upon this patient. In other words a rather bold attempt was made to create *de novo* a social life, the nucleus of which was formed by a group of individuals selected largely with a view to their favorable social reaction upon the patient. That this is very artificial the writer is willing to admit, and that it is more or less blameworthy to use individuals as social pawns he confesses to likewise, but there seemed to be little danger and the fascination of the game proved sufficiently strong to hide the defects of the system. Sometimes these people were led to meet the patient through the ordinary conventional means, more often not. Complicated schemes were devised through which meetings were arranged which on the surface appeared accidental. After the pivotal units were gotten together, the rest

followed even more rapidly than at times was altogether desirable. In this way very slowly a small group of people was formed of which the patient was an active and congenial factor. Among those who were selected were one or two whose robust personality and sane views told with great force upon the patient's weakened grip on the tangible business of life. She saw in them the positive evidence of the new viewpoint which it was the part of her physician to attempt to develop for her theoretically. The second part of the therapeutic scheme was accomplished with very little difficulty. The patient had a talent for verse which had enabled her to have published in some of the better magazines some of her work. The Atlantic Monthly, Scribner's, and the Century had printed some verse of hers, and one or two of them had attracted the attention of the editors. Some of you have very possibly seen specimens of her verse. These poems are very analytic and rather somber and they are of a kind that would never attract popular attention. When she came under my care, she had given this thing up for three years or more. It was part of the plan to make this activity the central part of her life in its larger relations. This was never lost sight of for one moment. Its importance was perhaps exaggerated from the very beginning. She was encouraged to attempt newer forms of activity in a literary way. Short stories, drama, sketches, a novel, etc. The physician in this instance established himself as her literary critic and confidant and at times supplied plots for the short stories. Through her published work a certain amount of recognition was obtained which was more effective than any other one thing in strengthening her growing feeling of importance and the lessening the tendency towards self-minimization which was so much in evidence before. This patient became somewhat sought after, that is, in a minor sort of way, and her sense of being somebody with a place created by her own effort seemed to act in the way a tonic ought to but never does. I might mention here the effect on the patient of a series of imaginary characters for one of the shorter stories and the effect on a writer of the mental diversion produced by following their necessary fate. I mean as an agency for directing the attention from the thought of the neurasthenic symptoms this can be made remarkably effective. The last part of the plan was fulfilled mainly through carefully planned talks at frequent enough intervals so that the thread of the thought might be continued. This is difficult to describe, but it consisted largely

in an effort to dissect former erroneous ideas and hopes and to substitute a saner conception and a more definite appreciation of the whole scheme of existence. Especial attention was directed to the creation of the objective attitude to happenings of daily experience. No description of this part of the plan is necessary. Everyone has had his to do. I wish to insist again, however, that to be of any service an effort of this kind must be carefully planned and consistently carried out and yet with enought tact that the patient is not aware that she is the object of a series of didactic lectures on her shortcomings.

The result. After a year and a half the patient, whose condition I have outlined in the begining of this paper, is a very much changed person. She herself confesses to a totally different feeling about herself. The fatigue symptom has almost disappeared. The hopelessness and morbidity are no longer in evidence. The patient is active, busy and fairly happy and reasonably contented. The testimony of her brother supports this view. At the last examination the objective hysterical symptoms had very nearly vanished—the subjective completely. As a therapeutic result I should consider this case an exceedingly favorable example of treatment. That she still has hysteria I am willing to admit, but as a member of society she does very well in spite of the neurasthenia and hysteria.

The limitations of the method outlined in this paper must be sufficiently obvious. It is only in rare instances, I believe, that such a combination of circumstances is met with as was constantly present in this case. For this very reason it is not a sufficient experiment to warrant any conclusions. It might be said, however, that experiments in therapeutics are usually made on the extreme instances of disease and that afterwards the more common varieties are the subjects of treatment. In this way perhaps this experiment in psychotherapeutics may be regarded. It establishes, I trust, the right of the neurologist to broaden the field of his therapeutic endeavor, and it implies the existence at least, of forces to be used in a curative way, that were not thought within the province of the physician. Above all it suggests that the technique or system of psychotherapeutics must in the long run be made up of the accumulated wisdom of many such experiments in each of which some definite thing might be found. To contribute something to this end has been the chief purpose of this paper.

MYOCLONUS-EPILEPSY WITH A REPORT OF TWO ADDITIONAL CASES.*

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This condition is one of the numerous forms of the epilepsies of which there has been but a small number of cases reported.

A review of 2150 cases of epilepsy admitted to the Craig Colony for Epileptics, since its opening in February 1896, shows but seven cases of myoclonus-epilepsy. The first of these was reported by L. Pierce Clark in *Archives of Neurology and Psychiatry*, Vol. II, 1899. Three of these were reported by L. Pierce Clark and T. P. Prout in their excellent paper in the *Journal of Insanity* in 1902; the fifth by L. Pierce Clark and myself, the last two being now reported for the first time.

The first case on record of paramyoclonus multiplex reported was that by Friedreich in 1881. A search through the earlier literature shows histories of cases which were undoubtedly the same condition. He describes a patient, aged 50 years, who developed quick clonic contractions of symmetrical muscles of extremities. These contractions resembling those produced by an electric current, ceased during sleep; increased by peripheral mechanical stimuli. They were diminished or entirely inhibited by voluntary movement. Muscles of trunk and face escaped. Nutrition and mechanical and electrical irritability unaltered. Increased knee jerks, no affection of sensation. No interference with motor power or coordination of involved muscles.

His case was a result of a severe shock, and the spasms, after lasting several years, rapidly ceased, recurring later and continued until death.

Many variations from this description have been seen in cases reported later. In some there is considerable locomotor effect, in others little or none.

Face and trunk muscles are frequently involved. Voluntary movements often increase the myoclonic movements instead of diminishing them. Knee jerks may be normal or diminished instead of increased.

It attacks groups of muscles which cannot be thrown into

*Read before the Rochester Pathological Society, May 2, 1907.

contraction voluntarily and movements show no coordination as occurs in movements produced at will. In the majority of cases the proximal segments of the limbs are most affected.

Mental excitement and physical fatigue often provoke the movements. Excess of indican has been present in some cases when the myoclonic movements were most marked. In other cases it was absent. The movements are of a type that cannot be produced voluntarily.

Some of the phenomena of hysteria may be present in myoclonus epilepsy as in ordinary epilepsy.

Clark in his article in *The American Journal of Insanity* in 1902 has compiled a list of 57 cases of myoclonus epilepsy reported up to that ttime. Since then several cases have been reported in the literature. The most recent being eight cases reported from the Colony for Epileptics at Bielefeld in a population of 2000 epileptics.

Aldren Turner in his recent work on Epilepsy reports but two cases of myoclonus epilepsy in one thousand cases of epilepsy that came under his observation.

Many of these cases have been confused with motor aura and incomplete seizures.

Unverricht in 1891 in his monograph, "Die Myoclonie," describes several cases of a family type.

Etiology.—The same causes which predispose to epilepsy, insanity, imbecility, etc., are found in these cases, viz., alcoholism, insanity, tuberculosis, epilepsy and other signs of family degeneracy. These patients themselves show many signs of degeneration.

The exciting cause, if one is given, is frequently one which has little or no bearing on the condition in question. In one of the two cases reported in this paper no cause is assigned except that patient was always "nervous." In the other jerkings appeared a short time after patient had measles.

Sex.—Males seem to predominate. In those at Bielefeld 5 were males, 3 were females. In the 57 collected by Clark in 1902 the ratio was 5 males, 3 females. In the seven at the Colony, 4 were males, 3 were females.

Most of the cases develop before adult life although some do occur later, one reported in this paper claiming that the myoclonic movements did not appear until she was over 30 years of age.

Turner's two cases were women and in them the myoclonus was present for many years before the first epileptic seizure.

Pathology.—Friedreich and Unverricht believed the disease to be due to excitation of the motor ganglia of the cord. Unverricht also considered an additional involvement of the cortical motor ganglia. Others have described the condition to lesions in the medulla oblongata, cerebellum, corpus striatum, optic thalamus and cerebral cortex. As has been frequently stated, it would seem if the lesion was in the spinal ganglia we would have trophic changes in the muscles.

Clark and Prout conclude that the lesions appear to be in the cerebral cortex involving the nucleus and the intra-nuclear network of cells of both sensory and motor types. Its pathogenesis appears to be an intoxication or an auto-intoxication of these cortical cells, probably brought about by a faulty chemotaxis of these same cells because of an inherent organic anomaly.

Clark reports a case from the Craig Colony in which an autopsy was made and a microscopical examination of the brain showed the following condition: General chromatolysis, absence of nuclear membrane and granular (often swollen) nucleus in every portion examined (Sections taken from both motor and frontal areas). He lays stress on the fact that the large pyramidal cells of the third layer are especially involved.

J. Ramsey Hunt believes that there is a coexisting degenerative affection of the cerebro-spinal axis, the epilepsy referable to the upper, the myoclonus to the lower centres. He found a marked hypertrophy of the muscle fibers in a case of myoclonus multiplex.

J. Risien Russell's hypothesis is that according to the phenomena, the motor neurones of both the brain and spinal cord may be affected, and that the neuroclonic state existing in the neurones of the anterior horns of the spinal cord or their homologues in the medulla oblongata and pons, may give rise to myoclonic spasms, whereas an altered state in the neurones of the cerebral cortex may be subsequently and secondarily induced, giving rise to epileptic seizures. Or again the altered condition of the cerebral cortex, and the consequent epilepsy, may be primary, and the neuroclonic state of the neurones of the spinal cord may appear subsequently.

This hypothesis of Russell's appears to be very reasonable

when the symptoms are given careful consideration.

Diagnosis.—This condition is confused at times with pseudo-myoclonic contractions (which the patients themselves characterize as "jerks"); isolated tics of epilepsy; post-hemiplegic choreic movements associated with epilepsy; choreic epilepsy; hysteria; and myoclonia in general paresis.

The characteristic grand mal seizure and movements which could not be produced voluntarily should be sufficient to exclude hysteria.

The pseudo-myoclonic movements, "jerks" are bilateral flexure movements of the upper extremities although cases are seen in which these premonitory "jerks" involve the lower extremities. Some are on the border line between epilepsy and myoclonus epilepsy.

Various tics in epilepsy are confined to the face; are not co-ordinate and usually voluntary or reflex emotional acts, acts whose general character continues unchanged although exaggerated by emotions.

One case admitted to the Craig Colony showed multiple tics involving especially face and right arm. These movements were constantly present to a greater or lesser degree but increased markedly 24 to 28 hours before seizures. Patient had a marked neuropathic family history.

In choreic paresis and in infantile spasmodic hemiplegia (without palsy) the convulsions begin and involve most frequently parts once paralzed. The choreiform movements are rhythmical and unilateral. Unilateral atrophy also occurs.

In general paresis the mental state, speech, pupils, etc., should make the diagnosis clear.

The diagnosis is made on the presence in an epileptic of bilateral, electrical shock-like convulsions occurring in paroxysms and involving no impairment of consciousness and at intervals the occurrence of grand mal seizures, epileptic in nature.

It may appear as a pre-epileptic or post-epileptic phenomenon or as a condition independent of the distinct epileptic seizure. In the latter case it is sometimes considered as an equivalent of the seizure.

Prognosis—Periods of freedom from myoclonic movements of variable length occur but there is no permanent cessation. The mental condition may change but little. Death may oc-

cur in status myoclonus or from intercurrent disease as pneumonia.

Treatment—This must be along general lines tending to place a patient in the best possible physical condition.

Chloral hydrate and amylene hydrate have a marked effect in controlling myoclonic movements. Bromides have an excellent effect in some cases.

Report of two new cases follows:

E. C. B., age 39 years. Single. Common school education.

Family History—Father living, aged 71 years. He is an alcoholic and has been frequently intoxicated during the past twenty-four years. Said to be a worthless character. No definite history of syphilis can be obtained but as he was an old alcoholic soldier and his wife had several miscarriages it would seem probable that he had syphilis.

Mother was a nervous woman who had frequent headaches, fainting spells and attacks of vertigo, but no fits or movements similar to those patient has. She died at age of 39 years. Assigned cause of death was confinement and Bright's disease.

Maternal grandmother died at age of 87 years. She enjoyed good health until late in life, when she developed some nervous trouble.

Patient eldest in family of five children. One brother died at 19 years from pulmonary tuberculosis. One sister was sickly and died at age of two years from some intestinal trouble. Two sisters married, both of whom are in poor health, exact condition unknown. One has no children, the other has had several who have been sickly and died young. She has one daughter living and well. One of the children who died, a son, had convulsions during his last illness at five years of age. He is said to have been a bright boy but frequently cried without apparent cause.

Two maternal aunts are troubled with some gastro-intestinal disorder, the exact nature of which cannot be ascertained.

Personal History—Patient was born at full term. Nursed by mother. Nothing can be ascertained as to delivery or early infancy of patient. No history of any traumatism. No history of difficulty in learning to walk. Had measles at six years. She is said to have been ill for three months with scarlet fever at age of nine years. During the period of this illness she had convulsions and was totally blind for an unknown period.

Patient claims that her first seizure, resembling a fainting spell, occurred when she was eighteen years of age. She

was at that time working as a clerk in a store and had to give up her position. The seizures recurred and became more severe until she had a general convulsion at age of twenty-two years. During this seizure she fell against radiator burning her right hand. Usually has no aura, but at times has a tingling in her fingers. Previous to her admission to the Colony she is said to have had seizures at intervals of from one week to two or three months. They occurred usually in series and were followed by a marked prostration and stupor. The first seizures in the series were severe and the later ones mild. Face and hands said to have been affected first. She had bitten lips and tongue during seizures, and received many injuries by falling at those times, viz., burns, fracture of nose, contusions about head and arms.

Patient complained of a pain and a "drawn feeling" in the head after seizures. At age of thirty-six she had a slight paralysis of left hand following seizures. This disappeared after two months. She has had as many as eight seizures in twenty-four hours. Is said to have been jealous, stubborn, depressed and at times hysterical.

Physical examination on admission to the Craig Colony, June 15, 1905. Brunette of spare build. Height 4 ft. 10½ in. Weight 71½ lbs. Has an anxious expression. Ears have adherent lobules. Small inferior maxilla. Palate has a narrow and moderately high arch. Moderate scoliosis. Slight facial asymmetry. Thyroid not enlarged. Skin and mucous membranes pale. No evidence of syphilis. Slight cyanosis of feet. Eyes—No ptosis, exophthalmus, nystagmus or strabismus. Twitching of eyelids. Cornea and conjuctiva are markedly sensitive. Vision—right eye, 20—40; left eye, 20—50.

Complains of an occasional tinnitus aurium. Gustatory sense normal. Olfactory sense impaired on both sides.

Chest negative. Has always been troubled with constipation. Appetite poor. Some pyrosis and distress of epigastrium after eating. Considerable prominence of abdomen in general, apparently due to accumulation of gas in intestines.

Breasts atrophied. At age of 27 years she had both ovaries removed as she was told this would cause her seizures to stop. Menstruation began at 14 years and was irregular up to time of operation. Complains of almost constant vertigo, suboccipital headache and pain in vertex. General feeling of weakness and exhaustion.

Tactile, pain, temperature, localization and stereognostic senses normal. Dermatographia absent. No anesthesia of ulnar nerve.

Slight abdominal and epigastric reflexes. Very active patellar jerks. Plantar is of lively flexion type. Active wrist and elbow jerks.

Patient right handed. Dynamometer shows no inequality between two sides. Handgrasp only moderately strong for her general physique.

Fig. 1. E. C. B. Holding inferior maxilla with right hand and left hand grasping clothing tightly, in an effort to control myoclonic movements.

No signs of paralysis except that in showing her teeth muscles on right are more active than on left. General musculature is poor. Sense of position normal. Because of presence

of myoclonic movements patient could not walk without assistance. Mentality fair. Speech interrupted but slightly by movements.

Shortly after her admission to the Craig Colony, she had a series of grand mal seizures which was followed by a state of mental confusion accompanied by illusions and hallucinations of sight and hearing. Disoriented. Very lachrymose and then would suddenly change and laugh in a meaningless way. She dressed herself and went to the table for her meals. At another time she had marked myoclonic movements following grand mal seizures. Seizures are usually grand mal in type and occur at intervals of several weeks.

Following is a brief description of a myoclonic period: Patient appears slightly agitated, is lachrymose. When walking with support there is a marked extension of right leg at knee from time to time. Very unstable equilibrium, especially when weight is borne on left leg. Bilateral clonic nodding movements, at times muscles on right side of neck drawing head to that side, occasionally similar movement of head to left. Smacking, tasting and munching movements of jaws. To and fro movements of tongue when mouth is open. Frequent marked clonic movements of muscles of mastication causing lower jaw to be depressed and then elevated. Patient holds inferior maxilla with her hand endeavoring to control movements. (See photograph.) Swallowing movements frequent. When mouth is closed there is a lifting of chin and compression of lips at frequent intervals.

Muscles of eyelids and forehead occasionally involved. Drank water without difficulty when glass was held to lips by nurse. No ejection of food from mouth. Active involvement of temporal muscles but only occasionally of occipito-frontalis. Complains of a "drawn sensation" in temporal and occipital regions.

When sitting or walking the muscles of back and back of neck are markedly involved in lightning-like jerks. No tremor of tongue. No nystagmus.

Can walk about room by balancing against walls, chairs, beds, etc., or having some assistance from nurse.

When lying on abdomen there is an occasional myoclonic movement of erector spinæ. Lying on back there is a marked myoclonic flexion of lower extremities, especially right. Right upper extremity, then left markedly involved. Flexors more than extensors.

Knee jerks exaggerated. Patellar clonus on right side. No ankle clonus or Babinski reflex. No increase in reflexes of upper extremities.

Abdomen prominent, walls are not rigid nor apparently involved in myoclonic movements. Peristaltic movements apparent in lower abdomen. No borborygmus.

Passive movements of extremities resisted. (Patient said she was endeavoring to exert some control over movement.)

No diaphragmatic grunt. No hiccough. Patient says she feels worn out and discouraged during myoclonic periods.

When not spoken to and allowed to lie quietly in bed, the movements are somewhat less frequent, but when spoken to become exaggerated.

She says she often has a sensation of tingling formication, followed by a feeling of coldness over entire body, but more in left arm and leg, especially former. Feels better when sitting up sewing, although when working at latter, she frequently sticks needles or scissors' points in fingers and has great difficulty in threading needle.

Urine examined during myoclonic periods shows no increase in indican. Sometimes urinates involuntarily during myoclonic periods. No impairment or loss of consciousness. Complains frequently of anorexia and insomnia.

Patient states that previous to her first seizure at age of eighteen years she was nervous, could not stand any excitement and had frequent crying spells. No myoclonic movements until she was about 34 years old. They first appeared in hands and feet, later in head. Movements have gradually become more severe and frequent. Movements are frequently more marked following seizures than preceding them. Marked vertigo accompanies periods.

She says the weather has no influence on the movements and this has been corroborated since her residence here. Electrical reaction normal.

This patient has not been entirely free from the movements at any time since her admission to the Colony.

E. J. W. Age 15 years. No occupation.

Patient is second child in family of three children (two boys and one girl, all of whom are living).

His brother, aged 11 years, has jerkings and has had "two or three fits."

Father died at 29 years from typhoid fever and is said to have had nervous prostration. Mother died at 23 years from tuberculosis. She had convulsions when a child. On his mother's side there has been a number of cousins who have had convulsions. Mother's people were "nervous." Father or mother was not intemperate. Maternal grandfather died at 40 years from tetanus. Otherwise grandparents negative.

Patient born at full term. Labor "quick and normal." Delivery natural. No injury during delivery. Weighed eight pounds at birth and was a strong baby. Had no spasms directly after birth nor was he paralyzed. Nursed by mother. No indigestion nor prolonged crying in infancy. Dentition began at four months and was not accompanied by convul-

sions. No pavor nocturnus. Began to walk without difficulty at age of fourteen months. Did not show any swollen glands or signs of rickets. No epistaxis. No injury in early life. Had pertussis at nine years and measles at ten; both ran ordinary course.

Patient says he started to school when he was ten years old, could not go earlier because he lived so far away from school.

First attack occurred when he was eleven years of age. He was driving a horse when it fell down and broke the harness and shafts. That night he had first convulsion just before going to bed. Supposed cause was fright. Second attack occurred three days after first. Attacks have occurred nearly every night during the last two years. Has had a few attacks in day time. Attacks are not as bad as formerly, but are more frequent. One attack is described, "He screeched and shook for about ten minutes." Feels badly in head after each attack. No aura. Vary in severity. Sometimes he says his right and sometimes his left side has a pain in it after a fit, more often the right. Patient is conscious in light attacks. No paralysis or aphasia following seizures. Has had seven attacks in 24 hours. Rather small eater. Patient says that for about a year preceding his fright he had what he called "jerkings." They began a short time after he had the measles.

Physical Examination—Rather slight. Hair brown and abundant. Poor musculature. Height, 3 ft. 9½ in. Weight, 74 lbs. Head well shaped. Palate high, some lordosis. Is flatfooted. Scapulæ stand far apart. No eruptions, scars or bruises. Skin and mucous membranes pale. No evidence of syphilis. Thyroid not enlarged. Eyes negative as also ears. Taste and smell normal. Chest somewhat flattened. Lungs and heart normal. Digestive organs normal.

Patient says he is not as strong as formerly. No vertigo except after seizures. Seldom has a headache. Following some attacks he has a pain on right side just below costal border. Tactile sense, slow in responding, seems diminished in all parts of body. Pain sense diminished. Temperature sense normal. Stereognostic sense poor. Reflexes negative. No evidence of any paralysis. Co-ordination and muscular sense normal. Has frequent jerkings of upper extremities, chiefly on right side, sometimes head jerked also. These movements occur frequently and are apparently more frequent when he closes his eyes. Slight tremor of tongue. Mental state fair.

November 1, 1905—Flexor muscles of leg are most often affected in convulsive movements. At times they are almost continuous changing from one group to another. No loss of consciousness. At times has great trouble in dressing and walking.

July 1, 1906—Continues to have much jerkings of his muscles, chiefly in arms, legs, head and lips. More on right than on left. They are worse in early morning, at which time he is quite helpless.

October 1, 1906—Movements are present much of the time, although some days he is entirely free. They are absent during sleep. All muscles seem involved with no distinct order of invasion. Sometimes upper extremities and sometimes lower. At times neck muscles. Always worse in morning.

January, 1907—Pale and anemic.

February 16, 1907—Myoclonic movements continue. At frequent intervals, especially in morning, although at times they are present during entire day. Recently received a severe contusion of tissues about left eye by falling during a seizure. Myoclonic movements involve trunk extremities and facial muscles. To-day, directly following a grand mal seizure, he had loss of plantar, patellar, wrist and elbow reflexes. Wets bed every night. Ordered strontium bromide grs. x, sodium bromide grs. v, chloretone grs. iii, t.i.d.

February 27, 1907—Is in a semi-stuporous state. Occasional myoclonic movements on right side. Chloretone stopped.

March 13, 1907—Cold, rainy day. Myoclonic movements are very marked. (They were practically absent for past two weeks.) They are general and involve principally the flexors. Muscles of abdominal wall markedly involved. No nystagmus. Pupils widely dilated and react but slightly to light. Some spasm of eyelids but not much of facial muscles. No impairment of consciousness. Reflexes not exaggerated, except plantar which is toe flexion in type. No ankle clonus. Flexors of toes are first involved, then foot, then entire extremity on each side. No apparent involvement of muscles of back. Movements increased when talking.

March 15, 1907—Marked myoclonic movements during early morning and up to 10 a.m., when he had a grand mal seizure which was followed by marked perspiration and complete cessation of myoclonic spasms for several hours, after which they reappeared and were very severe, but again ceased when patient was given chloral hydrate grs. x, potass. bromide grs. xv. Developed difficulty in swallowing and seemed to have a spasm of laryngeal muscles producing difficulty in respiratory act. Occasional loud grunt.

March 16, 1907—Abdomen is now negative. Patient had no myoclonic movements for several hours after yesterday's grand mal seizure. They returned late in afternoon and became more severe.

He developed difficulty in swallowing and seemed to have inspiratory difficulty. Auscultation over chest, negative. Grunt

heard occasionally as though respiratory muscles were involved in myoclonic movements.

March 28, 1907—Pupils well dilated. Wrist jerks exaggerated on both sides. No difficulty in swallowing fluids. Myoclonic movements involving sterno-cleido-mastoid, trapezius, deltoid and flexors of forearm and hand on both sides but not synchronously. Spasm of both eyelids is synchronous and bilateral.

No exaggeration of elbow jerks or knee jerks. No ankle clonus or Babinski reflex.

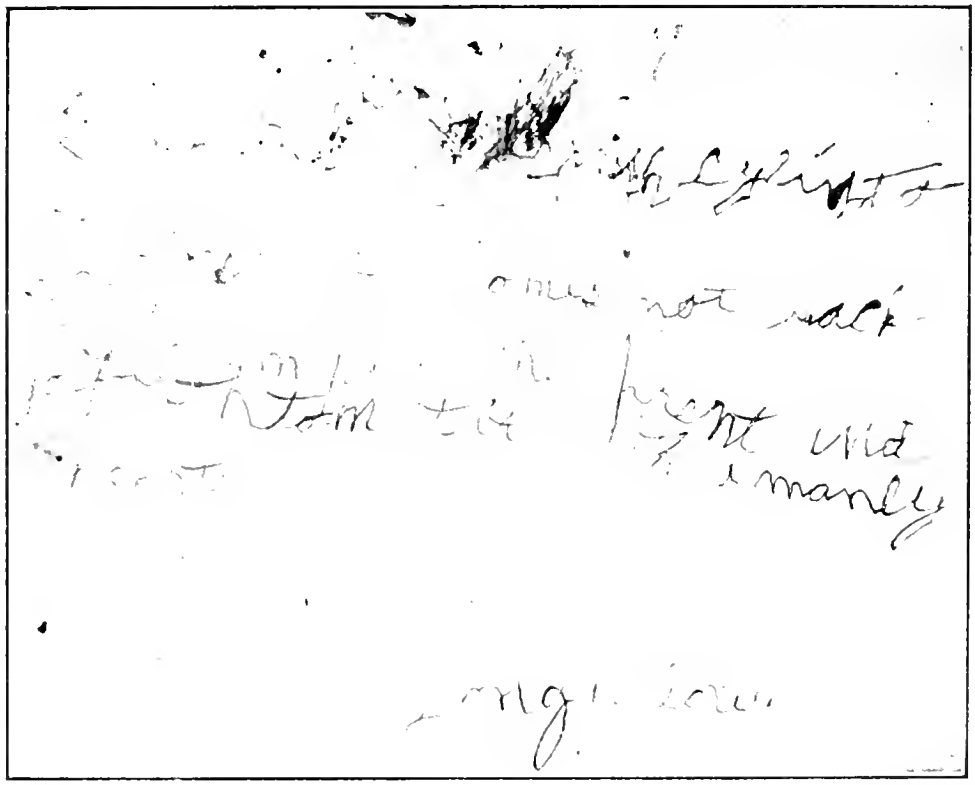

Fig. 2. Sample of writing of E. J. W., showing inability to control myoclonic movements.

Some borborygmus. No hippus.

March 29, 1907—Marked myoclonic movements to-day which involve muscles of back, abdominal wall, occipito-frontalis, right face, eyelids, flexors of lower extremities and all muscles of upper extremities. Right upper extremity affected much more than is left, arm being raised from side. Head turned toward left but not held rigidly.

Occasional grunt accompanies severe myoclonic movements. Facial muscles are not as markedly involved as are muscles of right arm. Some difficulty in speaking. Reflexes not exaggerated. Swallows without trouble.

Tapping muscles, excitement, etc., increases movements. Pupils normal.

General convulsion occurred about 10 a.m. Head turned markedly to left, pupils widely dilated, no cry before, but did occur after seizure (at time of cry patient appeared to have an atypical seizure as pupils dilated and there were some clonic movements of upper part of body). All parts of body involved. Saliva blown from mouth. Slight stertor and very brief period of cyanosis. Reflexes, tested immediately after cessation of clonic movements, were absent except for slight plantar flexion.

Flatus passed but no fecal matter or urine. Pupils returned to usual size directly after convulsive movements ceased. Moderate increase in pulse rate. Two minutes later reflexes were active but not exaggerated.

April 3, 1907—Myoclonic movements marked since 6 a.m. Mild seizure at 7.40 a.m. Thirty minutes after taking amylene hydrate by mouth all movements ceased and patient was able to sit up in bed and read.

April 5—Jerkings slight until 5 p.m., when they became very marked and he had a grand mal seizure at 5:15 p.m., after which he perspired very freely for several hours and was very restless. Myoclonic movements returned at 7 p.m.

April 6—Myoclonic movements continued very severe all night so that patient could not sleep.

April 9—Dark, rainy day. Marked myoclonic movements involving especially flexors of extremities both distal and proximal positions. Increased cremasteric reflexes. Diaphragmatic grunt. Slight lateral movement of eyeballs in some movements. Back muscles involved markedly also. Fibrillary twitching observed in posterior aspect of thighs. Consciousness unimpaired.

Seizures, both grand mal and abortive or atypical types, vary in frequency. Grand mal occurring from two to twenty-two times per month, the abortive form more frequently. Electrical reaction of muscles normal. At no time has there been an increase of indican in the urine.

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Excellent biographies are appended to articles of Clark, Hunt, Dana and Russell.

Society Proceedings

NEW YORK NEUROLOGICAL SOCIETY.

Dec. 4, 1906.

The President, Dr. Joseph Fraenkel, in the Chair.

A CASE OF SYRINGOMYELIA.

By Dr. William B. Noyes.

The patient was a young man of twenty-three, a straw-maker by occupation, whose mother died of some rheumatic complaint, and his father of some obscure complication of medical conditions. There was no history of syphilis.

Four years ago the patient first noticed a heavy feeling in the arms and legs, accompanied by weakness, which gradually became more marked. About two years ago he also began to develop pain in his legs. When Dr. Noyes first saw him, five months ago, an examination revealed marked atrophy of certain muscles of the upper extremities, notably the supraspinatus and trapezius. The patient complained of very severe pain along the course of the spine and around the abdomen, which has been quite constant. There were certain well marked areas of anesthesia and analgesia and of thermal anesthesia over the back, chest and upper extremities, although these areas of loss of sensibility to pain and to heat and cold did not entirely coincide with each other. In certain areas over the back and arms he could not distinguish between heat and cold. Fibrillary twitchings were well marked over the atrophied muscles. The knee jerks were increased. There was ankle clonus. There were no symptoms referable to the special senses; no bulbar symptoms. Babinski reflex was present. Arm reflexes were absent. He had an unexhaustible ankle clonus on the left side. His abdominal reflexes were normal. His eyes were normal, and there was no symptom of disease of any of the cranial nerves.

Dr. Noyes said he regarded the case as one of syringomyelia, although there was a possibility that it was one of meningeal tumor pressing on the nerve roots. The only reason for even suggesting any other diagnosis than syringomyelia is the uncertainty of the true value of failure to distinguish between heat and cold, where anesthesia and analgesia are present over the same areas. In this case there were undoubted areas demonstrated before the Society where thermo-anesthesia alone was present.

Dr. J. Arthur Booth said that from the symptoms presented by the patient shown by Dr. Noyes he thought there was no question of the correctness of the diagnosis of syringomyelia. The symptoms were those of an anterior and posterior poliomyelitis from a gliosis of the cord in the cervico-dorsal segment. The patient also shows a difficulty in walking, which had not been noted before.

A CASE OF DEMENTIA PARALYTICA IN A BOY 15 YEARS OLD.

Dr. M. S. Gregory.

This patient had been referred to the psychopathic ward of Bellevue Hospital for examination from the New York Juvenile Asylum with the statement that he was developing imbecility. The boy's present, as well as his antecedent history, had been furnished by his mother, who was a well developed, well nourished woman, about thirty-eight years of age. She was somewhat emotional, and readily became suspicious and irritable without provocation.

According to her statement, the family history had been negative, with the exception of one brother, a maternal uncle of the patient, who had been an inmate of the Manhattan State Hospital for the past six years, suffering from a mental disease of a paranoid type, with prominent religious ideas and hallucinations of hearing. The patient's father had been addicted to the use of liquor for many years; he had kept late hours, and, it was believed, dissipated and associated with women of low character. It was not known whether or not he had had syphilis. The mother had had ten children in all—nine by the patient's father, and one by her second husband. She had had two full term still-births, one between the second and third child, and one between the fourth and fifth child; the latter was said to have had a "water-head."

The patient was born on July 17, 1891. He was the seventh child, and two healthy children had been born since his birth. He was a healthy and well-developed infant, and the delivery was a normal one. The mother denied his having snuffles, coryza, skin lesions, etc. He walked at fifteen months, learned to talk at the usual age, and as a child was healthy and well. He entered school at the age of seven, and left at thirteen, making fairly good progress in his studies, and acquiring an average knowledge of the elementary branches. He was not considered backward in his class. At seven years of age, while at play, he was struck on the center of the forehead, and was unconscious for two hours following the injury. He had a small scalp wound, which healed promptly. He suffered from no headaches nor inconvenience after the injury.

At thirteen he left school and entered a box factory, where he worked for six months, giving satisfaction, as far as was known. He afterwards became a messenger boy in a theatrical agent's office, where he remained for four months. Later, he was employed in a bookbinding establishment, where he remained until the time of his commitment to the New York Juvenile Asylum, May 9, 1906, at the age of fifteen. During the time that he remained at home, his mother was certain that he showed no peculiar traits, being fairly obedient and industrious until three or four months prior to his commitment. Between the ages of thirteen and fifteen years he smoked cigarettes to excess, averaging from two to three boxes of Turkish cigarettes a day, and inhaling them. About April 1 he became unruly, indolent and frequently failed to come home at night. He also stayed away over Sundays, and did not give his earnings to his mother as he had previously done. He admitted having spent the money for cigarettes. About this time his step-father precipitated a quarrel which resulted in the commitment of the boy to the New York Juvenile Asylum for disorderly children.

At the asylum he was employed mostly out of doors, weeding the garden, mowing the grass, etc.; he did his work fairly well, but seemed to take little interest in it. About Aug. 1, 1906, while working in the

garden, he had a "sunstroke," followed by a convulsion and a stupor lasting twenty-four hours, and on the following day it was observed for the first time that he had some difficulty in speech. He "stuttered," hesitated in talking, and became irritable when he could not express himself. During the following month it was noticed by the attendant that he was unsteady on his feet and was unable, on account of his shaking, to do the work he had previously done. Finally, it was noticed by his mother that he showed marked tremor in writing home, and talked with much difficulty when she visited him.

On admission to the psychopathic ward on Oct. 16, 1906, a physical examination showed a well-developed and well-nourished boy; complexion fair; features regular; slight tendency to continual arching of the right brow, but no loss of power in the facial muscles; bridge of nose broad; teeth show some irregular ridging, but are not of the Hutchinson type; no glandular enlargement. The skin showed a peculiar condition; it was smooth and waxy in appearance, and there was an absence of hair over the entire body surface. The left pupil was slightly larger than the right, and both reacted slightly, if at all, to light. Both reacted to accommodation. The movements of the eyes were apparently normal. The patient complained of imperfect vision, but rough tests showed little impairment. There was no hemianopsia, and an examination made by Dr. Reese, of the Cornell University, showed the eye grounds to be normal. The elbow, wrist and knee jerks were slightly exaggerated, but equally on both sides; front cap and Achilles reflexes were present and lively. The superficial reflexes were about normal; no Babinski. There was no distinct impairment of power in any muscle group, so far as could be determined. The right forehead was more deeply corrugated than the left, but both sides were wrinkled equally well on examination.

There was some fumbling when attempts were made to touch the nose with the finger-tips with the eyes closed. There was pronounced lateral tremor of the extended fingers, and a fine tremor of the tongue, which was extended in a jerky fashion. Marked flicker of the muscles about the mouth was present on showing the teeth. Speech showed a well marked defect, and enunciation and articulation were indistinct and ataxic. Heart and lungs normal. An examination of the spinal fluid made by Dr. Hastings, of Cornell University, showed marked lymphocytosis.

Mental examination: The patient was quiet, composed and well behaved. His mood, as a rule, was mildly exhilerated, with occasional quick changes to irritability on insufficient grounds. At such times, however, he could be easily pacified. He felt contented and happy in a childish way, showing very little appreciation of his condition and situation. He had no expansive ideas nor well defined delusions. Memory showed slight impairment, and judgment was quite defective.

This case was presented, Dr. Gregory said, as one of interest mainly because of the age of the patient, he being the youngest of about 1,500 cases of dementia paralytica that had been admitted to the psychopathic wards of Bellevue Hospital during the past five years. The next youngest case was that of a girl of seventeen who had contracted syphilis when thirteen, and the third youngest a man of twenty-one whose father and mother were both syphilitic. The former had dementia paralytica and the latter tabes. Another point of interest was that, although very

young, the patient presented fairly typical somatic as well as mental signs of dementia paralytica.

In the consideration of the etiological factors in this case, the most stricking feature, perhaps, was the absence of the positive history of syphilis. However, the dissipated life of the father, as given in the history; the peculiar condition of the skin, which had been suggested by Dr. Collins as being a form of syphilitic cachexia, together with the presence of marked lymphocytosis in the cerebrospinal fluid might suggest the existence of specific infection. It was also worthy of consideration what part the head injury and cigarette smoking might have had, if any, in the causation and development of the disease.

Dr. Edward D. Fisher said the case was a good illustration of the type of general paresis in which the patient is in a fatuous, contented, happy condition; it was not an example of the expansive type.

Dr. L. Pierce Clark said that some four or five years ago, Dr. Mott, of Claybury, England, collected all cases of juvenile paresis on record, and the number at that time amounted to about 97. He considered it rather a rare type, and called attention to the fact that these cases commonly developed soon after puberty. The case shown by Dr. Gregory, Dr. Clark said, could not be counted as an instance of a particularly early case of the juvenile type. There seems to be no doubt but that the case is one of juvenile paresis and not an early development of the ordinary adult paresis, as Dr. Gregory appears to imply.

Dr. Charles L. Dana inquired if the boy had not some bad sexual habits, and he suggested the possibility of acquired syphilis.

Dr. Gregory said that they had found no positive evidences of acquired syphilis. Sexually, the patient was not as well developed as a boy of his age should be.

The President, Dr. Fraenkel, said that Dr. Sachs and himself have seen what was perhaps the youngest case of dementia paralytica on record. The patient was a girl of ten years, whose mother came to the clinic complaining of tabetic pains. At that time it was noticed that her little girl, who was then attending school, presented the same somatic picture as the mother; *i. e.*, unequal, stiff pupils and exaggerated reflexes; and six months later the child was admitted to the Montefiore Home with well marked symptoms of general paresis, which rapidly progressed to a fatal issue. The post-mortem examination corroborated the diagnosis. Patient was ten years old when she was admitted to the home.

Dr. Gregory said that he had seen a case of dementia paralytica in a child of seven years at Dr. Kraepelin's clinic in Munich last summer. That case, however, was of a different type from this boy, who presented many of the symptoms present in the adult type of the disease, which perhaps was due to the fact that his age closely approached the limit of the juvenile period. One of the interesting features of the case to which he had already called attention was the peculiar waxy condition of the skin, and the entire absence of hair on the body. He was uncertain as to whether this was indicative of syphilitic disease.

Dr. Fraenkel said that this type of velvety skin was usually looked upon as a form of trophic disturbance of the skin, and was not infrequently observed in tabes, either in a localized or generalized form. It was regarded as a tabetic or post-syphilitic dystrophy of the skin.

Dr. William M. Leszynsky said he did not think the absence of hair was an unusual manifestation of syphilis. He recently saw a man of

fifty who was in the tertiary stage of syphilis, and whose body, with the exception of his head, was entirely denuded of hair.

SARCOMA OF THE SPINE; PROBABLY EXTRADURAL.

By Dr. I. Abrahamson.

The patient was a male, thirty years old; married; a native of Russia and a painter by occupation. His family history was negative. The patient had pneumonia ten years ago. He denied venereal disease; there was no history of lead intoxication; he used alcohol and tobacco in moderation.

Seven years ago a mass was noticed in the spine, in the sacro-lumbar region. It had increased in size during the past two years. His present illness dated back two years, and was assigned to a fall from a scaffold, striking on his right shoulder and head. Four weeks after the injury he complained of a pain in the right lower ribs, posteriorly, burning in character; this was followed a week later by pain in the left lumbar region, in the axillary line; pains then occurred in the ball of the left foot, and soon afterwards in the ball of the right foot. He began to suffer from constipation and increasing difficulty in urination; *i. e.*, delay and interrupted flow; then a girdle sensation around the abdomen below the umbilicus, at first on the left side; at about the same time there was weakness and stiffness in the left lower extremity, together with numbness and a "dead" feeling over the same extremity; there was also diminished potency. He complained of no symptoms above the waist line excepting occasional dizzy spells, with blurred vision simulating diplopia. About this time the mass on the back began to increase in size, and the patient ascribed his symptoms to it. He remained in the hospital three months, and during that period his symptoms improved somewhat, his pain being less severe.

Examination showed the head, chest and upper extremities normal. There was no lead line on the gums. The pupils and reflexes of the upper extremities were normal. The abdominal reflexes were present, but the left lower abdominal reflex was much diminished. There was no clonus. The patient dragged the left lower extremity somewhat, and the foot was turned outward. Motor power was somewhat diminished. Tactile sensibility was normal. There was a belt of hyperalgesia on the right side from the umbilicus to the ribs anteriorly, and from the buttocks to the ribs posteriorly. There was hypoalgesia on the right side from the umbilicus to the groin, and sensibility was still more diminished over the right thigh, especially its outer surface. There was analgesia over the right upper outer leg and foot. On the left side there was hyperalgesia from the umbilicus to the groin. There was tenderness to percussion over the ninth dorsal spine, and upon moving the upper body from side to side the girdle sensation was increased. The electrical reactions were normal.

The mass over the spine was removed, and a microscopical examination showed it to be a spindle and giant celled sarcoma. The patient was given hypodermic injections of salicylate of mercury and increasing doses of iodide of potash, with very little improvement.

A study of this case, Dr. Abrahamson said, indicated the presence of a lesion of the spine involving from the tenth to the twelfth dorsal segments, mainly on the left side, probably posterior, and of extra-medullary nature. The likelihood was that the lesion was either extra-dural, and

large and flat, or else of the dura itself. It was sarcomatous. The case was of particular interest on account of the marked sensory symptoms, the Brown-Séquardian tendency and the minimum motor symptoms.

Dr. B. Sachs said that in view of the anatomical and pathological findings in this case, there could be very little doubt about the nature of the condition. He thought Dr. Abrahamson was right in suspecting a rather diffuse sarcomatosis rather than a small, localized tumor, on account of the extensive character of the motor and sensory involvement.

Dr. Sachs said there were a number of cases of spinal tumor on record in which pain was the most pronounced and for weeks and months the only symptom. The speaker said he was particularly interested in this subject, as he had within the past few years seen a number of cases of spinal cord neoplasm secondary to malignant disease elsewhere in the body. Within the past few months he saw two cases of carcinomatosis of the spinal cord which developed in a remarkably short period of time after radical operations for mammary carcinoma. In one instance, the spinal involvement occurred eight weeks, and in the other four months after the removal of a cancer of the breast.

Dr. Adolf Meyer thought it was questionable that a diffuse sarcomatosis, as the term is understood by Redlich and others, could produce such well-marked sensory disorders of so one-sided a character. Of course, it was probable that we had to deal with a tumor of fairly good size, but he could scarcely coincide with the view that the condition was a diffuse sarcomatosis.

Dr. B. Sachs said that by the term diffuse sarcomatosis he meant one or more flat tumors, and not a single compact one. A growth of the latter kind in the limited space of the spinal canal would doubtless by this time have given rise to very marked paralytic disturbances and of a more localized character than were shown in the case reported by Dr. Abrahamson. From that point of view the speaker said he would argue that the tumor was widespread in character and not limited, say, to one segment of the cord.

Dr. Edward D. Fisher said the symptoms in Dr. Abrahamson's case were rather characteristic of pressure on the spinal nerves, rather than on the cord itself. He did not see how such unilateral motor and sensory symptoms could be caused by pressure on the cord.

Dr. L. Pierce Clark asked Dr. Abrahamson how frequently tumors outside of the cord and extradural growths that had produced the Brown-Séquard syndrome were met with in literature. He understood that it was an extremely rare occurrence.

Dr. William M. Leszkynsky said that if the growth in this case was extradural, there might be some chance for its successful removal.

Dr. Charles L. Dana said that the immediate outlook for surgical intervention in extradural tumors of the cord was very hopeful. Quite recently he had had a growth removed from almost the exact region of that in the case reported by Dr. Abrahamson, and the result was very successful.

Dr. Abrahamson, in closing the discussion, said that as a rule, the extradural conditions did not give rise to the Brown-Séquard syndrome. The case he had shown was also particularly interesting on account of the comparatively little motor and the very marked sensory symptoms. The objective sensory symptoms were far in excess of the motor, and

indicated that the lesion was essentially one-sided, that it was most probably extradural, and rather diffuse.

A CASE FOR DIAGNOSIS; POSSIBLY SYRINGOMYELIA.

By Dr. Edwin G. Zabriskie.

The patient was a boy of nineteen, a native of Silesia, and a baker by occupation. No satisfactory family history was obtainable, but he had apparently enjoyed good health up to the time of his present illness. He used tobacco and beer moderately. About a year ago he fell on the sidewalk, striking his left elbow and causing a fracture of the olecranon. Shortly afterwards, his left hand became emaciated and weak, beginning in the middle finger and extending across the hand to the thumb, the condition taking about two months to develop. Since then the hand had been growing progressively weaker, and a month ago the right hand became similarly affected. The electrical reactions showed complete loss of response to both galvanic and faradic stimulation. The reflexes were present in the biceps and triceps, but the wrist reflex could not be elicited unless the muscle was struck directly. The knee jerks were unequal, the left being greater than the right. There was a well-defined Babinski. The patient had a pronounced scoliosis. There was decided analgesia and thermo-anesthesia in a well-defined area about the elbows, and the temperature sense over the rest of the body was also impaired. There was no vesical nor rectal disturbance. The speaker said he was inclined to regard the case as one of syringomyelia.

Dr. L. Pierce Clark said he thought the case was undoubtedly one of syringomyelia. He thought the speaker was trying to involve the peripheral trauma as a cause of the syringomyelia, which he considered unlikely to be the case, although not a few neurologists held that view.

Dr. Fraenkel said that some years ago he saw a patient with marked chronic degenerative disease of the cord. The symptoms were reported to have come on about three weeks after an accident. The speaker was at that time already able to make a diagnosis of amyotrophic lateral sclerosis. Subsequently, the case came to court, the patient claiming that his disease was the result of the injury he had sustained. Dr. Fraenkel said that he did not coincide with that view, and testified to that effect, but other physicians testified to the contrary, and the jury took their view of the case.

Dr. Noyes said that last spring he showed a case of amyotrophic lateral sclerosis in which the symptoms developed two months after the patient had sustained an electric shock from a third rail, which severely burned his hand. There was a possibility, however, that the symptoms might have been the result of lead poisoning, though no symptoms of lead poisoning were obtained, and the patient gave an indefinite history of weakness of the muscles supplied by the posterior interosseous nerve prior to the accident. There was a scar of an old stab wound inflicted many years before directly over the nerve, and there had been no increase of the paralysis before the electric shock. The symptoms had grown progressively worse quite rapidly, the muscles of the legs and neck now being affected, together with some symptoms of bulbar paralysis. Dr. Noyes said he was still inclined to believe that the electric shock was the cause of the symptoms in this case.

Dr. Zabriskie, in closing, said that while he was inclined to look upon

the case as one of syringomyelia, the relation of the symptoms to the accident was very significant. He had found several cases on record where a very definite connection was traced between an injury and the onset of syringomyelia.

REMARKS ON MYOCLONUS EPILEPSY, WITH REPORT OF A CASE.

By Dr. L. Pierce Clark.

The case reported was of the family type, and was briefly as follows: The patient was a woman, fifty-two years old, single, who was admitted to the Craig Colony for Epileptics on Sept. 30, 1904. Her paternal grandmother had always suffered from headaches. A paternal uncle had three children, one boy and two girls, all of whom suffered from myoclonus-epilepsy similar to that of this patient. The disease was known in the family as St. Vitus' dance and fits. Whether the epilepsy or myoclonus developed first in the cousins was not certain. The eldest of the myoclonus-epileptic cousins (female) married and had one child, who lived to an adult age and showed no signs of the disease or other nervous affection. The mother died of an unknown disease at fifty years of age. The two male myoclonus-epileptic cousins died of unknown diseases at the ages of twenty-five and thirty, respectively. A maternal granduncle was insane and had several children who were insane; one grandson (second cousin to the patient) died of paresis. Several maternal relatives had tuberculosis. The father and mother of our patient were first cousins. Both suffered from chronic rheumatism. The father was still living and was a fairly healthy man of seventy-five years.

The patient was a strong child. She had an ordinary attack of typhoid fever at ten years of age. She began school at five years of age and made the average progress of her grades until thirteen years of age, when she had a typical grand mal epileptic fit at night, without obvious cause. The myoclonus, which either immediately preceded or followed the fit, began in the arms first. Although it was more or less constant after its inception, its paroxysmal intensity at stated periods was certain. On the bad days, the myoclonus often interfered with her piano practice. It was interesting to note that the patient saw her myoclonic cousins a few months prior to the onset of her own disease, and she was much affected by the sight. The fits were rather severe in her fourteenth year, and on one occasion she had seven severe fits in one night. From fifteen to forty-two years of age the attacks were less frequent, averaging one a month. She was now having eight attacks a year; four grand mal and four petit mal, equally divided between day and night attacks. An aura of a cramp-like sensation in the right wrist was invariably present formerly, but it was now absent. For several years after the onset of her association disease she had increasing vertigo and myoclonic movements for two or three weeks before the genuine fits occurred, after which she was perfectly free from myoclonus for two or three days. The myoclonus never interfered with nor did it occur during sleep. Bromide and chloral, particularly the latter, improved her condition considerably during the thirteen years of their use, from the onset of the disease. She was now a Christian Scientist, and had taken no medicine for several years. Coffee had some quieting effect on the myoclonic movements. The patient had nocturnal enuresis until she was thirty-one years of age; this stopped with-

out treatment. Involuntary urination never occurred during the myoclonic or epileptic attacks.

Dr. Clark said that the epileptic attacks in this case were so classic in detail that they did not deserve mention. They had produced little or no mental enfeeblement. Some myoclonus was now present every day. The movements ranged in degree from slight fibrillary twitchings in individual muscles to that of universal involvement of all voluntary muscles. Extremes of heat and cold, mental stress and emotion excited the myoclonus. At first, the myoclonus often interfered with swallowing, and occasionally now, at the height of paroxysmal myoclonic days, foods and fluids were forcibly ejected at meals. She had broken her nose and received many disfigurements about the face due to her myoclonic attacks. At the present time the myoclonic movements might be observed to rapidly succeed each other in a series of brisk, shock-like muscular contractions typical of myoclonus. All the muscles of the trunk and extremities were involved in this play of spasm movement, bewildering to describe. The large proximal muscles of the extremities were most involved, together with those of the trunk proper. The movements of the muscles of the two sides were a little asynchronous, the body and head being hurled to the right frequently at the end of the complex movement. In the extreme paroxysm all the muscles of the face were markedly affected, and the diaphragm and respiratory muscles were commonly involved. At times, when standing, she was thrown from her feet by the shock-like contractions; if she remained in bed she was almost hurled from it by the violent contractions. There was never loss of consciousness during any myoclonic movements. Speech was commonly interrupted thereby by the violence of respiratory involvement, but continuity of thought was never broken. The great flexors of the trunk were most involved, and the contractions of the rectus abdominis were often painful. For several days following these severe periods of myoclonus a feeling of general exhaustion and muscle soreness existed. The nervous system showed no other abnormality, nor was there any hysterical stigma.

Dr. Charles L. Dana said that Dr. Clark was so familiar with the subject of myoclonic epilepsy, and had presented it so fully, that he hesitated to make any criticism or offer a different point of view. The speaker said that from an observation of several cases that had come under his care he was inclined to regard the myoclonic manifestations as a serious, progressive motor disorder, finally involving the whole body, so that for protracted periods many of the muscles were in a state of almost continual tonic contraction. In addition to the twitching of the muscles, there were, at the same time, certain irregular movements which were entirely similar to those observed in chorea. That fact had led him to look upon the disease as a degenerative myoclonus, and one that was closely related to the degenerative tics. When associated with epilepsy, the latter might be regarded as a terminal condition or an accidental occurrence, or the combination perhaps represented those forms which had a particularly serious family character. At all events, he was inclined to recognize a form of degenerative myoclonus which was distinct from the epileptic type.

Twenty years ago, Dr. Dana said, he reported a case of this character under the title of choreic tics. The term myoclonus epilepsy was then unknown. The onset of the disease in that case was very similar to that observed in Sydenham's chorea, the movements first being limited to one arm and gradually involving the entire body. There were also intercurrent

epileptic attacks, which were regarded as accidental. The patient died, and an autopsy showed changes in the cortical cells.

Dr. C. E. Atwood said he had seen two cases of myoclonus associated with epilepsy, and one of myoclonus without epilepsy. The latter patient was an inmate of a hospital in London, and was regarded by the physicians there as a case of multiple tic. The movements which involved the muscles of the trunk and extremities were violent and lightning-like in character, and the case corresponded very closely to the disease that had since then been so well described by Dr. Clark under the name of myoclonus.

Dr. Clark, in closing, said that in the near future he expected to show some biographic pictures which were taken of a well-marked case of myoclonus epilepsy, and these, he felt assured, would clearly differentiate this type of case from the condition known as multiple tic. The character of the movements in the two conditions was entirely distinct.

PHILADELPHIA NEUROLOGICAL SOCIETY.

December 21, 1906.

The President, Dr. D. J. McCarthy, in the Chair.

A CASE OF INCOMPLETE BROWN SEQUARD PARALYSIS.

By Dr. G. E. Price.

J. G., Italian laborer, aged 46, from the Philadelphia Hospital.

Eight years ago he had been stabbed in the neck by a fellow-countryman, the wound being followed by immediate loss of power in the left arm and leg, the right side of the body being unaffected. Movement of the shoulder was regained within a few days, and some motion of the fingers in a month. He was in bed 17 days and could walk in about one year from the time of the injury. Upon examination a linear scar three-quarters of an inch in length is seen, almost horizontal, and directly across the median line of the neck between the spinus processes of the sixth and seventh cervical vertebræ. The pupils are unequal, the right being the larger; both react to light and accommodation; there are no external ocular palsies. The patient has no motor or sensory involvement of the face, and no difficulty in swallowing; the tongue is protruded in the median line. His gait shows a stiffness and dragging of the left leg, the station is normal.

Of the upper extremities, the movements and reflexes of the right arm are normal, the left arm presents no weakness of the muscles about the shoulder, of the biceps or triceps, but the grip is distinctly weak. All reflexes of the left arm are plus; there is no inco-ordination of either extremity.

The trunk presents no signs of muscular weakness, the cremasteric and abdominal reflexes are preserved on both sides, the sphincters are unaffected.

In the lower extremities there is no muscular weakness or rigidity of the right leg, the left leg is distinctly weak and stiff. The knee-jerks and Achilles jerks are increased on both sides, especially on the left, ankle clonus is absent on the right side and present on the left; there is no Babinski or Gordon reflex on either side and no inco-ordination.

Sensation: Tactile sense, pressure sense and muscle sense are pre-

served over the entire body. Pain and temperature senses are preserved upon the left side, but lost over the entire right lower extremity and over the trunk as high as the costal margin anteriorly and two finger's breadths below the angle of the scapula posteriorly. The loss is distinctly defined at the median line of the body and includes the right half of the penis, and the right testicle. There is no astereognosis.

The patient also presents symptoms of a sharply defined localized sweating on the left half of the trunk, extending to a little below the hip and including the left shoulder, arm, forearm, and hand, though much less marked in the forearm and hand than in the other parts affected.

There is no evidence of muscular wasting in any part of the body.

A skiagraph taken by Dr. Kassabian revealed no abnormality of the bony structure.

Dr. F. X. Dercum asked whether the pupil on the side of the lesion was smaller. Dr. Price replied that it was.

Dr. Dercum said that it was rather an interesting fact that the patient's myosis was present on the side of the lesion or wound. Of course, the symptom itself could be explained only in a speculative way. Perhaps as has been attempted in the myosis of tabes, lesion of the posterior columns interferes with impressions coming from the general body surface which have a dilator action on the pupils. The absence of such dilator stimuli would, of course, result in a small or myotic pupil on the same side as the lesion in the cord.

Dr. Alfred Gordon thought the case very interesting. It reminded us clearly of Brown-Séquard's experiment when he produced line sections of the spinal cord and produced symptoms almost identical with those of the present case, excepting the areas of anesthesias and hyperesthesias which are usually present above the level of sensory disturbances. In hearing Dr. Price recite the case, some thoughts of medico-legal nature suggested themselves to Dr. Gordon. For instance, in cases of a stab wound in the back, the patient may present a paralysis of one side and sensory disturbances on the other. Acute disturbances of the function of the cord are usually looked upon very seriously. The great improvement the patient presents with an evident lesion of the cord, points to the great practical importance of the fact that patients of this character, in spite of distinct cord lesion might improve considerably.

A CASE PRESENTING SYMPTOMS OF CEREBRAL TUMOR, WITH RECOVERY.

By Dr. A. A. Eshner.

The patient was a man, 30 years old, suffering from vomiting, anorexia, bad taste, headache and vertigo. Gait and station were ataxic, and the man was unable to attend to his business. Pulse and temperature were normal. The man was a large eater, but he had not indulged excessively in alcohol or tobacco, and he denied venereal infection. Under observation, weakness of the extremities on the left side of the body developed, with increase in the reflexes. There was no change in the eyegrounds. Under treatment with increasing doses of iodid and mercurials, improvement gradually took place, leaving only a slight degree of weakness on the left side, not sufficient, however, to interfere with the pursuit of the man's usual avocation. The early symptoms in this case, namely: vomiting, headache, vertigo and ataxia, suggested the possibility of a new-growth involving the cerebellum. The subsequent development

of the left hemiparesis, with increase in the reflexes on the affected side, bespoke invasion of the motor tract on the right side. The absence of convulsions, of perturbation of consciousness, of sensory disturbance, of astereognosis, of mental derangement, pointed to freedom of the cerebral cortex, while the absence of changes in the eyegrounds and of palsy of ocular muscles and the escape of other cranial nerves indicated that the lesion must have been one of small dimensions. The afebrile course of the illness seemed to exclude an inflammatory process. The results of treatment raised the question of a possible syphilitic infection, while drowsiness pointed in the direction of meningitis or arterial disease.

A CASE OF HYSTERIA PRESENTING SYMPTOMS OF CEREBELLAR DISEASE.

By Dr. A. A. Eshner.

The patient was a tailor, 42 years old, who had difficulty in walking after the removal of several teeth, in conjunction also with fright from having remained alone for a short time in a house in which a close friend had died. The man walked like a drunken person, with a tendency to fall to the right. The right chest felt as if grasped in a vise, and there was pain on the right side of the head. There was tinnitus in the right ear, with impairment of hearing. Sensibility was less acute on the entire right side of the body than on the left. The visual apparatus was normal, and there was no lesion of the fundus. There was no sign of inflammatory disturbances in either ear. The muscular apparatus was normal. A laxative containing aloin, cascara, and asafetida was prescribed and hypnotic suggestion was practised, and recovery ensued in the course of three weeks. The disorder of gait, in conjunction with the feeling of unilateral weakness, suggested the existence of cerebellar disease, and the tinnitus and impaired hearing an aural origin. The suspicion of hysteria was strengthened by the absence of organic disease and it was established by the results of treatment.

Dr. W. G. Spiller said that the absence of early changes in the eyegrounds in the case with cerebellar symptoms should make one very cautious in diagnosticating tumor of the cerebellum. Among the earliest signs of cerebellar tumor is choked disc. Tumor in the motor region may exist a long time without any change in the eyegrounds, but a tumor in the basal ganglia, the pons, or the cerebellum is likely to cause early choked discs. Dr. Spiller stated that he had had two cases within the past few years which simulated tumor of the brain very closely. He was called to see a young woman about three years ago who had intense ataxia, violent headache, vertigo and other symptoms of brain disease. The symptom-complex, after careful study, seemed like hysteria, therefore she was placed in a private room at the University Hospital with a trained nurse, and under treatment by suggestion all the very grave symptoms of brain tumor disappeared in a few weeks, and the woman became perfectly well. The notes of this case had been given to Dr. Weisenburg and had been reported by him.

Last summer Dr. Spiller had a patient brought to him from a distance who was said to have symptoms of brain tumor, viz., optic neuritis, convulsions on the right side, weakness on the right side, and complete blindness in the left eye, and sensory disturbances which aroused his suspicions of hysteria. She had the history of having been struck on the left side of the head by a piece of iron which had fallen some distance. The

physician who brought her stated he had operated and found the bone of the skull exceedingly thick, he had cut through the dura and the parts seemed to him abnormal. It was possibly a case of hysteria added to organic disease. Dr. A. C. Wood operated and found the bone exceedingly hard and three-fourths of an inch thick over the parietal region.

Dr. Shumway had examined the eyes previously and had found no evidence of neuritis. Immediately after the operation the blindness of the left eye and hemianesthesia disappeared, and the patient improved rapidly and got almost entirely well. The brain was found protruding very slightly through the dura. Extreme care must be taken in diagnosticating such cases. Dr. Spiller said he had been called to a case within the past few days in which it was almost impossible to decide how much was hysteria and how much was organic, the man having been in a trolley car accident. He had symptoms which were largely hysterical, and yet probably some of his symptoms were due to an organic lesion.

A case in which the symptoms of Paralysis Agitans developed in the unparalyzed side of a hemiplegic.—By Dr. Joseph Sailer. (See this journal, page 425.)

Dr. Spiller said that the question of the effect of hemiplegia on tremor which Dr. Sailer brought up was one of importance. It interested him some years ago very greatly, and he studied at that time the literature on the subject. Paralysis agitans is not uncommonly unilateral. A case of this character is at present in the Philadelphia General Hospital. The effect of paralysis on tremor has been observed sufficiently often to show that a relation exists. Dr. Spiller said that in a case of malaria of the nervous system he had reported which had been under the care of Dr. Dercum, the tremor was exactly like the intentional tremor of disseminated sclerosis, and was only on one side. The malarial parasites were found in the blood vessels of the brain and spinal cord, and it was hard to understand how an irritation so general could produce a strictly unilateral tremor. One of the pyramidal tracts was slightly degenerated. Dr. Spiller explained the condition on the supposition that the slight degeneration of the pyramidal tract prevented the impulses caused by irritation from passing downward on the degenerated side to the degree that they did on the normal side and therefore these abnormal movements occurred on one side only. On looking up the literature he found that Dr. Sinkler had reported a case of disseminated sclerosis in which hemiplegia caused cessation of tremor on the paralyzed side. Mannaberg had reported a case in which there was hemiplegia and the patient had tremor during a malaria chill only on the non-paralyzed side.

Dr. T. H. Weisenburg said that some years ago in a clinical study of hemiplegia, he had examined 300 cases for skin eruptions. In those in which there was a general skin eruption, it was remarked that this was limited to the unparalyzed side. In one patient who had been paralyzed for many years, the patient himself remarked that he would tan only on the healthy side, and that the other would look paler than it should. Dr. Weisenburg had also observed that in many cases of peripheral facial palsy hardly any eruptions would occur on the paralyzed side in cases where acne was common. All of these observations seem to point to the fact that the patients are not as well nourished on the paralyzed side as they are in the other side, and in spite of this when any general skin disease appears, the par-

alyzed portions of the body seem to escape. This is rather a curious conclusion and opposite to what we should expect.

Dr. Sailer agreed that the point brought forward by Dr. Spiller was similar to the one that he had suggested for his case. He believes that paralysis agitans is a diffuse disease affecting all the superior neurones, not necessarily equally, and perhaps at first those on one side more than those on the other. In nearly all recorded cases in which disturbing factors have been absent, ultimately the disease has become general, and there appears to be no record of a case without at least bilateral rigidity. In the case reported he believed that he had not sufficiently emphasized the fact that the residual symptoms of the hemiplegia on the left side were exceedingly slight. Practically the only symptom was slight difficulty in carrying out complicated movements with the right arm.

A CASE OF APPERCEPTION.

By Dr. W. W. Hawke.

Apperception may be defined as sense-perception (of objective things) as influenced by subjective factors, especially by attention, association and memory. One reason for the title of the paper is that the case here presented shows phenomena regarding which there is room for question as to whether the hallucinations are of the real or pseudo type.

The patient is a single woman twenty-four years of age, born in Boston of Irish parents. Family history shows no evidence of mental or nervous disease in near or distant relatives. Patient started school at five years and completed grammar school at sixteen, then left school to go to work. Was employed in a mill for some years, but gave up her position last September on account of slackness of work, because she was less in need of employment than some of her companions. A week later she left home without notice to her relatives and came to Philadelphia, attracted to this city by newspaper accounts of the progress along educational lines, the ability of the surgeons, and especially the work of the Society to Protect Children from Cruelty. On reaching the city she secured domestic employment, but could not get along with her mistress, and after two weeks gave up the position. She felt helpless and confused, and went to the rooms of the Society to Protect Children from Cruelty to ask advice. She was referred to the Organized Charities of Philadelphia, and was sent to the Philadelphia Hospital. After her admission she worried because she was unable to pay for prolonged treatment, and worried also about the condition of the patients, feeling it to be her duty to help them as much as possible, and depressed because there was so little that she could do.

Patient stated that she had always been extremely fond of reading, and that she seemed to see the historical and fictitious characters about whom she had read, and to hear their voices. She dreamed as much during the day as at night, and heard voices of all those amongst whom she had been, especially if they were complaining in any way. It was a sort of repetition, and she heard them most when she was farthest away from them. She could also hear historical persons telling her what they had suffered. Stated that she had heard these voices for some years, usually at night, but sometimes in the day. When asked whether it was real or imaginary she replied, "It is real to me: I see it with my variations, I suppose someone else sees it with other variations." The vividness of her mental pictures, as she described them, seemed at first to indicate that

they were real hallucinations, but further questioning showed that she did not attribute objective reality to them. At one interview she stated that her power of imagination was not so strong as formerly, and she was afraid she was losing it. At another time she said that she could call up images at any time, and could make them seem as real as life, but did not allow herself to do it very often. "It's a pleasant pastime, and I used to do it a good deal; but I was afraid I might get to seeing things too much, so I stopped doing it except once in a while, and then I was careful to be sure what was real and what was imaginary. It's terrible to think they are real, as some of the patients do."

The patient was given Titchener's questionary upon types of mental imagery, and her answers show a wealth of images in all departments of sense, especially visual and auditory. When asked if she believed others could call up images as vivid as hers she replied, "Oh, yes, I think a great many people can, but not everybody; I think there are some who cannot." She believed that she could write magazine stories if only she had a quiet place where she could think, and once gave a brief outline of her favorite production. Her most noticeable sensory delusion is the tendency to see beauty in everything. She has many intellectual delusions, mostly of the expansive type.

Dr. Gordon thought the case exceedingly interesting from several standpoints, particularly from the standpoints of psychology and psychiatry. He had an opportunity to see the patient on many occasions. The first day she arrived in Philadelphia she went to the Society for the Protection of Children from Cruelty, which society referred the case to him for examination. He talked with her for about three-quarters of an hour. He thought at first she was an hysterical patient. She told him that she had read a number of books and that she acted in accordance with what she read. She came to Philadelphia because she read about the great work of the society. There was a great element of auto-suggestion, every act of others she thought was referable to herself and if she read anything she presented in her mind that she participated in the acts described. He found a number of hysterical stigmata at that time. He arrived at the conclusion that the case was probably one of hysteria with hallucinations in which auto-suggestion played the most prominent part. He saw her a second time and made a second examination and elicited in addition to the above symptoms also delusions of persecution, vague in character. He saw her several times in the Detention Ward of the Philadelphia Hospital. After a thorough study of the case he arrived at the conclusion that it was very probably a case of the paranoid form of dementia paranoides.

Dr. Dercum said that he saw the patient in a rather cursory way on one of his visits to the Insane Department at the Philadelphia General Hospital, and that he was very much interested in her case. He said that the members were indebted to Dr. Hawke for bringing her down and making so elaborate a report of her symptoms. The case was also interesting from the standpoint of classification He thought the case unquestionably one of dementia paranoides. It was a case with expansive and mystic ideas and it was a case, too, which closely approximated true paranoia; paranoia simplex, or the paranoia to which Kraepelin limits the term paranoia. In paranoia simplex there are no hallucinations whatever. The entire superstructure of the delusive beliefs is built upon other matter than hallucinations, either from actual observation or from

vivid mental pictures which could hardly be called hallucinations. The case was further interesting in proving that the sharp differentiations made in the whole degenerative group cannot stand; that there are transitional cases. This is a transitional case, it is not an ordinary case of paranoid dementia or of paranoia hallucinatoria. On the other hand, it is not a paranoia simplex, but occupies a position between paranoia simplex and paranoia hallucinatoria.

Dr. Hawke, in closing, said he thought the members had determined by their line of questioning what he meant by pseudo-hallucinations.

A CASE OF POLIOENCEPHALITIS IN A BOY OF EIGHT YEARS ENDING IN RECOVERY.

By Dr. David Riesman.

Dr. Riesman showed a boy of eight years, who had recovered from what had seemed to be an attack of polioencephalitis. Except for migraine in the father and brother, the family history was excellent. There had been no serious prior illness and only a few trivial falls that were without consequences.

On the morning of August 13, 1906, he awoke with double vision. On the 14th he was dizzy and staggered; felt a little nauseated, but did not vomit; perspired profusely; had a bad taste in his mouth; and spat a good deal of saliva. On the 15th, although able to play, he would often run to his mother and say he was dizzy and afraid he would fall; and twice he did fall. About this time left-sided ptosis appeared, and the gait became staggering. A neurologist who saw him made a diagnosis of brain tumor.

On August 24, when he first came under the care of Dr. Riesman, his condition was pitiable. He was almost helpless, and usually had to be carried. The face had a sleepy, expressionless look. The eyebrows and forehead were contracted: the left eye nearly closed by ptosis. The eyeballs themselves were fixed, owing to a complete external ophthalmoplegia. The head was bent forward and tilted to the right. There was risus sardonicus, especially when he tried to laugh. One could not then tell whether he was crying or laughing. He could not whistle, and speech was indistinct. He would begin a sentence fairly well, but as he progressed the words would become more and more slurred, until they ceased to be intelligible. There was some difficulty in swallowing, and liquids regurgitated through the nose. He staggered on walking, and was inclined to fall toward the right. After attempting to walk a few steps, which he did out of doors, he would soon tire and ask to be carried. About the end of August a paresis of the left arm set in—dynamometer left 10, right 35. There were no sensory disturbances, except astereognosis of the left hand. The knee-jerks were somewhat variable, as a rule not exaggerated; ankle clonus and Babinski reflexes were present, more marked on the left side. There was slight headache, and occasionally some tenderness on percussion just to the left of the occiput. No trouble with the sphincters; heart-sounds normal; pulse irregular—84 to 102; respirations 24 to 29; no fever at any time; lungs normal; abdomen soft; bowels obstinately constipated; appetite poor; vomiting occurred a few times, seemingly induced by food or medicine; was not projectile, and was preceded by nausea. Occasionally there were attacks of diarrhea with cramps, which were probably caused by the medicine he was taking—hydriodic acid. An eyeground examination made about the middle of August had shown no changes in the fundus; on a second examination, a slight congestion-

edema was found. The diplopia, which had lasted only one day—August 13—returned on September 8.

Early in September improvement began, first shown by a tendency to hold the head erect. The risus sardonicus became less pronounced, and finally disappeared; movement returned in the eyeballs, first in the upward, then in the inward direction. The external recti remained paretic.

At the time the boy was shown, he seemed to be entirely well, except for diplopia, which was due to persistent weakness of the abducens.*

The diagnosis of the case was attended with difficulty. A number of neurologists had seen him and had diagnosticated tumor. Dr. Riesman's first impression was that the case was one of myasthenia gravis, but more careful reflection led him to consider it polioencephalitis superior (Wernicke). The involvement of the left arm indicated that the process had also extended to the cord. The rapid and almost complete recovery seemed to be against the existence of a serious organic lesion and made the propriety of the word encephalitis somewhat doubtful.

As to the pathogenesis, nothing was known; it was easy to assume, but difficult to prove the existence of some toxic agent.

Dr. Gordon said he did not know how soon after his examination of this case Dr. Riesman made the examination. He was requested to make an examination and gave an opinion on the case last summer. When he examined the patient he had Dr. Perkins' negative report concerning the eyes. When Dr. Gordon examined the child he found that the patient walked with zigzag movements, with a tendency to walk always towards the right; he had distinct nystagmus, he had paresis of the external recti; when he examined him for station he presented a distinct Romberg sign, he had also ataxia of the upper extremities. He found distinct Babinski on the left with exaggerated knee-jerk, the knee-jerk on the right side was distinctly diminished. At that period of the case he did not have any marked difficulty in swallowing, but the relations told him that the child would have at times some difficulty. However, he ate his food. They gave Dr. Gordon a history of a fall with headache, vomiting, which he understood was not connected with the food. Having that picture before him, he concluded after excluding everything else that it was probably a cerebellar condition. He did not say tumor. Since then Dr. Gordon had not seen the case. At present the patient presents still some difficulty in walking, he has still a tendency to walk towards the right; the knee-jerk on the left side is different from the right; he has distinct Babinski on the left and edema of the disc with diplopia. The case presents undoubtedly some obscurity, but to say that it was a case of myasthenia gravis Dr. Gordon could not agree; he is more inclined to believe that the condition is organic.

Dr. C. K. Mills said that when he saw the case first it was to him as it has been to others, difficult of diagnosis. He thought, however, it was not clearly, in spite of the symptoms which pointed in that direction, a case of cerebellar tumor. Dr. Riesman and Dr. Mills at the time discussed the question of myasthenia gravis or bulbo-spinal paralysis, to which diagnosis Dr. Riesman was inclined and Dr. Mills thought with good reason; it seemed to him, at least, that it was the probable diagnosis. The case might be one of somewhat widely distributed polioencephalitis. If the patient's symptoms were due to a polioencephalitis he would probably have

*This weakness is gradually disappearing.

fared worse than in the outcome as now seen. After all we do not know exactly what myasthenia gravis is etiologically, and it is possible that a toxemia of some sort had to do with the origin of this case. It is well known that there are cases of myasthenia gravis lasting a comparatively short time, cases fatal after recurrences of attacks, cases of recovery and others of partial recovery.

Dr. Potts thought the symptoms described in this patient resembled very much the symptoms that were present in a patient he had in the Philadelphia Hospital a couple of years ago, and in which he made a diagnosis of polioencephalitis. Dr. Dercum agreed with this diagnosis. Typhoid fever was followed by ocular palsies, intense bulbar symptoms, in co-ordination of the extremities, weakness of the legs, increased knee-jerks, ankle clonus and the Babinski phenomenon. This patient afterwards died, but an autopsy was not obtained. The existence of permanent symptoms in Dr. Riesman's patient was certainly against myasthenia gravis as was also the existence of the ankle clonus and the Babinski reflex. He did not think that the symptoms coincided with the usual idea of myasthenia gravis. He was inclined himself to think of encephalitis in this case. It would have been of some interest and some value if a careful electrical examination of the muscles had been made.

Dr. Perkins stated that at the time he saw the boy his vision was absolutely perfect, but his general condition was so grave that it was impossible to estimate accurately the presence of a paralysis of the right rectus. There were no abnormal fauces conditions present. The case having come to him not by being referred by a physician, but because he had treated some members of the family, he was absolutely in the dark as to previous medical history. The boy had a hypermetropia of moderately high grade.

Dr. Dercum stated that we must bear in mind that the knee-jerks are unequal, that the left is exaggerated as compared with the right and that there is a distinct Babinski of the left side. These symptoms are not in keeping with myasthenia gravis. It is not improbable that some toxin acting upon the nuclei, and giving rise to polioencephalitis, also acted upon other nervous centers and in turn gave rise to the other general and local symptoms found in this case.

Dr. Spiller thought that because of the constancy of the symptoms in this case without periods of amelioration or exacerbation and without increase in intensity of symptoms after fatigue, the condition was unlike myasthenia gravis and that probably the case was one of basal encephalitis. He reported a case with Dr. Buckman, of Wilbesbarre, of myasthenia gravis confined to the ocular muscles, which showed exhaustion paralysis when an object was fixed. Dr. Buchman had written that complete recovery had occurred. Dr. Spiller did not believe that the Babinski reflex would exclude myasthenia gravis. Babinski had not said that his reflex was always the result of an organic change, but had spoken of perturbation of the pyramidal tract. Dr. Spiller believed that a functional disturbance of this tract sufficient to cause marked paralysis as in myasthenia gravis might produce the Babinski reflex.

Dr. Eshner said that at the time he saw the boy he thought the symptoms were unmistakably those of organic disease and he thought probably involved the cerebellum. He believed that there was a growth gummatous or gliomatous in character. The improvement that the boy showed was simply amazing. It would be interesting if Dr. Riesman would state the

treatment the boy had been given, how much iodide and mercury, if these were used.

Dr. Gordon said as far as the result of treatment was concerned, he could cite a case he had had for four or five years. A case of cerebellar tumor, the child is still living, in which large doses of the iodides had been given, up to 115 grains three times a day. The ataxia, headache, and the vomiting disappeared; the improvement under Dr. Riesman's treatment is not surprising to him if there is an organic condition.

Dr. S. F. Gilpin stated that in listening to the history of the case and the history of the recovery, he would like to suggest the diagnosis of multiple neuritis, even though the knee-jerks were plus and the Babinski reflex present.

Dr. Riesman, in closing, said that seeing how difficult it was at this time to make a diagnosis, it was not surprising that there should have been a great diversity of opinion at the outset. The case had come to him labeled with a variety of diagnoses that had been made by different members of the Neurological Society. The majority were on the side of brain tumor, the hopelessness of which condition had been explained to the parents, who in consequence were in the greatest mental distress. Dr. Riesman did not think it was brain tumor when he saw the case, because of the absence of headache, of vomiting, and of choked disc, one, or all of which could, with reason, have been expected to be present in a case showing such profound focal disturbance of the nervous system. His first impression of the case was that it was one of myasthenia gravis because the symptoms had come on in gradual sequence, and some of them had seemed to be aggravated as, for instance, the difficulty in walking and speaking, by effort. The first symptom had been ptosis, then had come drooping of the head, then ophthalmoplegia and paresis of the face, then paresis of the arm. In that diagnosis he had the approving counsel of Dr. Mills, who could not convince himself of the existence of brain tumor, and was inclined to attribute the symptoms to some toxic agent. At the height of the boy's illness, when his life seemed in peril, no one could have predicted the outcome of the paralysis in case of survival. The external ophthalmoplegia had been complete. At the present time the external rectus was still paralyzed; but Dr. Riesman questioned whether that would be permanent. Dr. de Schweinitz was of the opinion that the right external rectus was probably congenitally weak, but that there was a definite paralysis of the one on the left side. The vomiting had not been projectile, but had always been due to some discoverable cause. The speech had been decidedly a fatigue speech, as had been observed by the mother and the nurse. With regard to the Babinski reflex, Dr. Riesman did not think its presence could be used as a point against myasthenia gravis; the strongest argument against such a diagnosis was, aside from the incomplete character of the fatigue phenomena, the persistence of the ocular palsy. An affection of such a pronounced character as myasthenia could easily produce disturbances of the reflexes resembling those of organic disease. Although myasthenia was considered as not dependent upon an anatomic lesion, he could not conceive of a disease going from bad to worse and frequently ending in death, as having no organic basis. Neither the changes in the muscles that had been found, nor those in the thymus gland, could in themselves explain the condition. It seemed to him to be a toxic process, which produced some structural change of such a fine character that up to the present time it had not been possible to dis-

cover it. Returning to the subject of diagnosis, Dr. Riesman said that he had come to the conclusion that the case was one of polioencephalitis for the reasons already indicated. The treatment had been simple; the boy had been put at rest in the charge of a trained nurse, and had received cascara for the bowels and increasing doses of syrup of hydriodic acid. He had gained weight and seemed as well and bright as he has ever been, except for persisting double vision due to the abducens palsy.

Note.—There has been a decided improvement in the condition of the eyes since the foregoing remarks were made.

A CASE OF TABES DORSALIS WITH INVOLVEMENT OF MANY CRANIAL NERVES.

By Dr. T. H. Weisenburg.

This patient was seen with Dr. Wm. Zentmayer in the Wills Eye Hospital. The patient was 51 years of age, with no medical history of any importance and no specific history. His trouble began two years before he came under observation with a diplopia. Soon after this he had drooping of the left upper lid. These symptoms subsided in the course of two months and gradually disappeared. About the same time he began to have a twitching-like sensation in his right face. This sensation terminated with a pain in the right eye, and came on gradually. He was apparently well otherwise until two months ago, when he was taken to a hospital for involvement of the bladder and rectum. About the same time he developed a drooping in the right upper eyelid. He has had increasing girdle sense and numbness in both legs, and about the same time he began to stagger, especially in the dark or with his eyes closed.

Examination shows complete paralysis of both oculomotor nerves and also paralysis of the right fourth nerve. The ptosis on the left side is not complete. The motor fifth nerve is normal, but the sensory portion of the fifth nerve, however, is involved. Touch and pain sense as well as taste are disturbed in the anterior two-thirds of the right side of the tongue. Teeth can be pulled out on the right side of the jaw without any difficulty, not so in the left. The seventh nerve is partially weak, the other cranial nerves are normal. The pupils are partially dilated and react only to strong light stimulus. Both optic nerves are red gray. The fields are functionally normal for form, and slightly contracted for color. Power in both upper and lower limbs is about normal. Ataxia, especially with eyes closed, is very marked in all of the limbs. All of the tendon reflexes are lost. Sensation has disappeared over the front of the chest and soles of the feet. There is considerable hypotonia present in all of the joints.

The case is evidently one of tabes dorsalis, with disease of the right second, third, fourth, fifth and seventh nerves and of the second and third nerves on the left side. Ordinarily without the involvement of the cranial nerves there would be no question about the diagnosis of tabes dorsalis. With this rather unusual affection of the cranial nerves, the question arises whether this case is really one of tabes or of syphilis. All of the cranial nerves which are diseased in the present instance may be involved individually in tabes, and there is no reason why a diagnosis of syphilis should be made even though as many cranial nerves are diseased as happens in this instance.

AMERICAN NEUROLOGICAL ASSOCIATION.

Held in Washington, May 7, 8 and 9, 1907.

The President, Dr. Hugh T. Patrick, in the Chair.

Presidential address: Ambulatory Automatism. By Dr. Hugh T. Patrick. (See this journal, page 353.)

The Study of Reflexes of the Lower Extremities in Sixty Cases of Paresis, with a Special Reference to the Paradoxical Reflex. By Dr. Alfred Gordon. (See this journal, page 430.)

Dr. F. X. Dercum said he believed the paradoxical reflex has a distinct practical value. One of the cases in which he was able to study it in connection with pathological findings has, as Dr. Gordon has said, been already placed on record, but it was so important a case that a brief statement of the facts is still of value. The gentleman was a medical man who had been a member of Dr. Dercum's own staff at the Jefferson Hospital. He had had what proved to be an effusion or a hemorrhage in the membranes upon the right side. He had no distinguishing features so far as his reflexes were concerned to enable them to localize any lesion (the effusion was very slight in degree) except the presence of this paradoxical reflex, and it was distinct. Dr. Keen operated upon the right side, and a considerable amount of bloody serous fluid was evacuated. Immediately after the operation, as soon as the patient had recovered from the ether, he was tested as to the paradoxical reflex and it had disappeared. Some days subsequently the drainage became obstructed and the paradoxical reflex again appeared. There was no other guide at this time as to the localization of the lesion. Both knee jerks were somewhat exaggerated; there was no ankle clonus upon either side. The patient was operated upon again, at least the wound was opened and freely drained, and again the paradoxical reflex disappeared. It seemed to Dr. Dercum that the paradoxical reflex is one which obtains when the interference with the motor pathway is very slight. It may be lost or masked when the lesion is gross. In his judgment it is a very valuable sign.

Dr. Morton Prince said he was unable to add anything in regard to the clinical value of the contraction spoken of by Dr. Gordon, but he would like to say a few words upon our interpretation of this sign. It would seem to bear some relation to, if it is not identical with, Westphal's "paradoxic" contraction. If they are different it is unfortunate that Dr. Gordon should have adopted the same name, "paradoxic." Westphal included extension (dorsally) of the ankle, as well as of the toes. Dr. Gordon only describes the toe movement. This may possibly be due to the mechanical interference with the foot from the position adopted in testing. It has been forgotten that Erlenmeyer in 1880 maintained that Westphal's contraction was due to excitation of the calf muscles caused by stretching; Charcot and Richet (1885) held the same view and pointed out that in hysterical subjects dorsal flexion of the foot under certain experimental conditions could be induced by massage of the calf muscles. They concluded that when a muscle is stimulated its antagonist is simultaneously excited, the latter having thus a regulating function, and under certain conditions its action may become predominant. This spasm of the antagonist has been found in other muscles than the extensors of the foot and toes; *e. g.*, in the flexors of the knee and in the arm muscles.

In 1889 the speaker had become interested in this phenomenon and published some observations (with cases) upon it (*Boston City Hospital Reports,* 1889), but at that time had considered, perhaps erroneously with Charcot and Richet, the "paradoxical contraction" and that resulting from massage of the calf muscles as identical. He had given it the name *"antagonistic contraction."* There were other ways, besides those mentioned, of producing the contraction. It is possible that two different reflexes have been included under Westphal's; viz.: that following dorsal flexion of the foot and that from massage of the calf muscles. The former, the speaker had shown, was only an exaggeration of a normal phenomenon; the latter may be identical with Dr. Gordon's reflex. And yet it is significant that Dr. Gordon had not found this reflex in hysteria and functional diseases, while Charcot, Dr. Prince and others had found it in hysteria. Westphal had found his reflex in tabes with *motor weakness,* atypical multiple sclerosis (?) and paralysis agitans, while Dr. Gordon had not found his reflex unless the motor tract was involved. Dr. Prince had found "Westphal's" contraction in multiple sclerosis and functional troubles. The relation to one another of these different contractions, including Oppenheim's and Babinski's, elicited by different methods, needed further investigation.

Dr. P. C. Knapp said that in spite of Dr. Gordon's kindness in demonstrating the technique of his paradoxical reflex to him, he had to confess that he had often failed to obtain it, even in cases where the Oppenheim or the Babinski reflex was well marked. He cannot therefore speak absolutely pertinently with reference to this question of the prevalence of the paradoxical reflex in general paralysis, but he has seen a considerable number of cases in which the relations between the exaggeration of the knee jerk and the Babinski and Oppenheim reflexes were not consistent. Of course we all agree that those reflexes are indicative of change in the motor tract, just as the exaggeration of the reflex is often indicative of such a change, especially if the exaggeration amount to a true clonus. But the two do not seem to hold the same relation. In general paralysis, for example, Dr. Knapp had seen a case in which there was a complete absence of knee jerk. It was impossible to obtain the knee jerk in any way, even by reinforcement, and yet there was on one side a distinct Babinski reflex and Oppenheim reflex. In hemiplegia he had sometimes noted upon the paralyzed side a diminution in the knee jerk, often a knee jerk which could be obtained only by reinforcement, although there was a fair knee jerk upon the unparalyzed side, and yet in the paralyzed leg there were the Babinski and Oppenheim reflexes. It is perhaps unfair to include in this, as an example of this discrepancy, the existence of the Babinski and Oppenheim reflexes in cases of fracture of the spine where the knee jerk is lost, because there is a possibility that the reflex arc for the knee jerk is higher than that for the plantar reflex. There may have been a lesion there, although in one case, Dr. Knapp recalled that the fracture was much above the reflex center for the knee jerk. There are certainly a considerable number of cases in which the Babinski and Oppenheim reflexes are not in harmony with the knee jerk. Of course, in paresis it is quite rare to get a sufficient exaggeration of the tendor reflexes in general to amount to a true ankle or patellar clonus. It is worth while to bear in mind certain other methods of eliciting the knee jerk which often have a decided significance, especially in cases of unilateral lesion where it is desirable to determine any difference in the

reflexes on the two sides. The knee jerk obtained by striking above the patella instead of below it, the so-called tibial reflex obtained by striking upon the periosteum of the broad head of the tibia, the patellar twitch obtained by drawing down the patella with the fingers as in testing for patellar clonus and striking the finger a sharp blow, and the front-tap contraction, are forms of the knee jerk, and these methods should be employed. Dr. Knapp had very often found them to be of use in showing differences in the tendon reflexes where the ordinary test for the reflex was not significant.

Dr. H. M. Thomas said he had only one or two remarks to make, dealing particularly with the definition of the reflex. He feels strongly about giving names to signs and not describing them. As he understands the discussion by Dr. Prince of Dr. Gordon's paper, it was simply brought about by the lack of definition. Dr. Gordon gave no definition at this meeting. He has done so before. It seemed to Dr. Thomas that if we are going to use the term paradoxical reflex for contraction of the tibialis anticus, according to the method of getting the ankle clonus, it is extremely important to define what we mean. Dr. Knapp describes four or five methods of getting the knee jerk. Dr. Gordon did not say anything about the ankle reflex that Dr. Thomas heard; he said ankle clonus. Dr. Thomas does not know whether Dr. Gordon's cases had exaggerated ankle reflex or not; he supposes they had, and that it was not clonus.

Dr. P. C. Knapp asked to be permitted one word in response to Dr. Thomas's criticism. Giving the different grades of exaggeration there is first the simple knee jerk, as obtained by striking the tendon below the patella; in a slighter degree of exaggeration there is the knee jerk obtained by striking above the patella; in a third degree of exaggeration the patellar twitch obtained by pulling down the patella with the finger and striking it a sharp blow with the percussion hammer; the fourth degree would be the front-tap described by Gowers; the fifth would be the so-called tibial reflex, the kick forward in striking the broad head of the tibia, and the sixth would be the true ankle and patellar clonus.

Dr. F. X. Dercum wished to be allowed to say that he believed Dr. Gordon's reflex is an entirely new thing in itself, a new discovery. It has nothing to do with what was formerly known as the paradoxical reflex.

Dr. A. Gordon said in reference to Dr. Knapp's remark that in a number of instances gentlemen who wished to exhibit the reflex and have failed to do so called him to help them, and he obtained the reflex with the greatest facility. It is consequently a question of the exactness of the method. The patient is placed on a chair, and his feet (not the legs) are placed on another chair. The patient is told to relax his muscles, to make himself perfectly comfortable. The feet should be thrown slightly out, not forcibly, and the operator should place himself outside of the legs, place the soft part of the hand on the tibia in order to avoid irritation of the extensors. The tips of the fingers are placed on the middle of the gastrocnemius muscle. Then pressure should be made upon the middle of the soft part of the calf of the leg. Sometimes the examiner has to pass his fingers up and down (frequently he does not have to do that); and at the moment the reflex is present the toes will come up, particularly the big toe. The same can be obtained when the patient is in bed.

In regard to the explanation of the phenomenon, Dr. Gordon said he wished to answer Dr. Prince that at present he has on record 357 normal

cases without the slightest ailment, especially ailment of the nervous system, in which he has not been able to elicit the reflex of which he is speaking. In these cases pressure of the calf muscles of the legs gives no response at all or flexion, but in the cases where the motor tract is supposed to be involved, he very frequently has found this phenomenon.

He said he wished to emphasize one more point in regard to the value of this sign. As far as the relation of this reflex to Oppenheim's is concerned it is not an Oppenheim reflex at all or a modification of it. Dr. McCarthy, who at first expressed the opinion that it was perhaps a modification of Oppenheim's reflex has since retracted that opinion. It is a decided sign of involvement of the motor tract. He said he wanted to refer to another anatomical and clinical case. A boy entered the Jefferson Hospital with injury to his right temporal region; he had headache and was stuporous. He was put to bed, and on the opposite leg increased knee jerk was found, but no Babinski, no ankle clonus and no Oppenheim. Dr. Mills and Dr. Dercum examined the case and they saw that while all the other reflexes were absent, this reflex was present. Dr. Da Costa operated upon the right side of the skull and found marked pressure. After the operation the reflex disappeared, and Dr. Gordon has examined the patient many times since and has never been able to discover the paradoxical reflex.

(This case was reported before the Philadelphia Neurological Society and is to be published in the *American Journal of the Medical Sciences.*)

THE SENSORY SYMPTOMS AND THE SENSORY AFFECTIONS OF THE FACIAL NERVE.

By Dr. J. Ramsay Hunt.

The facial nerve like the trifacial is a mixed nerve; consisting of a motor root, a sensory root and ganglion.

In this communication the symptomatology of the sensory mechanism of the seventh cranial nerve is discussed, with the practical importance of this group of symptoms to clinical neurology and otology.

1. Preliminary remarks on the embryology and anatomy of the facial nerve.
2. Pain and sensory disturbances in facial palsies (Fallopian neuritis).
3. Herpetic inflammations of the geniculate ganglion.
4. Primary otalgia (neuralgia of the facial nerve).
5. Secondary otalgia (tabetic ear pains).
6. Reflex facial twitchings and spasms.

Dr. P. C. Knapp said he had had the opportunity lately of seeing two or three cases of facial paralysis very early in the onset of the disease. In them he found just for a day or two a very slight diminution of sensibility. It could only be obtained, as Dr. Hunt has suggested, by the comparative method of testing the two sides, and sometimes only by the comparison of exceedingly slight stimulus such as Frey's esthesiometer, and in a day or two even that difference had disappeared. He thought that if we could see the cases very early and test by the comparative method we should find sensory disturbance in facial paralysis more often.

Dr. H. H. Hoppe said he would like to ask Dr. Hunt whether he came across many cases of pain in the eyeball. Only a few days ago he (Dr. Hoppe) saw a patient who had been paralyzed some years before. The history was that the onset of the facial paralysis was accompanied by acute pain in the eyeball.

Dr. T. H. Weisenburg said that he always had thought that the facial nerve had also a sensory function. For many years he had followed a case of facial palsy in which he noticed that whenever the patient developed an ordinary coryza there would not be as much secretion from the paralyzed side as from the other. This he had observed in many similar cases of peripheral facial palsy. He had also observed that when, for instance, a general eruption like an acne would appear upon the body the paralyzed side of the face would escape. This freedom from general manifestations is also true of other so-called motor palsies. He had one patient, a hemiplegic, who would tan only upon the paralyzed side. It seemed to Dr. Weisenburg that the terms motor and sensory palsies were purely relative. Thus in a case with pure motor lesion there will always be some sensory symptoms. For instance, in hemiplegia there will always be loss of vasomotor tone, and this in a broad sense is a sensory manifestation. On the other hand, in cases of pure sensory lesion there are always some motor symptoms. As for instance, in a case of pure sensory involvement of the fifth nerve there will always be some difficulty in moving the muscles of the face on the involved side. In tabes, in which the lesions are almost wholly sensory, there will be some difficulty in movement, the so-called incoördinate movement of tabes.

Dr. L. P. Clark said that in the last two or three years he has been interested in the neurological surgery of the seventh nerve. He has noticed in a number of very old cases of facial palsy, even when by very careful sensory test no difference in the herpetic zoster zone is capable of detection, yet subjectively the patients state that there is alteration in the sensibility in that side. Heretofore he has supposed that the patients having the palsy would naturally think they ought to have a sensory defect also, but Dr. Hunt's explanation would be a complete justification of their contention.

Dr. S. I. Schwab said he would like to ask Dr. Hunt whether he includes deafness as a part of the syndrome in his cases. Dr. Schwab had an opportunity of observing a patient who had the herpes, deafness, vertigo and facial paralysis on one side, and the deafness was very distinct.

Dr. Hunt said in regard to Dr. Schwab's remarks, he of course includes deafness as a very important symptom in this syndrome. The simplest expression is the herpetic pain and the zoster, in the other clinical types there is facial palsy alone or in conjunction with auditory symptoms. These may be vestibular or cochlear or both. They are so marked in some cases the thought has occurred to Dr. Hunt that perhaps the sensory ganglia of the acoustic nerve may have to be brought into the realm of herpes zoster also, but he has explained these cases in his paper by an extension of the inflammation from the geniculate ganglion through the sheaths of the nerves.

In regard to the other questions, the tympanum canal, the auditory canal, the concha and the auricle have a very complex and a very varied innervation. This is from the auricular branches of the trigeminus and of the cervical and of the vagus nerves, and, he thinks also, from auricular branches of the facial. These all converge and innervate a very small area. As the auricular area of the facial nerve is small and the additional innervation so extensive, anesthesia probably would be of very short duration or very slight. There are also certain difficulties in examining the sensation of a canal like the auditory canal. Personally, he has never found a distinct anesthesia in the concha or the canal. He has, however,

only expected to find it in the early stage of severe facial palsy. Gowers, however, says distinctly that he has observed it in a number of cases, and Dr. Cushing says he has found in some cases a hypesthesia. There is no question as to the subjective sensation in the ear in these cases of facial palsy. If inquiry is made very definitely the patients say the ear seems full or there is something in it, which may be interpreted as a paresthesia.

In regard to Dr. Hoppe's case with pain in the eye, of course in cases of facial palsy or neuritis it is not infrequent to find pains in the trigeminal area, just as in affections of the trigeminal area it is very frequent to find a pain in the sensory area of the facial nerve. The anatomical connections of the two sensory systems he thinks afford a sufficient explanation.

ON THE SPLITTING OF AFFERENT FIBERS IN PERIPHERAL NERVES.

(Observations of Dr. Elizabeth H. Dunn.)

Reported by Dr. H. H. Donaldson.

In a frog, the ventral roots of the spinal nerves supplying the leg were cut within the spinal canal. This left the limb supplied only by the afferent fibers derived from the dorsal nerve roots. A study of the supply to the skin and muscles showed that the muscles were abundantly innervated with afferent fibers. A study of the numerical relations showed also that the afferent fibers must split in their course, in such a way that each division of the split fiber passed to a different segment of the limb. It could not be determined, however, whether any of the splitting fibers were so distributed that one division went to the skin, and the other to a muscle.

We do not know whether this splitting is more developed in the frog or in man, but if it occurs in the frog, it probably occurs in man also, and is an anatomical fact to be taken into consideration in cases of disturbed sensation, and possibly also has a bearing on the phenomena of "referred" pain.

Dr. Langdon said it seemed to him that this subject has a practical bearing of clinical import. For instance, in the question of the origin of referred pain. He wished to ask Dr. Donaldson if he will give us his individual opinion as to whether this splitting of fibers, so-called, is really a histological splitting of a neurone process, or perhaps more likely a rearrangement of the fibrillæ; in other words, does a new structure arise at that point of splitting or is it simply a separation of pre-existing paths?

Dr. Bullard asked how much of what Dr. Donaldson had said applies to man and how much to the frog and lower animals.

In answer to the first question, Dr. Donaldson said he should hesitate to introduce a discussion of the fibrilla hypothesis at this stage.

As to the second point, there are no direct observations showing that this observation does apply to man. There are some indirect observations based on the size of the nerve trunks both in the limbs and in the nerve roots, which seem to favor this, but he said he was careful to emphasize the fact that his observations had been made upon the frog. There is, however, at each turn a high degree of similarity in the arrangement of the nerves even between the frog and the higher vertebrates, and he feels very strongly that we shall be able to corroborate these relations in mammals and in man.

(To be continued.)

Periscope

Miscellany

Internal Hydrocephalus. W. G. Spiller and A. R. Allen (Journal A. M. A., April 13).

The authors discuss the causes of internal hydrocephalus, reviewing the literature, and remark that while the occlusion of the aqueduct of Sylvius is not infrequently mentioned as an etiologic factor in the text-books, it is rare to find references to actual cases of such occurence. They take the opportunity, therefore, to report a case in which the condition was induced by a probably congenital, almost total occlusion of the aqueduct. The subject was an elderly woman who was supposed to have been born hydrocephalic, but in spite of this and of an epilepsy that had lasted for years she had lived to the advanced age of 62, and retained her faculties fairly well developed. The aqueduct, while almost entirely occluded, was well lined throughout by a layer of ependymal cells which would not be the case had its occlusion been due to neuroglia proliferation, as in a previous case reported by Spiller. This, and the long continuance of the condition through the patient's whole life, indicates the congenital nature of the obstruction.

Cerebral Localization and the Study of Psychiatry, with Especial Reference to the Mechanism of Hallucinations and Illusions and the Classification of the Forms of Insanity. Chas. K. Mills, M.D. (The British Medical Journal, Sept. 29, 1906).

Coarse cerebral lesions when diffuse are effective in the production of hallucinations and illusions, but not if strictly focal, and it is evident therefore that the part played by the associative mechanism of the brain is most important in the symptomatology of insanity. The author's classification of insanities is into the teratological or abiotrophic and the pathological or acquired. Under the first would be classed congenital idiocy and imbecility, dementia præcox, dementia choreica and other presenile insanities, and senile dementia; also those less in degree, such as the melancholia-mania group, paranoia, hysteria, epilepsy and psychasthenia. The pathological insanities would include those due to diffuse destructive organic disease, as the forms of imbecility or mental defect due to post-natal causes, general paresis, syphilitic insanity, and traumatic insanity; also the insanities due to toxemias and exhaustion. C. D. Camp.

Paralysis of the Eye Muscles Following Spinal Anesthesia. Alvin. Ach, M.D. (Muench. med. Woch., March 26, 1907).

The etiology of the paralysis of the eye muscles, following spinal anesthesia is still obscure. Adam, who observed the first case of paresis of the muscles, thought the manifestations were due to hemorrhage in the nucleus of the sixth nerve. Loeser considered it to be due to the toxic action of the drug on the nucleus or nerve involved. He points out the affinity and selective action that some poisons have for certain nerves and nerve centers, as lues, alcohol, diphtheria and leal. Landow agrees with Loeser. Lang thought it to be perpipheral neuritis, whereas Baisch, in his case of double-sided abducens paralysis, was under the impression that it was an apoplectic insult. Ach is of the opinion that it is not due to the

toxic action of the drug on the nucleus in the floor of the fourth ventricle, and the anatomical relations make it nearly impossible for any solution to enter the fourth ventricle from below. His view is that the poisons act directly on the nerves. Paralysis of the eye muscles occured from four to eleven days after the operation and lasted from six to forty-three days in the case observed by him. He gives the following rules for spinal anesthesia: (1) The most harmless drug should be used. (2) Tropacocaine is better than stovain as the latter has a marked influence on the motor nerves. Use small doses. (3) Do not use concentrated solution. (4) Rest of patient after operation, with head and shoulders raised.

F. J. Conzelmann (U. S. Army).

The Pathology of Paralysis Agitans. C. D. Camp (Journal A. M. A., April 13).

Camp, after giving a review of the literature of the pathologic findings in the nervous system in paralysis agitans, reports the results of his examination of fourteen cases, in eight of which he was able to study the peripheral nerves and muscles, and in two the ductless glands also. The most constant lesion in the nerve centers was a fibrosis of the capillary blood vessel of the spinal cord, which by rendering them more prominent, caused them to appear more numerous. The posterior and lateral column regions seemed most involved. There was no degeneration of nerve fibers of the cord in any case, and in only two cases was this observed in the peripheral nerves with the Weigert hematoxylin stain; in two others there was a swelling of the myelin sheaths, accompanied in one case by swelling of the axis cylinders. There was considerable pigmentation of the Betz cells in the paracentral lobule, but in only two cases were distinctly degenerated cells observed. He discusses the various theories of the disease, and thinks that many of the pathologic conditions found, and to which the disorder has been attributed, are only coincident senile changes. The muscular theories are also discussed, and in nine cases in which he examined the muscles, one of them during life, he found pathologic changes. There was swelling of the muscle fibers, which were round in cross-section instead of polygonal, multiplication of nuclei, etc., his findings agreeing in the main with those of Schwenn, Schiefferdecker and Idelsohn, indicating, he is inclined to believe, a specific change in the muscles in paralysis agitans, though he admits that this is not yet definitely proven. Camp, in considering the pathogenesis of the affection, finds most reason to believe that it is due to a toxemia, and that this is connected with disordered parathyroid function. In the two cases in which he was able to examine the ductless glands he found the parathyroids in a decidedly pathologic condition; in both there was a peculiar fatty infiltration, especially in relation to the blood vessels. Such a finding, in connection with the experimental evidence as to the effect of parathyroidectomy and the therapeutic results of Berkley in the treatment of paralysis agitans with parathyroid extract, furnish, he thinks, strong proof that the parathyroids play an important part in the pathogenesis of paralysis agitans. His conclusions are given as follows: (1) Paralysis agitans is not a neurosis nor is it senility. (2) The anatomic basis of the symptoms, muscular rigidity, tremor and the symptoms dependant on them lies in the affection of the muscles. (3) The disease is probably a general toxemia, and there is evidence that it is due to alteration in the secretion of the parathyroid glands. The case histories of the patients of which Camp made the pathologic studies are appended to his article.

THE DELIMITATION OF GENERAL PARALYSIS OF THE INSANE. Julius Mickle (The British Medical Journal, Sept. 29, 1906).

In the delimitation of general paralysis with relation to degeneracy and mental degenerates, as regard symptoms the author divides the relevant cases into four groups. Degenerates with deliria, but not marked physical signs, simulating G. P. I. Recurrently alcoholized and deliriant degenerates simulating both the mental and physical signs of G. P. I. Original paranoiac degenerates simulating both the mental and physical states of G. P. I. Degenerates becoming true G. P. I.'s. Cases of general paralysis of the insane may be arrested in their course, the patient remaining a chronic mental defective. This is a fixed mental state and not a long remission, and is one of the terminations of progressive general paralysis of the insane though the descriptive term, progressive, does not apply. CAMP (Philadelphia).

THE PRE-INSANE STAGE OF ACUTE MENTAL DISEASE. Campbell Meyers, M.D. (The British Medical Journal, Oct. 20, 1906).

The stage referred to is "the weeks or months" which "often elapse between the time when any competent observer can notice the deviation from normal mental health and the time when the boundary line of insanity is reached in the onward progress of the disease." The symptoms are: Difficulty in sustaining intellectual effort and concentrating the attention, loss of will power and energy, morbid sensitiveness, emotionalism, morbid introspection, mental depression, loss of affection for friends or relatives, senseless fears, insomnia, and peculiarities in speech and handwriting. The author favors calling the condition "cerebrasthenia" and advocates its treatment in special wards in all general hospitals believing that such care would prevent many cases of insanity.

C. D. CAMP (Philadelphia).

TREPHINING AS A PALLIATIVE MEASURE IN TUMORS OF THE BRAIN. Herbert A. Bruce (Annal. of Surg., April, 1907).

Five cases are reported to show that the classical symptoms of brain tumor—optic neuritis, headache and vomiting—can be relieved or entirely removed by freely opening the skull and dura. As regards vision improvement depends upon the condition of the optic discs. Where secondary changes have set in improvement in proportion to the changes only can be expected. Where loss of vision is due to simple swelling of the discs the sight is not only saved, but improved.

COWLES (New York).

EXPERIENCES IN CEREBRAL SURGERY. Frank Hartley and James H. Kenyon (Annal. of Surg., April, 1907).

After a review of the lack of adaptability of instruments generally employed in brain surgery, Drs. Hartley and Kenyon describe a new hand motor from which power is derived for drilling bone and cutting osseous flaps by means of drills, burrs and disc saws. The advantage claimed for this device are safety, ease of manipulation, shortening the time of operation, lessening of operative shock, and making it possible to cut a clean beveled edge, which last brings autoplasty in cerebral surgery to its highest utility. The most striking point, however, is the fact that they can easily expose any cerebral area through an autoplastic flap having for its base the temporal fossa, the thinnest portion of the skull. This also

insures an abundant blood supply to the flap through the temporal or occipital arteries.

COWLES (New York).

OCCUPATION IN THE TREATMENT OF THE INSANE. T. J. Moher (Journal A. M. A., May 18).

The author calls attention to the need of a more systematic use of employment in the treatment of insanity. The percentage of patients who will not be benefited by occupation of some kind is very small, and if we exclude the physically disabled and the very advanced demented cases it is practically negligible. Acute maniacs in some stages and some exalted paretics are temporarily unable to work, but the duration of this condition can be very much shortened by careful and systematic effort. Some few patients absolutely refuse to work, and can not be made to by any effort. Occupation should be simple at the outset and the patient's temperament and predispositions should be studied in every case. It is not wise to restrict a patient to any one class of work, and his previous occupation and social condition should not be the only determining factor in the choice, though some can not be induced to undertake anything to which they have not been accustomed. Another thing not to be forgotten is to avoid asking certain patients to perform some of the more menial work that has to be done. The objections of friends can usually be successfully met by patient explanations, and the patients themselves are generally easily influenced by surroundings, and the facts that they are never asked to overwork and that the occupation is made pleasant for them induces even the indolently inclined to fall into line and accept willingly the tasks asked of them. If the fact that occupation is an important remedy is impressed on intelligent attendants and they are instructed how to apply this method of treatment in a skilful and systematic way, there will be little difficulty, Moher says, in keeping patients employed. His experience has taught him that it is unwise to offer any pecuniary reward for patients' work. They should not be allowed to get the idea that they are employed for the advantage of the institution rather than for their own good. In some cases it may be advisable to encourage patients to learn a trade with a view to their self-support after discharge. It is not always easy to say how occupation effects a cure. Besides the exercising and calling into action of disused brain centers in some cases, it acts by improving the physical condition. As a result of suitable outdoor employment we find that patients are less restless, sleep better, improve in appetite and become more normal in their secretions. While employed in the wards also, they become less noisy, less quarrelsome or destructive and better behaved generally. Mental improvement is often directly coincident with improvement in physical health. It is important that members of the medical staff regularly visit and observe the patients at their work, with a view of studying the effects in individual cases. But while admitting that the cure in many cases can be rightly credited to occupation, Mohr believes that its greatest good is conferred on the incurable cases by delaying dementia and adding to the comfort, happiness and general well-being of the chronic insane. Agricultural pursuits and caring for the grounds appear to be the ideal labor for the great majority of male patients, while female patients can be employed with advantage in the vegetable and fruit garden, weeding, picking berries, etc., in addition to the work performed in the day rooms, dormitories and dining rooms. They should also do fancy work, do all the mending, and assist in the industrial department in making of mats, mattresses, etc.

Book Reviews

ANANLYSE VON 200 SELBSTMORDFÄLLEN NEBST BEITRAG ZUR PROGNOSTIK DER MIT SELBSTMORDGEDANKEN VERKNÜPFTEN PSYCHOSEN. Von Dr. Helenefriederike Stelzner. Mit einem Vorwort von Professor Dr. Th. Ziehen. S. Karger, Berlin. 4 marks.

This is not only an interesting contribution to the subject of suicide, but it contains some important features usually overlooked in studies of this nature. Most older observations on the subject are taken up in the larger part of their pages with considerations of age, of sex, of race, etc., whereas the present volume very wisely discusses the whole problem from the standpoint of the underlying psychoses. Statistical inquiries are silent on this point.

From a careful analysis of 200 cases of attempted or successful suicide, the author has clearly brought out those mental disorders which are most likely to lead to suicide, in Berlin, at least. It is quite conceivable that other cities and other nationalities may present some variations.

The depressive psychoses—melancholia, in Ziehen's sense—show the greatest incidence of suicide. Of the 200, 65 were suffering from one of the forms of melancholia, climacteric, hallucinatory, hypochondriacal and periodic, as outlined by the chief of Berlin's psychiatric clinic.

The degenerative mental states, hysteria and the psychopathic constitution, contributed the next largest number of suicides; 32 in the group of 200. Other psychoses contribute but smaller numbers of suicides in the present study.

Thus in the lists there were 11 cases suffering from acute paranoia (Ziehen), including this same author's amentia; 24 cases were in chronic paranoiacs, 4 in senile dements. Dementia paralytica contributed 6, while dementia præcox is credited with 7. Suicide was attempted by 11 epileptics, by 3 imbeciles and alcoholism was responsible for 6 cases. In 31 there was no history of a distinct psychosis.

The author further attempts, by her clear and careful analysis, to show what may be the determining features leading to suicide in the varying psychoses. Thus in acute paranoia (Ziehen), the extreme anxiety due to being followed or oppressed by the hallucinations is the given cause. In chronic paranoid psychoses suicide is sought as a relief from the persecutory delusions. In dementia præcox, simple pathological impulsiveness seems to be the cause in the majority of the cases. As a rule, a logical reason for the attempt at suicide is rarely given by the patient suffering from dementia præcox.

Very superficial motives come to the fore in the study of attempts committed by imbeciles—simple quarrels with relatives and neighbors being sufficient. In hysteria and other psychopathic inferior states, a common cause of the attempt is largely dependent on some emotional shock, and is very frequently accompanied by a general theatrical mode of operation.

The study, notwithstanding its many excellent features, is incomplete in that only women were studied, and the cases were only those brought to a psychiatric clinic. Thus, wide generalizations are impossible to be

drawn from the material at hand. In spite of the drawbacks, however, the book is one of the best that have appeared in years. It is well worth reading. JELLIFFE.

ARBEITEN AUS DEM NEUROLOGISCHEN INSTITUTE AN DER WEINER UNIVERSITÄT. Herausgegeben von Prof. Dr. Heinrich Obersteiner. Band XIII. Franz Deuticke, Leipzig und Wien, 1906.

Vol. 13 of Obersteiner's Arbeiten comes with its usual rich collection of thorough and stimulating studies. We can but enumerate some of the more complete and noteworthy. Of the purely anatomical papers, E. Zuckerkandl contributes two extensive dissertations, one on the Anatomy of the Calcarine Fissure, and another upon the Gyrus Transitivus; M. Grossman has a study on the Intrabulbar Connections of the Trigeminus with the Vagus; E. Hulles one on the Sensory Roots of the Medulla, and P. Biach, a Study on the Comparative Anatomy of the Central Canal in Mammals.

Of pathological studies there are Cytological Changes Observed in Puerperal Eclampsia, by E. Pollak; Changes in the Spinal Ganglion Cells After Amputation of the Upper Extremities, by K. Orzechowski; one or two on Senile Changes in the Brain, and an extensive study by Marburg on Hypertrophy, Hyperplasia, and Pseudohypertrophy of the Brain. The collection is thoroughly representative and praiseworthy.

JELLIFFE.

ANNUAL REPORT OF THE CENTRAL STATE HOSPITAL OF VIRGINIA (FOR THE INSANE).

The old clinical classification is still maintained at this hospital, although the records are said to be arranged after Kraepelin. The superintendent recommends that each year one of the medical staff be sent to some medical center for a month for instruction in the most modern practice. He also suggests joint meetings, once or twice a year, of directors, superintendents, assistant physicians and other officers of Virginia State hospitals, at which papers should be presented and discussed.

THE THIRTY-THIRD ANNUAL REPORT OF THE CINCINNATI SANITARIUM.

An epitome of the medical results of the year shows the total number treated during the past year to be 302. Of these, 88 were under treatment at the beginning of the year, and 214 were new admissions. The daily average for the year was 93.15. Eighty-one patients were discharged recovered (37.37%); 73 as improved; 38 as unimproved. The mortality was 18 (5.96%). The medical administration has been strengthened by the addition of a pathologist and hematologist. A series of research observations on the relations of the *Bacillus paralyticans* of Robertson to paresis has been conducted in the clinical laboratory, and is made the subject of a separate illustrated paper accompanying the report proper. This research work is especially creditable to a private institution, which receives no State aid of any kind; and is an evidence of progressive methods, which merit the approval and continued confidence of the profession generally.

ATWOOD (New York).

PRIMER OF PSYCHOLOGY AND MENTAL DISEASE. C. B. Burr, M.D. Third Edition. F. A. Davis Co., Philadelphia, 1906.

In every hospital for the insane there is a need of a book that will give the nurses and attendants an idea of the real state of such patients as they are thrown in contact with. It is only by means of understanding

the nature of a mental change that the persons intrusted to their care become interesting and subjects of their sympathy and thoughtfulness, and are not only interesting because they do and say curious things.

Dr. Burr's book fulfills these wants to a marked degree. His chapter on psychology is good; it is not perhaps based on principles which meet the approval of all psychologists, but that is not a possible task.

The chapter on insanities is founded on the newer teaching, embraces dementia præcox and manic depressions, insanities, etc.

It is difficult in a small book to present such intricate diseases so as to be recognizable by nurses, but sufficient is given for developing their powers of observation and making them a valuable aid to the physician in studying the cases.

The chapter on the management of cases is sufficient as an outline of what should be accomplished in conditions of alienation, and the author's advice on the diplomacy of action with the insane is most creditable.

S. D. LUDLUM.

COLLECTED STUDIES ON IMMUNITY. By Professor Paul Ehrlich. Translated by Dr. Charles Bolduan. John Wiley & Sons, New York.

This neat volume of 586 pages, well printed and well bound, contains the greater portion of the studies on immunity published in late years by Professor Ehrlich and his collaborators. They are here made available to the English reading public for the first time.

As the leading worker in this singularly complex and intricate field—that of cellular metabolism—Ehrlich's views constitute at the same time the foundation and the superstructure. His ideas, as is well known, have developed along chemical lines of thought, and in the study of the fundamental biological phenomena the significance attached to chemical interpretations, rather than those founded on morphological considerations, is manifest. With this view modern science is in accord.

The lectures and studies here presented are invaluable, and should find a place in every student's library. To review them more in detail would be a work of supererogation. HOWARD (New York).

BEITRAG ZU LEHRE VON DEN PSYCHISCHEN EPIDEMIEN. Von W. Wyegandt. Carl Marhold, Halle a. S.

In this short, practical monograph of 100 pages, Weygandt discusses some of the important and striking phases of psychic infection. He first gives in considerable detail the life histories of a few carefully observed cases in which mental contagion was a prominent etiological factor. Using these few cases as illustrations he further elaborates his theme, and endeavors to show how such contagion may spread from simple foci, as outlined, to at first a few friends, and then to larger masses of people, and thus give rise to widespread epidemic conditions. That such widespread epidemics have been frequent in various communities the history of the middle ages amply verifies. Even in our own time, similar epidemics are not rare.

A most interesting portion of the monograph deals with the interpretation of the psychoses as seen in the histories studied. His second case he speaks of as one showing the signs, in classical fashion, of being "possessed." She would certainly have been in the middle ages an applicant for trial as a witch. In another, an involution or senile melancholia would have been interpreted in the same light.

In speaking further of those mental disturbances which seem to bear

most the stamp of a more or less defined psychosis, Weygandt states that for the most part they are best arranged under the groups of paranoid states, hysteria, and in a few instances the communicated type bears the ear-marks of a manic depressive psychosis. Naturally, in the case of twins, of members of the same family or in close relation a certain "anlage" may be posited to explain the tendency to similarity in the observed disturbance, but further one must bear in mind in the case of twins or members of the same family that what appears as a contagion may be simply the onset of a psychosis in a predisposed individual, not set up at all by reason of the contact with a similar affection in a near relative.

The monograph is not only interesting, but highly suggestive, and affords a glimpse at a mode of attack on the study of the psychology of the masses. JELLIFFE.

UNTERSUCHÜNGEN UND STUDIEN ÜBER DIE INNERVATION DES PERITONEUM DER VORDEREN BAUCHWAND. M. Ramstrom. J. F. Bergmann, Wiesbaden.

The peritoneal nerves arise partly as small branches of the intercostal and lumbar nerves which enter the lateral portions of the peritoneum, and partly by the joining of adjacent intercostal or lumbar nerves which enter in the neighborhood of the lateral borders of the recti muscles. The nerves form plexuses in the serosa and subserosa, and end in perivascular networks, in a network of non-myelinated fibers between the vessels of the serosa and subserosa, in end-bulbs and other lamellated nerve structures similar to the Vater-Pacinian bodies. This investigation shows that the phrenic nerve has nothing to do with the innervation of the peritoneum of the abdominal wall. The work is beautifully illustrated in colors.

C. D. CAMP (Philadelphia).

PHYSIOLOGY OF THE NERVOUS SYSTEM. By J. P. Morat, Professor of Physiology of the University of Lyon. Authorized English Edition. Translated and Edited by H. W. Syers, M.A., M.D. W. T. Keener & Co., Chicago.

We cannot attempt to do full justice to this large work of 676 pages in review form. All that can be done is to outline the function of the volume, to state wherein it differs from similar volumes, and how effectively the author's task has been performed.

To answer this last inquiry first, the work is an excellent one, of superior merit and integral worth. No recent modern writer has presented so lucidly and so well the current doctrines regarding innervation and its physiological consequences as has Morat. This means that, for the most part, the work differs from preceding ones of its kind, largely by reason of its newness and freshness, but further, a difference in looking at the problems is manifest. Morat's physiology is of great service to students of clinical problems of nervous and mental disorders. Stimulation or destruction of brain or cord areas is viewed in the light of the resulting physiological conditions, and treated in a simple and effective literary manner. It is an extremely useful volume. JELLIFFE.

THE PROPHYLAXIS AND TREATMENT OF INTERNAL DISEASES. Designed for the Use of Practitioners and of Advanced Students of Medicine. By F. Forchheimer, M.D., Professor of Theory and Practice of Medicine, Medical College of Ohio, Department of Medicine of the University of Cincinnati, etc. D. Appleton & Co., New York.

"In order to get the best therapeutic results," says Dr. Forchheimer, in

concluding his introduction, "the physician should be both scientist and practitioner." This sentence epitomizes the spirit of Dr. Forchheimer's book, in which one recognizes the voice of a master in the art of therapeutics, who is quick to recognize and use every aid which science has to offer. This work admirably fulfils its aim in presenting to the practitioner a working system which he can apply in private practice, leaving the consideration of such measures as require hospital treatment to other works.

In this systematic consideration of internal diseases, prophylaxis receives the same broad treatment that is bestowed upon the curative measures. The important hygienic measures which apply to the prevention of specific diseases in the community are outlined, and the subject of personal prophylaxis is considered in some detail. That "to the bacterial cause (of disease) we must add the chemical, physical and biological causes" is not lost sight of in the treatment of this most important subject.

In describing the treatment of each disease, Dr. Forchheimer first outlines the method which has proved most serviceable in his own hands, and then describes other measures which may be found of value, giving considerable attention to measures other than drugs, such as diet, hydrotherapy, exercise, etc. Complications and sequelæ are fully considered, and the care of the patient during convalescence is most satisfactorily described—a subject which is largely neglected in the great majority of textbooks on medicine. The author has made free use of the best literature, particularly in reference to those subjects which are new or which are still under discussion.

The convenient arrangement and full index make this work a very handy reference book, and in referring to it the reader may feel that he is appealing to very high authority. We know of few books which will prove as valuable to the advanced student of medicine or the practitioner as "The Prophylaxis and Treatment of Internal Diseases."

HOWARD (New York).

EPILEPSY. William Aldren Turner, M.D., London, England. 267 pages. The Macmillan Company, Limited, London and New York.

"Epilepsy—a Study of the Idiopathic Disease," by Turner, is based largely on Dr. Turner's experience as physician to out-patients at the National Hospital for Paralyzed and Epileptic, and as visiting physician to the Colony for Epileptics at Chalfont, St. Peter's. It is divided into twelve chapters in addition to appendices and an index.

Chapter I. deals with a definition, general considerations, inheritance, conditions underlying convulsions, and speaks of epilepsy as "an organic disease." Turner's definition of epilepsy embraces ninety words and seems unnecessarily cumbersome and to contain features that have no place in what purports to be a definition only. Turner defines epilepsy as "A chronic, progressive disease of the brain characterized by periodic occurrence of seizures in which loss of consciousness is an esential feature, commonly associated with convulsions and frequently accompanied by psychical phenomena of a well defined type; occurring generally in persons with a hereditary neuropathic history which shows itself in signs or stigmata of degeneration; running its course uninterruptedly or with remissions over a number of years, and terminating either in a cure, in the establishment of the confirmed disease, in delusional insanity or in dementia." The last lines seem to encroach decidedly upon prognosis and to have no bearing upon definition.

Chapters II. and III. deal with the etiology of epilepsy, the general

prevalence of the disease, its relative frequency in the sexes, and the age at onset.

Chapters IV. and V. take up the clinical side of epileptic fits; and here Turner seems to follow Binswanger in the classification of types. Much may be noted in this chapter similar to the writings of Gowers.

Chapter VI. deals with the mental states found in epilepsy, the epileptic temperament, and the various psychoses that precede, accompany or follow epileptic convulsions.

Chapter VII. deals with the miscellaneous phenomena of epilepsy, including its neuropathic associations, such as paroxysmal headache, chorea, the tics, myoclonus epilepsy and chronic nervous diseases; accidents due to epileptic fits, conditions that have been fully described in other works on this disease.

Chapter VIII. deals with pathology. A careful study reveals nothing that is strikingly new; yet the chapter is well and conservatively written, and seems up to date in all respects. "Changes resulting from epileptic attacks" are found in this chapter. Turner states the brain of an epileptic is "unusually bulky, its convolutions simple and its membranes to the naked eye are clear and not apparently thickened; they are not adherent to the cortex and are congested only in cases which die in the status epilepticus." The statement that the brain of an epileptic is "unusually bulky" and its "convolutions simple" does not seem to us to be borne out by facts observed in dead-houses in this country, where hundreds of epileptic brains have been studied.

Chapter X. takes up the diagnosis of epileptic convulsions, points the way in which they are to be distinguished from hysterical fits, from epileptiform convulsions and from aural vertigo.

In Chapter XI., prognosis and curability are well discussed. It is evident that Turner takes a more generally optimistic view of the curability of epilepsy than do most neurologists in this country. He gives the percentage of cures at ten to twelve, and states that no case is to be regarded cured until the attacks have been arrested for at least nine years. Just why Turner fixes upon this seemingly arbitrary period of nine years we do not quite understand.

The last chapter deals with treatment, taking up the bromids, the methods of their administration, as well as numerous other remedies. Prophylaxis is not overlooked.

Appendix "A" deals with incidence and mortality of epilepsy among European troops, native troops and prisoners in India for the years 1899, 1900, 1901, 1902 and 1903. So far as we have the data are new and are of more than passing interest.

Appendix "B" gives a list of "quack remedies" for epilepsy. We have known for more than fifteen years that substantially all quack remedies advertised as sure cures for epilepsy contained the bromid of potassium in some form and variously disguised. The prescriptions given by Turner of quack remedies fully bear out this assumption.

Appendix "C" comprises a list of the common foodstuffs according to their purin value.

Appendix "D" deals with epileptic colonies, describing the essential features of such communities.

Altogether Turner has written a very excellent book on epilepsy, restricted as it is to the idiopathic disease, and the volume is a welcome addition to the increasingly frequent literature on epilepsy. It contains numerous charts and illustrations. W. P. SPRATLING (Sonyea).

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HAVE THE FORMS OF GENERAL PARESIS ALTERED?*

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There has been considerable speculation in recent years regarding the variations of the forms of paresis. Many neurologists and alienists believe that the disease has undergone great modifications in types and in their proportionate frequency. Thus Paton, for example, believes that until recently the expansive form included the majority of the cases, but that now only one-tenth to one-fifth are of the expansive type; while the depressed type forms the majority of all cases, and the increase of the demented type is apparent only. Unfortunately Paton's generalization is based upon comparatively few cases.

Paresis with excitant and exalted delusions is considered by Brower and Bannister, on the other hand, as still the typical form of paresis, a statement to which most clinicians readily subscribe. The megalomanic type is held by Diefendorf (who also quotes Kraepelin to the same effect) as becoming much less prominent, until now it is encountered in the disease in less than one-fourth of the cases. The dementing form is also held by Kraepelin to be the prevailing type, forming two-fifths of all cases; while the depressed form exists in more than one-fourth

*Read at the thirty-third annual meeting of the American Neurological Association, May 7, 8 and 9, 1907.

of the cases of paresis. One gains not a little insight to our subject in Kraepelin's statement that the neurologist sees more dementing forms of paresis than the alienist on account of the absence of the grave mental symptoms which necessitate asylum care.

As a representative of the English view one notes with interest that Clouston holds that one-third of paresis belongs to the dementing form and that all the older physicians in asylums believe the type is increasing at the expense of the grandiose type. However, it is interesting to note that not a few English writers fail to diagnosticate paresis in the absence of euphoria during some stage of the disease, a view largely dependent upon Mickle's teaching two decades ago.

Italian, French and Russian alienists make no extended comment upon the modern views of variations of type in paresis. Indeed, it may be said that not many writers of to-day in any country even, make anything like a genuine attempt to differentiate types or forms of paresis, hence our task of a wide geographical interpretation must be somewhat imperfect. Our own experience in hospital, dispensary and private practice led us at first to believe an affirmative answer should be returned to the query title of this paper.

On account of the varying opinions respecting the types of paresis and their proportional frequency, we have endeavored to dispel the present confusion of the subject and bring the whole subject up for general discussion here by making a careful analysis of 3,000 cases of paresis covering the period of the last three decades. The material under immediate study was drawn from the asylums of the New York Metropolitan district at Ward's Island and Central Islip Hospitals. Only male cases were considered in the study.

We have found it practicable to subdivide paresis in but three forms: grandiose, depressed and simple dementing. The grandiose form embraces all cases in which euphoria and expansive delusions obtained, whether attended by motor restlessness or not. The depressed includes all cases that have exhibited depression and excessive emotional element, depressive delusions or hypochondria throughout the major part of the disease. The simple dementing form includes those cases that show primary progressive deterioration without further mental symptoms the existence of

which would permit their being placed in either of the other two types.

Our analysis naturally possesses the advantage of covering the entire course of the disease, as all the cases were under asylum care and the histories were very complete, and the diagnosis was confirmed by death in the majority of cases. One appreciates the advantage of viewing the whole course of the disease in classifying types of paresis, inasmuch as the grandiose complex is not infrequently established late in the course of the disease. It often suffers a preliminary cloaking of hypochondriacal depression. Again, etiological factors other than syphilis, such as alcohol, very often tinge the prodromes of the disease, giving rise to persecutory ideas, ideas of marital infidelity, etc.

The gradual increase of the simple dementing form during the last few years shows that some cases of cerebral lues are finding their way into this class. Many simple dementing paretics of earlier years were, moreover, lost in the former classification of terminal dementia. Not a few cases in old records show that acute melancholia terminating in dementia should have been classed as the depressive type of paresis. All these facts explain the natural numerical advance of this form of paresis.

The same explanation can not, however, be urged of the acute maniacal phases of insanity, as the agitated types of paresis, for which they might be mistaken, usually present the wildest extravagance of euphoria.

In considering our analysis by year periods one finds no distinctive feature in the curve-charts. Although there is considerable variability from year to year in the thirty-year period we have analyzed, there is no constant law deducible. If one groups the data in *five-year* periods, however, there appears to be a fairly constant ratio between the total admission, the total paretic class and the total grandiose type; in other words, the ratio is constant between the cardinal features of the disease study. This fact alone teaches us that the grandiose element in paresis is the true disease complex. The principle may be considered so firmly established that no immediate change in it may be looked for in the future. (Time may be saved if the charts are allowed in the main to speak for themselves.)

We found the euphoric syndrome in recent years less extrava-

gant, less grotesquely exalted. Patients in the last decade spoke of possessing, or hoping to possess, a few thousand dollars, instead of having billions of trillions as formerly. The frequent persecutory ideas of the grandiose state have a curious exalted trend and are strangely mixed with true euphoric concepts, even in the same sentence.

A percentage analysis of the grandiose type by five-year periods shows that there was a steady increase from 1877 to 1896, from which date there has been a gradual decrease to date. Fully 70 per cent. of paretics are of the grandiose type to-day. Even in mild euphoria the manner of grandiose reasoning in paresis is as characteristic as that encountered in the persecution of paranoia.

On the whole one may say that the depressed type of paresis has steadily decreased in frequency since the first five-year period (1877-81), at which time it was 15 per cent. of the total paretic class. It reached its minimum frequency in 1892-96, when it was about 10 per cent. It has increased slightly in the last ten years. During the last five years it has stood at about 12 per cent. of the total paretic data.

It is interesting to note in passing that while Bayle in 1822 first recognized paresis as a morbid entity characterized by ambitious delusions which he believed to be pathognomonic, it remained for Baillarger of Paris, nearly forty years after, to show that the depressive syndrome might exist in certain forms of paresis. The French school exemplifying the old adage, still persists to-day in making no provision for these cases in paretic classifications. There is often a strangely enlarged view even in the depressive phase of paresis. Patients believe they have murdered all the people in the world, have killed all the numerous members and relatives of their family, etc.

The greatest period of frequency for the simple deteriorating class of paresis was in the first five-year period of our analysis (36 per cent.), since which time there was a steady decrease until during the last ten years it has remained constant at about 17 per cent. As the type runs a rapid course, without remissions, and almost invariably ends in convulsions, the majority of the cases remain outside of asylums until late in the disease. Indeed, the greater part of the disease course is an extra-asylum state. This fact accounts somewhat for the opinion among neu-

rologists that the simple dementing form is gaining in frequency at the expense of the grandiose type. Difficulties in diagnosis here, as elsewhere in paresis, are largely removed if one depends more on the somatic signs than on any mental criteria.

In conclusion it may be said but three types are needed for analysis of paresis, and variations between types of late years are less great than formerly held.

The true explanation of the occurrence of a considerable number of depressed and simple dementing types of paresis rests upon a more exact analysis of cases and a recruiting of these two classes from the melancholias and dementias of former years. The real but slight variations in types are due to a better system of treatment and earlier diagnosis. Moreover, the specific treatment of syphilis, in paresis, has largely been discarded for more rational principles of hydrotherapy, dietetics and hygienic surroundings, both in and outside of asylums. The more prompt detection of the disease has made paresis a younger disease; more cases occur between 20 and 30 than formerly and fewer occur over the age of 50. In whatever light paresis is viewed we can hope for but little variation in the disease, inasmuch as the fixed and definite causes of paresis are syphilis, alcohol, sexual excesses and mental stress. We believe the relative percentage of the various types should be more generally expressed in textbooks, in order that readier comparisons and deductions may be made.

The whole subject under study here is far from being merely academic. The different types of paresis have a widely dissimilar prognosis. The determination of the true syndrome of the disease and its atypical forms is, therefore, a very practical and timely issue.

Finally, from our study of 3,000 cases we deduce that paresis is essentially a disease in which the grandiose type predominates in about 70 per cent. of all cases, the dementing form occurs next in frequency of 20 per cent., while the depressive form is found in but about 10 per cent.

THE SYMPTOMATOLOGY OF LESIONS OF THE LENTICULAR ZONE WITH SOME DISCUSSION OF THE PATHOLOGY OF APHASIA.*

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INTRODUCTION.

The views on aphasia recently advanced by Marie have served, among other things, to concentrate the attention of physiologists and neurologists upon the functions of the lenticular nucleus, to which body Marie has given an unusual role in the production of motor aphasia. It is a noteworthy fact that, in spite of the neurological activity all over the scientific world during the last three or four decades, the functions of the basal ganglia still remain largely undecided. Many speculations have been advanced regarding the functions of the thalamus, caudatum and lenticula; and many facts, anatomical, physiological and clinicopathological, have been contributed regarding these organs; but the comparative certainty of our knowledge as to the functions of the cerebellum, most of the cortical fields and centers, and many of the encephalic tracts of projection and association, is lacking with regard to these ganglia. The reasons for this state of affairs are not hard to see. Owing to the deeply seated position of the basal ganglia, it is difficult to experiment on them without involving or injuring other parts; and the lesions which are observed by the clinicopathologist are rarely absolutely limited to any one of these organs. Microscopical investigations of degenerations of these ganglia and of their incoming and outgoing tracts have not led to as clear inferences as in other regions of the brain.

Our paper is based in the first place upon a study of eleven cases with necropsies, in ten of which the lesion involved the

*Read at the meeting of the American Neurological Association, May 7, 8 and 9, 1907.

lenticular zone. In six of these cases the lenticula itself was involved. Other parts of the lenticular zone were variously implicated in the ten cases. It was possible, however, at least in some of the cases, in which regions other than the lenticula were involved, to arrive at an idea as to the part played by this body in the symptomatology presented by the patients.

For several years the writers of the paper have been collecting cases with necropsies in which lesions of the basal ganglia have been present. Most of the specimens from these cases are to be found in the Laboratory of Neuropathology of the University of Pennsylvania. In not a few instances papers have been published in which references have been made to some of these cases, and some use will be made of these published but personal data. In going over the specimens of cases, clinical and pathological studies of which have not yet been made, the writers found that they had in their possession more than twice as many specimens of cases as have been used in this paper, but owing to the imperfections of the clinical data, or because of the confusing character of the lesions, only eleven of these cases could be used with any advantage.

OLDER VIEWS REGARDING THE FUNCTIONS OF THE LENTICULA.

We shall first note briefly the views regarding the functions of the basal ganglia held by some of the older physiological and neurological teachers.

Fifty years ago one of the most relied-upon general works in physiology was that of Carpenter.[1]

An old view which is discussed but antagonized by him was that the thalami in some way were centers for movements of the upper extremity, and the striata for the lower. Carpenter also opposes the idea of Magendie that removal of the striata causes an irresistible tendency to forward progress, whilst the division of the peduncles of the cerebellum occasions the reverse movement.

Discussing the functions of the striatum, Carpenter says that, "According to Longet, Schiff and Lafargue, the results of re-

[1] "Principles of Human Physiology," by William G. Carpenter, M.D., F.R.S., F.G.S., Examiner in Physiology and Comparative Anatomy in the University of London, etc., fifth American from the fourth and enlarged London edition, Philadelphia, 1853.

moval of the corpora striata with the anterior upper part of the cerebral hemispheres, are for the most part negative; for the animal usually remains in a state of perfect stupor, although still retaining the erect position; and it is only when irritated by pinching or pricking that it will execute any rapid movements. No mechanical irritation of the corpora striata produces either sense of pain or muscular movements. No distinct evidences regarding the special functions of either of these ganglionic masses can be gained from pathological phenomena. So far as is yet known extensive disease of either the thalami optici or the corpora striata of one side, produces hemiplegia or paralysis both of sensation and motion on the opposite side. The same result very commonly follows an apoplectic effusion into the substance of either."

Broadbent,[2] writing in 1876, voiced the views which were held by many about the time and shortly after the time when the study of cerebral localization received its greatest impetus through the suggestions of Hughlings Jackson (1864) and the researches of Hitzig, Fritsch, Ferrier and others (1870-1875).

Discussing the position of the thalamus and striate bodies, with reference to the crusta and tegmentum, after indicating the existence of connections between these ganglia and the other parts of the nervous system, both lower and higher, he expresses the opinion that the striatum is a motor and the thalamus a sensory ganglion for the entire opposite half of the body. With regard to the striatum, like all the earlier writers not separating in his discussion the caudatum and the lenticula, Broadbent holds that this ganglion translates volitions into actions and puts into execution the commands of the intellect. "It selects, so to speak, the motor nerve nuclei in the medulla and cord appropriate for the performance of the desired action, and sends down the impulses which set them in motion. These impulses are transmitted through fibers, and the fibers must start from cell processes in the corpus striatum. A given movement, therefore, must be represented in the corpus striatum by a group or groups of cells giving off downward processes, which become fibers of the motor tract of the cord." When the movement is simple the cell group will be small and the fibers few; when complex the cell group

[2]Broadbent, Wm. H. British Medical Journal, April 1, 1876.

will be large and well defined and the descending fibers numerous.

"Words," says Broadbent, "which require for their utterance the simultaneous co-operation of the muscles of the chest, larynx, tongue, lips, etc., and the exquisite and rapid adjustment of their movements concerned in phonation and articulation, must be represented in the corpus striatum by very large groups of cells, and not in that of one side only but in both."

According to Ferrier[3] it is evident that destruction of the striatum produces a much more complete and enduring paralysis than destruction of the cortical motor centers alone, or of the pyramidal tracts which proceed from them.

He summarizes his views with regard to the corpora striata as follows: "It appears from these facts that the corpora striata proper are centers of innervation of the same movements as are differentiated in the cortical motor centers, but of a lower grade of specialization. The innervation of the limbs in all that relates to their employment of instruments of consciously discriminated acts is dependent on the cortical centers, while for all other purposes involving mere strength or automatism, primary or secondary, the corpora striata with the lower ganglia are sufficient. In man almost every movement has to be elaborately acquired by conscious effort through the agency of the cortical centers, and continues to involve the activity of these centers to a greater or less extent throughout. Hence the destruction of the ganglia of the corpora striata adds little if anything to the completeness of the paralysis which results from destruction of the cortical motor centers alone."

Ross[4] has contributed something to our more exact knowledge of the motor functions of the lenticula in a paper on what is now commonly designated as pseudo-bulbar paralysis, the title of his contribution, however, being labio-glosso-pharyngeal paralysis of cerebral origin. He cites a case from Oulmont[5] with the typical symptoms of pseudo-bulbar paralysis in which the motor and speech phenomena were apparently due to bilateral lesion of the lenticula.

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[3]"The Functions of the Brain," by David Ferrier, M.D., second edition, rewritten and enlarged, New York, 1886.

[4]Ross, James. Brain, July, 1882.

[5]Oulmont. Cited by Lépine, R. Mensuelle de Médecine et de Chirurgie, Vol. 1, 1877.

"An old hemorrhagic focus was found in the right hemisphere occupying the third or external segment, and a smaller focus situated in the second segment of the lenticular nucleus. Similar foci were found in the third and second segments of the lenticular nucleus of the left hemisphere. The internal capsule was not involved in either hemisphere. The medulla oblongata and pons were free from disease."

Ross cites a case of Eisenlohr[6] in which lesions were found in both striata (the lenticular nuclei?) and in the thalami.

Ross also cites the case of Kirchhoff,[7] published in 1881, in which lesions of both the caudatum and lenticula were found, but as the capsule also seems to have been involved in this case, it is not of as much value for our purposes as other cases in which the lenticula alone seems to have been implicated.

One of the most important cases of labio-glosso-pharyngeal paralysis or pseudo-bulbar paralysis from disease of the lenticula which has as yet been described is that reported by Ross himself. In this case the lesions described were as follows:

"The lenticular nucleus of each hemisphere was found post-mortem replaced by a well-defined cystic cavity, containing a clear, straw-colored fluid, the internal capsules being apparently uninjured. Microscopic examination failed to detect any evidence of disease in the nerve nuclei of the medulla oblongata, or descending changes in the pyramidal tracts in any part of their course."

In another case recorded by Ross the lesions present were described as follows:

"The anterior part of the lenticular nucleus of the left hemisphere presented two small cyst-like cavities, containing clear fluid. These cavities occupied the second and third segments of the nucleus, and one of them appeared to encroach to some extent on the knee of the internal capsule. The right hemisphere was preserved in spirit, and was not examined until a fortnight later, when a small cavity was found in the lenticular nucleus of this hemisphere also. This cavity occupied the anterior part of the nucleus, but did not appear to encroach upon the internal capsule." Some descending degeneration, probably due to lesion in the internal capsule, was present in this case.

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[6]Eisenlohr, C. Archiv. für Psychiatrie, Vol. IX, 1878, p. 43.
[7]Kirchhoff. Archiv. für Psychiatrie, Vol. XI., 1881, p. 132.

In a third case recorded by Ross a small cystic cavity was found in the left lenticular nucleus close to the border of the genu of the internal capsule. No secondary degeneration detectable by the naked eye was in the crura. A careful microscopical examination of successive sections of the crura, pons, and medulla oblongata, failed to detect any secondary degeneration of the pyramidal tracts. The bulbar nuclei were healthy. This case, which was complicated with disease of the spinal cord, presented typical pseudo-bulbar paralytic symptoms.

Ross held that his cases did not prove that the lenticula is an independent center for the regulation of all movements of articulation and deglutition. He was also inclined to negative the view that the lenticula is a ganglion of interruption between the cortex and the parts below, and looked with favor on the idea that in the cases of labio-glosso-pharyngeal paralysis recorded by him the symptoms were due to lesions interfering with the paths of conduction from the cortex, although at least two of his three cases seem to have been instances of lesions isolated to the lenticula, these causing disorders of speech and some paresis of the face and upper extremity. His view would appear to be in a general way that of Dejerine that the cases of pseudo-bulbar paralysis attributed to lesions of the lenticula, either unilateral or bilateral, are probably dependent upon undetected lesions of the pyramidal tracts or cortex.

THE VIEWS OF DEJERINE.

In his Anatomy of the Central Nervous System[8] Dejerine in several places goes rather fully into the question of the functions of the lenticula, but his results would seem to be included in the assertion that this ganglion plays only a negative part. He is mostly concerned with denying its connection with the motor and other portions of the cortex by projection fibers, appearing to hold that the lenticula plays no part in the control of movement or in the function of speech. In order that his views with regard to the functions of the striate bodies may be understood, it will be necessary to include a reference to his opinions as to the anterior limb of the internal capsule. Lesions of the anterior seg-

[8]Dejerine, J. "Anatomie des centres nerveux." Vol. II., fasc. 1, Paris, 1901.

ment of the internal capsule, says Dejerine, are not manifested by any appreciable motor disturbances when they occupy only the anterior half or three-quarters of this segment. These fibers are, he believes, frontothalamic projection fibers. He says that as soon as the knee of the capsule is reached, a distinct symptomatology appears,—that of paralytic phenomena on the opposite side of the body. Secondary degeneration of this part of the capsule is also seen following cortical lesions of the Rolandic zone. Dejerine has shown that the area of degeneration in the anterior portion of the posterior limb of the capsule is correspondingly more posterior, according as the lesion is higher and higher in the Rolandic cortex, his own observations confirming in this respect the physiological observations of Horsley and Beevor on the internal capsule of the monkey.

When the knee and the adjacent portion of the posterior limb of the internal capsule are involved in a lesion, a paralysis of the inferior facial nerve and of the half of the tongue on the opposite side is observed. The fibers arising from the frontal operculum and Rolandic operculum pass through the knee and the adjacent portion of the posterior segment of the capsule. Facial paralysis of capsular origin presents the same features as that of cortical origin, the superior facial is not entirely intact.

When the lesion is bilateral and symmetrical, the clinical picture will be that of pseudo-bulbar palsy. The anatomical localization of the paralysis has been and still is differently interpreted. It is known that this may be of cortical origin (bilateral lesion of the Rolandic operculum), subcortical, capsular, pontile, or bulbar, situated in the course of fibers arising in the motor zone which control the nuclei of the motor cranial nerves. Dejerine does not accept the localization of those who have their views based upon cases in which the lesions were supposed to be limited to the lenticula. In one place he says emphatically that the lenticular nucleus does not send fibers into the foot of the cerebral peduncle and does not receive any from the cortex. Further on in the same work, however, he modifies this statement by saying that the *putamen* does not receive any fibers from the cortex, but that the globus pallidus may do so. The lenticular fibers of projection, he holds, belong to the system of strio-thalamic and subthalamic radiations.

Dejerine states that symmetrical lesions (hemorrhage, soften-

ing, either in the putamen or in the lenticular nucleus as a whole) are frequently observed in the necropsies of the aged, who have not presented any symptoms of pseudo-bulbar paralysis during life. He recalls the fact that Oppenheim and Siemerling had shown in 1886 that when they observed a bulbar palsy during life in these cases of symmetrical lesions of the lenticula, there were always also foci of softening in the pons and oblongata. According to Dejerine in all the cases in which there was absence of all pontile or bulbar lesions appreciable by the microscope, the pseudo-bulbar palsy seems to have resulted not from the localization described as lenticular by some; but when these cases are studied by the method of serial microscopical sections, the bilateral and symmetrical lesions of the lenticula are found always to have involved the knee and a portion of the posterior limb of the internal capsule.

He speaks of tracing clearly in the retro-lenticular portion of the internal capsule fibers which traverse the globus pallidus. He says that these may appear to be cortico-lenticular fibers, but suggests that inasmuch as they cannot be followed in the corona radiata, except immediately above the retro-lenticular segment, they may rather be lenticulo-caudate fibers, that is caudate fibers of projection or termination.

Dejerine asserts that in cortical lesions, either extensive or confined to small areas, when the material is treated by the method of Weigert and Pal, the globus pallidus does not appear to undergo any modification, either in the form of atrophy or disappearance of fibers, similar to that which is seen in the thalamus in the same case. He acknowledges, however, that we cannot assert that the globus pallidus possesses in such cases as many radiating fibers, or as dense a network of fibers as in the normal state.

It is further stated by him that in two cases of recent cerebral disease, in one of which the lesion was extensive and in the other limited, examined by the method of Marchi, the globus pallidus and the corpus Luys both received fibers of projection from the cerebral cortex.

In still another place Dejerine states that the striatum, especially the caudate nucleus and the putamen, at least in man, do not receive direct cortico-striate fibers; that they receive only cortical fibers of projection traversing them on their way to the

internal capsule (cortico-thalamic, cortico-pontile, cortico-bulbar, cortico-spinal fibers). It seems to be well demonstrated, according to Dejerine, that cortical lesions, whatever may be their situation, their extent, and their duration, do not affect the caudatum and the putamen as they do the thalamus. This contrast existing between the striatum and the thalamus, says Dejerine, was well recognized by Gudden in 1872 as the result of experimental observations. Other well known investigators cited by him have observed atrophy of the striatum following cortical ablation experiments, but this atrophy results probably from the degeneration of fibers of passage and collaterals or injured cortical neurones, and is not comparable with the atrophy, even extreme, in the thalamus under the same circumstances. The very slight diminution in size of the striatum sometimes seen in man following old lesions of early childhood is, in Dejerine's opinion, the analogue of the diminution in volume seen in all these cases in the anterior corresponding half of the encephalon, in the anterior horn of the opposite side of the cord, and in the opposite cerebellar hemisphere. It results as much from degeneration of the collaterals which the system of cortical projection fibers sends to the striatum and the region of the tegmentum, as from the functional inactivity of this region. Dejerine[9] has expressed his views regarding the lenticula also in a recent paper.

Some others, (Schwalbe, Edinger, Sachs, Bechterew, Marinesco), Dejerine remarks, deny the existence of cortico-striate fibers of projection, but depending on the homology of development of the striatum and of the cerebral cortex established by Wernicke, attempt to prove the existence of a cortico-striate fasciculus of association, and as such describe the occipito-frontal fasciculus and collosal fasciculus of Muratoff.

THE VIEWS OF MINGAZZINI.

In the appendix to his monograph, Mingazzini[10] discusses and antagonizes the opinions of Dejerine.

The arguments of Dejerine, says Mingazzini, appear far from decisively proving that the lenticular nucleus does not possess

[9]Dejérine J. L'Encephale, No. 5, May, 1907.

[10]Mingazzini, Giovanni. "Sulla sintomatologia delle lesioni del nucleo lenticolare." 1902.

motor fibers, and that therefore a lesion of the same is incapable of producing paralytic disturbance. The argument that the secondary degenerations show that the lenticula does not send fibers into the foot of the cerebral peduncle and does not receive any from the cortex, and its fibers of projection must belong only to the system of the strio-thalamic and sub-thalamic radiations, according to Mingazzini, has little value, since he (Mingazzini) believes that he has shown in the beginning of his monograph that lesions of the lenticula, according to the points at which they occur, seem to give rise to a diverse symptomatology.

Mingazzini believes that it is not true that in every case in which lesion of the lenticula produces permanent disturbances, there must be existing concealed destruction of the internal capsule. He believes that the arguments of Dejerine might just as well be turned around; in other words, it is just as likely that lesions of the lenticula might be present and escape detection in those cases in which capsular lesions are discovered as the reverse. The arguments of Dejerine that because lesions of the pons and bulb are frequently found in pseudo-bulbar paralysis, therefore these are probably present and the real cause of the paralytic phenomena in cases apparently due to lesions of the lenticula, is easily overcome, according to Mingazzini, if we remember that the symptomatology of cerebro-pontile pseudo-bulbar paralysis is much richer than pseudo-bulbar paralysis of pure cerebral origin; in other words the picture of the two diseases is different.

Mingazzini insists on the point that only certain parts of the lenticula transmit motor and sensory fibers or have motor or sensory functions, and it is therefore easy to understand how lesions may sometimes be found in these ganglia in the old or in others without motor symptoms.

Mingazzini does not accept the opinion of Dejerine with regard to the cases collected from different authors, in which atrophy of the lenticula occurred after lesions or arrests of the cerebral lobes or convolutions, but believes that these cases show that anatomical and physiological connection exists between some part of the cortex and the lenticula. Mingazzini lays particular stress upon the researches of Dejerine, already referred to in our analysis of Dejerine's views, in which he found degenerated fibers in the globus pallidus as the result of cortical lesions, since Min-

gazzini says it shows that these ganglia receive fibers of projection from the cerebral cortex.

Leaving his criticisms of Dejerine, let us turn now to Mingazzini's positive statements of his own views based upon personal clinico-pathological observations, his pathological findings being both gross and microscopic. In his monograph he first calls attention to the manner in which the question of the function of the lenticula has been largely passed by, or has been altogether slighted by writers on neuropathology and clinical neurology. He refers particularly in this connection to well known authors like Gowers, Oppenheim, von Monakow, and Brissaud. Unlike Dejerine, who attacks the problem of the functions of the lenticula in his great work on the anatomy of the nervous system, with the result of negativing its functional importance, the other authors referred to by Mingazzini have contented themselves with slight or imperfect allusions to the problem of lenticular disease.

Mingazzini, in this monograph, gives details of nine cases of lenticular lesion with necropsies observed by him, and refers to other cases in a preceding publication. We have gone carefully over the notes of these cases, and also the table in which the topography of the lesion and the disorders of articulation and of motility in the face and limbs are summarized. These cases would seem to leave little doubt of the correctness of the conclusions drawn by Mingazzini. One criticism is worth mentioning, however, regarding some of them. Several of these patients were quite advanced in years; some over seventy and others between sixty and seventy. In several of them also abnormal conditions of the meninges and cortex or subcortex were present, as opacities of the membranes, adhesions with decortications, shrinkage of the convolutions, and in one or two instances, actual softening. Making, however, due allowance for these findings and their possible bearing upon the symptoms in limbs, face and speech, we believe that the most important of Mingazzini's conclusions are to be accepted. We shall give these somewhat at length, translating and summarizing his remarks, omitting quotations, as in order to save space we shall not follow his language consecutively and with exactness.

A focus of disease, even of small size, involving only the lenticular nucleus, according to Mingazzini, never fails to mani-

fest itself with motor disturbances. Usually these are dissociated or total paralyses, to which are sometimes added irritative symptoms.

Facial paralysis is very rarely of lenticular origin. Mingazzini has not observed any case of it. He refers to the one recorded by Ross, to which we have already directed attention. Cases of unilateral facio-lingual paralysis and of bilateral facio-lingual paralysis such as is frequently seen in pseudo-bulbar paralysis without involvement of the limbs are also rare. Mingazzini gives three instances of what he regards as facio-lingual paralysis of lenticular origin. In the first the putamen on the left and in the second the putamen on the right in the neighborhood of the genu was softened; in a third a focus of softening was present in the globus pallidus. Mingazzini refers in this connection to the cases of Lépine and Halipre[11] which with others have been criticised by Dejerine.

Mingazzini refers to a form of paralysis intermediate between the pure facial and the brachio-facial paralysis which he had previously reported. The patient presenting this symptom-complex, besides having paralysis of the inferior portion of the right facial, was affected with athetoid movement of both hands. Many lacunar foci were found in the left putamen.

A single example of pure brachio-facial paralysis is recorded. The case was that of a patient affected with paresis of the seventh nerve and of the superior limb of the right side following softening of the anterior portion of the left lenticula.

Facio-crural paresis is very rare indeed. Mingazzini says that he has never seen a pure case of this dissociated paralysis, but refers to a case previously recorded by him in which was present oscillatory tremor of the left superior limb with paresis of the left inferior facial distribution and the left lower extremity. Two foci of loss of substance as large as a millet seed were found; one in the external part of the right putamen and the other in the external member of the globus pallidus. In two cases Mingazzini has observed an irritative paretic disorder of the upper extremity from partial destruction of the lenticular nucleus on the opposite side. In one of these cases the limb affected was spastic; in the other it was the seat of clonic move-

[11]Lepine, R. Op. cit.

ments. In another case in which softening was found in the middle part of the left putamen, the patient was seized occasionally with clonic movements of the hand and the forearm of the opposite side.

While the forms of hemiplegia, with or without irritative phenomena, are sometimes present in lesions limited to the lenticula, the more frequent syndrome, according to Mingazzini, is motor paresis of the entire half of the body. He records five such cases in the monograph we are analyzing and refers to others previously recorded. The paralytic disturbances often pass without being observed by the patient, says Mingazzini. They apply to the physician complaining of paresthesia, he continues, or of pain along the paretic limbs, or else of vertiginous conditions, or weakness of memory, symptoms evidently due to a diffuse cerebral arterial sclerosis. It is only a minute objective examination, he says, which will reveal certain hemiparesis. While the attack of paralysis may be ushered in by an apoplectic stroke with unconsciousness, the symptoms accompanying this are not so severe and persistent as in ordinary cerebral hemorrhage. The patients are often syphilitic, and probably because of this the attacks are frequently preceded by headache and febrile symptoms. As indicated, much of the paralysis disappears, but a residue can always be found by a careful examination. The paresis or paralysis of the facial is almost always limited to the inferior distribution of the nerve. The superior extremity assumes peculiar attitudes. The forearm tends to bend on the arm, and the hand on the forearm, but these conditions or attitudes are only rarely spastic in character, as is observed in capsular lesions. This point seems an important diagnostic one to the writers who have observed cases presumably of lenticular lesion in which the non-spastic limbs assumed special attitudes, simulating those seen in spastic hemiplegia. The three portions of the upper extremity are usually about equally affected with the impairment of power. Gross oscillatory tremors may result from attempts to use the limb. In the lower limb, as in the upper, the paresis is usually about equally distributed in its three parts. It is rarely, if ever, a complete paralysis. The patient feels as if his paretic leg would give way; sometimes he can walk and sometimes not; when he walks his steps are slow and short. The leg of the paretic side is a little flexed on the thigh, the foot striking the

ground, without, however, performing the classic movement peculiar to capsular lesions.

Mingazzini lays stress upon the fact that the majority of his cases with the syndrome of hemiparesis could be referred to softening of the putamen, situated near, and in correspondence with, the anterior limb of the internal capsule and not with the posterior portion. The lesions being somewhat removed from the pyramidal tracts, therefore their effects when these lesions are hemorrhagic cysts cannot be attributed to pressure upon these tracts. In addition, Mingazzini points out that in most of his cases the lesions were foci of softening which did not exercise pressure on neighboring parts. A still further point in favor of the paralytic disorders being due to lenticular lesions and not to pressure exerted upon the capsule is the fact that these affections, even if they are not marked, are persistent and continue until death. Although partial paralyses, they are not transitory.

The motor function of the lenticula, says Mingazzini, deserves to be called supplementary if it is borne in mind that the motor disturbances in comparison to those produced by the destruction of the pyramidal region of the internal capsule are very mild. Mingazzini does not attempt to decide whether the motor fibers which originate in the lenticula join with those of the internal capsule; whether the neurones of which they are part originate in the motor cortex or in the lenticula itself; and whether if they originate in the lenticula they come in contact with neurones derived from the cortex. These are matters for future research to determine, especially by the studies of experimental and other degenerations. His claim is simply that the lenticula is a motor organ of some sort; that it possesses real motor function. He refers to Johansen's faradic excitation of the lenticula as confirming his own results and views.

Disturbances of speech, according to Mingazzini, are often associated with lesions of the lenticula. He says with some reserve that he believes the dysarthric disturbances dependent on lenticular lesions arise only when these lesions are situated on the left side. He has recorded cases in which the dysarthric disturbances were absent even when the lenticula was injured on both sides. His explanation of such observations is that the fibers destined for the movements concerned in speech run in circumscribed zones, as it is true that the fibers destined for the face, the

upper and lower limbs, run also in circumscribed zones; in other words, that the motor zone of the lenticula is subdivided as is the cortical motor zone.

Discussing in detail some of his observations regarding speech, Mingazzini says that disturbances of articulation under the form of elisions, initial pricking and bad pronunciation of the dentals were signalized in one case in which the external member of the globus pallidus on the left was destroyed; and in another case in which there was a softening of the left putamen. But it was impossible to find any disorder of speech in still another case in which there was a hemorrhagic focus on the external margin of the right putamen. Disturbances of articulation were found in a case in which there was a loss of substance which occupied the more external limit of the left putamen, on a level with the middle part of the colliculus caudatus; and these were not found in another case in which the hemorrhagic focus occupied the medial and inferior part of the globus pallidus of the left; and not in still another case in which the lesion was in the base of the putamen and in the middle part of the external capsule of the left. These results demonstrate again that the lesions of the right lenticular nucleus have no effect on speech, and that those of the left lenticular nucleus give rise to disturbances of articulation only if situated at fixed points.

VIEWS OF SCHÄFER, VON MONAKOW AND OTHERS.

Coming to one of the most recent physiological works, the views of Schäfer,[12] regarding the functions of the striatum (lenticula and caudatum) are stated in the following manner:

"Cajal describes axones of many of the pyramidal cells of the frontal lobe as passing to the corpus striatum. After destruction of the frontal lobe in dogs and monkeys, Marinesco found numerous degenerated fibers in the corpus striatum and especially in the caudate nucleus. On the other hand, the corpus striatum receives numerous fibers from the thalamus and the sub-thalamic region, and probably sends out centrifugal fibers downwards, along the motor tract; but these have not hitherto been satisfactorily traced; some appear to go to the substantia nigra. The

[12] "Text Book of Physiology," edited by E. A. Schäfer, LL.D., F.R.S., Vol. II., Edinburgh and London, 1900.

corpus striatum is generally believed to act as a center for the higher reflex movements, and to be in close association with the Rolandic area, but the experimental grounds for this belief are still lacking. Morphologically, the corpus striatum is regarded as a part of the cortex."

Von Monakow[13] says that a lesion taking in almost the entire caudate nucleus, leaving the internal capsule free, or even the lenticular nucleus, does not of necessity cause a persistent hemiplegia. He continues that Reichel[14] has shown that a symmetrical softening of both lenticular nuclei may occur without clinical manifestations. As a rule, hemiplegia with the involvement of the facial and hypoglossal occurs in extensive lesions of the caudate and lenticular nuclei. It is seldom complete and only persistent when the internal capsule, that is the premedial portion of it, is implicated. Some areas of softening, either in the lenticula or in the caudatum, usually are latent. Irritating foci, for example tubercles, cause occasionally early hemiplegic contraction. Aside from the hemiplegia,—though together with this in connection with lesion of the caudate and lenticular nuclei—occasionally occur hemianesthesia, tremor, forced laughter, choreiform movements, vasomotor disturbances (elevated temperature, reddening of the skin, etc.) and central pain in the opposite half of the body, especially in the arm. But all these phenomena, as a rapid glance through the literature on focal diseases shows, may occur without direct lesions of the ganglia of the forebrain. Occasionally they appear following lesions in the neighborhood of the posterior internal capsule, in the posterior portions of the thalamus, and in the tegmental region. Nothnagel, for instance, says von Monakow, attributes vasomotor disturbances accompanying hemiplegia to involvement of the posterior internal capsule. Numerous observations speak, however, for the closer relation between elevation of temperature in the paretic extremities of one half of the body and disease of the caudate nucleus in the opposite half brain. Experimental investigations also are favorable to this view. We know that irritation of the caudate nucleus in animals may be accompanied by pronounced, though transitory, elevation of temperature. Various investigators have observed similar elevations of temperature on the opposite side to the foci

[13]Von Monakow, C. "Gehirnpathologie," second edition, Wien, 1905.
[14]Reichel. Wiener Medicinischer Presse, 1898. "Diseases of the Caudate Nucleus and Lenticular Nucleus." Cited by von Monakow.

of disease in man. In such cases edema of the skin was also present in the extremities. In all these cass th lesion, even microscopically, was not limited strictly to the caudatum.

Choreiform movements, says von Monakow, were years ago associated occasionally by this or that investigator with disease processes in the caudate or the lenticular nucleus. Ellischer, Flechsig and others have seen in those who have been affected with chorea and have died, peculiar, strongly refractive, often mulberry-shaped, bodies in the lymph sheaths of the vessels in the portions of the brain mentioned. Wollenberg found similar formations in the lenticula of persons who had not suffered from chorea, and therefore disputes the causal relation between chorea and these bodies. Hebolt observed tremor in the extremities in a case of lesion of the putamen. Such isolated observations, according to von Monakow, are of little value so long as they relate to the chance occurrence of focal disease. At all events, he says, a uniform relation has not yet been established between choreiform movements and disease processes limited to the caudate or lenticular nucleus.

Deroubaix,[15] in a paper on spasmodic laughing and weeping, reports one case with necropsy which is of great interest in connection with the pathology of this emotive syndrome, and with the general discussion of lesions of the lenticula. The patient was a man thirty-eight years old who had had a stroke causing left hemiplegia, but without any aphasia or dysarthria. He exhibited spasmodic laughter followed by weeping and accompanied by associated movements in the paralyzed left arm. Death was from a second stroke. Necropsy showed a large area of softening which had destroyed the lenticular nucleus, the anterior limb of the internal capsule, and all the white matter of the frontal lobe and of the central region, to the cortex on the right side. The thalamus, genu and posterior limb of the internal capsule were not affected.

Some of the conclusions drawn by Deroubaix are as follows: That the fibers concerned with the function of emotive expression probably pass outside of the pyramidal fibers like those concerned with the function of coordination and tonus; that the thalamus is the seat of the automatic movements of emotive expression, es-

[15]Deroubaix, M. A. Journal de Neurologie, Vol. XI., No. 5, 1906.

pecially of laughing and crying; that laughing and weeping are under the control of the cortex; that the cortico-thalamic fibers pass in front of the knee of the internal capsule, probably by the lenticular nucleus. Deroubaix therefore did not attribute the spasmodic laughing and weeping in his case to destruction of centers in the lenticula, but of fibers passing from the cortex through the lenticula to the thalamus. He speaks especially of the facio-thalamic bundle.

With regard to this explanation it is not altogether acceptable, as Mingazzini believes he has shown and as facts supplied by others indicate, that the lenticula is a motor organ and has distributed through it zones and centers of particular kind for movements of various parts of the body, including the movements connected with the expression of emotion. This being the case, destruction of such centers, especially if on both sides, would give rise to such a lack of control of the movements of expression as to cause the involuntary or spasmodic laughing and weeping.

In one or two of our cases of lenticular lesion involuntary emotion was present.

In connection with the question of speech disorder from lenticular lesion it will be observed in the case of Deroubaix that the lesion was right sided.

Pagano,[16] by injecting solutions of curare into the caudate nucleus has concluded that:

(1) Excitation of the anterior third and of the middle third of this nucleus provokes in dogs something very like the emotion of fear. This effect is best brought out when the injections reach the internal half of the organ. All the characteristics of this emotion are present, the gesticulations, the play of the physiognomy, the cardiac and respiratory phenomena, the actions of the intestines, and of the bladder, the state of the pupil, and the effect of threats and noises all go to form conclusive evidence of this emotion.

(2) The excitation of these points, but especially of the middle third, provokes a strong erection of the penis which appears immediately after the injection and persists until death.

(3) The excitation of the anterior extremity of the nucleus

[16]Pagano. Riv dipatol. nerv. e. Ment., July, 1906. Reviewed by W. W. Ireland in the Journ. of Mental Science, January, 1907, p. 175.

produces an agitation which presents the appearance of fear modified with anger.

(4) The excitation of the posterior third of the nucleus provokes a series of manifestations of anger; the grinning and barking, the readiness to attack and bite, and the whole attitude leave no doubt as to the nature of the emotion.

(5) The excitation of the outer part of the anterior third of the nucleus, besides some emotional disturbance, provokes in a greater degree intestinal and vesical phenomena.

The nucleus caudatus according to these experiments is a center of some of the emotions.

Pagano has shown the existence of distinct motor centers in the cerebellum by the same method.

THE NUMBER AND CHARACTER OF THE CASES STUDIED BY THE WRITERS.

The cases which follow are a few out of a considerable number which have come under the observation of the writers, in which lesion of the lenticular nucleus was found at necropsy. In the majority of these cases the lesion, usually a hemorrhagic cyst or an old area of softening, has not been confined to the lenticula; indeed, in most of them several parts of known functional importance, and others whose functions are still in obscurity, were involved in the destructive area of disease. The reason of this is of course easy to recognize, and resides in the fact that the blood supply to the great basal ganglia is from branches of the middle cerebral artery, which also sends branches to various regions both of the cortex and of the interior of the brain. The writers have realized the same difficulty that has been realized by others in reaching conclusions regarding the functions of the striate bodies, because of the infrequency of lesions isolated to them. Even in the cases chosen to illustrate some of the points of this paper, the lesions present were scarcely in a single one absolutely limited to the lenticula. In two or three, however, this was so nearly the case that they have felt justified in drawing some inferences regarding lenticular function.

The points which the writers have attempted to elucidate or at least on which they have tried to obtain some light through the study of their cases, circle around the subject of the mechanism of cerebral speech and that of the representation of motility.

CASE I. *Apoplectic Attack Causing Right Hemiplegia and Loss of Speech—Partial Recovery of Motility and Speech—Some Mental Slowness and Confusion—Dysarthria—Partial Word Dumbness—Word Deafness Present but only Slightly Marked—Hemorrhagic Cyst involving the entire Lenticula, the white matter of the Insula and the anterior part of the First Temporal Convolution, and slightly the Posterior Limb of the Internal Capsule.*

M. S., aged forty-eight years, white, born in Ireland, was admitted to the men's nervous wards of the Philadelphia General Hospital August 21, 1903.

On admission the patient complained of headache and also of more or less weakness and helplessness in his right leg and at times in his right arm.

No family history and no history of important illness previous to that producing his condition on admission could be obtained.

The patient made a statement that he had had fits after an injury to his head, after which he had also developed some difficulty of speech.

He also said that three months ago he came home feeling in his usual health, and after supper became dizzy and fell on his face. He said that he did not know anything for two weeks, but when he recovered his mind, he found that he could not use his right leg or arm and could not speak at all. He remained unable to speak for about two months, since which time his power of speech had gradually returned, but he still spoke with difficulty.

The patient was a hearty, robust-looking man. His tongue was moist, slightly coated; pulse of good quality. His heart action was irregular. The lungs showed good expansion.

The pupils were equal, reacted well to light, but slowly to accommodation.

Examination made September 16, 1903, showed but little loss of power in the right arm or leg; the grip on the right side was, however, slightly impaired. Station was good and gait steady. The reflexes were normal. No note as to sensation was recorded.

On February 24, 1904, a note was made by one of the physicians in attendance which stated that in his last apoplectiform attack he had some spasm in the right arm and leg; and also that he had sensory aphasia; and although he did not miss any words he occasionally missed a syllable.

On February 25, 1904, the record stated that his gait was fairly good, but slightly stiff; that he dragged the right foot slightly. The knee-jerks were spoken of as "spastic" with slight attempts at clonus on both sides; station was good with the eyes open or closed; the pupils responded to light and accommodation; no palsies of ocular muscles were present; the tongue protruded to the right; the naso-labial fold was slightly deeper on the right

side. The grip was now said to be better on the right than on the left side.

The patient's mentality was noted as "slow"; he required more time to comprehend questions. He could not read nor write because of lack of education. He appreciated the purposes and uses of objects. He recognized a book and named it as "book." He called a gold watch a "dollar." When pointed to the time, which was 12.20, he said it was "after two"; he could not specify the minutes. He could say the days of the week slowly. When told to close his eyes he grasped his hand; when asked to point to a chair, he picked up his hat. He easily confused the name of one object with that of another. He recognized what spectacles were and said that he needed them himself; he could not, however, name them. He could not name

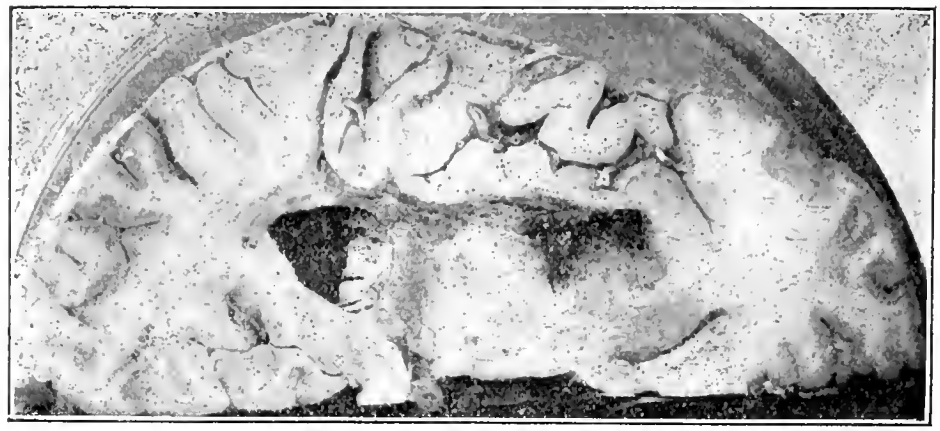

Fig. I. Cyst of the Left Lenticular Nucleus and External Capsule. Motor Aphasia Incomplete (Case I).

a lead pencil. He recognized a pen, could not name it at first but after a little effort was able to name it. He was also asked to name various other objects, as a key, a ring, and gloves, but failed to do so; but he named some objects of wearing apparel correctly.

Astereognosis was not present.

On December 21, 1905, he was found in a state of collapse and the following facts were noted: His eyes were turned upwards and oscillated slightly from right to left; the face was drawn slightly towards the left side. The right arm and leg dropped lifeless when raised. There was some spasticity in the right arm. The reflexes were exaggerated. On December 22, 1905, the patient was conscious again and seemed to understand all that was said to him and a note said "were it not for his partial aphasia would be able to answer intelligently."

The heart sounds were very good, the second sound being exaggerated, especially the aortic. Pulse at the wrist was strong,

of rather high tension and moderately rapid. The patient did not respond to pin pricks. Upon catheterization only one half ounce of urine was obtained.

On December 23, 1905, the patient was in fairly good condition at 10 A.M. At 11.15 he was found rigid, with legs extended and arms held close to the body, the forearm being flexed on the arm. His face was markedly cyanosed. The jaw was rigid, respirations had ceased and the pulse could not be felt at the wrist. The heart sounds could not be heard. A minute or so later the patient relaxed and was dead.

A section made at the level of the upper part of the basal ganglia revealed a cyst in the left hemisphere, which had implicated the middle and posterior part of the left lenticular nucleus, and extended to, but did not involve, the cortex of the island of Reil. It extended ventrally to 0.5 cm. (1-5 in.) from the anterior limit of the lenticula. It extended to, or possibly slightly involved, the posterior limb of the internal capsule. At a level 0.5 cm. (1-5 in.) lower the cyst extended from its posterior end into the anterior part of the white matter of the first temporal convolution, but did not involve the cortex, nor the posterior half of the first temporal convolution.

The temporary right hemiplegia with residual very moderate hemiparesis was probably due to the lenticular lesion, although it is possible it may have depended on the slight involvement of the internal capsule. The temporary, somewhat complete, aphasia, was probably due to the effects of this lesion on neighboring parts, some of the motor defect in speech being, however, probably due to the lenticular lesion. The partial word dumbness could not have been due to lesion of Wernicke's zone, as only the anterior part of the first temporal convolution was involved. As the posterior part of this convolution is considerably higher than the anterior part, the fibers from the former could not have been greatly involved by the extension of the cyst at a low level into the anterior part of the first temporal convolution. It is more probable that some of the symptoms are to be attributed to injury of fibers in the external and extreme capsules which stand in relation to the posterior part of the first and second temporal convolutions.

CASE 2. *Apoplectic Attack causing Right Hemiplegia and alost complete aphasia—Only a few Recurring Utterances retained—Marked Word Deafness at First—Probably some Word Blindness which soon Disappeared—Power of Humming Melodies retained—Continuous Improvement under Systematic Training—Spontaneous Language largely regained—Ability to write with unparalyzed arm acquired—Mentality practically unaffected—Death after four years from Thrombosis of Superior Mesenteric Artery—Dura thickened and adherent—*

Cyst destructively involving the Insula, the Lenticula, the Anterior and Posterior Limbs of the Internal Capsule, the External part of the Thalamus, and the White Matter of the Anterior Half of the First Temporal Convolution at a little distance from the Cortex—The Area of Broca and Subjacent White Matter and Wernicke's Zone not involved.

This case is one, the clinical details of which were in part given by one of us, Dr. Mills,[17] in a paper on the treatment of aphasia by training, published nearly two years before the death of the patient. A contribution giving some facts regarding his condition and attempts made at training him for his aphasia before he was seen by Dr. Mills has also been published by Mrs. Kate Herman.[18]

In the account which follows the writers have summarized the most important facts bearing upon his aphasia and hemiplegia from the papers just referred to, and also from communications received from the patient's former secretary, Miss Dorothy C. Falk, and his former professional and business associate, Dr. H. C. Hays. As this contribution is intended to deal more especially with the functions of the lenticula and the symptomatology resulting from a lesion of this ganglion, the writers have thought it better to concentrate attention upon the facts demonstrating his type of aphasia, rather than upon the efforts at re-education of his language and the results of these efforts. The case is unusually valuable because of the fulness of the history obtained from the different sources to which reference is made, and also because of the intelligence and education of the patient which allowed a more complete record of his speech defects.

The patient was a physician, forty-five years old, residing in one of the western states. In July, 1902, he had an attack of right hemiplegia with almost complete aphasia. He was first under the instruction of Mrs. Kate Herman from June 15, 1903, to July 15, 1903. At the beginning of this period his vocabulary consisted of only four expressions, namely "yes," "no," "but," and "O God." "Yes" and "no" were commonly used correctly. He sometimes added the word "but" to "yes" or "no" apparently when the "yes" or "no" did not seem to him to express his meaning, or in order to give some force to what he was trying to say. At other times he would use the expression "O God." He often used pantomime in an unmistakable manner. When he got possession of a few useful words and phrases he discontinued this expletive. Dr. Mills does not recall his

[17]Mills, Chas. K. "The Treatment of Aphasia by Training." Journ. of the Amer. Med. Association, Dec. 24, 1904.

[18]Herman, Mrs. Kate S. "Heilung der Aphasie durch den Artikulationsunterricht." "Eos." Vierteljahrschrift für die Erkenntniss und Behandlung jugendlicher Abnormer, Heft 3, 1906.

having used it when he was under his care. His memory for persons, places and occurrences was good. He was often very insistent in his efforts to make himself understood. His instructor was soon convinced that his periods of concentration should be short and his exercises for improving his language varied. Word deafness was quite marked at this time, but was not complete. He seemed at times to be partially word blind, but this defect was probably comparatively slight if it really existed, as even early in the efforts to train him he was much helped by seeing the words which were used in training him either in script or printing. It was noted that he could assist himself in understanding what was read from a newspaper by tracing the words with a pencil as the paper was being read.

When he heard music which he knew, he would hum the melody and occasionally articulate some of the words of the song. One day when he was trying to relate an historical occurrence he could not pronounce the word "Maryland" intelligibly until it fortunately occurred to him to murmur the song, "Maryland, my Maryland."

His voice was not impaired,—a rather interesting fact in connection with the functions of the lenticula. He was trained systematically after the manner used with dumb children. The formations and combinations which he found most difficult were those with which such children struggle most, namely, *k*, *g*, and *ng*; *th* also gave trouble. He was made to repeat the names of objects in his environment, and his teacher states that the patient would succeed one day in using such words as *house*, *cow*, *tree*, *bird*, etc. and at another time would be unable to recall these, unless helped a little at the beginning. Gradually verbs were taken up and longer and longer sentences tried. With his first instructor, as later with Dr. Mills, it was found that he experienced his greatest difficulty with adjectives, pronouns and articles, and words generally which expressed no mental image.

He was taught to write with his left hand, making continuous and notable progress. He repeated aloud what his instructor wrote for him.

When he first consulted Dr. Mills in October, 1903, his right-sided paralysis, though still marked, was much improved. No loss of sensation and no affection of the bladder or bowels were present. The face was the seat of a moderate right-sided paresis, the tongue not deviating to either side. The deep and superficial reflexes were of the usual type found in cerebral hemiplegia —exaggerated knee-jerk with front tap and ankle clonus and the Babinski response being present on the right side.

When tested with Wyllie's physiologic alphabet, it was found that he could repeat the vowels *a*, *e*, *i*, *o* and *u* well, having been previously trained in their pronunciation, but he could not remember them from day to day. In like manner he could repeat

the consonants of the alphabet, but could not recall them spontaneously. He was partially word deaf, having regained word hearing to a considerable extent by time and training. He could understand most familiar expressions, but if a strange or unusual word were used he did not understand it. He was also apparently partially word blind, having relearned to read considerably before coming under observation. With his left hand he could write the words that he was able to read. He could not understand the meaning of such prepositions as *of, to,* and *for,* if pronounced or read. He could not spontaneously write these words when asked, but could copy them.

In connection with a study of his reacquisition of language, particular attention was paid to the degree in which he regained different parts of speech. It was noticeable that in his efforts to talk he chiefly used his nouns and verbs, occasionally employing a pronoun. The words which expressed the qualities of things, which join and relate things to each other and to actions,—in other words, adjectives, adverbs, conjunctions and prepositions,—were still almost entirely eliminated from his vocabulary. Auxiliary verbs like *to have,* and *to be,* in all their modes and tenses, were apparently without any meaning to him. The word *is* or *was* occurring in a sentence seemed to worry him. In brief, this patient was a hemiplegic aphasic, the motor aphasia having at first been almost complete; the sensory aphasia partial. An appreciable degree of recovery from his aphasia had occurred from the first under the method of training by repetition and by efforts at spelling, reading and writing.

Persistent efforts at training were made. The measures employed were the facial and other gymnastic methods used with the deaf and dumb, the method of repetition from dictation, of copying, the use of Wyllie's physiologic alphabet, of a phonetic reader, a language primer and grammar. An interesting point in the history of the attempts at this man's re-education in speech was the fact that he made much use of a small dictionary. He made considerable advance by looking up the definitions of words, and when he found words and expressions in the definition which he could not comprehend, these in their turn were looked up in the appropriate places.

After the patient's return home communications were received from time to time regarding him. He steadily improved, although at first his progress was comparatively slow. In a short time he was reading his newspapers and magazines and understanding fully what was in them. Six months after he left Dr. Mills word was received that he was acquiring more voluntary expressions every week. A noticeable feature now was that when he was told to say anything instead of repeating it verbatim, he would express the idea in his own words. He read much and played whist and other games, and had just finished transacting

some business for the institution, the first which he had done of any importance since his illness. A little later, in describing his improvement, the physician associated with him in his work, said that his use of words to express an idea was much more general than had been the case heretofore. He now expressed in his own words answers to questions in the form of a sentence rather than a single word, and it was noticeable that he was able to use a different word to express the same idea. At times he made sentences of considerable length, using nouns, verbs, adjectives, prepositions, etc. Thorough and systematic training with the assistance of his instructors and members of his family was kept up, the patient helping the work very much by his own determination and originality in methods. He continued to use his dictionary. His teaching was much along the line of both what is known as the word method and also that of spelling by using each letter as in the older method.

Many of the details of the methods used to improve his language, and of the time spent in these efforts will be better reserved for an additional paper. With regard to the re-education in language this case is the most interesting that has ever come under our observation. Nearly four years elapsed between the time of the apoplectic attack causing his hemiplegia and aphasia, and his death. During much of that time with interruptions sometimes extending over a few weeks, or even months, the earnest, formal retraining of his speech was carried on, and even when this systematic instruction was not resorted to, the patient, a man of much originality and energy, was always trying to re-educate himself in the use of language. His former secretary calculated that during the four years he was given about one and one half years of training. As a rule only one to two hours daily were given to formal instruction.

It is interesting to note in connection with the question of intellectual deficit in aphasics, which has become so prominent through the views of Marie, that this patient retained throughout his mental vigor and general capacity. His deficiencies seemed to be entirely those conditioned by his defects of speech, meaning by this, of course, his word deafness, his early slight or moderate word blindness, and his difficulty in correlating concrete concepts with each other as well as his very pronounced motor aphasia. About nine months after his attack the sanitarium, of which he was the proprietor and chief physician, burned down. Measures were at once taken to rebuild it, and as bearing on his mental condition, his secretary wrote Dr. Mills that he was always able to figure on any proposition just the same as before his illness but he could not speak it, all his figuring for his sanitarium being done in a very accurate and clearsighted manner.

The improvement in his speech was continuous up to the time of his last illness.

He had no second apoplectic attack. He had, however, three distinct epileptiform seizures, at long intervals, during the last two or three years before his death. No increase either of his paralysis or of his aphasia occurred as the result of these attacks.

His general mental state was good. At times there were indications of moroseness, some irritability of temper and impatience when he was not immediately understood, and occasion-

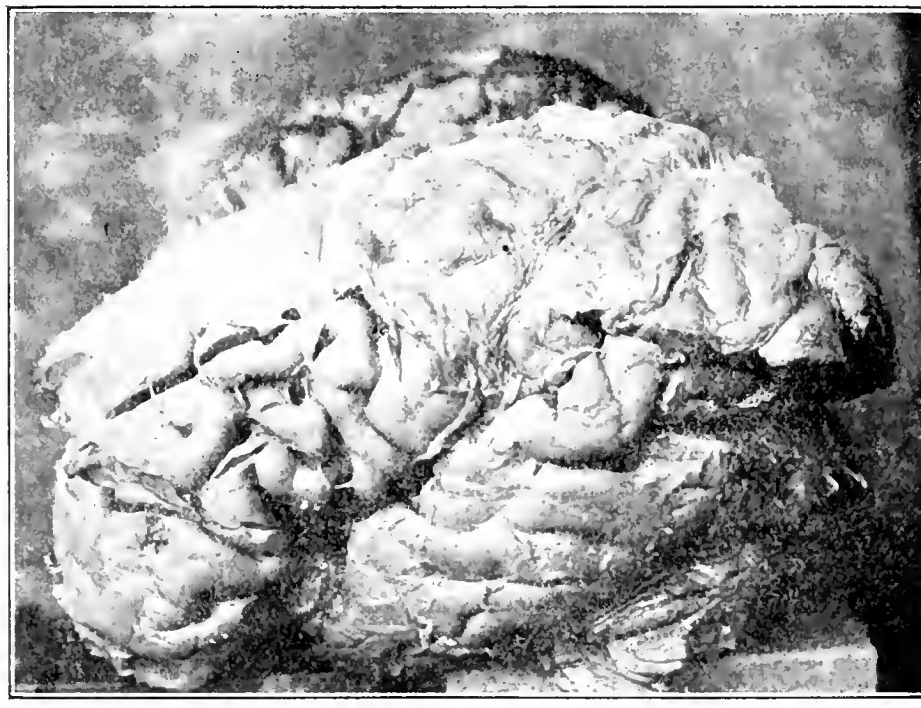

Fig. 2. A case of marked motor and sensory aphasia. The area of Broca is intact and the subcortical white matter was not involved in the primary lesion (Case 2).

ally when much discouraged, he spoke of suicide. He showed no marked change in disposition which could be noted as altogether different from his former self. His general physical health remained good.

The conditions and symptoms preceding his death could in no way be attributed to the cerebral lesion causing his aphasia and hemiplegia, although the changes found in the aorta and the superior mesenteric artery might have some relation to the alterations in the vessels of the brain which were present at the time of the cerebral attack. He died about four years after the onset of his aphasia and hemiplegia, from thrombosis of the superior mesenteric artery, as determined at the post-mortem

examination. His symptoms were those of shock and great abdominal pain.

The necropsy was made by Dr. Philip Hillkowitz of Denver, Colorado, at which place the patient was at the time of his death. We are much indebted to Dr. Hillkowitz for the account of the gross findings. The body was well nourished, with considerable adipose tissue; muscular development good; the heart showed

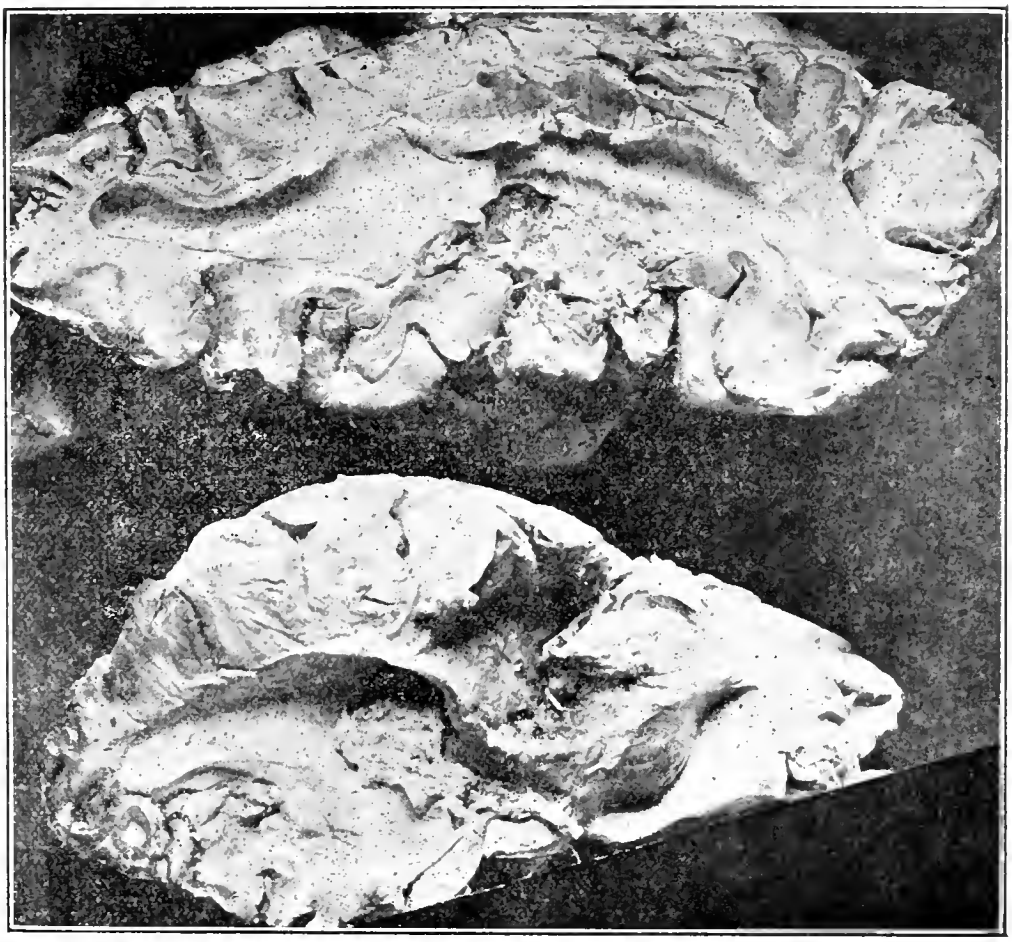

Fig. 3. The lenticular nucleus and the island of Reil were destroyed. The sclerosis extended anteriorly to the white matter of Broca's area, and posteriorly to the posterior extremity of the island of Reil (Case 2).

slight thickening of the mitral valve; numerous small plaques were found on the aorta, above the valves and around the opening of the coronary arteries. Marked hyperemia was present around the opening of the right coronary artery. The lower lobe of the left lung was strongly adherent to the chest wall and diaphragm. The left kidney was very much puckered on its surface. A slight scar was found on the external border, about 1 cm.

(2-5 of an in.) in diameter, extending into the interior. The cortex was slightly diminished and was pale in color; the right kidney was similar to the left with the exception of the scar. The intestines showed a dark purple color extending all over both the large and small intestines. The superior mesenteric artery showed a thrombosis which could be followed in the branches of the vessel.

The dura was much thickened and adherent in many places to the membranes beneath. The membranes over the occipital lobe were largely congested. An elliptical cavity 5 x 2.5 cm. (2 x 1 in.) was found in the left cerebral hemisphere. The description of the parts destroyed by this lesion is given in the following paragraph after careful study of the specimen by Dr. Spiller, the brain having been forwarded, after hardening in formalin, to the Laboratory of Neuropathology of the University of Pennsylvania by Dr. Hillkowitz, who however had made an examination at the time of the necropsy. The description of the position and extent of the lesion differs in only one particular from that furnished by Dr. Hillkowitz, although this is one of some importance. Dr. Hillkowitz speaks of the lesion as involving a portion of the inferior frontal convolution (presumably meaning by this the third frontal). Although the cyst extended close to the third frontal it did not involve the cortex of this convolution.

A large cyst was found in the left cerebral hemisphere. In a horizontal section through the lateral ventricle and upper part of the basal ganglia, the sclerosis was found to extend to 1.5 cm. (3-5 in.) from the cortex of the convolution posterior to the ascending limb of the fissure of Sylvius. The area of Broca and subjacent white matter were intact, although fibers passing to or from the foot of this area were probably implicated. The cyst had destroyed the island of Reil, the whole of the lenticula, the anterior and posterior limbs of the internal capsule and the external part of the thalamus. The head of the caudate nucleus was very slightly affected. The gray matter of the first temporal convolution was not involved, although the white matter was involved at a little distance from the cortex.

The part of the first temporal convolution destroyed was the white matter of its anterior half. To be exact, the posterior border extended to the beginning of the innermost part of the middle portion of the first temporal convolution, in a section made through the upper part of the basal ganglia.

This case, although one of great value in connection with the discussion of the subject of aphasia, loses some of its importance as regards the functions and symptomatology of the lenticula, because of the fact that not only was this ganglion destroyed, but so many other functionally important regions. Any speech or paralytic disorder if dependent upon the lenticular

lesion, was probably covered by the effects of destruction of other parts. Broca's convolution was not involved, as was not the Rolandic operculum.

THE LENTICULAR ZONE OF MARIE AND MOTOR APHASIA.

Some light is thrown on several of the points of great interest in connection with the current discussion of the subject of aphasia, springing out of the somewhat revolutionary contributions of Marie, by the clinical and pathological facts observed in the case just described. Marie has laid much stress upon the lenticular zone. In defending himself against those who have charged him with giving too much importance to the lenticula in his theories of the pathogenesis of motor aphasia, he insists that the lesions producing the train of symptoms to which he has called attention are not due to isolated lesions of the lenticula, but to it and to those portions of its environment which he includes in this lenticular zone. It is important, therefore, to know just how he defines this zone.

In a horizontal section of the brain, he says, a transverse line drawn from the anterior angle of the insula to a corresponding point of the lateral ventricle will give its anterior border, while a line from the posterior angle of the insula to a corresponding point of the lateral ventricle will give its posterior limit. Thus he includes within this zone more than half of the thalamus, both limbs of the internal capsule, the greater part of the caudate nucleus (all except a part of the tail), the lenticular nucleus, and the island of Reil with the subjacent white matter. Marie does not attempt to distinguish between the symptoms of lesions of the striatum and of the white matter and cortex of the island of Reil. He defines his position concisely as follows:

A lesion implicating only the zone of Wernicke causes the aphasia of Wernicke;

A lesion of the lenticular zone causes anarthria, which is merely the pure motor aphasia of the writers, believed by them to result from a lesion of the white matter beneath Broca's area with the cortex intact. Articulate speech alone is affected, internal speech is preserved; the patient reads and writes and understands without difficulty spoken speech.

A lesion implicating the zone of Wernicke together with the lenticular zone causes the aphasia of Broca (great impairment

or loss of spontaneous speech, of reading and writing, and more or less impairment of the comprehension of spoken language).

The case just given fulfills these requirements for the aphasia of Broca. The patient was motor aphasic and agraphic in large measure and had great impairment of the power of reading, According to widely accepted opinion, the case should have exhibited also pure word dumbness and pure word deafness, because the lesion did not extend to the cortex either in Broca's area or Wernicke's zone.[19]

[19]Marie, Pierre. Revue de Philosophie, 1907.

(To be continued.)

Society Proceedings

AMERICAN NEUROLOGICAL ASSOCIATION, HELD IN WASHINGTON, MAY 7, 8 AND 9, 1907.

The President, Dr. Hugh T. Patrick, in the Chair.

(*Continued from page 542.*)

INTRADURAL RESECTION OF THE POSTERIOR ROOTS OF A NUMBER OF SPINAL NERVES, FOR THE RELIEF OF INTRACTABLE PAIN.

By Dr. George W. Jacoby.

Historical. General belief that the operation is dangerous is erroneous. Questions of technique which are of neurological interest. Tabulated cases. Indications for operation. Report of a successful case in which all the roots which go to make up the brachial plexus were resected. Sensory change after operation. Head's observations. Conclusions.

Dr. P. C. Knapp said that in spite of Dr. Jacoby's paper his own experience has forced him to look with considerable hesitancy upon this operation for the division of the nerve roots. He had reported two cases of division of the roots before this Association several years ago. In one case death occurred upon the table. The patient was a very stout and muscular man in good physical condition at the time of the operation. Much blood was lost in getting into the spinal canal. In the second case, also for amputation neuralgia, the operation proved temporarily successful for the relief of the neuralgia, but although four or five nerve roots were divided there was no special loss of sensibility which could be made out in the stump, the stump being close to the elbow, so that there were the areas of a number of nerve roots there which could be tested. The man at the time the case was reported had developed a spastic paraplegia which came on rather gradually after the operation. After the case was reported another operation was done, the laminectomy wound opened, and there was found no indication of any hemorrhage or any special pressure upon the cord. The pain had returned, though never to the same extent in the stump. Further roots were divided, but nothing was found which could be relieved, and the man was in a most deplorable condition, still suffering from pain in the stump in the one arm, and with a most extreme grade of spastic paraplegia of the legs. There was considerable disturbance of sensibility in the legs, but it was about equal on the two sides. His statements in regard to the degree of sensibility, however, were rather untrustworthy, so that it could not be very exactly mapped out, but his condition was distinctly deplorable. In view of these two cases and of Dr. Prince's, which Dr. Prince will probably speak of, Dr. Knapp said they felt in Boston considerable hesitancy about recommending division of the posterior roots for the relief of intractable pain.

Dr. H. H. Hoppe said the experience he had had of resection of the posterior roots is limited to one case, but that case was an especially disastrous one. The woman had suffered from intractable pains in both legs for three or four years, and a tentative diagnosis of extradural tumor was made. It was decided that at the operation if no extradural tumor was found the posterior nerve roots were to be cut off. Laminectomy was performed and the posterior roots were divided on both sides of the spinal cord. After the operation there was absolute loss of sensibility in both

legs, the reflexes were abolished and the pain continued. During the operation the cord seemed to be entirely normal. There was no swelling in the cord, in spite of the fact that all of the pain was concentrated and localized in both lower extremities. The operation was followed by complete paralysis of both lower extremities, which at the time seemed to confirm Sherrington's opinion that complete loss of sensation in all of its qualities leads to loss of motor power. Twenty-four hours after the operation this woman developed acute trophic sores on the heels and died seventy-two hours after the operation. No autopsy could be obtained, but the fact was very striking that this woman's pains were not relieved by the division of all the posterior roots on both sides.

Dr. Morton Prince said he has always felt that in suitable cases this operation is one which he should not hesitate to recommend, but he thought the case must be a justifiable one. By this he meant that the pain must be of such a severe character as to justify the risk, which his own experience showed was considerable. In his case (one of brachial neuritis) which had just been referred to by Dr. Knapp and was included in the statistics of the reader there was as a result of the operation complete relief of the pain in the arm. This was fine, but that was not the end of the patient's troubles. After the operation there developed a Brown-Séquard paralysis attended by most shocking pain in the leg. The cause of this secondary paralysis can only be inferred to have been caused by pressure of some kind, while the pain may have been due to an ascending neuritis. The patient's troubles and trials were so great that as a final result of the operation he shot himself; so that it cannot be said that so far as the ultimate results were concerned the operation was a success in this case.

Dr. Prince said there was another point which he thought must be borne in mind, and that is, that when the pain has persisted for a long period of time there is a tendency for the patient to become hysterical and develop hysterical pains. Dr. Weir Mitchell, it will be remembered, called attention to this fact in his work on the injuries of nerves. The patient becomes worn out mentally and physically with suffering, and the pain after a while spreads and becomes both in degree and extent far out of proportion to the injury. Dr. Prince thought that in the case Dr. Knapp spoke of a very intense hysterical condition developed, creating pain beyond that which could be accounted for by the affection of the nerve. The persistence of pain after the resection of the nerves may often be explained in that way. He thought that this tendency must be taken into account to explain the failure of this operation to relieve pain. If the nerve roots are cut there must be cessation of pain in that district unless the pain is of an hysterical character, or unless an ascending central neuritis has developed.

Dr. P. Zenner said he merely wished to mention a case never reported which he had under observation several years ago. This was a case of tumor involving all of the brachial plexus. The tumor was shown post-mortem. The man had intractable pain. He had had symptoms probably for eight months before Dr. Zenner saw him; pain, then anesthesia in the ulnar region, and paralysis of all the muscles below the elbow. He had become addicted to morphia on account of his intense suffering. In this case after a preliminary operation seeking for the tumor, which was not found, laminectomy was performed and the roots of only three nerves, the first dorsal and the last two cervical roots were resected. For a little while the patient had complete relief from pain, so that one might suppose

that all the nerves related to the affected area had been resected, but soon his pain returned, and he said his suffering was as great as ever. This was doubted. He lived six months or longer, and finally died of a fatty heart. The operation appeared to have done no harm in this case. It did not impair his general health, excepting that he always said he was disabled, that he could not hold his head as well, etc. But the operation seemed to have done no harm to the man, and while it was supposed that his pain might be explained in the way Dr. Prince has just spoken of, still it is more probable that the pain returned or was as severe as ever because not sufficient roots had been resected.

Dr. F. R. Fry said that Dr. Mudd had recently operated for him on a case where the relief from pain was marked. The patient had a large sarcoma in his left thigh. The right thigh also contained considerable amount of the growth. The distribution of some of the pain regions led him to believe that the cord might also be involved. The operation was done for the purpose of severing all the sacral and lower lumbar sensory roots. Owing to the unusual amount of fluid in the wound and the general condition of the patient the lowest roots were missed. Three days later the patient was again placed on the table; the field was dry; the patient's condition good and these roots were readily recognized and cut. The patient was entirely relieved from the severe pains which morphine was failing to control. He died two months later from exhaustion. Dr. Fry thought there would be an advantage in a two stage operation in some cases of this kind.

Dr. H. T. Patrick said the crucial points in this question are the surgeon and his technique. He has seen a few of these operations performed, and it makes every difference who does the operation and how it is done. He was quite impressed with the technique of Sir Victor Horsley a year ago. This surgeon had gone so far as to have special spectacles made; strong convex lenses combined with prisms, so that the strain of convergence would not be too great. By this means he could readily see the small vessels supplying the posterior roots, and Dr. Patrick thinks it is reasonable to believe that Sir Victor Horsley was right in supposing that cutting these small vessels going into the cord had a great deal to do with the seriousness of the operation from the surgical standpoint.

Dr. G. W. Jacoby said he was glad the paper had brought out much discussion. It seemed to him the stand Oppenheim takes in the last edition of his text-book should not be allowed to go unchallenged. Naturally the cases must be selected. The indications for the operation must be precisely drawn. The source of pain must be in the nerve roots or in the ganglia. Then, having drawn the specific indication for operation, Dr. Jacoby agrees with Dr. Patrick absolutely that it is a question of the surgical technique. If the technique is beyond criticism, then from what Dr. Jacoby has seen he does not believe the danger of these cases is any greater than it is in an ordinary laminectomy. He does not believe that the section of the nerve roots themselves adds to the danger of the operation. The small sensory defect after the section of a large number of roots in other cases than his own will bear a great deal of discussion. The fact that a number of cases have not been relieved at all is, he thinks, explicable by what Dr. Prince has said. The majority of these cases are morphinists, and whether they have not been relieved of their pain because of the craving for morphia, or whether you would call it a persistence of hysterical pain, is a question upon which he cannot enter now.

Sensory and Motor Disturbances in Parts Above the Distribution In-

volved by Definite Organic Lesions of the Spinal Cord. By Dr. T. H. Weisenburg. (See this journal, page 434.)

Dr. F. R. Fry said he would like to ask Dr. Weisenburg what importance he attached to certain predisposing factors in these and similar cases; for instance, syphilis and other infectious diseases, arteriosclerosis, etc.

Dr. C. K. Mills said this paper of Dr. Weisenburg's interests him particularly with regard to the question of the occurrence in stages of certain symptoms as the result of a co-terminus necrosis due to arterial obliteration, which seems to him to be one of the points of the paper. It is of considerable clinical and pathological importance in connection with that general but often vaguely considered subject of arteriosclerosis. He believes it is true, as Dr. Weisenburg has stated, that in an old case of transverse myelitis, especially if it is complete (that is, completely transverse), or when it is not quite complete, there is a tendency, for whatever reason, to the very slow obliteration of blood vessels near the point where the original necrosis or where the acute myelitis resulting in destruction has taken place. There is a distinct clinical type of arteriosclerotic disease which has not, Dr. Mills thinks, been well and systematically presented, although he could certainly not say that it has not been considered, and this is the type of disease in which we have both cerebral and spinal symptom-complexes which simulate others which may be due to tumor or to some other lesion, as to syphilitic inflammation. There is a clinical type of disease in which, for instance, we have a definite form of hemiparesis with or without hemianopsia and with or without other cerebral symptoms lasting for a definite time, and then remaining for a time in *statu quo* so far as the symptoms are concerned; next in a period of two or three months increasing by steps rather than by insidious progression. Undoubtedly the increase is due to a peculiar form of obliteration of neighboring vessels. Dr. Mills said he had had several of these cases; three operation cases of this kind, one of which was studied microscopically after the operation by Dr. Spiller. These cases should be regarded not as myelitis or encephalitis, but rather as a peculiar form of necrosis occurring in successive stages.

Dr. W. G. Spiller said he was rather inclined to doubt whether this ascending form of disturbed sensation will be found in every case, and only further investigations will determine that. Dr. Weisenburg spoke of lesions occurring in various parts of the cord and of the vascular changes resulting therefrom. In early life the cord has a remarkable recuperative power, and fewer fibers and nerve cells may perform the function usually performed by a larger number, and it is possible for recovery of function to take place; later, as years pass by, the vitality of the cord is lessened and the fibers and nerve cells can no longer perform the function so perfectly, and there is a progression of the symptoms in that way, as seen in certain cases of poliomyelitis, etc.

Dr. Weisenburg in closing said in answer to Dr. Frey's question as to what influence syphilis has, he was careful to select cases for his paper in which such disease was excluded, but he thought that syphilis, as well as alcohol, if present, would cause the disease process to be more marked.

As regards Dr. Mills' comment, Dr. Weisenburg stated that he had taken up the question of secondary degeneration in this paper because it had a direct bearing upon the subject. He thought that the questions relating to it were very important, and should be further investigated.

NEW YORK NEUROLOGICAL SOCIETY.

Jan. 8, 1907.

The President, Dr. Joseph Fraenkel, in the Chair

DEMONSTRATION OF THE RESULTS OF THE RE-EDUCATIONAL TREATMENT OF TABES DORSALIS.

By Dr. H. S. Frenkel, of Heiden, Switzerland.

Dr. Frenkel said that in order to complete his theoretical explanations of the re-educational treatment in tabes dorsalis, he had tried to get some demonstrable results by starting the treatment in a few hospital cases of different stages of ataxia. Through the kindness of the President of the New York Neurological Society, Dr. Joseph Fraenkel, he was able to show three different cases of tabic ataxia treated in the Montefiore Home, with the most attentive and intelligent assistance of Dr. E. A. Fruchthandler, the assistant physician of the Home. While the results in these three cases were perhaps not striking, on account of the short duration of the treatment and the nature of the cases, they demonstrated very well some important improvements, and also the lines to be followed in the further treatment.

The first patient was a man, sixty-two years old, whose present illness began seven and a half years ago, when he noticed pain of gradually increasing severity and of a shooting character in both legs. This was diagnosed as sciatica. At the end of two years he could not walk in the dark, and for the past two and a half years he could not walk without a cane. The interest in this case, Dr. Frenkel said, lay in the marked hypotonia, which was almost limited to the left knee. An examination showed that the difficulty of the gait was due not so much to the ataxia as to the hypotonia, and its unequal development in the knees. This change rendered these patients especially unfit to balance the weight of the body. In unilateral hypotonia the act of walking was rendered more harmful than in a case of equal knee-hypotonia, as in the former condition the patient was inclined to rest the weight of the body on the hypotonic side. By doing this, the concavity grew more and more, and the re-educational treatment, as ordinarily applied, not only failed in its purpose, but was directly dangerous and actually contra-indicated. The proper procedure in these cases was to have a special appliance made before the re-educational treatment was instituted. The principle of the appliance was very simple, but its execution was rather difficult. A marked improvement in the gait of this patient was noticed when he wore the appliance.

The second patient showed by Dr. Frenkel was a man, forty-three years old, who showed all the classical symptoms of tabes dorsalis. His history dated back six years. In 1904 he was first unable to walk in the dark, and since then he had been compelled to use a cane. His gait had improved markedly under the re-educational method of treatment. He was able to walk without the aid of a cane, and in the course of four or six weeks, the speaker said, he would probably be able to walk in the dark.

The third case shown by Dr. Frenkel was a man, forty-nine years old, who had suffered from tabes since 1900, and for the past five years he had been confined either to bed or to a chair. On Nov. 10, 1906, when Dr.

Frenkel first saw him, the ataxia was very marked. In order to raise him from his chair it required the assistance of two nurses, and when he attempted to remain on his feet his knees would give way under him and he would fall to the ground. Treatment was begun on the following day. At first, the patient was able to stand only for a second or two; then the period gradually lengthened, and on Dec. 11, he stood without assistance for two minutes and ten seconds. Three days later he arose from his chair without assistance, and balanced himself for a few seconds without falling. On Dec. 25, he was able to lift his foot and put it down without falling. He was now able to take a few steps, and there was a slow but gradual improvement in his condition.

In conclusion, Dr. Frenkel again emphasized the fact that this method of re-education applied without a thorough knowledge of its principles and dangers was criminal.

Dr. George W. Jacoby said that in connection with the appliance shown by Dr. Frenkel he wished to call attention to the fact that within the past few years a relative of the orthopedist Hessing had settled in this country, and the high standard of the work he turned out was worthy of the appreciation of the profession. For example, in order to obtain an exact fit, it was his plan to build his apparatus upon a model of the part.

Dr. Jacoby said he could not let this opportunity pass without expressing his appreciation of the work done by Dr. Frenkel since his arrival in this country. While the progress made by the patients shown at this meeting was apparently not very startling, they certainly showed marked improvement, which could be appreciated by those who had been able to watch these cases from the time when treatment was first commenced.

Dr. Frenkel, in closing, said that while Hessing made some very wonderful appliances, they were intended for orthopedic rather than for neurological conditions.

In reply to a question as to whether these exercises increased the tabetic pains, the speaker said that on the contrary, some of the patients took them in order to relieve the pains.

Dr. Frenkel said that he agreed with Dr. Jacoby that while the improvement in the cases he had shown was not startling, still they furnished a good example of what might be expected from this re-educational method of treatment. In the third case, for example, the patient had not been able to stand for five years, and now, within a period of less than two months, he was able to stand and even to lift his foot. Standing was the first essential in these cases; the treatment of walking was secondary. People who could learn to stand could always learn to walk, but you could not teach a man to walk who was unable to stand. A far advanced case of this kind, therefore, was a more instructive object lesson than it would be to show a score of milder cases whose locomotion had been improved by this method.

A CASE OF SUBCORTICAL CYST: OPERATION AND RESULT.

By Dr. William M. Leszynsky.

The patient was a school girl, twelve years old, who was referred to him on April 24, 1905, by Dr. Francis Todd, of Paterson, N. J., with the following history: For the previous five months she had often stumbled while walking, and had occasional jerking of the left hand and arm, frequently dropping things held in that hand. She was supposed to have chorea. Two months ago, when she came under Dr. Todd's care, she complained of occasional general headache and twitching of the fingers

of the left hand, and of the facial muscles on the same side. Twice or thrice daily the left thumb would suddenly become flexed and adducted, the left eyelids twitching simultaneously. These attacks usually lasted a few moments, and were at once followed by weakness of the hand, which remained useless for about five minutes. Subsequently, the left arm became paralyzed, and later the left leg, and the spasmodic attacks ceased. A month later, the headaches became more intense and were accompanied by vomiting. Diplopia began five weeks ago, and still continued. During the last few weeks the headache had been more severe, and was attended with vertigo and mental confusion. She was in constant fear of falling. She had never had a general convulsion nor lost consciousness. She vomited four or five times daily, and her symptoms were getting progressively worse. She slept well; her memory was good; appetite normal; bowels constipated. She had never menstruated.

The patient was born normally at full term, of healthy parents, and up to the time of the present illness she had been in good health, excepting chicken-pox in the sixth year and occasional attacks of "night-terrors." There was no history of a fall or injury of the head. Her father and mother were living and had seven healthy children. Three others died in convulsions during infancy. There was no family history of tuberculosis, cancer, epilepsy, syphilis or rheumatism.

The diagnosis was made of a subcortical tumor in the Rolandic area on the right side, and an early operation was advised. Iodide of potassium was being administered, and its use was continued. When Dr. Leszynsky first saw her, her gait was hemiplegic, and she dragged the left leg in walking. The pupils were equal, reactions normal; paresis of the right external rectus and homonymous diplopia; bilateral optic neuritis of four diopters, with numerous retinal hemorrhages. About three months later, when he again saw her, he was informed that the headache, vomiting and vertigo had entirely subsided. Recently, there had been occasional tonic spasm of the left upper and lower extremities, and one general convulsion followed by coma. Her mental condition was good, and she was increasing in weight. Both pupils were dilated and she was totally blind. The facial paralysis had disappeared; the left upper extremity was in the same condition as at the first examination. In the left lower extremity there had been added loss of muscular sense, pseudo-clonus, and Babinski plantar reflex.

On July 22, 1905, the skull was opened by Dr. John C. McCoy at the Paterson General Hospital, and a large subcortical cyst was found in the right Rolandic area. The cyst was evacuated and drained. She left the hospital nine weeks after the operation, and her improvement was progressive.

The operation was performed seventeen months ago, and resulted in complete relief from the headache, vomiting and convulsions, and a subsidence of the hemiplegia. Her general health was excellent. At the seat of the operation there was a pulsating hernia cerebri, one by two inches, projecting with the bone flap over two inches above the level of the skull. She still dragged the left leg, but was able to walk without assistance. There was no rigidity. The knee jerk was exaggerated and ankle clonus and Babinski were unchanged. Had the operation been performed at the proper time, blindness would have been prevented. In all probability, the cyst was of gliomatous origin, and from the history of recent local convulsive attacks, a gradual recurrence of the previous symptoms might be expected.

Dr. Charles L. Dana said the results obtained by the evacuation of the cyst in the case presented by Dr. Leszynsky were very similar to those reported by Dr. Cushing after the decompression operation. In those cases there was a subsidence of the symptoms for a year or more, and the operation was not followed by the production of a hernia cerebri, as in this case.

Dr. Leszynsky said that one of the marked features of this case was the complete flaccid paralysis of the left arm, from which the patient had entirely recovered. In the operation done by Dr. McCoy the cyst was not only evacuated, but it was drained for some time. The operation was certainly more radical than the decompression operation of Cushing, to which Dr. Dana had referred.

A CASE OF ENCHONDROMA OF THE SELLA TURCICA.

By Dr. L. Pierce Clark.

The patient was a boy, seventeen years old, the eldest in a family of two. His family and personal history were negative. When he was four years old a growth appeared on the sixth rib, at the juncture of the rib and sternum. It grew slowly, without pain, and five years thereafter it was removed by Dr. Charles McBurney at the Roosevelt Hospital. Dr. Eugene Hodenpyl, who examined the specimen, found the growth to be a typical enchondroma. A second, third, fourth and fifth growth appeared, several months apart, on the left wrist, at the end of the radius, on the upper end of both tibia, and on the costal ends of all the ribs on the left side. Similar tumors were now to be found on all the long bones of the body. They ranged in size from a very small to a fair-sized orange. The patient and his relatives expressed the belief that the tumors come and go, disappearing spontaneously after a certain size is obtained, and in proof of this statement the patient showed several sites which were apparently formerly occupied by growths that had disappeared, and which still showed the remnants of ruins of former tumor formation.

During the past two years the patient had been gradually growing stiff and weak, and a progressive spastic quadriplegia was now fairly developed. The spastic palsy began in the right side. For the past four months the feet often fell asleep at night. There were no sensory defects. During the past few months the patient had had paroxysmal frontal headache. The left hand and foot had steadily enlarged during the past year in an acromegalic manner.

An eye examination, made by Dr. Henry H. Tyson, showed that there was a blurring of the nasal half of the optic discs, with hyperemia and slight swelling of the discs, and that the veins were enlarged and slightly tortuous. The field of vision was contracted on the temporal side for form and color.

The diagnosis in this case, Dr. Clark said, was multiple enchondroma, one of which was at present growing from the sella turcica, causing pressure on the pituitary body, the chiasm and the crura.

Dr. I. Abrahamson said an interesting feature of this case was the absence of myxedema and infantilism. With a destructive lesion of the pituitary body we should expect symptoms of either myxedema or infantilism.

A CASE OF PERONEAL MUSCULAR DYSTROPHY.

By Dr. Clark.

The patient was a boy, ten years old, whose paternal grandfather had

asthma, and whose paternal grandmother died of diabetes. His maternal grandfather died of cancer. No member of his family ever had any form of dystrophy. The boy's past history was unimportant, with the exception of the fact that dentition was delayed and accompanied by fever. He was a vigorous child and learned to walk at one year of age; he then stopped walking for several months without apparent cause. Between the ages of three and seven years he suffered from measles, pneumonia, diphtheria and scarlet fever, and from all of these he had apparently made an uneventful recovery.

Nothing abnormal was noticed in the patient's muscles until eight months ago, when he began to turn the left foot out and drag it in walking, and walked flat-footed with the right. This condition slowly progressed. Six months ago he began to experience difficulty in going upstairs, and this had steadily increased. The patient now presented a marked degree of left club foot of the varus type. The right foot showed a slight grade of talipes valgus. The peronei on the left side were weak and atrophic, as was the outer part of the soleus; on the right side these muscles were hypertrophic, but weak in contractile power. The left quadriceps was much weaker than the right, although both were deficient. The knee jerks were absent, and all the affected muscles showed diminished sensibility to both currents. There was, however, distinct R. D. in the left peronei. There was a moderate degree of lordosis. The entire musculature of the shoulder girdle was slightly affected with hypertrophy, weakness, fibrillation or fasciculation, and the right infraspinatus was especially prominent in enlargement. The muscular fasciculation induced a sort of choreiform movement in the hands and fingers. There was an extreme hypotonia in the fingers, showing advanced disease in the small muscles of the hands.

This case, Dr. Clark said, was an example of the Charcot-Marie-Tooth type of peroneal dystrophy and progressive muscular atrophy—a transition case embracing the pseudo-hypertrophic, the neuritic and central degenerative symptoms of the mixed type. It presented symptoms of the three affections, but was nearest to the peroneal dystrophy type.

Dr. J. F. Terriberry, in speaking of muscular dystrophies, said that for the past eighteen months he had had under his observation a child, about four years old, in whom the muscles of the face and upper extremities were very much atrophied, while those of the lower extremities were hypertrophied. The electrical reactions were all changed; still they maintained the normal formula. The mother stated that the child had been weak from birth, and it was now unable to stand or to raise the arms above the head. Its cry was very feeble, showing that the body muscles were also probably involved.

This case demonstrates that all of the so-called types of muscular dystrophy may be found in the same child at the same time.

Dr. Clark said the case referred to by Dr. Terriberry was possibly a combination of the pseudo-hypertrophic type and the Erb juvenile atrophic type. The speaker said the case he had shown was quite distinct from any of the seven types of juvenile atrophy described by Erb. Among the many thousand cases of muscular disorders that had been seen at the Vanderbilt clinic during the past ten years, this was only the second case of this particular type. The speaker said he wished to call special attention to the central and neuritic features of the case, in contradistinction to the pseudo-hypertrophic picture that was also presented.

A CASE OF CHRONIC PROGRESSIVE OPHTHALMOPLEGIA.

By Dr. Isador Abrahamson.

The patient was a man, twenty-seven years of age; single; a butcher by occupation, and a native of Germany. His family history was negative, with the exception of the fact that his mother had been paralyzed for years. The patient was anemic as a child, and had diphtheria when he was twelve years old. He smoked much, and drank beer excessively in former years. Eight months ago he had a small ulcer on the penis, which left no scar. He had no secondary symptoms of syphilis. His eyes were apparently normal up to six months ago, the date of the onset of his present illness. At that time he complained of pain in the left knee, like rheumatic pains. There was also general formication, and he was easily fatigued. Soon afterwards, there was pain in the left upper extremity, and immobility of the eyes. He could not rotate them, and the eyelids could not be raised. His speech was slightly altered and he felt tired when he spoke for a time, although his voice remained unchanged in pitch, etc. There was slight difficulty in swallowing. He also complained of delay in urination, with diminished potency, and constipation.

A physical examination showed a ptosis of both eyes; the pupils were equal, and reacted normally. The ocular movements were very much limited from side to side; also upwards, and to a lesser extent downwards. Convergence was preserved; divergence was considerably diminished. The superior oblique muscle seemed to be least involved. The lines on the right side of the face were flattened, and there was some weakness on that side. The mouth was drawn somewhat to the left, and hearing and bone conduction were diminished on the right side. There was weakness of the pharyngeal musculature. The tongue was protruded straight; there was no tremor; motor power in the upper and lower extremities was normal. There were no fibrillary twitchings; Romberg absent; knee jerks lively; Achilles lively; no clonus; sensibility normal; electrical examination negative. The disease was rather sudden in its onset, but made very little progress. Specific treatment and little or no influence up to the present time.

Clinically, Dr. Abrahamson said, the condition was a progressive chronic ophthalmoplegia. Whether it was of luetic nature, or indicated the early stages of a high tabes the future could only decide, yet the integrity of the pupils in spite of the extensive involvement of the ocular muscles seemed to negative the latter.

A CASE OF CONGENITAL MYOTONIA (THOMSEN'S DISEASE) ASSOCIATED WITH OPHTHALMIC MIGRAINE.

By Dr. Charles E. Atwood.

The patient was a Scotchman, twenty-three years old; single; a bookkeeper by occupation. He complained of left-sided headache, with left ptosis; also stiffness and weakness of the voluntary muscles, especially after rest, and becoming better with use. One sister, aged thirty-five, has had the same muscular disorder since childhood, but to a less marked degree than the patient. His oldest brother, when about ten years old, "could not start quickly to run." Otherwise, the family history was negative.

The patient, as a child, had pneumonia and incontinence of urine. At

nine years he had little strength to stand, and the wind from a passing football knocked him down. At the age of eleven or twelve he noticed that he went upstairs slowly, and could not lift heavy things. He also began to suffer from attacks of migraine on the left side, with vomiting, etc., occurring every six or eight weeks. He was also very constipated. At seventeen, the left eyelid began to droop during these attacks. When at play, he noticed that after resting it would be hard for him to start again, and he began to fear to go into a crowd because he could not move quickly enough to protect himself. These symptoms had persisted since, the migraine occurring intermittently for a week at a time about every six weeks, and when the patient was first seen at the Vanderbilt clinic he was suffering from one of these attacks of migraine, associated with paresis of the left third nerve, which disappeared in a few days. Other cranial nerves were free. The eye muscles affected were the lavator palpebræ, the left superior and inferior recti, and the inferior oblique.

The patient was well formed, and showed excellent development of muscles, without local hypertrophies or other external defects, the strength not being proportionately great. After rest, the voluntary muscles showed stiffness and rigidity or spasticity, both flexors and extensors and other groups being affected. The condition gradually wore off with exercise of the various muscle groups, only to recur again after a period of rest. There were no sensory changes to heat, cold, pain or touch. The right knee jerk was normal; the left diminished. He showed plainly the well-known myotonic (persisting) reaction of muscles to percussion and also the Erb (persisting) reaction after prolonged faradic stimulation and removal of the electrodes. Co-ordination and position and muscle sense were apparently unaffected. There was no Romberg; no astereognosis. There was no ankle clonus or Babinski. The patient was not paralyzed at all, but there was a feeling of resistance or persistent contraction which he had to overcome in performing any act. Mental excitement exaggerated the stiffness of the muscles. Ergograph tracings by Dr. Scripture showed an incomplete relaxation of both flexors and extensors at the initial of the muscle movement. Electric stimulation of the abductor indicis showed the same thing.

Dr. George W. Jacoby said he believed he was the first one in this country to demonstrate a case of congenital myotonia, and in connection with that disorder he thought it well to emphasize a little more strongly the myotonic reaction, because after all the diagnosis would depend upon that. In the case shown by Dr. Atwood, there was apparently no question in regard to the correctness of the diagnosis, although the history was a complicated one and contained some reference to a cerebral trouble. The case would probably have to be classed in Erb's second group, in which it was questionable whether the muscular disorder was primary or secondary.

In a typical case of myotonia, the three cardinal signs upon which to base the diagnosis and which constituted the myotonic reaction, were the persistence of the muscular contractions to the faradic stimuli, the same persistence to mechanical excitation, and the persistent contracted state of muscular groups after voluntary action.

Dr. Clark said that in looking over the literature of myotonia, he was surprised to see that hypertrophy was so little dwelt upon by Hale White, Thomsen and others. Writers agreed that at most the muscle state could easily be embraced in calling it the athletic type.

The possibility that the case shown by Dr. Atwood was not of the

congenital type was worthy of considerable attention, in view of the fact that the patient had suffered from paroxysmal migraine and constipation for many years. Possibly, the muscle state here was of toxic origin, which also had some bearing on typical Thomsen's disease, as had been demonstrated by Goldflam and others for family periodic paralysis.

Dr. Terriberry said he had reported two cases of myotonia occurring in the same family, both girls. In neither of them was there noticeable hypertrophy of the muscles. He was inclined to think that hypertrophy in these cases was a less marked feature in females than in males.

ANTERIOR POLIOMYELITIS WITH INCREASED KNEE JERK.

By Dr. Atwood.

The patient was a girl, three years old, who had enjoyed good health and had been able to walk at the age of two years. Last June, *i. e.*, six months ago, she had an attack of fever lasting two days, following which she has limped slightly in the right leg. The muscles permanently affected are the posterior tibial. In these there is reaction of degeneration. The knee jerk of the affected limb is greater than that of the left, and the case is interesting on this account.

A CASE OF ACUTE POLIOMYELITIS IN AN ADULT.

By Dr. Atwood.

The patient was a man, thirty-three years old; a college student. His family and previous history were negative.

In August, 1906, nearly five months ago, he had a fever of several days' duration, accompanied by certain subjective symptoms, and the gradual onset of paralysis in his right arm, beginning in the shoulder and upper arm and culminating in the hand and finger muscles after thirty hours. An examination showed that sensation to heat and cold, pin prick and cotton were preserved. There was marked wasting of the right upper arm, shoulder and extensor surface of the forearm; also slight wasting of the right anterior tibial. The hand and affected portions of the arm were cooler than the left arm to the touch. The position of the hand was peculiar, on account of the extensor paresis. The muscles affected were the pectoralis major (upper portion), the supra and infra-spinatus, the deltoid and coraco-brachialis, the biceps, triceps, the extensors of the wrist and the long extensors of the fingers. There was also weakness of the pronators and supinators, and of the abductor pollicis.

This case, Dr. Atwood said, was thought to be of some interest on account of the comparative rarity of acute anterior poliomyelitis in the adult, and of the clearness of the subjective symptoms. In connection with the treatment of this case, the speaker inquired regarding the advisability of nerve transplantation.

Dr. Leszynsky, in discussing Dr. Atwood's first case of poliomyelitis, with increased knee jerks, said he had seen a number of cases in children where in the early stage of the disease the knee jerk was entirely absent on the affected side, but gradually returned and remained active, while the peroneal group of muscles was paralyzed and remained so.

Dr. Clark said it had occurred to him that we had a very simple explanation for the exaggeration of the knee jerks in certain types of poliomyelitis; *i. e.*, those affecting the lower posterior group of muscles

of the leg. As was well known, these muscles were represented in groups of anterior horn cells at the side of the anterior horn, and, as was also well known, the central branch of the anterior spinal artery supplied more than the gray substance of the cord; in fact, it supplied nearly all of the lateral limiting layer that was situated between the gray substance and the lateral pyramidal tract. Injury to the fibers of the lateral limiting layer destroyed the controlling connection between the first and second segments of the motor system, and then, as in hemiplegia, we might expect to find an unrestrained reflex center in the anterior horn, giving rise to an exaggerated reflex. That the injury outside the gray matter was confined to the lateral limiting layer was evidenced in that the exaggerated reflex in these cases did not bring the Babinski sign with it, or other definite symptoms of serious injury to the pyramidal tract.

Dr. Clark believed that a sharp distinction should be drawn between the ordinary cases of poliomyelitis, in which the reflexes returned in the repair process, as cited by Dr. Leszynsky, and the type Dr. Atwood showed. The latter had been very carefully studied from their *very inception,* and had presented the unique reflex exaggeration from the first stroke of the disease.

Dr. Terriberry, in reply to Dr. Atwood's query as to the advisability of nerve transplantation in these cases, said that although some excellent results had been obtained in nerve grafting, he believed that in suitable cases much more prompt and efficient repair to loss of muscle balance could be obtained by muscle transplanting; he instanced a case of substitution of the sartorius for a useless quadriceps extensor which he had seen recently, with a most happy result, and this was but one of many very excellent results of attempts at muscle balancing that he had seen at the Hospital for Ruptured and Crippled Children in this city.

The following officers were elected for the ensuing year: President, Dr. Charles L. Dana; First Vice-President, Dr. B. Sachs; Second Vice-President, Dr. L. Pierce Clark; Corresponding Secretary, Dr. Hallock; Recording Secretary, Dr. E. G. Zabriskie; Treasurer, Dr. Graeme M. Hammond.

NEW YORK NEUROLOGICAL SOCIETY

February 5, 1907,

The President, Dr. Charles L. Dana, in the Chair.

DISCUSSION OF APHASIA, WITH PRESENTATION OF CASES

By Dr. B. Sachs

The subject of aphasia is of extreme interest at the present moment, owing chiefly to the articles that had appeared recently from the pen of Marie, which, at least in some important respects, threatened to revolutionize our past conception of speech disorders. For the past half century we had accepted the dictum that motor aphasia was due to a lesion of the third frontal convolution. Marie had shown that that dictum was based on the slimmest possible evidence; he questioned the existence of a pure motor aphasia, and the possibility of differentiating anatomically between various forms of aphasia. He doubted whether there were any cases of pure motor aphasia, and also, whether in so-called sensory aphasia there was really a loss of the true perception of words, or whether it was more or less an intellectual defect. It was very difficult, Dr. Sachs said, to make out exactly what Marie meant by the term "intellectual defect"; whether he meant a defect in single or collective concepts, or in general intellectuality.

In connection with this subject, Dr. Sachs showed a woman, 39 years old, who he said represented the most complete example of loss of motor speech that he had met with in many years. The case was not one of pure motor aphasia, as there was just as marked a defect on the sensory side as on the motor, and although the woman had a most complete loss of language, she retained her musical sense to a remarkable degree. The history of the case was that about four months ago she had sustained a right hemiplegia, which was clearly due to embolism. At the same time, she suddenly developed this complete motor aphasia, combined with sensory aphasia. It was impossible to determine whether there was also word blindness or agraphia, as no reliable information could be obtained concerning the woman's mental attainments prior to her illness. She was very emotional, and while her speech was limited to jargon, it was easy to demonstrate that her musical sense was very good. There was no hemianopsia nor hemianesthesia; no apraxia.

Dr. Sachs said he was impressed with the fact that sensory aphasia, pure and simple, was of extremely rare occurrence. This had particularly impressed him in connection with otitic temporal abscess, where we should expect to find sensory aphasia, pure and simple, according to Wernicke's theory. As a matter of fact, the speaker said he had never seen a case of temporal abscess in which sensory aphasia was not also associated with some form of motor speech disturbance.

A CASE OF PRE-SENILE DEMENTIA WITH APHASIC SYMPTOMS

Presented by Dr. Leslie Meacham for Dr. Dana.

The patient was a man, 61 years old; a court photographer by profession, who was first seen at the Cornell University Medical College

Dispensary on February 1, of the present year. His father had died of apoplexy at the age of 64; his mother had died of tuberculosis. Four brothers and sisters were alive and well; one had died from nephritis and five from various diseases. The patient was the father of eight children; six of these had died at or soon after birth, and his wife had had one miscarriage.

The patient had pneumonia ten or twelve years ago. He denied all venereal history, and had been very temperate in his habits. For some time he had been troubled with shortness of breath, and about three years ago he had an attack of dizziness. He did not fall nor become unconscious, but for several days afterwards his speech was thick, and for six or eight weeks he had much difficulty using his left hand and arm. There was also some difficulty in walking, and the left hand had remained permanently weak. He had suffered much from indigestion, and had practiced gastric lavage, but had discontinued it after this seizure.

In September, 1905, he awakened one night about midnight with very severe pains in his head. He also had severe pains in his stomach, which continued for two or three days, and for the relief of which he was given codeine by his physician. Since that time he had had much trouble with speech, and had been unable to write any letters, but he was still able to read. He had become timid about going out alone, fearing he might become lost. He had continued to direct his business, but had been unable to use the camera in the preparation of legal exhibits. He had also become unable to get correct scales, and was uncertain about the use of the chemicals employed in the development of photographs. During the summer of 1906 he engaged in active exercise in order to reduce his weight and relieve his shortness of breath. In September, 1906, after certain exercises, he complained of feeling dizzy, and this persisted for about two weeks. After this he found that he was no longer able to read, as he would frequently lose his place, and could not grasp the meaning of what he read. His speech became more difficult, and he had some trouble in understanding what was said to him. He was even unable to read his prayers, which he had read daily during his entire life. He was still able to give instructions as to the proper conduct of his business, but could not converse with customers. At times, he had outbursts of laughter or tears, and became irritable when he could not make himself understood.

Examination showed that the facial innervation was unequal, probably as the result of his old hemiplegia. There was some drooling from the left corner of the mouth. Gait and station were normal, with the exception of slight uncertainness and some vertigo. The pupils were equal, and reacted to light and accommodation. There was no hemianopsia; no atrophy nor tremor of the tongue. Taste and smell were normal, but he showed some hesitancy in being unable to recall the names of substances. Hearing was apparently dulled, but was equal on both sides, and he understood names of objects producing sounds. The palate reflex was present. The deep reflexes were active; the superficial ones were present, but the left plantar was diminished and sluggish. There was a systolic heart murmur, with accentuation of the second sound, and some cardiac irregularity. The patient's voice was rather low-pitched and somewhat monotonous. He hesitated, then spoke a few words rapidly, followed by another hesitation, and stated he was afraid

that he would forget what he wished to say. His wife asserted that he had always had some trouble in speaking. He could not picture words. He could spell, but could not tell the number of letters in words of even three letters without counting on his fingers. He could repeat the Lord's prayer correctly, but brokenly and hurriedly. Repetition was much affected; voluntary writing was bad and copying even worse. He had a clear understanding of the use of articles, and there was no astereognosis. He could carry out simple instructions, and could usually do two things, but never three. For many years he had been a member of a number of secret orders, but he could no longer recall the passwords. His urine contained a trace of albumin and some mucus and quamous epithelium; no casts; no sugar; it was acid, with a specific gravity of 1,010. He habitually drank large quantities of water.

The president, Dr. Dana, said the case shown by Dr. Meacham was not presented as one of distinct aphasia, but rather as one of pre-senile dementia, although the patient lacked many of the characteristic symptoms of dementia, while on the other hand he showed many of the characteristics of mixed aphasia. He regarded the case as a form of aphasia associated with senile dementia. The symptoms of dementia were apparently limited to the language zone. The case was certainly rather unique, especially in connection with the recent dictum of Marie that aphasia was associated with an intellectual deficit.

A CASE OF APHASIA.

By Dr. C. L. Dana and Dr. J. Ramsay Hunt.

The patient was a man, 45 years old, who was brought to Bellevue Hospital on November 26, 1906. He was suffering from complete motor aphasia, together with a moderate degree of right hemiplegia. He was unable to express himself or answer the most simple questions. Upon admission, the light reactions of the pupils were somewhat stiff, and it was thought for a time that the case might be one of aphasia associated with early paresis. In the course of a few days, however, his speech disturbance began to clear up; he was able to say a few words and then a few sentences, and this improvement has been progressive. There were some sensory disturbances on the right side, which still persist in a moderate degree, and he has also evidence of a right hemianopsia. On admission, he could understand and was able to carry out single directions, such as those to close the eyes, etc.

From a friend who visited the patient at the hospital it was learned that the latter, for several weeks prior to the onset of his attack, had complained of headaches and a distinct weakness in the right leg. The patient was now able to speak fairly fluently; he was still unable to make himself understood or to name certain objects. He has alexia and agraphia. The case was regarded as one of occlusion of the posterior cerebral artery, probably syphilitic in origin. (There were large pigmented scars along the tibia in both legs, and a somewhat doubtful history of a primary lesion.)

Dr. Dana said that while this patient had fairly good voluntary speech, and while his general intelligence was excellent, there was a jargon of sentences which made his speech unintelligible. He was unable to write. He understood words and sentences, and did what he was told. His worst defect, apparently, was the anomia, which applied not only

to objects seen, but also to objects felt and heard. His ability to read was absolutely gone. The case was one of pure sensory aphasia, with anomia as its dominant feature, and with it was associated agraphia, and, as was usually the case, alexia. The lesion in this case was regarded as a thrombosis, or perhaps a small hemorrhage.

In considering this general subject of aphasia, Dr. Dana said he was inclined to believe that the new theory of Marie regarding the localization of these speech disorders was really much nearer correct than the views that had been accepted by the profession for half a century or more. While these disorders were the result of a lesion in the so-called aphasic area, he believed it would be wise to either cut out the Broca convolution from the zone of language, or else regard the latter as only more or less distantly associated with the real aphasic area, and of minor importance in that connection. The frontal lobe governed, apparently, the centers for memory of the movements of articulation rather than the true centers of speech. It controlled, apparently, certain forms of disturbance of speech, such as stuttering, but was not intimately associated with the higher speech centers.

Dr. Joseph Collins said that the views of Marie seem at first sight to be more revolutionary than they are in reality. The most far reaching effects of Marie's conception are in the psychological conceptions of the development of speech and its production. There is no doubt that the schematic or diagrammatic representation of the speech areas has been carried too far, and the definiteness with which verbal memories and visual memories, and memories of movements of the spoken and written words have been allotted to certain small areas has been overdone, but this does not justify the overthrow nor the attempt at overthrow of that which has been definitely proven in regard to the localization of the different forms of memories, the awakening of which constitutes the initiation of articulate speech. Marie's views are sufficiently definite and his position in the scientific world sufficiently firm to demand that his claims be verified or denied. This verification or denial cannot be through expression of personal opinion, but by study of patients and of the lesions which are found in their brains after death. Much that Marie claims is by no means new. For instance, everyone who has much to do with aphasia will agree with him that there is in all aphasic individuals a diminution of intellectual capacity which amounts to real enfeeblement. Anyone who would claim the contrary could scarcely have an adequate conception of the enormous structural lesions that are at the basis of the particular lesion that gives rise to the aphasia. In the vast majority of cases of aphasia there is widespread arteriosclerosis, and the nutrition of the brain and its dependent psychological functions are proportionately disordered.

Although Marie has brought forward much evidence to show that many cases of so-called motor aphasia, i.e., aphasia due principally to the loss of memories of articulation, is dependent upon a lesion of the posterior areas, i.e., of the first temporal and the inferior parietal convolutions, plus anarthria; although Dr. Collins for one, was willing to admit that in some instances this condition is the one that exists and which accounts for the symptoms, still there are cases on record in which the lesion is closely limited and confined to the allotted seat of memories of articulation, viz., to Broca's convolution, and in these cases the symptoms that occurred were typical of motor aphasia. Dr. Collins

referred particularly to the cases of Ladame and of Bernheim. One such case as either of these is as good as one hundred to demonstrate that motor aphasia may be the result of a lesion so situated.

Finally, a word in regard to Marie's conception of anarthria. First, it must be admitted that Broca's convolution sends no projecting fibers directly into the motor tract, and that therefore a lesion of Broca's area does not cause anarthria. Second, Marie's idea of anarthria and its relation to destruction of the lenticular area is not materially different from Dejerine's idea of subcortical motor aphasia, or pure motor aphasia, as he calls it. Finally the only way of settling this question is not academically, but at the bedside and at the post-mortem table, and that work we are called upon to do.

Dr. B. Onuf said that frequently, in cases of aphasia, the autopsy showed very extensive lesions, making the cases unfit for differential diagnosis, inasmuch as such lesions caused the involvement of so many functions that it was difficult to determine which part of the lesion had impaired this function or the other. As proof of this statement Dr. Onuf adduced three cases (two of aphasia and one of dysarthria) which had been clinically observed by Dr. Fraenkel and himself, and examined post-mortem in several sections. In all three of these cases the lesions had been very extensive, destroying a large part of the hemisphere.

The question of the role of the third frontal convolution, which Marie denied had any relation to the function of speech, had been carefully investigated by Drs. Fraenkel and Onuf, and embraced the study of 104 cases of aphasia, with autopsies, collected from the literature. This had shown on what a remarkably small number of clear-cut cases the view of the speech functions of Broca's region was based. Only five of the 104 cases showed a lesion confined to Broca's area, while in two cases there was a slight encroachment on adjoining regions. In all the other cases, Broca's region was either not involved, or there were marked additional lesions of other cortical regions or of the sub-cortex. Of the seven cases first referred to, only two showed clearly a lasting aphasia, while in the others it was either only temporary or not sufficiently long observed to say whether it was temporary or permanent. Nevertheless, the fact that all of these seven did show aphasic disturbances, whether temporary or permanent, was in confirmation of the importance of Broca's center for the function of speech.

To elicit further evidence regarding the function of Broca's center, Drs. Fraenkel and Onuf tabulated, graphically, all the cases of purely cortical lesions in their collection, according to the scheme of Naunym, which consisted in having the surface of the brain divided up into squares of equal size, and registering the lesions on that diagram in the following manner: Whatever squares were covered by the lesion in a particular case received one dot. If a given square fell within the domain of the lesion of a second case, that square received a second dot, and so forth, so that the squares showing the greatest number of dots indicated the greatest number of cases in which those particular regions had been affected. The result of this registration showed such a marked accumulation of dots in the region of the foot of the third frontal convolution that the view of the important role of that region for the function of speech was amply confirmed.

Marie's views regarding aphasia, as set forth in his first article, ex-

perienced a considerable elaboration, modification and crystallization in his second article, making some things clear which did not appear so in the first article. The exact meaning, for instance, of the word "anarthria" was by no means clear from the first article, but was distinctly defined in the second one. It appeared that some of his differences of view from those of other writers were partly in name only. Anarthria, for instance, was substituted for the term aphasia motrice pure of other authors, and was acknowledged clinically but given another anatomical basis by Marie. That basis was a lesion of the so-called lenticular zone, *i. e.*, of the zone consisting of the white substance situated between the cortical convolutions of the insula and the lenticular nucleus, and of the outer layers of the lenticular nucleus itself. Marie gave no definite explanation why such lesions should cause anarthria.

The researches of Drs. Fraenkel and Onuf, on the other hand, endeavored not only to find an anatomical basis for the aphasia motrice pure, but also to explain the mechanism of the lesion. Their results spoke against the existence of a direct speech pathway leading from Broca's area of the internal capsule, inasmuch as lesions of the internal capsule were relatively very rare in sub-cortical cases of aphasia in the 104 cases they had collected. On the other hand, the lesion mostly met with in those cases was one of the medullary layer of the third frontal convolution, and next in frequency the region around the upper margin of the lenticular nucleus in its anterior part was involved. This strongly confirmed their theoretical considerations that these two regions formed part of an association pathway connecting Broca's area with those centers situated at the base of the central convolution which innervated the muscles concerned in the function of speech; *i. e.*, the muscles of the tongue, larynx, pharynx, lips, etc.; briefly, the articulo-motor muscles. They confirmed their view that Broca's center was a higher co-ordinatory center, presiding over the articulo-motor centers just mentioned, grouping those innervations of the latter which were necessary for the purpose of articulation.

Dr. Joseph Fraenkel said that by accepting Marie's views we gave up a great deal of what we had for years thought to be the best established truths of neuro-physiology. Personally, he did not hesitate to say that Dr. Onuf and himself had done a great deal of work in connection with aphasia, and for some reason that work had received no notice. During his residence at the Montefiore Home he had seen and thoroughly studied a goodly number of all forms of aphasia, and had witnessed the autopsies of a considerable proportion of them. As a result of this he believed:

1. That we must clearly know and define the terms we are using, and appreciate the fact that aphasias are all disturbances of speech resulting from an interference with the psychic mechanism of speech, and that anarthrias are all disturbances of speech which are the result of interference with the somatic or mechanical mechanism of speech.

2. That there undoubtedly exist—rarely, to be sure—forms of pure motor aphasia, cortical and sub-cortical.

3. That rarely would the lesions producing these forms of aphasia be so strictly localized as not to encroach upon neighboring territories, and even so, the underlying disease would to an extent interfere with the other functions of the brain, thus producing some intellectual impair-

ment, which would appear in every case at some time or other.

4. That their investigations had shown the existence of the lenticular association zone, which in their belief was the sub-cortical speech path, and which led, when diseased, to what they called sub-cortical motor aphasia, and what Marie called anarthria.

In connection with Dr. Sachs' case, the speaker said he wished to mention that Edgren had shown that the musical concepts and pictures were stored in the second left temporal convolution, and that therefore the preservation of this faculty in the patient presented by Dr. Sachs was not surprising.

Dr. L. Pierce Clark said if Marie's theory that an intellectual deficit was always associated with aphasia should be proven, it might have an important bearing from a medico-legal aspect, particularly regarding the will-making capacity of apoplectics.

Dr. Sachs, in speaking of the case of aphasia he had shown, said that if Dr. Collins had been present at the time, he could not possibly have made the statement that the patient did not have sensory aphasia, or that it was simply a case of motor aphasia.

This general subject of aphasia, Dr. Sachs said, was one of the utmost importance, and in spite of the statement made by Dr. Collins, and the quotation from Marie's recent article, the reference made by that writer to an intellectual defect in aphasics was not very clear, particularly from a psychological point of view. Personally, he believed Marie meant that the defect was entirely in the conception and perception of speech, and did not necessarily imply a distinct mental defect. Mental deterioration could be safely denied in a large number of cases of aphasia, and the speaker cited such a case that was at present under his observation at Mt. Sinai Hospital. The case was one of motor aphasia, pure and simple, in a man of considerable intelligence, and there was not a single indication of any mental deterioration.

In a close analysis of cases of motor aphasia, Dr. Sachs believed that the chief difficulty was not connected with the concept movements, but was simply a difficulty in directing the movements of speech. He thought there were very few cases in which the actual conception of movements that were necessary to effect articulation was absent; the difficulty was simply an inability to use the organs of speech. He was perfectly willing to substitute the word anarthria for the French term aphasia motrice pure, and if that was to be the outcome of Marie's doctrine, he considered it a distinct step in advance. It was unfortunate, he thought, that the work of Onuf and Fraenkel in connection with aphasia had not received more general attention.

Dr. Dana said that this subject of aphasia would probably come up before the Society for discussion again at some future period, and that at present it was impossible to arrive at any absolute conclusion regarding it. The entire question should be taken up in a receptive mood, and carefully re-studied. In the course of his teaching, Dr. Dana said, he had at times been somewhat embarrassed by the practical inutility of the older methods of presenting the subject of aphasia, and he had never been quite able to make the text-book descriptions of these speech disorders tally with his clinical findings. He was inclined to agree with those who believed that the entire subject should be studied from a little different point of view, which implied, essentially, that the cases should be studied as we saw them. We practically never saw cases of either pure

motor or pure sensory aphasia, and it was very difficult to explain this difference to students.

Dr. Dana said he agreed essentially with the views expressed by Dr. Sachs that the Broca convolution was not in the same class as the rest of the zone language; that it was of a lower type, and while cortical disease in that region might cause aphasia, it was a loss of articulation and not of the concept of language. He did not like the term anarthria.

ADDRESS OF THE RETIRING PRESIDENT.

By Dr. Joseph Fraenkel.

The retiring President, in a brief address, thanked the members of the Society for their support during the past year. He said that the gloomy views that had been held out in regard to neurology in recent years had not been realized: on the contrary, the outlook in this branch of medical science was brighter than ever, particularly in connection with the clinical investigation and study of function, a field that had been too much neglected in the past.

ADDRESS OF THE INCOMING PRESIDENT.

By Dr. Charles L. Dana.

Dr. Dana said that since his first experience in that office there had been many intensely interesting phases in the evolution of modern neurology. The finer anatomy of the nervous system was then a matter of active discussion, because it was just being unfolded. The exploitation of the neurone theory, the localization of the cortical and spinal functions, the changed views of neuro-pathology, the better descriptions of the various nervous diseases, the new diagnostic methods and signs and the portrayal of new types furnished a constant succession of interesting themes and topics for discussion. There was now a feeling that all of this was more or less completed, at least in its larger outlines, and to a certain extent this was true, but it was no more true than for other specialties, or even for internal medicine.

Dr. Dana said that all over the world where neurological societies existed he had found that the main body of the work was in the clinical presentation and study of cases, and this was the line along which the New York Neurological Society would have to continue. We could not tell at what moment the deeper knowledge of physical forces, of chemistry, radio-activity, etc., would evolve new problems and rich illumination. The time had come when the neurologist had to keep in closer touch than ever before, perhaps, with the laboratory, especially in the solution of functional neuroses and psychoses.

Among other topics touched upon by Dr. Dana in his address was the duty of the neurologist in relation to public economic questions; the great necessity for a hospital for nervous diseases in New York City, especially for the middle classes; a study of the nervous and mental diseases incident to our American mode of life; nervous diseases and diseases in general in relation to the occupations of the poor; and the proper enlightenment of the public in regard to the evils of the various forms of faith cure and charlatanry. The speaker said he looked upon the psychic side of neurology as still quite a fresh field. There was a whole world of subjective complaints that had heretofore been dismissed with impatience, but which really deserved

study and classification. The neurologist had also been silent on the subject of alcoholism as a social evil and cause of neuroses. In short, the neurological conscience needed quickening, and the time had come when neurologists had to take a more important part in the world as physicians. Otherwise, they would sink into insignificance as mere technical experts in the occasional correct diagnosis of tumors and obscure organic lesions that they could only point out and not cure.

REMARKS ON THE CURABILITY OF A RARE FORM OF NOCTURNAL PETIT MAL EPILEPSY BY USE OF LARGE DOSES OF BROMIDE, WITH NOTES OF FOUR CASES.

By Dr. L. Pierce Clark.

Dr. Clark stated that rapidly recurring nocturnal petit mal was a rare form of sleep epilepsy which occurred both independent of and in connection with grand mal seizures. The general exhibition of the type was the same, whether associated with grand mal or not. Herpin called this type "intractable" epilepsy, defying all medication. No writer had made extended comment on the condition. Bromides, as ordinarily administered, invariably increased the attacks. That fact had caused many observers to diagnosticate the state as a form of hysteria, a disorder of sleep such as pavor nocturnus, somnambulism and the like.

Dr. Clark said he had seen but four cases of this type of epilepsy in a material of several thousand. Aside from the peculiarity of the attack, the epilepsy in which the seizure occurred did not differ in causation or course from idiopathic grand mal. However, its termination under the specific treatment was quite different. The attacks of nocturnal petit mal invariably occurred while the patient slept, either by day or night. It usually began as soon as he fell asleep. There might be as many as 300 separate and distinct attacks in a single night. The patient usually awoke from a deep sleep, the eyes widely open, the pupils dilated and irresponsive to light, the head moving from side to side. There was an anxious and furtive look in the face, which might be either congested or very pale. In a few seconds after the onset, the patient executed some incredibly rapid movements of the hands or feet, but with no clear intent or purpose such as was seen in somnambulistic states. For instance, the patient might drum on the bed, kick aimlessly into space, or beat his head, thigh or chest in a senseless way. He might spring into the air or turn somersaults rapidly. Sometimes, the patients exhibited attention when spoken to, but they made no coherent reply. Unless disturbed, the patient soon passed into normal sleep at the end of the attack.

Dr. Clark then reported in detail four cases of nocturnal petit mal, all of which were relieved by large doses of bromides. In one case where 60 to 120 grains of the drug daily were not only ineffectual in controlling the attacks, but actually increased the number threefold, the attacks ceased entirely after a daily dosage of 400 grains, continued for five days. The dose was then gradually decreased to 230 grains daily, at which amount the medication was sustained for several months. The patient had no more awakenings nor epileptic manifestations whatever, and he was now in excellent mental and physical health, which was in marked contrast to his former hopeless state

Dr. Clark said the principle of high dosage of bromide might be stated as a form of hyperbromidism, bromide intoxication or poisoning. The drug was given in a steadily increasing dose, as was the iodide in specific disease. The patients received a high rectal irrigation once or twice daily to reduce the amount of congestive catarrh, eliminate toxins, and for its diuretic effect. They were also given prolonged hot packs and baths at night for diaphoretic effect, and cold douches, sprays and cold drip sheet treatment in the morning. Vigorous deep massage was steadily maintained to cleanse the muscle circulation. Incipient heart failure might be gauged by the second pulmonic sound and the degree of splitting of the second tone; the latter was easily controlled by strychnia and digitalis. Diet was carefully regulated, and no meats were allowed. In brief, the object aimed at was extreme impregnation of the cortex, while the somatic effect of the bromide was reduced to a minimum.

The speaker said that while it was highly gratifying that this peculiar and rare type of sleep epilepsy was most amenable to amelioration or cure by extreme bromide medication, the care and attention that such cases must necessarily be given during the course of the treatment could not be too thoroughly insisted upon.

Dr. B. Sachs said that under a recent ruling, the State Commission in Lunacy took the ground that insane persons could not be harbored in any home or clinic that was not regularly licensed. By this ruling, Dr. Sachs said, an injustice might be done in many instances not only to the physician, but to the patient. He thereupon moved that a committee be appointed to communicate with the State Commission in Lunacy in regard to the matter. This motion was carried, and a committee of three was appointed by the President to investigate the matter and report at the next meeting of the Society.

Periscope

Revue Neurologique

(Vol. XV. No. 1. Jan. 15, 1907.)

1. Crises of Petit Mal, with a Paramnesic Aura. Illusion of False Recollection. J. SEGLAS.
2. True Tactile Aphasia. ERNEST JONES.

1. *Paramnesic Aura in Petit Mal.*—There were sudden attacks in which the patient had the impression that he had been exactly in the same circumstances before. These false recollections in this case were an aura to an epileptic petit mal attack, pallid face, eyes fixed and staring, etc. On gradually recovering from this state there was an amnesia, more or less complete, for the entire period of the crises, including the period of the false recollection.

2. *Tactile Aphasia.*—Raymond and Egger showed before the Neurological Society of Paris a patient who was unable to recognize objects placed in his hand. Raymond named this condition "tactile aphasia." Claparede suggested for this condition the name of "tactile asymbolia," reserving the name "tactile aphasia" for a condition where there is only an inability to *name* the object. He considered the occurrence of such a case would be very improbable inasmuch as our memory of words is much more connected with the other senses such as vision, audition, etc. The author reports a case of true tactile aphasia, the first reported. The patient had traumatic hysteria with anesthesia. In the progress of recovery he passed through four stages; first, complete anesthesia; second, he could feel the objects in the hand, but had lost the sense of localization and could not tell their form; third, could perceive the form of objects and name their attributes, but he did not know what the object was; for instance, a piece of money was "a piece of metal, round and flat, but he did not know what it was," this phase corresponds to the "tactile asmbolia;" fourth, he recognized the object and knew its use, but simply could not name it, true tactile aphasia.

(XV. No. 2. Jan. 30, 1907.)

1. The Lesions of the Spinal Roots, the Root Ganglia and Nerves in a Case of Friedreich's Disease. Studied by the Method of Ramon y Cajal. J. DEJERINE and ANDRÉ THOMAS.

1. *Friedreich's Disease.*—A typical case of Friedreich's disease of 22 years duration. The authors calls especial attention to the preservation of all forms of sensation in the limbs up to a short time before death. Macroscopically the spinal cord showed a general diminution in size, a gray color of the posterior columns and of the lateral columns in the region of the crossed pyramidal tracts extending from the sacral region to the medulla. There was a marked grayness of the posterior roots which was in marked contrast to the anterior roots and the pia was thickened and irregular on the posterior surface of the cord, particularly in the lumbar region. Examination by the osmic acid method showed normal anterior roots, but marked atrophy of the posterior roots; there was nothing, however, to indicate an active process of degeneration. The atrophy was present in the fibers of the posterior root ganglia, and distally

to the junction of the anterior roots. In the cervical region the atrophy of the fibers proximal to the ganglia was more marked than in those distal to the ganglia, but in the lumbar region the process was about equal on each side of the ganglia. Lesions of the peripheral nerves were similar to those in the posterior roots and were more marked in the cutaneous than in the muscular nerves. Studies of the spinal cord by the method of Ramon y Cajal showed that axis cylinders had completely disappeared from the posterior columns except in the cornu-commissural and posterior radicular zones.

(XV. No. 3. Feb. 15, 1907.)

1. Paralytic Ptosis in Hysteria. CH. SAUVINEAU.
2. Paralysis of the Abducens Nerve and the Cerebral Arachnoid Space. F. LEVY and A. BAUDOUIN.

1. *Ptosis in Hysteria.*—The first observation was in a child of eleven years who after punishment developed a bilateral ptosis. There was no sign of blepharospasm. She was cured by one treatment with a slightly painful faradic current. A recurrence was cured by massage of the lids and suggestion. In the second case, in a child thirteen years old, there was a phlyctenular conjuctivitis. On the left side there was blepharospasm, on the right a paralytic ptosis with no signs of blepharospasm. The ptosis was cured in one seance by suggestion, but the blepharospasm persisted.

2. *External Motor Ocular Paralysis.*—A transitory paralysis of the abducent nerve may follow spinal anesthesia. The author reports two instances of a similar occurrence following the deep injection of cocaine for the relief of facial neuralgia.

(XV. No. 4. Feb. 28, 1907.)

1. Two Clinical Observations of Pseudobulbar Palsy Without Paralysis of the Limbs. HENRI LAMY.
2. The Pathogenesis of Mercurial Tremor. GUILLAIN and LAROCHE.

1. *Pseudobulbar Palsy.*—A man, aged sixty-two, developed a sudden dysarthria and paralysis of the lower portion of the right side of the face. There was also a remarkable anesthesia of the mucous membrane of the pharyngo-palatine region. The tongue protruded slightly to the right side. There was no psychic trouble, no difficulty in deglutition and no paralysis of the limbs. There was spasmodic laughter. The second case was very similar. The author thinks that the lesion in each case was in the external portion of the right lenticular nucleus. The facts suggest a lesion in a territory which presides over the function of articulation of words and phonation.

2. *Mercurial Tremor.*—The author concludes that the tremor produced by mercurial intoxication is not hysterical, and may endure a long time after the cessation of the exciting cause. The symptoms would indicate a lesion of the cerebellum or of the cerebellar tracts.

A. R. ALLEN (Philadelphia).

Brain

(Vol. 29. No. 115.)

1. On Some of the Nervous Complications of the Specific Fevers. SIR THOMAS BARLOW.
2. The Development of the Vertebrate Nerve Cell: A Cytological Study of the Neuroblast Nucleus. JOHN CAMERON.
3. Miner's Nystagmus. A. CHRISTIE REID.

1. *Nervous Complications of Specific Fevers.*—This is pre-eminently a clinical paper, and one, though discursive, filled with much wealth of experience. Speaking of diphtheritic neuritis, Barlow reports instances of similar nature occurring after measles. Acute ascending paralysis due to measles he also reports, as well as hemiplagic and disseminated sclerosis syndrome pictures. The specific infectious organism in anterior poliomyelitis is unknown. Barlow suggests that there is little doubt that measles, scarlet fever, typhoid and even chicken pox are to be reckoned with. Disseminated encephalomyelitis as a complication of the specific fevers is mentioned and stress laid upon transverse myelitis in the same connection, Multiple neuritis and hemiplegia are also discussed as secondary to the infectious fevers.

2. *Development of Vertebrate Nerve Cell.*—An extensive and detailed embryological paper copiously illustrated, from which the author's summary may be quoted in full: (1) The scanty cytoplastic investment which the neuroblasts possess during the early development stages takes little part in producing the abundant cytoplasm of adult nerve cells. (2) The latter is to be regarded partly as a product of the metabolic activity of the neuroblastnuclei during metabolic life. (3) One of the earliest indications of this metabolic activity is shown in the lower vertebrates (*e. g.*, fishes and amphibians) by an ingestion of yolk particles at one definite pole of the nuclear wall. For this the title of assimilative pole has been adopted. (4) The ingested yolk is elaborated within the nucleus and stored up as chromatic material which first shows near this pole. (5) The ingestion of the yolk is further evidenced by a progressive increase in the size of the nuclei, which occurs during the period when the absorptive process is going on. (6) The rudiment of the axis-cylinder process arises in the form of successive extrusions of nuclear achromatic substance from the assimilative pole of the neuroblast nucleus. This pole thus becomes the axon pole. (7) It is to be clearly understood that the term achromatic is applied throughout to a fluid substance which is nuclear in origin, and is in the nascent condition strongly resistent to the action of all coloring agents. This term is therefore applied in contradistinction to the chromatic or staining elements of the nucleus. It is possible that several distinct substances may be included under this title of achromatin; but from its very nature it has as yet defied attempts at learning its composition by means of microchemistry. (8) The assimilative or axon pole of the neuroblast nucleus has always a definite direction. Thus it looks towards the ventricular cavity in the cerebral hemispheres, and forwards as well as outwards in the anterior cornua of the spinal cord. (9) Not only the axon rudiment, but also a great part of the cytoplasm of the future nerve cell is produced from the neuroblast nucleus, owing to the continued exercise of the achromatin function. (10) The nuclear metabolic product is extruded mainly from the axon pole. This would account both for its gradual accumulation in this region and also for the eccentric position of the nucleus in the embryonic nerve cell. (11) The material which is

given off from the neuroblast nucleus is achromatic only in the nascent condition. It very soon, however, undergoes a characteristic process of re-chromatization which affects the extruded perinuclear substance in a definite manner and gives rise to the neuro-fibrillary network and the Nissl bodies. (12) The neuroblast nuclei exhibit structural changes as evidence of their metabolic activity. The most striking is a disappearance of the accumulated chromatic material from the axon pole, which occurs during the period that the axon rudiment is being given off. The chromatic element is thus re-transformed into nascent achromatic element, in which condition it is discharged from the nucleus. (13) In some regions, the retina, cerebral hemispheres and ganglion habenulae, of lower vertebrates, the neuroblasts exhibit in addition a marked reduction in size during the period that this achromatic element is being discharged. (14) Three types of neuroblasts may be recognized. For these the names of A. B., and V-neuroblasts have been adopted. (15) The a-neuroblasts retain their embryonic character more or less. They probably form a reserve of young nerve cells. (16) The b-neuroblasts become invested by a very scanty envelope, their process thus appearing to pass directly from the nuclei. (17) The v-neuroblasts become transformed into actively functioning nerve cells, and become endowed with an abundant investment which forms the adult cell-cytoplasm. (18) It is important to note that the nerve cells begin their life history as an a-type of neuroblast, and pass successively through the b and v-types. (19) Several observers have shown that the cytoplasm of nerve cells is rich in nuclein compounds. This seems to suggest that a part of it at least is derived from the nucleus as shown in this research. (20) The nucleus not only gives part origin to the protoplasm of the nerve cell, but also furnishes a fresh supply of material to replenish the latter, when occasion demands, throughout the life history of the individual. The nucleus is thus the nutritive center for the nerve cell. (21) It is thus obvious that a modification in our present acceptation of the Wallerian law of degeneration will be necessary. The latter ought therefore to read somewhat as follows: An axon when cut off from its nutritive center, the nucleus of the nerve cell, soon exhibits degenerative changes.

3. *Miner's Nystagmus.*—The author puts forward a new hypothesis to account for this disturbance. He terms it the equilibration disturbance theory. His conclusions may be summarized as follows: The etiology is complex, the factors of most importance are (1) Conditions tending to do away with yellow spot fixation; *e. g.,* feeble light; (2) Conditions tending to disturb the equilibrium of the body; (3) Conditions in which some more or less rythmical series of movements is performed by the head and body, the eyes remaining fixed; (4) The onset is hastened by debilitating influences; *i. e.,* alcoholism, influenza, accident. JELLIFFE.

News and Notes

In the death of E. Mendel neurology has lost another striking figure. He died on the 23rd of June from chronic nephritis. Mendel was born in Schlesia and took his first university studies at Breslau. He finished in Berlin and Vienna, taking his examination in 1861. He soon built up a large general practice in Pankow, a suburb of Berlin. After the Franco-Prussian war, in which he served, and was wounded, he founded a private sanitarium for nervous and mental diseases at Pankow, from which material he contributed important studies on the Early Diagnosis of Paresis, 1880 and Mania, 1881. In 1882 he founded the *Neurologisches Centralblatt*, and in 1884 was made Extraordinary Professor. His lectures were so well attended that a special auditorium was built for him. He built up a large consultation practice, being particularly aided by the fact of his great teaching ability and because Westphal, who held the chair, was more or less of an invalid. His was an optimistic and good-humored personality. He was genial and stimulating, and his loss will be felt.

Post-Graduate Courses in the Psychiatric Clinic in Munich. From No. 2. 4-24. 1907. as follows:

1. Alzheimer: Normal and pathological anatomy of the brain cortex; 27 lectures.
2. Gudden: Anatomy of the central nervous system; 6 lectures.
3. Kattwinkel: Neurological demonstrations; 9 lectures.
4. Kraepelin: Clinical and forensic demonstrations; 28 lectures.
5. Nitsche: Methods for clinical examination of patients; 5 lectures.
6. Plant: Research in sero-diagnosis; 3 lectures.
7. Rehm: Cyto-diagnosis from examination of the cerebrospinal fluid; 3 lectures.
8. Rüdin: Facts and problems of degeneration; 6 lectures.
9. Specht: Experimental psychology, clinically considered; 8 lectures.
10. Specht: Criminal psychology; 8 lectures.
11. Weiler: Physical methods of clinical examination; 5 lectures.
12. Visits to institutions.

The courses will be given daily from 8 to 12 A. M., and 4 to 6 P. M.

Cards are issued for the entire group at 60 marks. Application before Sept. 1.

The International Congress of Physio-Therapye.—The Committee of the Congress to be held in Rome next October for the consideration of physical remedies in the treatment of diseases have arranged special transit facilities for members of the Congress and their families with the following companies:

Societa veneziana di navigazione a vapone.
La Veloce.
Lloyd Italiano.
Navigazione generale Italiano.

The last named have agreed to a reduction of 30 to 50 per cent. The advantages proffered by the other companies can be learned through any transportation agency. The Committee have also concluded an especially favorable tariff for their visitors at the best hotels in Rome and other Italian cities to which excursions will be made at very reduced rates. The Secretary of the Congress is Prof. Colombo, Via Plinio, Rome.

VOL. 35. OCTOBER, 1907. No. 10.

THE
Journal
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Original Articles

LOSS OF COMPREHENSION OF PROPER NAMES.*

By Frank R. Fry, M.D.,

of St. Louis.

On April 27th, 1906 (one year ago) I examined with Dr. J. W. Dreyfus of Louisiana, Mr. A. B., a gentleman 40 years of age. Seven days previously he had spent the evening rolling ten pins and attending an informal banquet. He had drunk "a good deal." Late at night four of his friends accompanied him to his home, leaving him at the front door. He was found in bed the following morning in a dazed state with most of his clothing unremoved. Dr. Dreyfus was sent for. The patient although confused and stupid was able to respond to questions with some intelligence. He was complaining of pain and soreness on the left side of his head and face. A large area of the scalp above the ear was swollen and tender. There were blood stains on the pillows, which were traced to a hemorrhage from the right ear (the side opposite to the trauma). On the front steps of his residence there was quite a pool of blood which is supposed to have come from the same source. The ear was irrigated and there was no subsequent escape of blood or any other kind of fluid.

The patient cleared up rapidly within the next few hours and it was then evident that he could recall only to a limited extent the events of the evening of his injury. (These were learned subsequently from the companions who escorted him home and

*Read at the thirty-third annual meeting of the American Neurological Association, May 7, 8 and 9, 1907.

from others). What was still more noticeable, and especially distressing to the patient, he could not recall the names of the members of his family and most intimate associates.

I had obtained the above account, in greater detail, before I began my examination, one week after the injury; hence from the start I was alert to discover the character of his speech defect and to use due care in preserving a record of the examination, etc. When I was introduced he repeated my name, greeting me with a few courteous words spoken cordially and freely. I remarked to him that he did not have the appearance of being ill. He replied (almost verbatim): "I guess I am not very sick but I have been through a little experience in the last few days that is not very clear to me. To-day I am feeling stronger and better than any day yet and I feel that my head has cleared up a great deal, but I cannot yet remember all that happened to me the other night, which the doctor has no doubt told you about, and I cannot remember names at all. I cannot remember the names of any of the boys down at the bank (his place of business)." After what I had been told concerning him I was quite surprised to hear him speak at this length with none of the usual aphasic difficulties. Carrying the conversation farther I found that he not only spoke with fluency but very intelligently, and I soon noticed that he was deliberately avoiding the necessity of employing proper names in his conversation. When there was urgent necessity of one he appealed to those about him for it. I also noticed that when a name was supplied to him he accepted it on faith, as it were. It was quite evident that he was not positive about any proper name. At the time I saw him he was just beginning to venture on the given names of three or four persons and the manner in which he used them showed that he was never sure of any of them. I finally made some tests of which the following dialogue is a sample:

"Who was George Washington?" He smiled blankly and remarked: "Anything along that line gets me, Doctor, I cannot tell anything about it."

"Who was the first president of the United States?"

"Of course I should know, but I can only say you understand how to catch me."

"Where is Washington, D. C.?"

"No, I cannot tell."

"What is the capitol of the United States?"

"No, I cannot get anything along that line."

He was then tested in a similar manner with a number of names of familiar persons and places. In each instance when asked to repeat the name he always did so with no difficulty. And what was quite surprising, he wrote names to dictations accurately and without hesitation and did not gain any comprehension of them by so doing. When asked to write his own name he did so, and explained that he had recently re-acquired the ability to do so.

This peculiar disability lasted for about three weeks; at least at the end of that time names were coming back rapidly enough to give him an assurance that he would recover.

The above description hardly conveys the picture that this case presented to the observer, that of an alert, intelligent, fine appearing young gentleman conversing fluently, hardly hesitating or tripping on a word, and yet with as little conception of proper names almost as though he had never known any.

The loss of substantives, and especially the proper names is conspicuous in all aphasic cases but I never before have seen it so completely and strikingly limited to the one class, and I felt that this instance was rare enough to merit recording.

In attempting to at least partially consider the condition present in B.'s case it is necessary to keep in mind the distinction between memory and recollection.[1] B. had not lost the memory of proper names, for when he wrote, by dictation, names like "Chicago" and "Mississippi," promptly and accurately, the memory pictured in some form must still have persisted. The difficulty was, according to our modern theories, that by none of the intercortical connections or associations could the conceptions be recollected. In the case of proper names these associations are necessarily complicated. It is a trite observation that those who readily recall persons' names have an enviable gift. In some, as we know, it is a gift comparable almost to an unusual musical or mathematical faculty. In the majority of individuals, however, the recollection of proper names is a relatively difficult matter, and in some of us is only accomplished by multiple associative processes or ideas as numerous and almost as varied as the multitude of physiognomies which we would undertake to label with their respective baptismal stamps. And especially

as we grow older our efforts to retain a respectable cognominal vocabulary become a source of amusement, as well as chagrin, to ourselves and to others. These well-known facts, however, cannot altogether explain the total loss that was present in this case. And for that matter, the whole literature of aphasia reminds one of the difficulty of explaining some of the unusual pathological defects of the language faculty which now and then appears. These irregular phenomena cannot be taken care of by the usual diagrams and theories. On this point Oppenheim remarks: "In constructing these diagrams originally the question of *individual* variations was not thought of. One person uses his visual memory pictures in speech more than another; another, his auditory, speaking, reading and writing through the sound images; and a third the motor. According as one or the other of these predominates does the effect of the disease vary: the loss of a certain center will cause a hardly noticeable defect in one and a severe and persistent disturbance in another."

The history of this subject, like that of all difficult ones, well illustrates the usual tendency to elaborate hypotheses on insufficient data and misinterpreted phenomena; theories which are not only entertaining but instructive, and yet not to be taken too seriously, for we may at any time be called on to abandon them for something more convincing. In fact we are just now confronted with a proposition of this kind from a most eminent source. I refer to the recent observations of Marie.[2] They are, to say the least, quite revolutionary, yet they must of necessity influence our views of aphasia for some time to come.[2]

The singular loss of one class of conceptions in our case is somewhat comparable to certain authentic cases where patients quite word-blind are still able to recall mathematical characters and to do mathematical calculations; or to other instances of patients who having a command of two languages lose entirely the recollection of one of them and retain the other. If the loss had not been confined entirely to the one class of conceptions we could readily classify the case as one of "intercortical sensory aphasia,"[3] and conceive of a temporary difficulty in conduction so slight as to permit the stronger class of stimuli to carry, but failing for a time to convey the feebler or less insistent ones. Another suggestion is to be thought of; namely, that this was not a case of aphasia at all, but merely a transient psychic or so-called

hysterical condition. There were, however, no other symptoms that would tend to support this view.

On physical examination I found a large area of the scalp above the ear quite tender to pressure and boggy to the touch, evidently the site of a contusion which was rapidly subsiding. There was no abrasion of the skin. He had been having a great deal of pain in this locality, but at the time of my examination he was only feeling occasionally a slight headache on this side of which he complained very little. He also confessed to a slight vertiginous sensation at times. For several days following the injury the vertigo had been pronounced. The supraorbital nerves were not tender (the left possibly slightly so). The pupils were equal and responded to light and accommodation. There were no visual disturbances. There was a suspicion that the right side of the face was a shade smoother than the left, but a paresis of none of the muscles could be demonstrated. The deep and superficial reflexes of the body were everywhere symmetrical. There were no subjective or objective sensory symptoms. The sensibility of the fauces was normal. The pulse was from 62 to 70. There had been no elevation of temperature after the first day or so.

His occupation has been, for a number of years, cashier of a bank. He has had a good education, leaving school when about twenty-one, and has since been actively engaged in business, of which he has made a very good success. In recent years he has been drinking, in a convivial way, too much and too often, but he could not be considered an "alcoholic"; nor has he been one at any time. He is a person of fine appearance, showing no signs of dissipation, and has an active and alert mind.

Within the last few weeks I wrote him asking him to answer a number of questions. He replied (in part) as follows: "I am glad to report that my health at the present time is very good. While I still have some trouble in the memory of proper names, it is limited. My greatest trouble as a result of my accident last spring seems to me to be a shortened vocabulary. Very often in dictation or in conversation I am halted by an inability to bring to mind a word (the most common word) I wish to use,"

He further states, in answer to one of my questions, that before the accident he had had no especial difficulty in remembering proper names. He also states that at no period of his life had he

been required to write proper names to any great extent, but simply to the extent demanded in the usual routine of business.

REFERENCES.

(1) My attention was recently called to some well expressed sentences covering this point by Dr. Charles W. Burr, as follows:

"It is well not to use the words memory and recollection synonymously but to distinguish clearly between them, using the former to mean the storing up of sensations or ideas in the brain but not in consciousness. In the diseased states in which for a time power of recollection is lost, but later recovered, memory is not really at fault, the 'imprints upon the brain' of the original sensations or ideas have been retained, but the ability to bring them into consciousness, power of recollection, has been temporarily suspended." ("A Case of Loss of Memory" by Charles W. Burr, *American Journal of Insanity*, January 19th, 1907).

(2) Marie maintains that Broca's convolution—the third inferior frontal—plays no special part in the function of speech. Careful and very extensive pathological and clinical observation leads him to deny the existence of a special word-hearing center in the first temporal convolution, and to reject the current explanation of word-deafness. He regards the different aphasias as different degrees of the same disease. The essential difference between the motor aphasia of Broca and the sensory aphasia of Wernicke, according to Marie, is that in the first the patient is unable to speak, whilst in the latter he can speak, more or less badly. In other words, Broca's aphasia is simply Wernicke's aphasia minus the power of speech. The essential fact of aphasia, of whatever variety, is insufficient comprehension of speech. When to this is superadded anarthria, due to a lesion in the neighborhood of the lenticular nucleus, we have Broca's aphasia. The intellectual processes of speech are usually localized in the left hemisphere, whilst anarthria may be produced by a lesion of either lenticular nucleus. (*The International Medical Annual*, 1907).

(3) When the association fibers between the memories of sight and the memories of sound are severed a condition of aphasia results which is characterized by an inability to recall the name of a thing seen and to picture to the mind the appearance of a thing named. Yet the name is recognized when heard and the object is recognized when seen. This condition has been described under

different names by different observers. Freund named it optical aphasia or transcortical aphasia, and these terms are used by the Germans. I prefer the term intercortical sensory aphasia as less obscure and misleading. A patient suffering from this type of aphasia has not lost his memory pictures, for he is able to recognize anything once heard or seen. He can, therefore, hear, understand, and read; but if he is asked to call to his mind some place or person whose name is given *e. g.*, Lake George, Lake Como, President McKinley—he cannot do so. The impulse started from the word-hearing center cannot reach and arouse the visual memories; nor can the association be made in the opposite direction, for if he is shown an object or a person—a watch, a chain, or some familiar face—he cannot recall the name, though he recognizes it when heard. (*"Organic Nervous Diseases,"* M. Allen Starr, M.D., etc., 1907).

THE SYMPTOMATOLOGY OF LESIONS OF THE LENTICULAR ZONE WITH SOME DISCUSSION OF THE PATHOLOGY OF APHASIA.

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(*Continued from page* 588.)

THE ROLE PLAYED IN THE PRODUCTION OF APHASIA BY LESIONS OF DIFFERENT PARTS OF THE FIRST TEMPORAL CONVOLUTION.

In the discussion of the effects of lesions of the lenticular zone and the zone of Wernicke* in the production of aphasia, the importance of each portion of these zones must be carefully considered. It is only in this way that we can arrive at reliable conclusions on the mooted questions of the localization of the representation of the different elements of cerebral speech. The first and second temporal convolutions necessarily hold a prominent place in this discussion. It has been usually held that the higher auditory area, the so-called center for word hearing, is situated in the posterior portion of the left first temporal convolution, or of the first and second temporal convolutions, the focus of representation being about opposite the posterior upward turning of the horizontal branch of the Sylvian fissure. As a rule more stress has been laid upon lesions of the cortex than of the subcortex in the causation of word deafness, although of course the pure word deafness of Lichtheim and Dejerine has been attrib-

In the two cases, the histories of which have just been given,

*It might be well to give here Marie's definition of this zone. According to him it consists of the supramarginal and angular gyri and the feet of the first two temporal convolutions. Its limitations cannot be accurately determined, and it is impossible to say whether the symptoms accompanying a lesion of this zone are caused by the alteration of the cortex or the subjacent white matter; Marie inclines toward the importance of the white matter.

utable to injury of subcortical tracts proceeding to the cortical center from the basal centers and the auditory periphery.
word deafness was undoubtedly present and somewhat persistent, although not permanent. In both of these cases the zone of Wernicke, as described by Marie, was not involved in the destructive lesion, this being confined to the white matter of the anterior portion of the first temporal convolution.

It is not necessary to call into service the theory of diaschisis of von Monakow to explain the word deafness in these cases. When word deafness is a symptom, the subcortex of the anterior portion of the first temporal convolution being diseased, while the cortex and subcortex of the posterior halves of the first and second temporal convolutions escape, the aphasic phenomenon finds its explanation in destruction of the paths of transmission to and through the external and extreme capsules to the insula and the center of Broca. One of the writers, Dr. Mills,[20] has reported a case, now well known in the literature of the subject, which shows that word deafness may result from a lesion limited to the posterior portion of the first or of the first and second temporal convolutions, chiefly to their cortex, and reference will next be made to two cases previously recorded by Dr. Spiller.[21]

Case 3. *Fracture of the Skull in the Temporal Region—Contusion and Disintegration of the Inframarginal and Supramarginal Regions—Hemiplegia, Hemianesthesia and Aphasia Nearly Total at First—The Disappearance of Paralysis and Improvement in Motor Aphasia, Marked Word Deafness and Paraphasia Persistent—Operation with Improvement—Relapse—Auditory and Visual Hallucinations—Persecutory Delusions—Necropsy Showing Destructive Lesion Chiefly Involving the Zone of Wernicke (the Supramarginal and the Posterior Portions of the First and Second Temporal Convolutions).*

The patient, a man twenty-nine years old, had suffered from a fracture of the skull which left him at first completely hemiplegic and hemianesthetic on the right side and almost totally aphasic. He recovered largely from the paralysis and to some extent from the speech disturbance, but word deafness and paraphasia were pronounced. He was probably word blind.

An operation revealed a stellate fracture with contusion and

[20]Mills, Chas. K. "Lesions of the Superior Temporal Convolutions Accurately Locating the Auditory Center." University Medical Magazine, Vol. 4, November, 1801.

[21]Spiller, William G. "Lesions of the Left First Temporal Convolution in Relation to Sensory Aphasia." Review of Neurology and Psychiatry, May, 1906.

disintegration of brain substance, probably of the first and second temporal convolutions. Word deafness persisted, and later the patient became insane with auditory and visual hallucinations and delusions of persecution. The patient died in the insane department of the hospital and necropsy showed an area of sclerosis involving the posterior part of the first and second left temporal convolutions, and also part of the parietal lobe. It extended to the posterior part of the insula and to the optic radiations.

The necropsy showed the reverse picture of the lesions in cases 1 and 2 so far as the temporal lobe was concerned, the sub-

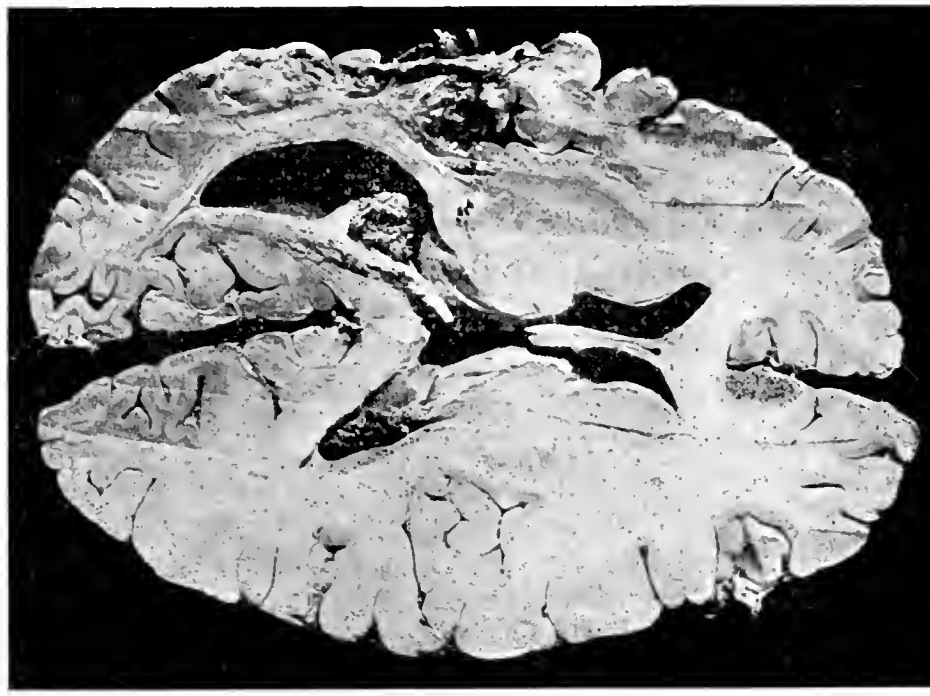

Fig. 4. Complete destruction of the first temporal convolution, with marked sensory aphasia. The lesion extends to the lenticular zone. (Case 3.)

cortex of the anterior halves of the first temporal convolutions being destroyed in these cases. Word deafness was pronounced but not complete. It would seem, therefore, from these three cases that either a destruction of fibers passing from the posterior part of the left first temporal convolution by way of the external capsule, or of these fibers plus a lesion of the cortex of the posterior part of the left first temporal convolution may cause word deafness. In the first two of these cases the word deafness was less persistent. It is to be noted that the sclerosis in case 3 involved the upper part of the middle portion of the left second temporal convolution.

Case 4. *Apoplectic Attack twenty-three years before death causing Hemiparesis and Disorder of Speech—A Second Apo-*

plectic Seizure fifteen years later—Slight Paraphasia and Paragraphia (Occasional Omission or Misuse of Words in Talking or Writing)—Word Deafness not present for several years before his death—No Facts as to Word Hearing at the Time and immediately after his aploplectic attack—No Word Blindness or Letter Blindness—No Mental Impairment except some Defect of Memory—Was Ataxic and had other symptoms of

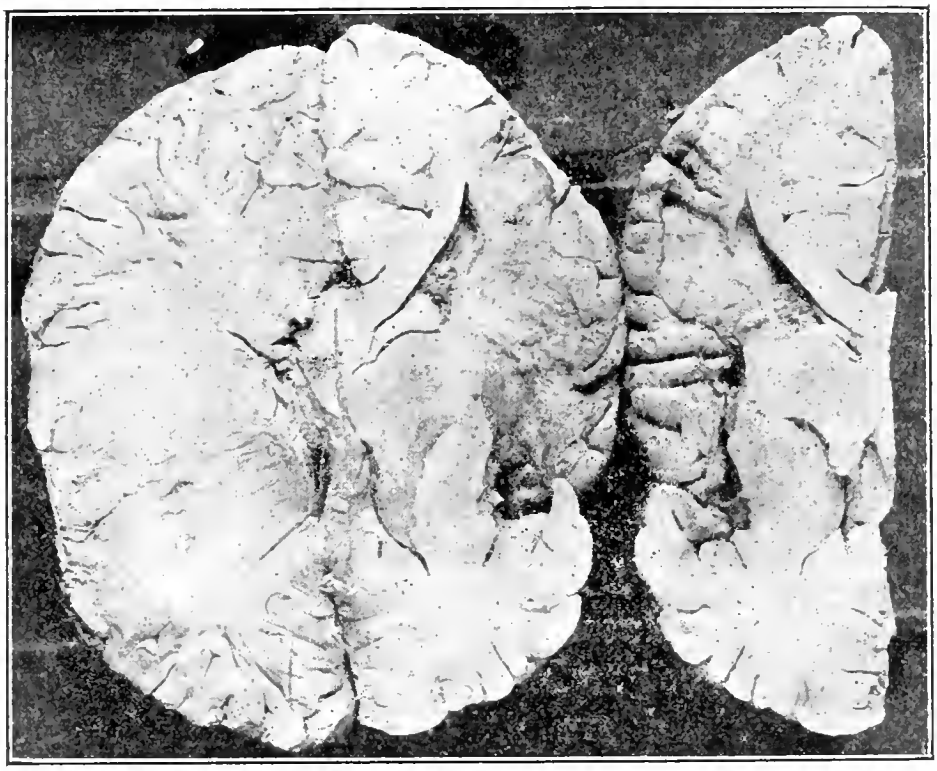

Fig. 5. Complete and long-standing destruction of the left first temporal convolution without marked persistent sensory aphasia. The destruction extends to Marie's lenticular zone. (Case 4.)

Tabes—The Microscopic Findings of Tabes—Destruction of the Entire Left First Temporal Convolution except in the Angular Region—Destruction of the Insula except at its anterior pole—No Involvement of the Lenticula.

A man, about sixty-one years of age at the time of his death, twenty-three years before this time had an apoplectic attack. He was left with some disorders of speech and impairment of power in the right half of the body. About eight years before his death he had a second seizure. He became partly aphasic, could not write without making mistakes, walked with difficulty, and at times had difficulty in swallowing and in urination. He also had spells of hiccough and vomiting. He had cramps or twitchings in the right leg.

The patient, at least in a late period in the history of his case, had no difficulty in understanding words. He was sometimes at a loss for a word, although he could talk with comparative ease. He was liable to misuse words in writing, sometimes misspelling, but these defects were but little marked. He was sound mentally, except that his memory had failed somewhat. He was right-handed. He read constantly, evidently not being word or letter blind. He was ataxic and had other symptoms of tabes.

The left first temporal convolution was destroyed in its entire length except at the angular and supramarginal gyri. The sclerosis extended to the posterior horn of the lateral ventricle. It destroyed the cortex of the island of Reil and the white matter immediately subjacent except in the extreme anterior end of the island. It did not involve the lenticular nucleus. The lesion was very similar to that in the brain of the patient in case 3, except that it extended a little more inwards and considerably further forward, but left the supramarginal and left second temporal convolution intact. It did not cause persistent word deafness, at least the patient was not word deaf at the time his case was studied. About his condition as to word hearing at the time of his attack and in the earlier years after it, no record was made.

In the original report of this case, one of us, Dr. Spiller, has suggested that the only explanations for the preservation of word hearing in this case are that the center for this function was largely in the posterior part of the left second temporal convolution, or that the right first temporal convolution had been unusually well developed during the patient's entire life, and was capable of assuming the function of the destroyed left first temporal convolution. It seems improbable that the small portion of the angular and supramarginal gyri preserved could explain the retention of word hearing.

The preservation of mentality in this case, as in case 2, is of interest in connection with Marie's views as to intellectual deficit caused by lesion of Wernicke's zone. The history of the case emphasizes the fact, to which attention has been directed by one of us[22] in a recent paper, that the degree of intellectual deficit in cases of lesion of this zone is largely conditioned by the intelligence and general capacity of the patient before the attack causing the aphasia.

THE INSULA AS A DISTINCT AND IMPORTANT FUNCTIONAL AREA.

One of the results of the acceptance of the views of Marie is to underrate the importance of the insula as a functional area or as a portion of a functional area concerned with speech. The

[22] Mills, Chas. K. The Journ. of Nerv. and Ment. Dis., Vol. 34, July, 1907.

effort should in the first place be made to distinguish between the functions of the insula and those of the lenticula.

Some of the contributors to our knowledge of aphasia, and especially some of the earlier writers of the period beginning with Broca's discovery in 1861, have gone so far as to regard the insula as the sole cortical motor center for speech, a view which must fix our attention in connection with some of the findings given in this paper, although we are inclined to the opinion that it is only a part, but it may be a large part, of this cortical motor center. The numerous cases of lesion of Broca's convolution with motor aphasia, even if the lesion in many of these cases was not confined to this convolution, cannot be set aside. In one of our own cases (case 5) the lesion causing the motor aphasia was nearly equally distributed between the insula and Broca's convolution, Wernicke's zone, the lenticula and the capsules not being involved.

For the literature of the insula in its relations to speech we must refer our readers to such well-known works as those of Bateman, Wyllie, Dejerine, and others.

Dr. E. A. Spitzka[23] has contributed something of interest in this connection from the standpoint of the anatomist and morphologist. He has directed attention to the redundancy of the preinsula in the brains of highly educated men,—men with great capacity for language both as a means of verbal expression and of reasoning.

The island of Reil is embryologically very different from the striatum. The latter rises from the base of the brain and is separated by a fissure, as Edinger says,[24] from the mantle or pallium, whose wall becomes thickened relatively late. The striatum develops in all vertebrates, but it is only in the higher that the pallium has a development of importance. The pallium later contains the cortex, on which depends all mental processes. In a figure of a frontal section through the brain of a human embryo at the age of two and one-half months, given by Edinger, the striatum rises free from the base of the brain, distinctly separated by a fissure from the external wall of the lateral ventricle. At a later embryological period (about the fourth month)

[23]Spitzka, E. A. Amer. Anthropologist, N. S., Vol. 5, October-December, 1903.

[24]Edinger, L. "Vorlesungen über den Bau der nervösen Centralorgane."

it is divided into the caudate and lenticular nuclei by the fibers of the internal capsule growing through it. The caudate nucleus projects free into the lateral ventricle, as does the lenticular nucleus also; but in later embryonal life the small fissure between the latter and the wall of the cerebral hemisphere becomes so narrowed as to be invisible; but even in adults the wall of the hemisphere can be separated from the outer part of the lenticular nucleus without tearing of fibers. This region of the early fissure is sometimes of importance in the fully developed brain. Hemorrhage occurs here very readily, and the blood, if not too great in quantity, fills the space between the wall of the cerebral hemisphere and the outer division of the lenticular nucleus. Edinger believes that the striatum has some of its fibers in the anterior limb of the internal capsule.

These remarks of Edinger explain why the external capsule is so often the area occupied by a hemorrhage or a cyst. In one of our cases (case 6) a cyst was found in each external capsule which was a *locus minoris resistentiæ*.

In another case studied by us,—not included formally in the cases given in this paper,—in which the lenticula was in an early stage of red softening, no separation of the lenticula from the island of Reil was seen when the brain was first opened, but during the process of hardening the putamen contracted from the white matter of the island, leaving a fissure in the external capsule.

The blood supply of the lenticula and of the insula and its subcortex seems to be distinct. We have preparations from a case (case 7) in which occlusion of the middle cerebral artery occurred after the supply to the basal ganglia had been given off. The point of thrombus formation is clearly demonstrated. The softening implicates the island of Reil and subjacent white matter, but leaves the lenticula intact. The patient could not speak during three weeks, but seemed to understand.

From these facts we may conclude that the function of the island and its white matter is distinct from that of the lenticula, and cases in which both structures are implicated may be wrongly interpreted.

Case 5. *Apoplectic Seizure causing Aphasia without Paralysis of Face or Limbs—Aphasia of Motor Type—A Few Recurring Utterances—Marked Paralexia—Some Retention of*

Writing—No Impairment of Articulation or Vocalization—An Old Cyst Involving only the Cortex and Subcortex of the Caudal Portion of the Left Third Frontal Convolution and the Insula.

Although the following case has been used by one of us, Dr. Mills,[25] in a previous article, and has also been referred to by Gordinier,[26] it has so much value on the negative side of the discussion of the functions of the lenticula, as this ganglion was not involved in the lesion, and also so much on the positive side of the consideration of the functions of the insula and Broca's convolution, both of which were destructively implicated, that we have thought it important to reproduce it in connection with our present series of cases. There is scarcely to be found in the literature of aphasia a more instructive case. Neither Wernicke's zone, the lenticula, nor the internal capsule was involved, and yet the patient showed motor aphasia of typical form with the retention of some power of writing and with the retention (or reacquirement, as the case was not seen at an early period after the apoplectic attack) of the power of reading silently.

The third frontal gyre and the insula were neatly involved in the same circumscribed lesion, the second frontal convolution, the internal capsule, the basal ganglia, and all other portions of the brain not being the seat of any old lesion.

The following citation and summary are taken from the paper in the *American Journal of the Medical Sciences*:

"This patient, after an apoplectic seizure, developed aphasia, apparently without preceding paralysis or any other symptoms of focal lesion like visual blindness and hemianopsia. When examined about nine years after the onset of the aphasia, he had almost complete inability to name persons and objects which he was able to recognize through all his special senses. He had also a marked form of paralexia. When he attempted to read, although he understood what he was reading, he repeated an absurd formula of a few phrases. He had limited spontaneous speech, even using short sentences without concrete nouns correctly. He could write many single words correctly, sometimes misspelling, however. He held his pen or pencil correctly and wrote with ease and firmness."

Many examinations of this patient were made. The case was clearly one of aphasia with marked verbal amnesia and general disorganization of language on its dynamic side. Grammatical expressions and combinations were lost. The patient could speak only in single words, or in short phrases, or very short sentences. What he said, however, he articulated and enunciated clearly.

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[25]Mills, Chas. K. Amer. Journ. of Medical Sciences, September, 1904.
[26]Gordinier, C. H. Idem, 1903.

He had no paresis of the organs of articulation, enunciation or vocalization.

The only lesion to be seen in the lateral aspect of the left hemicerebrum was confined to the hinder part of the third frontal convolution. The lesion was probably part of an old hemorrhagic cyst. When the brain was sectioned at the level of the insula the insula was found to be largely destroyed by a lesion which was continuous with that in the caudal part of the left subfrontal gyre. Other parts were not involved, as shown by

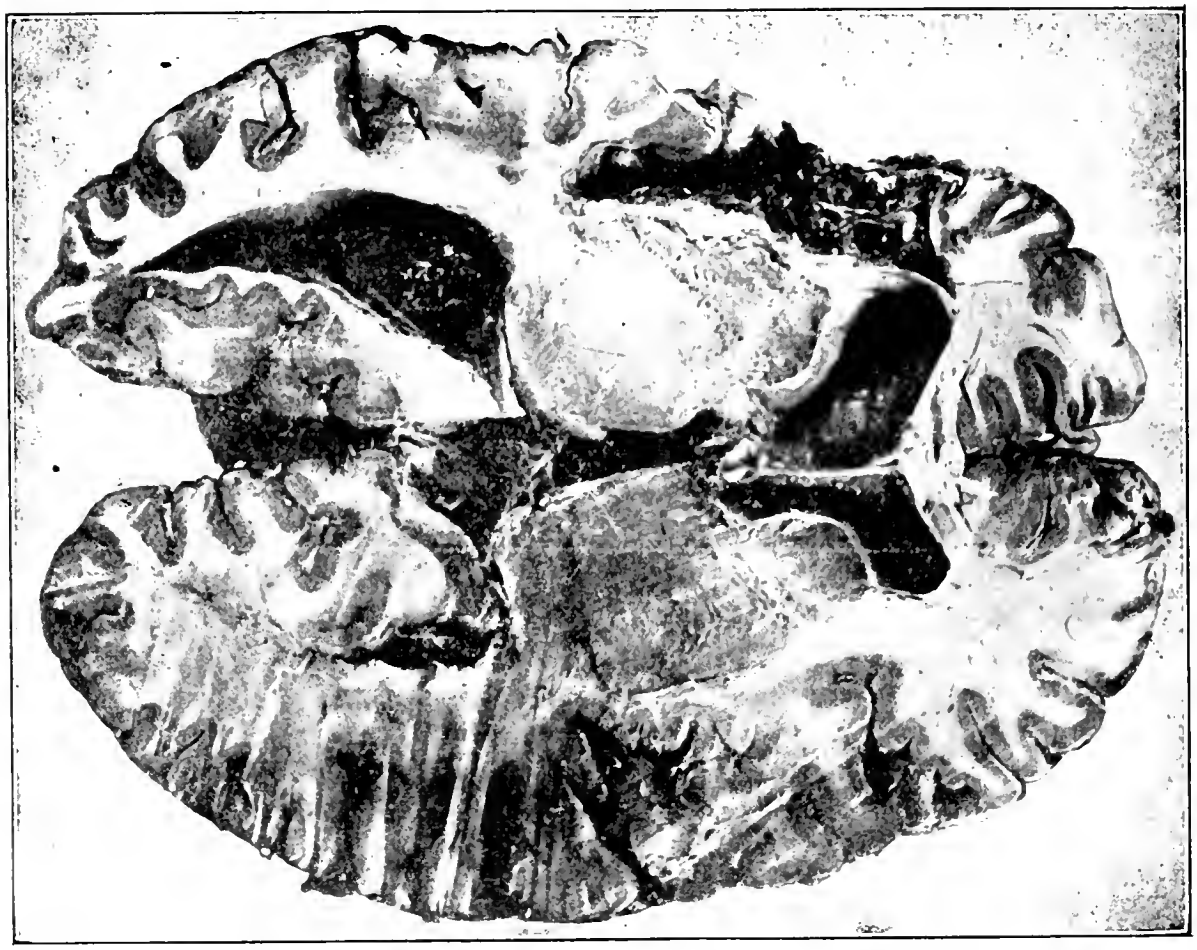

Fig. 6. Case of motor aphasia without hemiplegia. The primary lesion involves the left third frontal convolution and the insula. (Case 5.)

this section and also a subsequent one made a little lower than the first.

It is worth while to refer in this connection to one of the most recent and valuable contributions on the pathology of aphasia —a paper which forms a part of the general discussion which has arisen since the promulgation of the views of Marie. In this paper, in addition to many important considerations regarding

focal lesions and the pathology of aphasia, Dejerine[27] gives the details of two previously unrecorded cases studied both macroscopically and in microscopical serial sections. In these cases the primitive lesions causing the aphasia were confined to the cortex and subcortex of Broca's convolution and to the second frontal convolution with a slight involvement of the insula and extreme capsule in one case. The zone of Wernicke, the lenticula and the capsules, except as mentioned concerning the extreme capsule, were not implicated if we exclude processes of secondary degeneration.*

The first of the two cases reported by Dejerine in this paper was one of motor aphasia, at first very marked, the patient retaining only "yes" and "no" and interjections. Any evidences of sensorial aphasia were very temporary. No paralysis, orolingual, facial or of the limbs, was present. The patient was motor aphasic without being dysarthric. Writing could not be tested, as the patient had lost his right arm by amputation and had not learned to write with his left hand. As time progressed, and before his death two years after the apoplectic attack, he had regained largely the power of speech.

The necropsy showed a lesion similar in some respects in its limitations to case 5 of our series, the insula, however, not being nearly so much involved. In Dejerine's first case the lesion destructively involved about two-thirds of Broca's convolution and to a very moderate degree the anterior portion of the insula and the related segment of the extreme capsule. The caudate and lenticular nuclei were not attacked by the primary lesion, as were not the Rolandic operculum and the external and internal capsules. Serial microscopic sections showed some involvement of the second frontal and of the corona radiata, with a considerable portion of the frontal lobe anterior to the precentral convolution. Degeneration was distinctly marked in the knee of the callosum. The temporal lobe was nowhere diseased. Softening was observed in the first and second frontal convolutions of the right hemisphere. The degeneration involved the anterior segment of the internal capsule, the fibers of passage through the striatum, and the occipito-frontal bundle. Degeneration of the anterior

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[27]Dejerine. J. L'Aphasie Motrice et sa Localisation Corticale. L'Encéphale, No. 5, May, 1907.

*We understand by Broca's area, which has been described differently by writers on aphasia, that corticosubcortical area which surrounds the ascending branch of the Sylvian fissure, including its bifurcations. This makes Broca's convolution include what Dejerine calls the orbital portion, the cap and the foot of the convolution. The foot of this convolution is the part situated between the precentral convolution and the horizontal and ascending branch of the Sylvian fissure.

fibers of the extreme and external capsules was also present.

In the second case of Dejerine, the patient, a woman seventy years old, had a left hemiplegia and some years later an attack causing motor aphasia. This motor aphasia as described was typical and persisted. The patient retained only a few expressions, such as "yes," "no," and "good day." She was not word deaf, word blind or letter blind, or if word blind at all only to a limited extent. She understood all that was said to her, even when she was spoken to rapidly. She could read what was placed before her, but had difficulty if the sentences were long or complex. She could not write, except her name, although she held the pen correctly. She could, however, copy and transfer print into script.

We shall omit the lesions found in the right hemisphere in this case, except to say that they were such as to account for her left hemiplegia and perhaps some other phenomena. In the left hemisphere a yellow plaque was found, so situated as to destructively involve the cap of Broca's convolution, and also in part the second frontal convolution. Serial microscopical sections showed that this primitive lesion did not involve the insula, the external or internal capsules or the basal ganglia. The white matter of the third and second frontal convolutions was considerably diseased. Degenerations were present much as in the first case, involving, for example, the anterior segment of the internal capsule, the radiate fibers to the thalamus and the knee of the callosum. The zone of Wernicke and all other parts of the left hemisphere were intact.

This case clearly shows that Broca's aphasia (motor aphasia of persistent form) may be due to a lesion involving the third and second frontal convolutions and the subjacent white matter in the primitive lesion, all other parts escaping.

The first of the two cases is of similar import, although the insula and extreme capsule were to a slight extent included in the lesion, and the patient recovered largely from his motor aphasia. This recovery may have been due to the restoration of the parts of Broca's convolution and the insula not included in the lesion, or as Dejerine suggests, the fact that the patient had lost his right upper extremity some twelve years before his death may have had something to do with the unusual facility with which the convolutional areas of the right hemisphere, homologous to those of Broca in the left, assumed the representation of motor speech.

Case 6. *Right Hemiplegia with Exaggerated Reflexes on the Paralyzed Side—Attacks of Vertigo—Linear Cyst in the External Capsule of Each Hemisphere—Area of Sclerosis in the Middle of the Foot of the Left Cerebral Peduncle—Right Pyramidal Tract in Cervical Region Degenerated.*

The notes of the following case were meager.

E. D., aged fifty-seven years, white, German, was admitted to the Nervous Wards of the Philadelphia General Hospital November 14, 1902. He had been a strong man and had worked as a sailor.

When admitted to the hospital he stated that he was paralyzed on the right side six years ago. He said that he had had attacks of vertigo every day for the last six months, in some of which he fell to the floor. The eyes reacted normally to light and in accommodation. On the right (paralyzed) side knee-jerk was excessive, slight on the well side. No ankle clonus could be obtained, but a slight Babinski response was present on each side.

The next notes in this case were not made until September

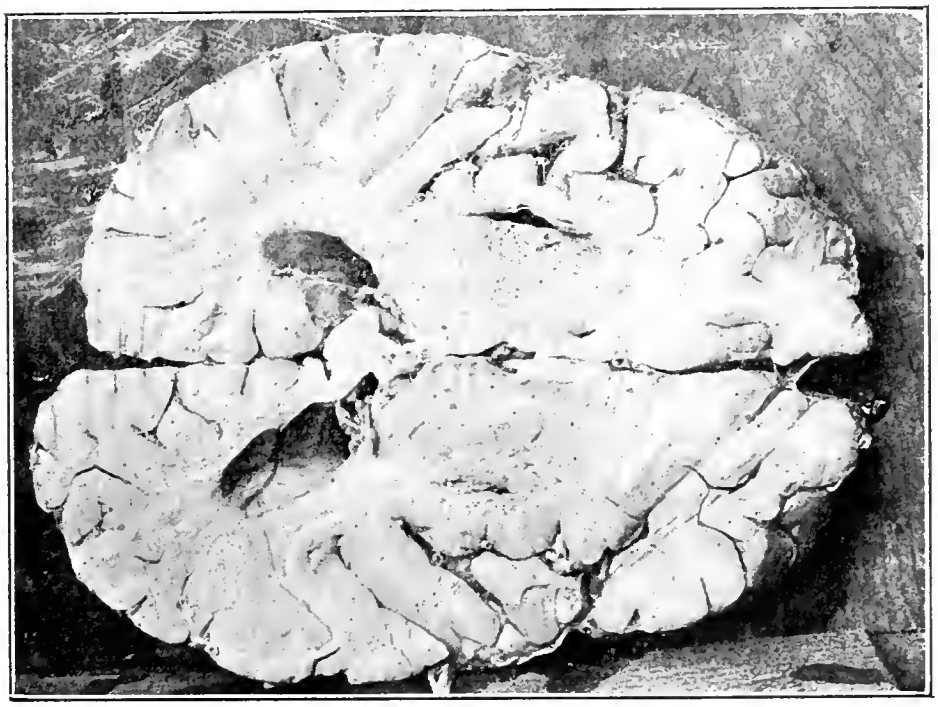

Fig. 7. A symmetrical cyst is present in each external capsule in the region of least resistance. (Case 6.)

11, 1903, when the records state that the patient was in a stuporous condition, unable to answer any questions and he did not seem to understand anything that was said to him. He had great difficulty in swallowing.

On September 15, 1903, the notes stated: He became weaker this evening and was no longer able to be out of bed. Incontinence of urine and feces was present. He died September 20th. The fresh spinal cord seemed to show sclerosis of both right and left pyramidal tracts. The brain showed some areas of softening of the right caudatum.

A horizontal section through the cerebral hemispheres nearly

at the upper border of the lateral ventricles showed a small hemorrhagic scar in the right hemisphere probably cutting some of the fibeers that form the knee of the right internal capsule. This may have developed shortly before death. A linear cyst was found in the external capsule of each hemisphere in a section made through the upper part of the thalamus and lenticula. The cyst on the right side to the naked eye measured 2.5 cm. in length and 0.5 cm. in its widest portion (1 in. in length by 1-5 in. in width). It did not extend into the cortex of the island of Reil and encroached very slightly on the lenticula. Its anterior end was 0.5 cm. (1-5 in.) behind the anterior end of the lenticula. Its posterior end reached very nearly to the posterior end of the lenticula. The cyst on the left side was smaller than that on the right side. It was in exactly a corresponding position except that its anterior end was 1.5 cm. (3-5 in.) behind the anterior end of the lenticula. Its posterior end extended to the posterior end of the lenticula. It also encroached very little on the lenticula. Neither cyst extended into the capsule, and neither extended downwards as far as the level in which the cerebral peduncles appeared distinctly formed, nor did they change their relative positions. No cause was detected for the right hemiplegia.

Sections of the cerebral peduncles and of the cervical region of the spinal cord made by Dr. T. H. Weisenburg showed a small area of sclerosis in the middle of the foot of the left cerebral peduncle. The right crossed pyramidal tract of the cervical region of the cord was degenerated. The lesion producing this degeneration has not been found, unless it was the cyst in the external capsule, which was doubtful.

The history in this case is very meager, but we have retained it in this series because the notes record that the patient stated that he had been paralyzed in the right side six years previously, and made other assertions. He therefore probably was not motor aphasic, though we could not assert that his speech was normal. An old lesion, a cyst, was found confined to each external capsule and claustrum, and yet persistent or complete aphasia seems to have been wanting. The right hemiplegia might be attributed to this cyst in the left external capsule, but inasmuch as a small area of degeneration was found in the middle of the foot of the left cerebral peduncle, it seems possible that some other lesion in the left cerebral hemisphere has been overlooked. The lenticula on each side, to the naked eye, seems to have escaped.

This case, after all, is of chief importance as illustrating the points made when discussing the views of Edinger and others as to the embryonal, and even later, separations of the insula and its subcortex from the basal ganglia, the inference being that they are functionally, anatomically and morphologically distinct.

Case 7. *Right Hemiplegia with Aphasia—Deep Reflexes but*

little changed—Some Hypesthesia—Stereognosis not studied —Aphasia of Motor Type—No Word Deafness—Thrombus of Left Middle Cerebral Artery just above the place where the Vessels to the Basal Ganglia are given off—Softening invoking the Cortex and Subcortex of the Insula, the Foot of the Precentral and Postcentral Convolutions, the Entire Parietal Lobe, the Fibers to the Lower Part of the Callosum, the Optic Radiations to a small extent—The Lenticula and the Temporal Lobe were not implicated in the Lesion.

A. H., sixty years old, colored, was admitted to the Philadelphia General Hospital December 15, 1904, and died December 29, 1904. The only history that was obtainable was that she had not spoken for five days and since that time had been paralyzed in the right half of the body.

On examination the patient was found lying in bed with her eyes closed, making no attempt to move or talk. The face was drawn to the left over one-half of its extent. There was marked over-action of the occipito-frontalis of the left, while on the right movement of this muscle was very slight.

The eyes were directed anteriorly without deviation; the right pupil was contracted, and apparently immobile. She could not be made to accommodate; the left pupil was fixed; the media of both eyes were very clouded, especially on the left, which seemed to show a cataract. Arcus senilis was marked. She could not be made to move her eyes in any direction when told to do so, although she evidently understood the command, as she made an effort to obey by a slight turning of the head.

The tongue could not be protruded. She opened her mouth when asked to put out her tongue, which was pushed to the left and was slightly tremulous and somewhat coated.

The right arm was apparently completely paralyzed and fell helplessly when raised. The response was very feeble on tapping the triceps and biceps tendons. The right leg, which was paralyzed, gave a slight knee-jerk but no ankle clonus, although there was a suggestion of dorsal flexion of the great toe on plantar stimulation.

She slightly moved her paralyzed foot and leg occasionally when stuck by a pin. Attempts to elicit ankle clonus occasionally resulted in a flexion of the thigh on the pelvis. The left leg gave a very slight knee-jerk. No ankle clonus and no Babinski response were determined. This limb was moved somewhat more when stuck by a pin than was its fellow. There was no positive result on either side to stimulation by the Oppenheim method.

Under date of December 18, 1904, the clinical notes stated that there was no change in the patient's condition. She understood what was said to her, but could not answer. Her bladder and bowels were incontinent. Her pulse was very irregular and she

slept most of the time. She was mentally dull. Temperature at this date was still down, but respiration was up to 50; pulse was at 150. The right chest gave bronchial breathing and great impairment about its middle in the midscapular line. Both sides were full of large bubbling and smaller sized rales. She could be aroused by vigorous questioning, but could not answer, although she tried. She was very weak. She died December 29, 1904.

At the necropsy the following pathological conditions were found in the various organs:

Congestion of the lungs; chronic interstitial myocarditis; chronic congestion and interstitial splenitis; chronic congestion and interstitial nephritis with infarction and hemorrhagic and healed infarct of the kidney; chronic congestion and fatty fibrosis of the liver and general arteriosclerosis. The brain and spinal cord were removed for hardening and further investigation.

Later this investigation showed a thrombus occluding the left middle cerebral artery just above where the arteries to the basal ganglia are given off, so that the latter arterial supply was intact. A large area of softening was found which implicated the cortex and white matter of the island of Reil, leaving a narrow margin of white matter intact on the lateral aspect of the lenticula; the latter ganglion was not affected. The foot of the precentral and postcentral convolutions were softened, but the extreme anterior part of the island of Reil escaped. The softening extended very close to and probably slightly involved the optic radiations.

At a higher level and just above the lenticula the softening extended inwards so as to involve slightly the fibers going to form the lower part of the callosum.

The precentral convolution was softened only in its lower part, but the postcentral was more involved together with almost the entire parietal lobe. The temporal lobe was intact.

This case therefore showed very clearly that the arterial supply of the island of Reil is distinct from that of the lenticular nucleus, and that the lenticule may escape although aphasia is pronounced.

Cast 8. *Apoplectic Attack causing Right Hemiplegia and Loss of Speech—Tongue protruded to the right—Right Hemianesthesia—Memory poor—Partial Recovery of Speech—Aphasia Motor in Type—Reflexes exaggerated on the paralyzed side—Linear Hemorrhagic Scar in the Left External Capsule—Lesion Involving also the Insula and Anterior Limb of Internal Capsule, the Lenticula and Posterior Limb of the Internal Capsule.*

E. J., fifty years old, colored, born in Virginia, an expressman, was admitted to the nervous ward of the Philadelphia General Hospital in the service of Dr. Charles S. Potts, on June 12, 1903.

No family history or previous history of importance was obtainable. The patient denied having had syphilis.

Several weeks before admission he fell unconscious in his wagon and remained unconscious for three days. It was then found that the entire right half of his body was paralyzed. At first he was unable to make his wants known by talking, but speech gradually improved. His wife stated that for several weeks before the paralytic attack he had complained of headache.

On June 14th, two days after admission, it was noted that he could not move his right arm and leg, that his speech was slow and his memory apparently poor. In general appearance the patient was lean and weak. The pupils were equal; the light reaction was normal, the accommodation reaction poor. The pulse was intermittent, the chest normal, the abdomen tympanitic.

Under date of June 17th notes were made that he was anesthetic on the entire right side, the anesthesia extending about one inch beyond the median line on the left side. The conjunctiva on the right side was anesthetic. The patient was so stupid that it was difficult to get from him satisfactory answers. He could not see a quarter of a dollar when it was brought toward him from the right, but he recognized it at once when brought from the left, or when it was held in front of either eye. Dr. Howard F. Hansell, one of the ophthalmologists of the hospital, however, reported no hemianopsia. On the right side he could recognize cold, but invariably called *hot* "cold." He could not distinguish any difference between two test tubes containing hot and cold water when they were placed on the right side, but could at once when they were placed on the left side. He complained of much pain when the right arm or leg was moved, but these limbs could be pressed or touched with impunity and he did not complain of pain in these parts while they were at rest.

He had use of all parts of the left arm, although power in it was somewhat impaired. He could also move the left leg properly, but with some diminution of power.

Further examination showed absence of sensation in the right side to above the costal margin, except on stroking with a sharp point. Sensation was also absent in the right arm and upper part of the body, except on very deep sharp pressure. Sensation was present on the left side. The knee-jerk was increased on the right side and still more increased and spastic on the left. The biceps jerk was increased on each side. The tongue, which was tremulous, was protruded slowly and to the right.

On June 22d examination showed that hemianopsia was absent. The Babinski reflex was present on the right; the plantar reflex was present on the left. No ankle clonus was present on either side. The tongue protruded decidedly to the right, with tremor. The naso-labial fold was lessened on the right; the

mouth was very slightly drawn to the left. The pupils were equal, contracted to light and very slightly to accommodation.

Arcus senilis was present. The arteries were thickened and tortuous, the right radial being somewhat beaded. The pulse was intermittent, its tension moderate. The heart was irregular in action, the second sound being accentuated, the first sound clear but not proportionately loud. Considerable atrophy and contracture were present in the right hand; the patient complained of pain in moving his right leg. He had frequent micturition and had control of bladder and rectum.

He apparently understood all that was said to him, but was not always able to find words to answer. When the proper answer was repeated to him, he recognized it. There was therefore apparently some motor aphasia.

On July 15th he called the hot test tube "cold" on both sides, and still had complete absence of power in the right arm and leg.

On July 25th it was noted that he had made some improvement during the preceding five days, but on this afternoon he became very suddenly prostrate, dyspnea became marked, and the pulse weak and rapid. He died during this day at a time not mentioned in the clinical record.

Necropsy showed the dura and pia normal. The upper aspect of the right cerebellar lobe was edematous, grayish yellow in color with distinct loss of substance. Inspection of the ventricles was negative. The vessels at the base of the brain were sclerotic. The specimen was removed for hardening and further examination.

Other pathological conditions present were: Fatty degeneration of the liver and myocardium, thrombosis of the pulmonary arteries, chronic pleuritis, edema and congestion of the lungs, chronic nephritis, slight prostatic enlargement, fatty degeneration of the kidneys, especially of the right, deformity of the right foot, and ulcer on the right ankle.

A horizontal section made through the cerebral hemisphere at the level of the upper part of the basal ganglia showed a linear hemorrhagic scar in the left hemisphere in the external capsule. It extended forward as far as the anterior limit of the island of Reil but not beyond, its forks sending off a branch cutting the anterior limb of the internal capsule. It implicated the greater part of the left lenticula, and extended very near to the posterior limb of the internal capsule, although apparently it did not implicate this limb at this level. The cortex of the island of Reil was not involved. The lesion extended downward in the same relative position about 1 cm. (2-5 in.) from this level. The upper part of the scar, in a section from a higher level, involved the posterior limb of the internal capsule, and as the island of

Reil is very short at this level the scar extended forward here beyond the anterior end of the island.

It will be noted that the lesions in this case involved both the external capsule, the anterior limb of the interior capsule, the lenticula, and to some extent the posterior limb of the internal capsule; also slightly the island of Reil. It is therefore difficult to draw any positive inferences with regard to the lenticula; the motor aphasia may have been due to lesion of the external capsule and the island, or to the lenticular lesion.

This man, several weeks after the beginning of his right hemiplegia, had slow speech; he apparently understood all that was said to him, but was not always able to find words to answer.

Fig. 8. A hemorrhagic cyst is present in the left lenticular nucleus. Some motor aphasia persisted. (Case 8.)

When the proper answer was repeated to him, he recognized it. There was apparently some motor aphasia, but it was by no means complete, and yet a large part of the putamen was destroyed.

The occurrence of the hemorrhage into the external capsule of one side is interesting in connection with what has been said of lesions thus located and of the anatomical and functional separation of the basal ganglia from the insula.

Case 9. *Left Facial Paresis—Deviation of Tongue to the Right—Spasticity of Right Upper Limb and of the Lower Limbs—Babinski Response on Both Sides—Spasticity Present and Deep Reflexes Exaggerated on the Left—Incontinence of Urine and Feces—Involuntary Laughing and Weeping—Mentality Dull—Speech Indistinct but no true Motor Aphasia Present—A Small Cyst in the Left Putamen extending into the White Matter of the Insula.*

R. S., sixty-two years old, white, German, was admitted to

the nervous wards of the Philadelphia General Hospital October 24, 1905.

Examination showed the left naso-labial fold to be less marked than the right. The tongue protruded to the right. The brow was wrinkled equally well on the two sides. The pupils were unequal, the left being larger than the right. The ocular movements seemed to be preserved, but the patient's mental condition rendered the examination of these movements a somewhat uncertain matter. On attempting to close the eyes, the left was not so well closed as the right, the edges of the left being separated by quite a little distance as compared with the right. The pupils reacted to light and in accommodation.

The patient seemed to have considerable strength in both upper extremities, all movements being preserved. A suggestion of spasticity was present on the right side. The reflexes of the right upper limb were increased; the left were not increased. The patient had lost control over the bladder and bowels. Movements of the legs were restricted and appeared to be spastic, but it was here again, as with the ocular movements, difficult to say how much was paralysis and how much failure to understand. Both knee-jerks were plus and the Babinski response was obtained on each side. The ankle jerk was also much increased on each side. The biceps jerks were present on both sides.

Under date of December 18th additional notes were made as follows: The right knee-jerk was plus; the left plus, plus; the ankle jerk was plus on the right side and also on the left; Gowers' front tap was present on the left side and absent on the right; ankle clonus was absent on both the right and the left sides.

Stimulating the sole of the foot gave very uncertain results. On the right side at times extension of the toes appeared to be present, and at other times, flexion. On the left side the tendency seemed to be towards the extension or Babinski response.

Power in both upper limbs was much diminished. Movements were much better performed with the right upper extremity than with the left. Fine movements were very poorly and clumsily performed. Movements were very ataxic, especially those of the right side. Marked tremor was present when the fingers of each hand were extended. The limbs on the right side were spastic, and contractures were present. The patient could move the right leg, but not nearly so well as the left. Sensation to touch, pain and temperature seemed to be undisturbed.

On December 27th further notes were made. The patient's condition was not so good as it had been. The nurse stated that the patient had occasional spells of laughing and crying; her mentality was not clear; speech was not distinct, and it was only with difficulty that she could be understood. She had no trouble with chewing or swallowing; she had no dribbling of

saliva. The pupils were unequal, both being larger than normal, the left larger than the right. Extraocular movements were normal. It was impossible to tell the condition of the light reflex and accommodation and convergence. Apparently the seventh nerve on each side was not involved. Apparently ptosis of the right upper lid was present. The other conditions were as noted in the preceding observations.

The bedside notes contain a statement without date made by Dr. Spiller that the patient's speech was very indistinct, but that she was not aphasic.

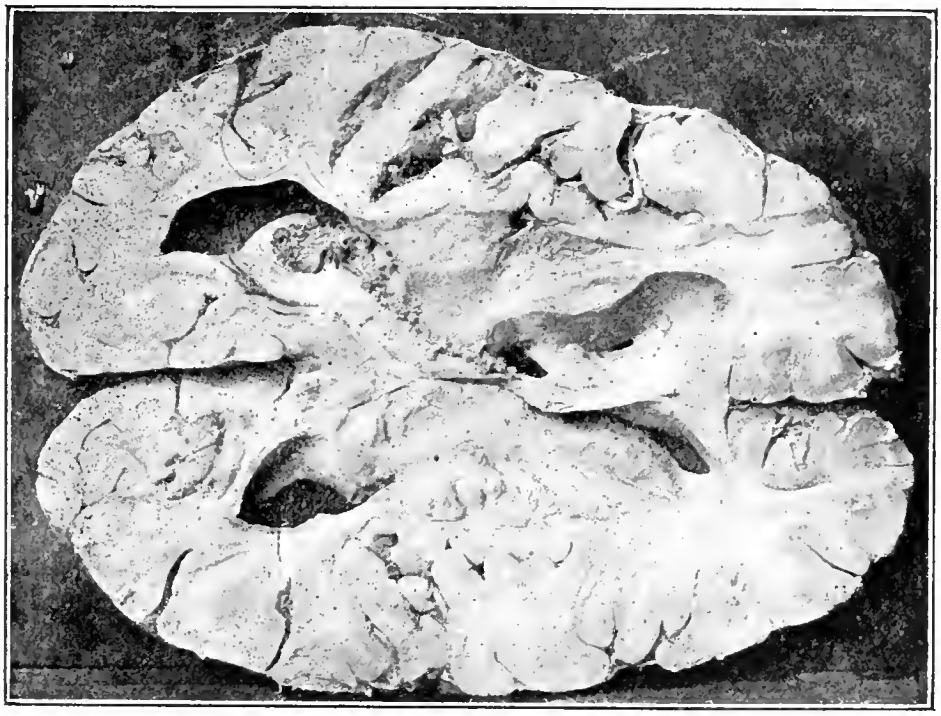

Fig. 9. A small cyst is present in the left external capsule, extending into the lenticular nucleus. The speech was indistinct, but not aphasic. (Case 9.)

On February 4, 1906, while eating her dinner, she suddenly turned to the attendant who was feeding her and said that she had enough. Immediately after this she gasped for breath, became cyanosed and fell over dead.

At the necropsy the pathological findings showed the presence of hypertrophy of the heart; chronic passive congestion of the spleen; chronic interstitial nephritis; passive congestion and fatty infiltration of the liver and general arteriosclerosis. The skull appeared to be thicker than normal. The brain and spinal cord were retained for subsequent investigation.

The gross lesion found was a small cyst 0.5 x 0.5 cm. (1-5x1-5 in.) in the middle of the left putamen in a horizontal section through the upper part of the basal ganglia. This cyst

did not extend into the cortex of the island of Reil, but implicated the white matter in the middle of the island.

It would seem in this case that the anarthric or dysarthric affection of speech and the impairment of power and spasticity and exaggerated reflexes on the right side might be fairly attributable to the lenticular and subcortical lesion. The speech disorder in particular is similar to that which seems to have been observed in other cases of limited lesion of the putamen—not a true motor aphasia, but a speech disturbance more nearly allied to that which is seen in pseudo-bulbar paralysis from capsular lesions. The woman was sixty-two, and some of the symptoms may have been caused by senile changes in the brain, but this finding is important, viz., a small cyst interrupting the fibers in the left external and extreme capsules, and extending into the putamen in about its middle portion.

Case 10. *Apoplectiform Attack without Unconsciousness—Speech Lost for Two Hours, then Recovered—Hemiparesis of Right Lower Face—Deviation of Tongue to Right—Paralysis of Right Upper and Lower Extremities—Reflexes increased on right—Sensation not impaired—Destructive Lesion involving the Left Lenticula and both the Anterior and Posterior Limbs of the Internal Capsule—Absence of Persistent Motor Aphasia.*

A. W., white, thirty-six years old, was admitted to the nervous wards of the Philadelphia General Hospital December 27, 1898. The family and previous history of the patient contained no fact of importance, except that her mother died of dropsy at the age of forty-two. The patient used alcohol moderately.

Five weeks before admission the patient had an attack of influenza from which she recovered. Three weeks before admission she was taken with weakness while on the street and felt three times. While unable in these spells to stand or walk, she remained perfectly conscious. Her speech was affected for two hours, during which time she was unable to talk. On attempting to move the right arm or leg she was unable to do so.

Examination showed a fairly well nourished adult. The pupils were equal; the irides reacted to light, and in accommodation and convergence. The extraocular muscles and the eyelids were not paralyzed. The muscles of the right side of the face appeared to be partially paralyzed, this being especially noticeable in the lower part of the face. The tongue deviated to the right on protrusion.

The right arm and leg were completely paralyzed, but there was no limitation of movement in the left extremities. The biceps and triceps jerks were increased on the right side and absent on the left; both knee-jerks were increased. Slight patellar clonus was present on the right side; ankle clonus was absent. The

plantar reflexes were increased on the right side. Sensation was nowhere impaired.

The pulse was frequent, small, and fairly strong. The heart sounds were normal; the second sound was accentuated.

Examination of the lungs showed nothing on palpation and percussion, but on auscultation at the right apex was heard a slight prolongation of expiration, especially noticeable at the middle lobe posteriorly.

An occasional note regarding this patient was made between her admission and November 17, 1903, simply showing that the patient was a right hemiplegic. On November 17, 1903, examination showed that the woman was weak and her breathing shallow, but that she was conscious. Both the right upper and lower extremities were completely paralyzed. Knee-jerks were present on both sides, prompt on the right; the Babinski response could be elicited on the right, but was absent on the left; neither ankle clonus nor patellar clonus was present. The tongue protruded slightly to the right. No old contractures were present on either side. Sensation was not impaired.

Notes were made at this date that the patient had not slept well. She was constantly muttering and picking at the bedclothes. She could, however, be aroused and made to answer questions intelligently. Respiration was increased, but not labored. The tongue was heavily coated. Examination of the urine was negative. Resonance was impaired over portions of the middle and lower lobes of the lungs on the right side; breathing was distinctly tubular over an area of the middle lobe. On November 18th the patient developed edema of the lungs; the heart became very rapid and weak. She died November 19, 1903.

The following conditions were found post mortem: Croupous pneumonia with red hepatization and right-sided slight hydrothorax; slight chronic diffuse nephritis and acute parenchymatous nephritis; also chronic passive congestion of liver; slight fatty degeneration of liver; slight chronic thickening of the pia arachnoid; atheroma of thoracic and abdominal aorta.

A horizontal section was made through the left cerebral hemisphere, just at the upper border of the head of the caudatum and thalamus, so as not to remove any of these structures. At this level a cyst was found occupying the entire area of the lenticula and encroaching upon the anterior and posterior limbs of the internal capsule and the white matter of the island of Reil. The cortex of the latter region was intact. The head of the caudatum and the thalamus, so far as could be determined by the naked eye, were not implicated. The cyst did not extend forward or posteriorly beyond the limits of the lenticula. It measured 3.5 cm. in length and 1.5 cm. in width (1 2-5x3-5 in.).

A horizontal section made through the cerebral hemisphere

1 cm. (2-5 in.) below the previous section showed the lower termination of this cyst. It was directly beneath the center of the cyst as shown in the previous section and measured 1.5 cm. long by 1 cm. wide (3-5x2-5 in.). Sections of the spinal cord made by Dr. Weisenburg showed intense degeneration of the right crossed and left direct pyramidal tracts.

The lesion present in this case, although in large part in the lenticula, was not confined to it, extending both into the anterior

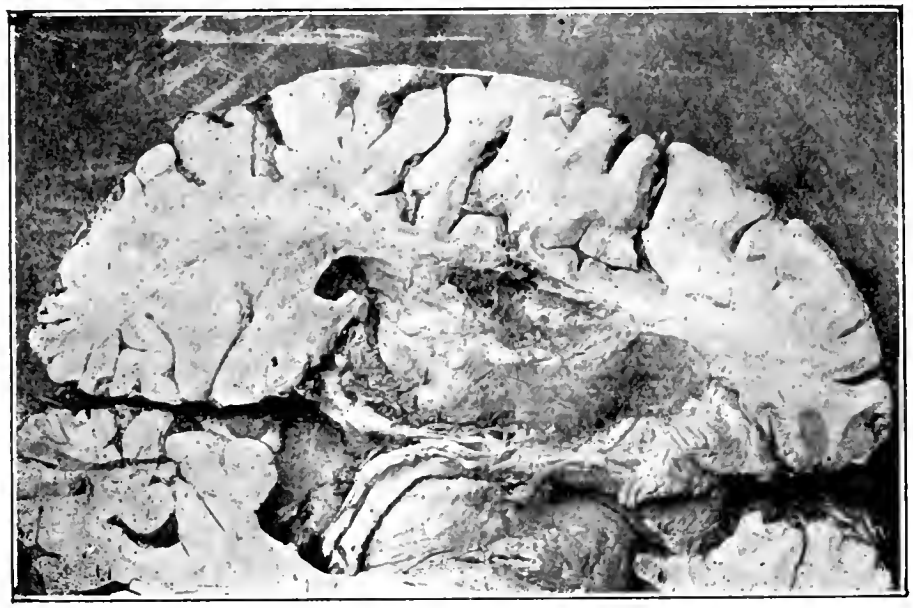

Fig. 10. Complete destruction of the left lenticular nucleus and anterior and posterior limbs of the left internal capsule, without persistent motor aphasia. (Case 10.)

and posterior limbs of the internal capsule. We cannot therefore draw any positive conclusions as to the part played by the lenticular lesion in the production of the hemiplegia. The case is chiefly of value in throwing some light upon the effect of so large a lenticular lesion on the function of speech. Speech, while affected at first, was soon recovered, and the patient continued to speak intelligently. She was not a motor aphasic.

This in connection with the first case of this paper shows that complete destruction of the left lenticula does not necessarily cause complete and persistent motor aphasia. While the notes on the condition of speech in this case are not as complete as might be desired, they indicate very clearly a recovery, in part at least, of the motor speech function, and we have the statement from Dr. T. H. Weisenburg that when a resident in the hospital he frequently conversed with the patient, and that she was able to speak. We cannot conclude, however, that speech was normal.

Case 11. *Aploplectic Attack causing Complete Right Hemi-*

plegia with Hemianesthesia and Complete Aphasia—Disappearance in large part of Speech Affection and Hemiplegia—Persistence of Hemianesthesia—Right Lateral Anopsia—Dyslexia—Partial Word Deafness and Partial Word Blindness—Deviation of Tongue to the Right—Right-sided Spasticity and Contractures and Exaggerated Reflexes—Large Cyst involving Left Extreme Capsule, Claustrum and External Capsule and Destroying the Left Lenticula and Carrefour Sensitif, also involving Optic Radiations—The Anterior Half of the Posterior Limb of the External Capsule and the Anterior Limb of the Internal Capsule not involved.

A case which was first recorded by one of us, Dr. Mills,[28] in a paper which was written in collaboration with Dr. G. E. de Schweinitz, is of some value in connection with the study of the motor and speech functions and the question as to the sensory functions of the lenticula. This patient died in the wards of Dr. F. X. Dercum, in the Philadelphia General Hospital, and the case was made the subject of a paper by Drs. Dercum and Spiller,[29] on permanent hemianesthesia resulting from destruction of the *carrefour sensitif* and the lenticula without implication of the thalamus, the case appearing to demonstrate that the cerebral sensory fibers are located chiefly, if not altogether, in the area of the *carrefour sensitif,* or it may be that some sensory fibers pass through the lenticular nucleus, as held by Edinger. The writers define the *carrefour sensitif,* following Verger, as comprising the last third of the posterior limb of the internal capsule in the most anterior portion of the opto-striate region.

The hemorrhagic apoplectic attack which left the cyst found at necropsy occurred many years before the death of the patient. Immediately after the attack the man was completely paralyzed in the right arm and leg, and also completely aphasic and hemianesthetic. He recovered his speech very largely in about two months and the motor paralysis largely disappeared in about three months. The hemianesthesia remained and was extremely marked up to the time of the patient's death. The man was paralyzed in 1892. Four years later, in 1896, when the clinical report of Dr. Mills and Dr. de Schweinitz was made, he had right lateral quadrant anopsia; absence of Wernicke's symptom; dyslexia; right hemiparesis; partial right hemianesthesia; and partial word deafness and word blindness. His tongue was slightly tremulous on protrusion and deviated a little to the right. With regard to his aphasia, the patient stated at

[28]Mills, Chas. K., and de Schweinitz, Geo. E. The Philadelphia Hospital Reports, Vol. III., 1896.

[29]Dercum, F. X., and Spiller, William G. The Amer. Journ. of the Med. Sciences, N. S., 123, March, 1902.

this time (1896) that from the first he knew what he wanted to say, but could not put it into such words as would be understood by his hearers. He could recognize objects, but could not pronounce their names. He could not read writing or print, because everything ran together; he recognized the letters, but could not pronounce them. He understood sentences, but could not read them. In 1896 he had almost completely recovered from his aphasia, although his voice was a little thick and difficult to understand.

In 1899 there was present a spastic hemiplegia of the right

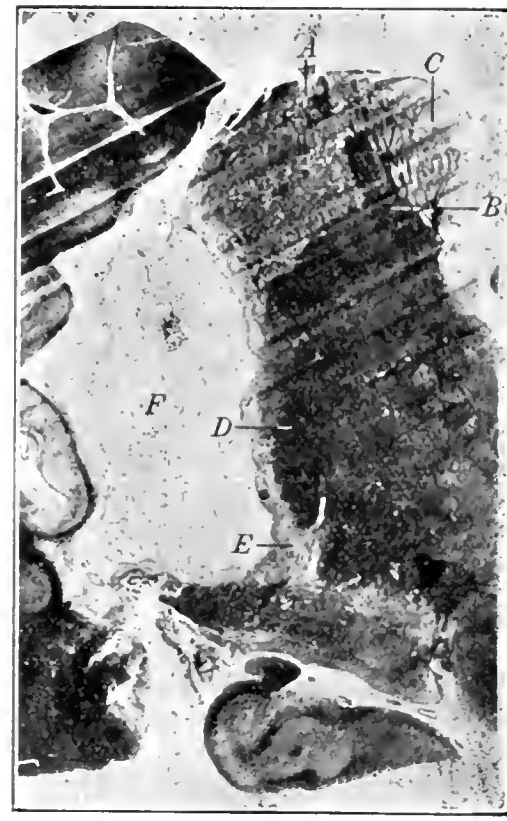

Fig. 11. A. Portion of the lenticular nucleus not destroyed. B. Anterior limb of the internal capsule. C. Head of the caudate nucleus. D. Posterior limb of the internal capsule. E. Carrefour sensitif. F. Cyst in the lenticular nucleus. (Case II.)

side, moderate in severity, with a moderate degree of contracture of the right arm and spastic rigidity and exaggeration of the knee-jerk on the right side. The right side of the face was only slightly involved. The hemianesthesia appeared to be everywhere complete, and was associated with right-sided homonymous hemianopsia.

"A cavity, probably resulting from an old hemorrhage, 2.8 cm. in depth from above downward, and 2.5 cm. in its longest diameter. (1 1-8 in. x 1 in.), was found in the extreme

capsule, claustrum and external capsule of the left side. The left lenticula appeared to be almost destroyed and the extreme posterior part of the posterior limb of the internal capsule and the optic radiations were involved in the destructive lesion. The anterior half of the posterior limb of the internal capsule was not implicated in the cyst. The anterior limb of the internal capsule was entirely normal. The thalamus was not implicated at all in the cyst, and the external medullary lamina of the thalamus was distinct and normal in appearance. The external nucleus seemed to be of about normal size. A transverse cut passing through the hypothalamic region showed the anterior half of the posterior limb of the internal capsule apparently normal. The lenticular nucleus, except in its extreme ventral portion, was destroyed.

"The portion of the thalamus that showed atrophic change was the pulvinar, and this alteration was the result of the destruction of the optic radiations."

In addition to the macroscopical examinataion of the speecimen in this case, careful microscopical investigation by serial sections was also made, this confirming the fact that the anterior half of the posterior limb of the external capsule and the entire thalamus were not involved in the lesion, while the *carrefour sensitif* and the lenticula, with the exception of its most ventral portion, were destroyed.

It would seem fair in this case to refer the moderate spastic hemiparesis which persisted until the death of the patient partly to the lenticular lesion, as the motor subdivision of the posterior limb of the internal capsule was only a little involved. The early aphasia which disappeared in two months was probably due to compression or partial destruction of some part of the lenticula concerned in speech phenomena, which must be the anterior portion, the compression of the neighboring speech areas in the temporal lobe and to the destruction of the insula. We are inclined to refer the permanent hemianesthesia chiefly to destruction of the *carrefour sensitif,* although we do not deny that sensory fibers may pass through the lenticula. The disturbance of both phonation and articulation which is referred to in the statement that his voice was thick and difficult to understand, was probably dependent upon the lenticular lesion, as the genu and anterior half of the posterior limb of the internal capsule were not destroyed.

Both from our study of the literature of the subject and from the analysis of the personal cases given in this paper, we find is somewhat difficult to form positive conclusions as to the symptomatology of lesions of the lenticular zone.

We believe, however, that a few general conclusions may be drawn:

1. Lesions restricted to the lenticula apparently do not cause sensory symptoms;

2. Motor symptoms probably result from lesions situated in certain parts of the lenticula: speaking generally, the lenticula may be regarded as a motor organ;

3. Anarthric or dysarthric speech disorders result from lesions of some portion of the left lenticula, which probably contains centers which are concerned with the movements which make speech possible;

4. Destructive lesions of certain portions of the lenticula probably cause a paresis of the limbs or face;

5. The paresis or paralysis caused by destructive lesions of the lenticula differs from that produced by capsular lesions, the impairment of power not being so severe and not being so characteristic in the former as in the latter case;

6. The paresis or paralysis which is caused by lenticular lesions differs from that produced by cortical lesions in that it is less likely to be dissociated; although dissociated lenticular paresis may occur;

7. While the loss of power which results from a destructive lenticular lesion is permanent, it is usually not intense;

8. Persistent true motor aphasia, as this form of speech disorder is generally understood, is not caused by a lesion restricted to the lenticula, no matter what its size or destructiveness;

9. The insula, cortex and subcortex play an important part in speech phenomena, one entirely different from that played by the lenticula and the internal capsule;

10. The insula is a part of the cortical motor center for speech, Broca's convolution probably forming with the insula the entire cortical motor center for speech;

11. Motor aphasia may be present without a lesion of the left third frontal convolution;

12. The lenticula forms too large a portion of the cerebral hemisphere to be regarded merely as a vestigial organ.

HAVE THE TYPES OF GENERAL PARESIS ALTERED?

By L. Pierce Clark, M.D.,
of New York.
and
Charles E. Atwood, B.S., M.D.
of New York.

(*Continued from page* 557.)

Charts omitted from article published under above caption in last issue, September, 1907, in which 3,000 cases of general paresis were analyzed by types.

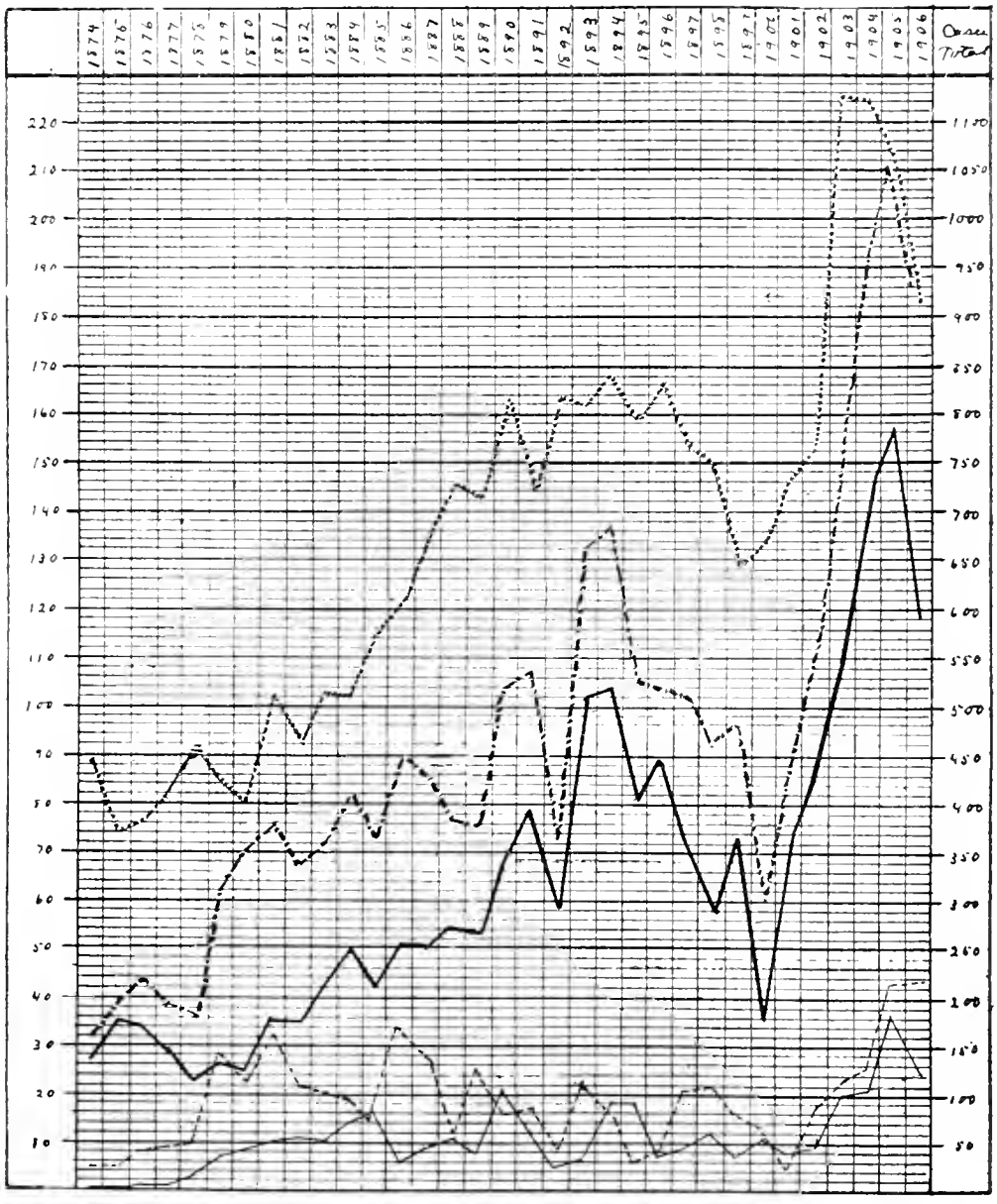

Chart No. 1.

Analysis of 3,000 paretics by yearly periods. The foremost (dotted) line indicates the total insane admitted. The second (dotted) line, the total paretics admitted. The third (heavy continuous) line, the number of paretics of grandiose type. The fourth (dotted) line, the number of simply dementing type. The lowest (light continuous) line, the number of paretics of depressed type.

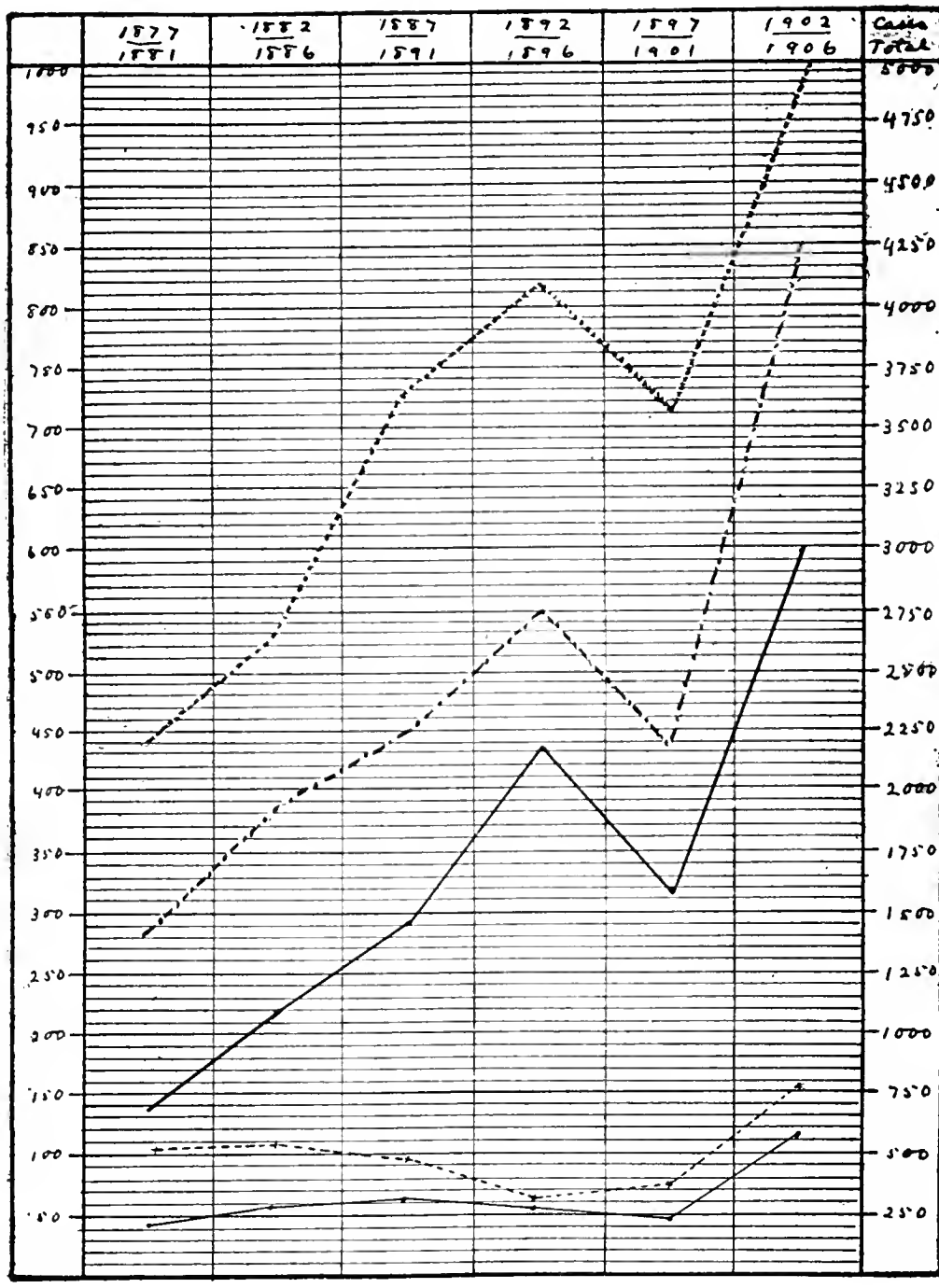

CHART NO. 2.

Analysis of 3,000 paretics by five-year periods. For detailed description see Chart No. 1.

Society Proceedings

AMERICAN NEUROLOGICAL ASSOCIATION.

Held in Washington, May 7, 8 and 9, 1907.

The President Dr. Hugh T. Patrick, in the Chair.

(*Continued from page* 592.)

SOME FORMS OF ACRO-CYANOSIS AND THE RELATION OF ACROCYANOSIS TO RAYNAUD'S DISEASE, ERYTHROMELALGIA, OSLER'S DISEASE AND OTHER CONDITIONS.

By Dr. Lewellys F. Barker and Dr. T. J. Sladen.

The paper reports a case of chronic anesthetic acrocyanosis with gangrene of the end of two toes in one foot and malum perforans of the great toe of the other foot. Instances of acrocyanosis in connection with the other syndromes mentioned are given, and the relations of allied symptom-complexes are discussed. (The paper will be published in this journal.)

Dr. A. Gordon said that two or three weeks ago he exhibited before the Philadelphia County Medical Society a case almost identical in its clinical manifestations to that of Dr. Barker and Dr. Sladen's, and his conclusions were almost identical with those of these gentlemen. It was a case of a young man of twenty-two, who had a redness of both hands. He was a carpenter by occupation. There was no alcoholic history whatever. He noticed about five weeks prior to his coming to Dr. Gordon gradual on-coming redness of the hands, limited just at the level of the wrists Of course the first thought was that of erythromelalgia, but the patient presented a number of little ulcers or scars which were the result of old ulcerations. Dr. Gordon thought of Mitchell's disease and of Raynaud's disease. However, a close examination showed that while apparently the case presented some features of erythromelalgia, on the other hand it presented signs which were antagonistic to Mitchell's disease. Dr. Gordon concluded that the case could not be classified as either of the two forms. It is allied to these two types, and it goes to show that the two extreme types of erythromelalgia and Raynaud's disease are not always present. There are many cases of so-called intermediary types.

Dr. E. Riggs said he was very much interested in Dr. Barker's paper because of a recent experience of his own. A man of forty-seven years of age had had for several years what he supposed were rheumatic pains in the feet. When he came under Dr. Riggs' observation there was a large ulcer of the left big toe, a slight congestion of the foot, and a cyanosis of the little toe. Dr. Riggs did not know how to classify the case. He did not regard it as one of Mitchell's disease; he could not regard it as typical of Raynaud's condition. However, the toe amputated itself, the pain disappeared (Dr. Riggs forgot to say that the pain was atrocious), and the man made an apparent recovery.

Dr. A. R. Allen said that in a case which Dr. J. K. Mitchell and he had reported before the American Association of Physicians last year the symptoms were very much like those in Dr. Barker's case, but they found over 20,000 white blood cells, with a distinct destruction of red blood cells, and 13 per cent. eosinophiles. There was intense itching, which was rather paroxysmal, coming in in the night. The urine showed a large amount of urobilin. During the hours of itching Dr. Allen examined the blood at intervals, and also examined it during the daytime, but there was no difference. He said he would like very much to know what the blood picture in Dr. Barker's case was.

Dr. H. H. Hoppe said he arose briefly to refer to a case of symmetrical erythromelalgia which he saw in a child four or five years of age, which was not typical. Both hands and both feet were affected, congested, red and swollen, with a formation of blebs; these blebs varied from the size of a split pea to the size of a ten cent piece. The special feature in this case was a subjective sense of pain and great heat, so that the child carried a small bucket of water around to put her hands in. The peculiar feature of the case is that the child recovered after six weeks under iodide of potassium.

Dr. P. C. Knapp said he wished to speak of a case of a young woman who had been under his care a year previously, who presented a very marked cyanosis of both hands, gradually diminishing on the arms, although there was a distinct congestion as high as the elbow, with the formation of very small ulcerations upon the tips of the fingers which were slow in healing. In this case there was no pain and very little sensory disturbance. There was one method in treatment tried at the suggestion of Dr. Cushing; namely, the putting an Esmarch bandage and tourniquet on the arm for a number of minutes each day and then letting it out in order to flush out the artery. The method, however, gave practically no relief.

Dr. W. G. Spiller said he had been forced to very much the same conclusion that Dr. Barker had reached, that these cases shade into one another more or less. In a case of erythromelalgia reported by Dr. Weir Mitchell and himself a number of years ago the blood vessels and the nerves of the big toe, which was amputated, were very much diseased. In another case of erythromelalgia that was under the care of Dr. Spiller, the big toe was amputated and the blood vessels and the nerves were very distinctly diseased. It is very important to determine how the nerves are examined. Degeneration if slight may entirely escape detection. Dr. Spiller believes that if the examination is properly made the peripheral nerves will in every case of erythromelalgia be found diseased in the limbs that are affected.

Dr. Spiller cautioned against operations on the feet of persons affected with vasomotor trophic neuroses. In the case which Dr. Mitchell and he had reported the big toe was amputated, and it looked as though the wound would never heal. In the other case to which he had referred it looked also as though the wound would not heal. He said that in the past two or three weeks he had seen still another case in which an operation on the foot had proved disastrous. The treatment which he thought would give most relief where pain in the foot was intense was either a resection of the nerve at the lower part of the leg or possibly stretching, but he would prefer the former.

Dr. J. J. Putnam said he thought it was very extraordinary, as Dr.

Barker had said, how many of these affections there are and how they run into each other, and yet on the whole, although the types are numerous, they are nevertheless types. The cases run pretty true in their own line. Each individual case remains as it was in the beginning. He said they had recently received at the hospital two cases in children which must be included in this general group—more especially as they are Hebrew children —where both arms were red almost up to the elbows, intensely red, the color fading slowly away, but there was no pain or sensory disturbance. Dr. Putnam's experience with the tourniquet had been a little more favorable than Dr. Knapp's. He had one patient with an ulcer on the side of his foot whose case belonged in this category, where the tourniquet was applied and he was put to bed. It was not possible to say whether the relief which followed was due to the tourniquet or to the rest in bed or to both, but it did seem as though the tourniquet was beneficial. His experience had been like Dr. Spiller's with regard to amputation. Frequently after amputation the wound does not do well.

Dr. Putnam said he would like to say a single word about the subject of acroparesthesia, of which he had seen a large number of cases. In the first description of this disorder, which he had written twenty years or more ago, he had suggested that there was perhaps a vascular change which involved the circulatory supply of the nerve endings, and this hypothesis still seemed to him admissible. It is certain that the skin sometimes changes color and that the muscles become stiff, so that vasomotor phenomena must be reckoned with. Yet these signs are not constant. The only constant feature is the paresthesia itself, but this could also be referred to a vasomotor change, if it could be shown that the nerve or nerve-endings have an independent circulation. This is possible, although the proof is lacking.

Dr. J. R. Hunt said he had seen several examples of the class of case that Dr. Spiller referred to, in which there was a great deal of pain and a great deal of redness in the extremities, more particularly in the toe, especially the big toe, and these cases were very often called erythromelalgia, and they do present many of the symptoms of erythromelalgia. In the cases he had seen, however, there was very marked defect in the pulsation of the pedal arteries, and he had regarded them as belonging to that very unusual type of endarteritis occurring in young adults of obscure origin. In these cases the changes in the peripheral arteries are so marked that any amputation near the site of the gangrene would be absolutely inadequate and healing delayed or impossible, and surgeons in such cases usually go above the ankle or the middle of the thigh; in fact they often do the operation without checking the hemorrhage, and they go on operating until they get a certain flow of blood which will presuppose the healing of the flap. These cases it seemed to him should be somewhat separated from the type Dr. Barker has described, particularly from the acrocyanosis and acroparesthesia and pure types of Raynaud's disease. He had seen one case which would come more particularly into this group, the case of a very young girl with marked scleroderma. She had repeated attacks of acrocyanosis, paroxysmal crises in the tips of her fingers and toes, as well as in the tip of her tongue, and on several occasions there was distinct substance loss. Just before her death she lost one or two fingers and he thought a toe.

Dr. F. X. Dercum said he did not think that the cases of sclerodactyly belong to these purely vascular or peripheral nervous cases, as would ap-

pear to be the case with erythromelalgia and allied states. Sclerodactyly is only a phase of scleroderma. It has distinct relations with morphea, it seems to be primarily neurotic, and he thought it important, while admitting relations between the various forms of peripheral dystrophies to keep them well defined.

Dr. L. F. Barker said he thought he should emphasize again the entire absence of pain in their case. The part was not at all painful; it was anesthetic.

As to the blood count, when the patient came to the hospital he was suffering from bronchitis, and had a slight polymorphonuclear leucocytosis, but after the bronchitis disappeared the blood findings were normal.

With regard to the Esmarch bandage for treatment he had had no experience, but he said he would like to point out the value of the Esmarch in diagnosis. If we have, for instance, in diabetes a gangrenous toe, we all know how useless it is to amputate the toe, at any rate until we have watched it for some time. Von Eiselsberg in these cases puts on an Esmarch from the toe up to the thigh, leaves it on ten or fifteen minutes and then removes it, and usually finds that while the blood returns very quickly to the whole upper thigh, the leg below the popliteal space remains pale. In that case if he amputates at all he does so above the knee; in other words, the application of the Esmarch and watching what happens afterwards gives, he believes, a clue to the seat of the arteriosclerotic disease. Dr. Barker said they had confirmed this in diabetic cases in the Johns Hopkins Hospital.

(To be continued.)

PHILADELPHIA NEUROLOGICAL SOCIETY.

January 22, 1907.

The Vice-President, Dr. J. W. McConnell, in the Chair.

BULBAR PALSY IN MULTIPLE SCLEROSIS.

By Dr. T. J. Orbison.

Dr. Dana and Dr. Spiller first called attention to the rarity of the disease in this country. In Starr's experience it occurs once in about 370 cases. The case exhibited is from Dr. Spiller's Polyclinic Hospital service. The ordinary type of the disease begins with numbness and increasing weakness of the legs. The gait is wabbly, rather than ataxic. The feet seem to wander about as in drunkenness. Oppenheim describes it as "vacillation." It is due to an irregular contraction of the muscles of the trunk, and the entire body sways in the attempt at locomotion. Tendon reflexes are increased and there may be a Babinski reflex on either side or on both. There is tremor in the hands, so much so, at times that a glass of water held to the mouth will be so shaken that its contents will be spilled. Bulbar symptoms often appear early, the speech is scanning rather than mumbled. The tongue may not be atrophied. Scanning is due to ataxia or intention tremor of the muscles of speech production. There are certain mental symptoms suggesting paresis that are often noted. All of the above symptoms, except the Babinski reflex, are seen in the case exhibited.

Dr. F. X. Dercum said that unequal involvement of the two hands and wrists is, of course, not uncommon. Sometimes the inequality is very decided, but he had never seen a case limited absolutely to one side. He recalled one patient where the difference between the two sides

was very great, and in this instance the patient gave a history that one side was distinctly paralyzed for a period of weeks before the other side was involved. Dr. Dercum did not, however, see him during this time. He thought it probable that palsy begins on one side and persists more frequently as a one-sided palsy for a time than we are in the habit of noting.

PROLONGED STUPOR RESULTING FROM ALCOHOLISM, FOLLOWED BY AMNESIA AND CONVALESCENCE.

By Dr. A. C. Buckley.

Male, age thirty-six, clerk. Excessive user of alcohol for fifteen years. Two years ago, after an over-indulgence, suddenly developed delusions of persecution and self-accusation, after which he passed into a state of excitement followed by stupor. He remained in bed in a state of absolute passiveness for about nine months, during which time he occasionally roused to say a few words and again would relapse into his former state. For several months there followed a state in which was complete absence of volitional action, while reflex physical response to stimulation was present. The latter state abruptly terminated by the patient suddenly beginning to talk; the following day he began to perform volitional acts, talked some, but speech was limited on account of a total amnesia for all past events. Later, he was able to recall what was said to him, also all that was told him about himself, his family and his surroundings, but could not by association recall any event of his previous life. His memory of recent events is good. His present knowledge is solely the result of experiences since his awakening. Objects the uses of which were at one time familiar to him, he recognizes as seeming familiar, but he does not know why they should be familiar. He is now in process of re-education; he has difficulty at times in remembering new facts which he has learned. He is able to write his name, which he has been taught to do; he can copy with difficulty.

UNILATERAL WRIST-DROP FROM LEAD.

By Dr. W. G. Spiller.

A man who was not alcoholic, and who had worked as a painter four years, developed in October, 1906, weakness of the extensor muscles of the right fingers, the palsy beginning in the forefinger. The extensors of the wrist were not affected. At the present time all extensor power in the right fingers is lost. Four or five weeks ago the extension of the first finger of the left hand became impaired. This finger is the only portion of the hand affected. Sensation is intact. Unilateral wrist-drop from lead is of very unusual occurrence.

Dr. Potts said that his recollection of a case mentioned by Dr. McConnell was that while the patient was under his observation only one side was affected. While he thought the trouble was due to lead, he had hesitated to make a positive diagnosis, because there was also a history of alcoholism. A point in favor of lead was that the supinator longus muscle reacted well to the faradic current, while the extensor muscles showed reaction of degeneration. The man as a house painter had been, of course, exposed to lead. The fact that the other arm became affected after he came later in Dr. McConnell's charge would seem to settle the diagnosis in favor of lead.

SPINAL CORD CHANGES FOLLOWING A SECONDARY GENERAL ANEMIA WITH RECOVERY.

By Dr. J. W. McConnell.

A young man of about forty years had a sudden gastric hemorrhage. A few days afterward he began to have diminution of vision and became totally blind in a few days. Soon after he had numbness in his limbs, this being followed by weakness. Within a short time he presented symptoms of a postero-lateral sclerosis. Blood examination then showed the changes of a grave anemia, with first an optic neuritis followed later by an optic atrophy. In the course of several years, during which time he was under constant observation, all of the sclerotic symptoms disappeared, leaving only the optic atrophy.

Dr. Spiller stated that he was much interested in the patient, as the man had been in his service at the Philadelphia General Hospital at the time the symptoms were very grave. He was paralyzed in his lower limbs especially, so that he was unable to stand. The case was interesting in other particulars. The man said he had vomited about a quart of blood and had passed blood by the bowel, but he really did not know how much blood he had lost. Such a case as this seems to show that the spinal cord changes may be the result of anemia. This man was healthy until the loss of blood occurred. He could not have had much degeneration of the spinal cord, or he would not have recovered as he had done. Dr. Spiller thought the optic neuritis might be the result of severe anemia, as it occurs also in chlorosis, although he was not aware that it had been observed in anemia. Certain mild forms of optic neuritis are hard to distinguish from atrophy.

Dr. Dercum thought it questionable whether the fluid that fills the vessels after such a severe anemia is not itself toxic. It seems difficult to explain the optic neuritis by a mere anemia. It is also interesting to note that this man had a Babinski reflex, which has since disappeared.

Dr. C. W. Burr asked if the patient had paralysis of bladder and rectum on admission.

Dr. Burr called attention to the fact that there had been some instances of women becoming paralyzed after bleeding in childbirth in precisely that way. He had never heard of a real optic neuritis following hemorrhage.

Dr. Weisenburg stated that he knew this case very well, as the patient was admitted while he was on duty. He did not believe that he had seen a case of spinal cord change as a result of pernicious anemia recover, and that there were few, if any such instances in the literature. In the present case the blood changes were those of a grave anemia and not of the pernicious type. The occurrence of an optic neuritis is extremely rare. Only recently Dr. Weisenburg had written upon this subject and had found no similar instances in the literature. Optic atrophy, a result of hemorrhages, is not an infrequent occurrence, and is not difficult to explain. The only reference that he had been able to find in the literature regarding optic neuritis was in Norris and Oliver's book. These authors mentioned the fact that sometimes in the course of a pernicious anemia there appears what seems to be an albuminuric retinitis. It may be that in this present instance the optic neuritis may have been a forerunner of the above condition. Another

possibility is that of a coincident optic neuritis as a result of another cause.

Dr. Potts thought that the relation of an anemia to optic neuritis had been shown, at least in the form of anemia known as chlorosis. A number of cases had been reported, among others by Patrick, in which the eye symptoms were such that for a time it was doubtful if the patients did not have a brain tumor. To be sure chlorosis is not a simple anemia, but it is one of the forms of that condition.

Dr. Burr called attention to the fact that this man did not have chlorosis. He had a sudden large hemorrhage and immediately after the loss of a great deal of blood he became paralyzed in both legs.

UNILATERAL OPHTHALMOPLEGIA WITH PARESIS OF VOLUNTARY UPWARD ASSOCIATED OCULAR MOVEMENT.

By Dr. J. T. Krall.

The patient was white, male, age forty, occupation fireman. He said he had a chancre seven years ago. His vision was good until six months ago, when during the night he was suddenly seized with severe pain in the head, as though something had burst. He could move his arms and legs, and there was no loss of sensation or motion in any of the extremities, and he was not unconscious. After this the right upper lid drooped and he could not move his eyes. He also had diplopia, dizziness and a feeling of dullness in the head. When examined, on June 12, 1901:

O. D., external appearance negative. O. S., ptosis of upper lid, not complete, he could forcibly lift the eyeball 3 mm. It could be moved a little down and out, but not inward and beyond the median line. Upper motion lost entirely, either alone or in association with O. D. Secondary deviation of O. D. outward. Cornea clear; pupil dilated, regular, round, 6 mm., did not react to light, accommodation or convergence.

Ophthalmoscopic Examination: O. d.; media hazy, nerve outlines indistinct, red in color. Perivascular neuroretinitis. O. S.: Cornea clear, media hazy; hyalitis. Nerve red in color; hazy; outlines veiled; vessels slightly congested and tortuous. Retina smooth, woolly, neuroretinitis. Some whitish plaques to outer side of nerve. O. D. V.=6/9 ??. O. S. V. =6/12 (by holding lid open). The sight of neither eye was improved with lenses.

Accommodation: O. D.+4.25 D. O. D.=3.25 D. Fields were concentrically contracted for form and color in both eyes about one-half the normal.

When examined on December 3, 1906, Dr. Krall noticed that the patient had a peculiar, fixed, stary, expressionless countenance. Head was thrown well back and upper lids forced open as far as possible. Examination showed O. D. normal. Excursions possible in all directions, pupil reaction normal. O. S.: The excursions were good except a trifle limited upward and inward, but in association with O. D. neither eye moved upward beyond the horizontal. All other associated movements normal. Voluntary associated movements upward not so good

as when following an object. On attempting to follow a moving object upward O. D. moved further than O. S. by a series of lateral motions. The von Graefe test was negative, there being no movement of the eyeballs either upon inserting prisms or removing them.

Ophthalmoscopic examination showed a decided improvement in the condition of both eyes. The neuroretinitis had subsided. Media clear. Accommodation: O. D.=3.50 D. O. S.=3.25 D. (about normal for age, forty-five). O. D. V.=5/7.5. S. myd.+.25 C. ax. 90°=5/5. O. S. V.=5/7.5. S. myd.+.25 D. S.×.25 C. ax. 90°=5/5. The vision had improved as is shown, and with a low correcting lens gave full vision. Fields for form were normal in both eyes. Slightly contracted for color. Music balance at 5m.=exophoria 8° and L. H. 1°. Muscle balance at 33 cm.=exophoria 14° and L. H. 1°.

Dr. Weisenburg said that he had examined the patient with Dr. Krall. The case was a very interesting one and it was rather difficult to understand why after a number of years following a unilateral ophthalmoplegia the other eye should be involved. Two possible explanations can be given: one that the nucleus of the oculomotor nerve has become diseased; the other, that we have here symptoms which are due to disuse, the latter being probably the case. This is also borne out by the fact that the movements of the eye are much better in so-called involuntary action than in voluntary movement.

Dr. Dercum asked why the affection could not have been a polioencephalitis superior. Polioencephalitis does distinctly begin on one side and at times is even limited to one side. It is not necessary to fall back on a theory of hemorrhage in a case of this kind.

AN UNUSUAL SYMPTOM IN CHOREA.

By Dr. G. E. Price.

A report of three cases of chorea from the Neurological Dispensary of St. Christopher's Hospital, presenting marked hypersecretion of saliva with more or less constant dribbling. Two of the cases were in girls and one in a boy; the ages being six, twelve and fourteen years respectively. All three cases had one prior attack of chorea, the intervals between the attacks being from one to three years. In each instance the choreiform movements were general, the tongue and muscles of mastication being especially affected. There were speech involvement and some degree of mental disturbance in all, but no history of fright, rheumatism or scarlet fever could be obtained in any of the cases. Two of the patients had systolic murmurs at the cardiac apex; in one case the heart sounds were normal. One child was poorly nourished and anemic, two were in fair general physical condition. None of the cases presented hysterical stigmata. One of the patients was included in the series through the courtesy of Dr. Luther C. Peter, the case occurring in his service.

THE CLINICAL RESEMBLANCE OF CEREBROSPINAL SYPHILIS TO DISSEMINATED SCLEROSIS.

By Dr. William G. Spiller and Dr. Carl D. Camp.

The case reported illustrated the difficulty in diagnosis which may exist. A young man, positively denying during several years, syphilitic infection, presented marked ataxia of gait, intention tremor of the limbs, and a month or two before death, of the muscles of the face; scanning

speech, at first normal pupillary reactions, later Argyll-Robertson pupils, unequal pupils and pallor of the temporal side of the right optic nerve. Remissions did not occur during the years he was in the hospital. Nystagmus was not observed. The lesions found were those of meningo-encephalo-myelitis, consisting chiefly of round cell infiltration, and degeneration of the posterior columns of the cord. It may be that some cases of multiple sclerosis are overlooked by superficial examination, but on the other hand there seems at present a danger that much will be called multiple sclerosis that in reality is some other disease.

THE NEURASTHENIA OF AUTOINTOXICATION.

By Dr. T. J. Orbison.

On the one hand is Oppenheim with those with him who explicitly deny that neurasthenia may be due to autointoxication. On the other hand are Bouchard and those who actively support the affirmative side of the question. There is a middle ground occupied by Osler and Musser, teachers who do not deny the possibility of this cause, but who do not teach it in their books. This paper supports the affirmative side of the argument and gives cases in support. The intestinal tract is an ideal laboratory for the manufacture of poisons; the mucous membrane is a secreting one; the bile and urine have been proven to be active poisons; the blood itself is a carrier of poisons. Given an excess in the poisons or a decrease in the expulsion of them, it is reasonable to suppose symptoms may arise that are truly neurasthenic in character.

Dr. Guy Hinsdale said that as we saw cases of the graver type of mental disturbances due to autointoxication he saw no reason why we should not have cases of neurasthenia due to the same cause.

THE PHILADELPHIA NEUROLOGICAL SOCIETY.

February 26, 1907.

The Vice-President, Dr. J. W. McConnell, in the Chair.

PARALYSIS OF THE SIXTH NERVE, COMING ON DURING AN ATTACK OF TYPHOID FEVER.

By Dr. J. H. Lloyd.

Dr. J. H. Lloyd showed this patient, a negro woman, a school teacher, aged twenty-nine years, who had been admitted to his wards in the Methodist Episcopal Hospital in January, suffering with typhoid fever. When admitted she was about at the end of the second week of the disease. The fever pursued a regular, uncomplicated course until the twenty-seventh day, after which her temperature remained practically normal. For a part of the time the patient received the Brand treatment, having in all ten tubbings during a period of four days. For the remainder of the time she was sponged. There was only slight delirium, and not much diarrhea. The urine for a while presented some albumin and a few casts. There were no nervous symptoms of special importance, except a little headache when the patient was admitted, and later the slight confusional delirium just mentioned. The Widal reaction was positive. The patient first noticed diplopia when she came out of her delirium, that is, before the fever ended; hence

in about the third or fourth week. She was then observed to have internal strabismus of the left eye, caused by paralysis of the left external rectus muscle. There were no cerebral symptoms, no headache nor any other paralysis, no rigidity nor signs of meningeal involvement.

Dr. Veasey examined the patient later and reported paralysis of the left sixth nerve. The patient saw double images on following the moving object to the left beyond, or a little beyond, the middle line, and the images tended to get further apart. There was no paralysis of accommodation, nor of any outer muscles of the eye. The fundus was normal. This patient had been under treatment for her eyes at the dispensary of the hospital before her typhoid fever, but at that time had no paralysis of her sixth nerve; so the onset of the trouble during the typhoid fever is quite clear. Since leaving the hospital, about ten days ago, the paralysis has decreased, and while still apparent it is not so noticeable as it was formerly. This, of course, is of good augury, and shows that the prognosis is favorable, just as in cases of diphtheritic paralysis, and that the patient will probably make a satisfactory recovery. Such cases must be very rare. This is the only one of the kind Dr. Lloyd had ever seen, and the literature is scanty. He had found only one reference to a somewhat similar case, but associated with double ptosis. Dr. de Schweinitz has stated that paralysis of accommodation after typhoid fever is not uncommon, but that other ocular palsies are very rare. The analogy of this case to some cases of post-diphtheritic paralysis will probably strike everybody, but in post-diphtheritic paralysis there is usually paralysis of accommodation, as well as nasal speech, dysphagia and loss of power in some of the extremities with abolition of the knee-jerks, none of which symptoms have been present in this patient. Besides, in this patient, who was under careful observation during most of her fever, there was no evidence of a diphtheritic process. Neither had there been any polyuria, as is seen in some cases of sixth nerve paralysis. Nor was it possible to make out a specific history.

NEW CLINICAL SYMPTOMS IN HEMIPLEGIA AND TABES DORSALIS.

By Drs. T. H. Weisenburg and C. C. Manger.

In a series of hemiplegias it was noted that the palpebral fissure on the hemiplegic side was larger than on the sound side. This symptom was looked for in about ninety cases and found in about eighty. It is probable that this was due to the drooping of the lower lid. This sign has not heretofore been noted, and is of some importance in the instant recognition of paralysis. It is, of course, also present in peripheral facial palsy.

For some time Dr. Weisenburg has noticed that patients with tabes dorsalis have a remarkable similarity of facial expression. So much so that it has been possible in many instances to recognize patients with this disease on sight. The composite picture shows this very easily. There is a paleness of the face and puckering of the brow and drooping of the upper lid and the corners of the mouth, and a peculiar expression around the eyes which is rather difficult to describe but easy to recognize. Besides, in a series of thirty-five cases, all men, the eyes had been found to be either blue or gray. This does

not hold true in women. The occurrence of blue or gray eyes in such a large percentage of cases examined is rather important and while this number is very small, still it is suggestive.

Dr. Charles W. Burr said any study of a possible relation between the color of the eyes and the occurrence of tabes to be of any value would have to include a very large number of patients. It was necessary also that the race of the patients and not merely their place of birth should be considered. Dark-eyed people were not as common at Blockley as light-eyed.

Dr. W. G. Spiller said that the widening of the palpebral fissure in hemiplegia, to which Dr. Weisenburg alluded, brought up the interesting question of paresis in the upper branch of the facial nerve distribution in hemiplegia. When Dr. Spiller was in Vienna in 1893 the paresis of the upper part of the face in hemiplegia was well recognized. In almost every case of hemiplegia where the face is implicated there is at first some involvement in the distribution of the upper branch of the seventh nerve. It is usually of transitory duration. The widening of the palpebral fissure to which Dr. Weisenburg alluded, probably depends on paresis of the orbicularis palpebrarum muscle.

Dr. Sailer called attention to the value of composite photographs for the purpose of determining the physiognomy of disease. The opportunities at Blockley for making such photographs are excellent, and they might be of great service in the conditions under discussion, that is, tabes dorsalis and hemiplegia. In the latter the difference in the palpebral fissure would probably be more accentuated in such a photograph than in any individual case.

Dr. D. J. McCarthy thought the involvement of the lids was due to secondary contracture pulling down the inferior lid. Dr. McCarthy also said that cases of tabes in the negro had been frequently reported to the Society. In all these cases, of course, the eyes were dark.

Dr. Spiller said in regard to the patients presented, they were old, and it is common to find in the aged a little drooping of the lower lid in paresis of the upper branch of the facial because of the loss of the elasticity of the skin.

Dr. Weisenburg in closing agreed with Dr. Burr that 35 cases of tabes in which blue eyes constantly occurred did not demonstrate much, but he thought that the fact that it occurred in so many cases was rather interesting. In the study of these cases, the race of the patients was considered, but there was no definite relation found. Dr. Weisenburg's main object in bringing this matter before the Society was, if possible, to obtain further information upon the subject, and also to stimulate further observations in the color of the eyes in tabetic cases.

Dr. Weisenburg did not agree with Dr. Spiller that the widening of the palpebral fissure was the result of the paresis in the upper part of the face, but thought that it was due to the paresis of the lower eyelid. He did not agree with Dr. McCarthy that this sign is due to secondary contracture as in secondary contracture we should have the opposite condition, that is lessening in the width of the palpebral fissure.

INCIPIENT TABES WITH SEVERE PAINS IN THE NECK.

By Dr. W. G. Spiller.

The patient was a male, 55 years of age. He had ataxia of gait, Romberg sign, difficulty at times in urination, numbness of the hands, very

feeble reaction to light, and gray degeneration of the optic nerves. The patellar reflexes were not diminished, possibly were a little prompter than normal; this is unusual in tabes but has been known to occur when the reflex collaterals in the lumbar region are not implicated. The pain in the shoulder was very striking, it occurred in severe attacks every few minutes, extended up the back of the head about as far as the ear on the left side and over the front and back of the upper part of the trunk on the left side. Sensation was diminished in this area. Spinal syphilis might be thought of, but Babinski's sign was not obtained. The pains in the left side of the neck resembled the shooting pains of tabes.

Dr. McCarthy thought these attacks of pain suggested the pain of cervical pachymeningitis. He asked if it was present from day to day.

Dr. Spiller replied that the patient had it constantly with exacerbations at times.

Dr. McCarthy reiterated that the type of pain was suggestive of the early stage of cervical pachymeningitis, but the great degeneration of the optic nerves and the ataxia of the lower limbs he had never seen in any cases of pachymeningitis he had examined. He had watched the crises of tabetics but they were not continuous as he understood the use of the term. They came and exhausted themselves and were not of the continuous type of neuralgic tic like Dr. Spiller's patient presented.

Dr. C. K. Mills said he had seen this man when he first came to the hospital and lectured on him on one occasion. He thought the case one of so-called high tabes. He had seen a considerable number of cases of this sort. He had also seen some cases of cervical hypertrophic pachymeningitis and forms of syphilitic meningitis in the cervical region. Confirming the diagnosis of high tabes were such symptoms as the ataxia, the condition of the pupils, and the atrophy of the optic nerves. Cases of high tabes vary considerably in their symptomatology. He had seen a case a week previously in a young man, a private patient, who had pains somewhat similar to the pains suffered by this man, but not similar in their continuance. The pains occurred at intervals but not almost daily. The patient had pains about the chest; he had lost the knee jerks and Achilles jerks and had dilated pupil on one side. It was a question in all these cases of the intensity, and above all of the peculiar distribution of the lesions. He had seen all sorts of commingling of phenomena in connection with dominating cervical tabes. He could recall 10 or 12 other cases; some with knee jerks lost, and Achilles jerks retained, others with knee jerks and Achilles jerks present, and so on through a considerable list of similarities and differences.

Dr. Sailer said he remembered some years ago seeing a commercial traveler passing through the city, who came into his hands suffering from tonsilitis. He discovered that he also had tabes dorsalis. The patient had continuous severe pain in the right arm, so severe that it practically disabled the arm unless he took huge doses of potassium iodide. There were no gross motor disturbances in the arm, no ataxia nor any sensory disturbances, simply the pain which compelled him to keep the arm quiet.

He remembered another case he saw with Dr. Musser a good many years ago, a man with tabes dorsalis evidently of the superior type. He had laryngeal crises, diplopia, ptosis of one eyelid, and Argyll-Robertson pupil. The case was typical, excepting that the knee jerks and the

Achilles tendon jerks were more lively than normal. There was, however, no ankle clonus.

LESION OF THE CAUDA EQUINA PROBABLY UNILATERAL.

By Dr. W. G. Spiller.

H. C., a male, thirty-one years old, was injured eighteen months previously by a bale of cotton falling against the abdomen. He was unable to work for about three weeks, but then returned to heavy work, feeling not quite so well as formerly. About a month after returning to his occupation, while lifting a bale of cotton, he felt something give way in the right inguinal region, and at the same time he heard a tearing sound. He immediately felt weak and limped on the right lower limb, but walked home, a distance of about two blocks, and went to bed. After one day he got out of bed but remained at home about a week. He then returned to heavy work, but he has not been so strong as he was before the injury.

After the accident he lost control of the bladder, so that when he coughed or exerted himself, the urine would escape. This condition gradually became worse until now he has no control of his bladder and has been wearing an urinal about a year. Sexual desire is not weakened, but only the dribbling of urine prevents the sexual act. During the past two months he has noticed that the rectal sphincter functionates feebly and that when there is a call to stool it is urgent. His gait and station are good. The lower limbs are well developed but the man thinks he is weaker than he was before the accident. The left side of the scrotum, left side of the perineum, and the left buttock near the anus have fully normal sensation to touch and pin-prick, whereas the right side of the scrotum except the upper outer portion, the right buttock in a small area near the anus, and to a less degree the right side of the perineum, show diminution of sensation to touch and pin-prick. The right side of the penis also is less sensitive than the left side. The sensation of the testicles is normal. The patellar reflexes are prompt, but the Achilles reflexes are slight. Babinski's sign is not present. The upper portion of the body is not affected. The lesion must be in the lower sacral roots, and probably confined to one side because of the unilaterality of the disturbance of sensation in the supply of these roots. This unilaterality also is contrary to a lesion of the conus. It is a question whether the vesical and rectal incontinence can be caused by an unilateral lesion but it seems probable. The cause of the symptoms was probably stretching of the lower sacral roots of one side by excessive straining, as has been seen also in lesions of the sciatic nerve.

Dr. Mills thought this a very interesting case. The only thing that suggested itself was as to how this stretching could occur in a case of this kind without other injury. That is, what were the exact mechanics of the process. It was difficult to understand how, with the nerves of the cauda equina, in the absence of accident locally interfering with them in some way, this stretching could be brought about.

Dr. McCarthy referred to a case he saw three or four years ago in Dr. Spiller's clinic of a man lifting a heavy weight, the case later coming to autopsy, in which there was a lesion of the cauda equina. It was bilateral. There was paralysis of the rectum and bladder (the rectum afterwards recovered). There was distinct sensory disturbance around the anus and scrotum. Dr. McCarthy's own impression was that

it was not due so much to stretching as to a localized hemorrhage either within or around the cord, without any special grounds for such an opinion. In that case there was no pain as he remembered it.

Dr. Spiller said he did not think we could accept a diagnosis of hemorrhage of the conus in this case, for the reason that the lesion was chiefly unilateral. If a hemorrhage is in the conus it must cause bilateral symptoms, as the conus is very small. It is also improbable that the hemorrhage is in the roots of the cauda equina, because we could hardly suppose that a hemorrhage around the roots would be confined to one side. The cauda equina roots are anchored as they pass through the dura, and if there is a forcible over-stretching of the back with the feet firmly planted one can readily believe there might be stretching of these roots. Dr. Spiller said he had found in the Berliner Klinische Wochenschrift a few weeks ago, a description of a man who was striking with a heavy instrument: he missed the object and the blow went farther than he intended and caused paralysis of the sciatic nerve from the stretching of the nerve. It did not seem necessary to assume that there was hemorrhage here.

Dr. Spiller also said that the paralysis of the bladder in the case with unilateral symptoms was not complete at first. He was able to retain urine excepting when he coughed or was under unusual exertion. It was an interesting question as to whether the bladder could be paralyzed from an unilateral lesion. The rectum had not been absolutely paralyzed but its action was very imperfect. To establish the unilateral involvement he depended more upon the area of sensation. We must assume that the bladder and rectum are innervated from the lower sacral roots, and we know that the area of anesthesia which this man had was in the distribution of probably the fourth and fifth sacral roots.

A CASE OF INTERMITTENT CLAUDICATION.

By Dr. D. Riesman.

T. W., married; 40 years of age; native of Russia; occupation, potter. For a year and a half typical attacks of intermittent claudication characterized by cramp-like pains coming on after walking a short distance and compelling him to sit down; after a few minutes' rest is able to proceed, but walking is again interrupted as before by cramp in the calf muscles. No pain at night; occasionally cramp in the toes and sensation of burning on inside of leg and in soles, with pain under toe nails; at times pains in back, arm, and wrists; changes in weather have no influence. Huge varicose veins, which in patient's opinion have nothing to do with present trouble; slight degree of flat foot. Tendon reflexes normal; no Babinski reflex; no ankle clonus; plantar reflex feeble; tactile sensation not disturbed; pupils unequal; react to light. Absence of pulsation in dorsalis pedis and posterior tibial arteries. Comments upon this case and upon arteriosclerotic ischemias in general.

Dr. McCarthy said that in respect to the cerebral type of exhaustion paralysis, two or three years ago, he discussed before the College of Physicians a condition which he called intermittent exhaustion paralysis of cortical origin. This was before the recent discussion on the senile type of intermittent claudication was published. Last year he

presented the subject more in detail before the American Neurological Association without publishing it. At that time he called attention to a syndrome which differed considerably from the intermittent peripheral paralysis with spasm, and called attention to the condition of intermittent hemiplegia occurring in cases of marked cerebral arteriosclerosis, these cases going to autopsy, diagnosed as uremic, although they could not be explained on that ground. His attention was first called to this condition in a case of migraine in which there was extensive arteriosclerosis without kidney involvement. In this just as in the peripheral cases, there is a condition of deficient nutrition to brain centers due to a very marked condition of arteriosclerosis. This condition he tried to explain in this discussion must be distinctly differentiated from cases with uremic symptoms, on the one hand, and the sclerotic cases in which the pons or basic centers are involved. In these cases the symptoms are much more prolonged. In the condition of which he was speaking they may last 24 hours. His own feeling about these cases of peripheral intermittent claudication was that they could not be altogether explained on the deficiency of the blood supply. There was some change in the muscles. A damming back of the blood with increased total blood supply in the extremity was a much more potent factor than the deficient blood supply. In the Phipps Institute the leg muscles had been examined in practically every case autopsied. Very often they came across extensive grades of arteriosclerosis, and in one case only, of intermittent claudication. We must consider whether a parenchymatous change in the lower extremities rather than a deficient supply from the vessels is responsible; i.e., a pathological change in the whole extremity in the arteries, veins, muscles and to a certain extent in the tissues.

Dr. Spiller stated that he understood Dr. Riesman to say that the only important differential feature in this central claudication according to Dejerine, is the condition of the pulse. Dr. Spiller stated that Dejerine emphasized the fact that in the central type of claudication the reflexes may be exaggerated after exhaustion and lost at other times; there may be a Babinski sign after exhaustion. He thought hardly anyone who had studied the subject of intermittent claudication believed it due solely to the arteriosclerosis. Erb stated that there must be something in addition to the arteriosclerosis. Arteriosclerosis often occurs without intermittent claudication.

Dr. Spiller said transitory hemiplegia from arteriosclerosis is well known. He had spoken of it himself repeatedly during many years.

A CASE OF ALTERNATING UNILATERAL EPILEPTIFORM CONVULSIONS ASSOCIATED WITH CORTICAL CEREBRAL DEGENERATION.

By Drs. Charles W. Burr and Carl D. Camp.

An elderly man suddenly fell unconscious in a right-sided convulsion. For the remainder of his life, a few weeks, he continued unconscious and had recurring epileptiform convulsions, sometimes confined to one side, sometimes to the other, and occasionally passing over slightly to the other side. The immediate cause of death was lobar pneumonia. At autopsy the calvarium and dura mater were found to be entirely normal. The pia over the Rolandic region and anterior

thereto was much thickened and milky in appearance. The anterior horns of the lateral ventricles were contracted and showed numerous adherent bands. The floor of the lateral ventricle was distinctly thickened. There was no softening or hemorrhage anywhere within the brain. The superior longitudinal sinus contained an ante-mortem clot. The pons, medulla and spinal cord were normal. The thickening of the pia was due to an overgrowth of the connective tissue without round cell infiltration. Many of the Betz cells of the cortex were markedly degenerated. The case is interesting on account of the occurrence of the convulsions due to primary disease of the cortical cells. It is also interesting on account of the nature of the convulsions themselves, sometimes occurring on one side, sometimes on the other.

Dr. Spiller stated that some years ago he had a case similar to this. The man had unilateral convulsions first on one side of the body and then on the other caused by an extradural hemorrhage in the occipital region. The patient was operated upon and the hemorrhage removed. There seemed to be irritation first on one side of the brain and then on the other.

NEW YORK PSYCHIATRICAL SOCIETY.

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Dr. Allan McLane Hamilton in the Chair.

THE PSYCHOGENETIC FACTORS IN SOME PARANOIC CONDITIONS, WITH SUGGESTIONS FOR PROPHYLAXIS AND TREATMENT.

By Dr. August Hoch.

Dr. Hoch pointed out that among the paranoic states there were cases, and that they probably represented a large proportion, in which the psychogenesis could be clearly traced, when the facts of the cases were really accessible. The theory of the development of paranoic states Dr. Hoch summarized briefly as follows, stating that besides basing his ideas upon facts of his own studies he had been influenced by the work of Adolf Meyer, Freund, Bleuler and Jung:

Every person has certain points on which he is especially sensitive. He has ideas or complexes of ideas which are associated with very strong feelings. These complexes refer either to personal defects, shortcomings, limitations, or to feelings of guilt, remorse, shame; on the other hand to certain longings and desires. We may, therefore, generally speaking, say that they belong either to the realm of self-assertion or to the sexual sphere, in the broadest sense of the term. Now most people are able to get square with such things, partly because their nature is such that these feelings never reach anything like a great intensity, or partly because they have a healthy way of dealing with these matters.

Other people do *not* get square with such difficulties. They do not acquire balancing, healthy habits, such as a healthy turning away from one's difficulties to outside interests, or a habit of unburdening or a certain aggressiveness and the like. While then such undercurrents, as we may call these complexes, when they are of any intensity have themselves

a tendency to set narrower and narrower limits to the interest and to create a certain fascination, they often become a menace to the sanity of mind, also because they are not balanced sufficiently by sound mental tendencies. In this way there develops a growing disharmony which gradually, or sometimes under the influence of acute causes, physical or mental, may suddenly lead to an unbalancing of the mind, when, finally the undercurrents break through to the surface.

But the mind, even in the cases in which the undercurrents are not handled properly, makes certain miscarried attempts at readjustment. Thus, the feelings of defect and the longings do not come to the surface as such, but are transformed; the former give rise to a general suspiciousness and delusions of persecution, probably for the same reason that we are inclined to blame everyone else except ourselves, when anything which we do goes wrong; the latter give rise to ideas that the innermost longings are fulfilled. And there are still other forms of such miscarried adjustments.

We see then that we have two things, the undercurrents and the abnormal manner of dealing with these undercurrents, upon which we should lay stress as important in the causation of these paranoic states. To a certain extent this division is of course artificial and the two principles often enough overlap greatly. Then again, it is difficult often to find a correct or definite formula for that which we have called abnormal mental habits, or difficult to pick out from among the complex fabric of mental reactions, those which are disastrous or estimate the dangers of certain combinations, or to correctly guage the value of saving traits. Naturally it will often be a combination of traits rather than single traits which we have to consider, and while we speak of some reactions as dangerous mental habits they may exist in certain combinations in which they are sufficiently safeguarded.

It is also very evident that other causes than an unhealthy manner of dealing with the undercurrents may enter into the causal constellation as well—such as influences which increase the strength of the undercurrents or influences, which, in other ways than those indicated, lessen the resistance, such as the action of alcohol, the menopause, and the like.

These principles were demonstrated by means of careful analysis of four cases and certain indications for treatment were discussed.

Dr. H. R. Stedman, of Boston, was inclined to lay more stress on the influence of heredity in affecting the progress of genuine paranoia than did Dr. Hoch. Numbers of cases of the disorder were seen in patients who had been sensibly brought up and who were treated affectionately by their families, nothing being left undone to make their surroundings congenial and their lives smooth and happy, yet in spite of it all they developed paranoia. Little could be hoped for, he believed, in the way of materially modifying the psycho-genetic factors so as to make any real impression on these cases of typical paranoia, a disease arising on a defective constitutional basis and gradually and logically developing into an inflexible system of delusional thought and conduct.

He thought, however, that after the disease had developed, when family, friends, and a normal environment had proved powerless to influence the disease and the patient was sent to the hospital, his condition was more susceptible of improvement than is generally thought to be possible. He had not infrequently found the paranoiac to be rendered

decidedly more manageable and his life made far more comfortable by regular friendly and explanatory talks, answering his questions, making the endeavor to set him right, and satisfying such of his minor demands as were not wholly unreasonable. The fact that many of them are hopeless and cannot be reached at all by such means—in fact only become worse in consequence—accounted, he thought, for the tendency that exists to pay them as a class little or no systematic attention, such as Dr. Hoch adopts with his cases. Dr. Stedman questioned if the reader had not chiefly in mind the paranoid state rather than the paranoic, that symptomatic, persecutory, condition so often found in dementia præcox. If so, he was wholly in accord with his view that much might be done in the way of prophylaxis. Dr. Hoch's masterly analysis of the psychogenetic conditions in his cases showed this plainly and he believed it to be due to the fact that the morbid direction of their thought had become less impaired than in the true paranoiac. Dr. Stedman felt the same confidence that he had expressed at length several years ago, that not a few cases of this kind when recognized early by the psychiatrist while yet the patient is comparatively comfortable, may be saved from an attack by well-directed medical oversight and guidance and regulation of his habits and surroundings. He attached little importance to the menopause as a special causative factor in insanity, as individual experience and statistics seem to show quite conclusively that paranoia develops to the same extent in both sexes during the period of life in which the menopause occurs.

Dr. Charles L. Dana had been interested in Dr. Hoch's analysis, which was instructive as showing that in a certain group of cases of paranoia conditions might be improved by careful therapeutic effort. He had not been in a position to carry out this method of treatment, which could not be very successfully employed by those not connected with institutions. He agreed with Dr. Stedman as to the importance of hereditary taint in all these cases, and that a goodly proportion of paranoiacs develop in spite of careful bringing up. Few of these patients could be influenced unless they were taken in hand very early. He had been much interested in two or three cases of paranoia which illustrated that the undercurrent does not always break through in a way that particularly disturbs the mental make-up or general life of the patient. Such a case was a woman, about fifty years of age, now under his care, who was first seen by him when she was forty years old. She was married and the mother of two healthy children. About fifteen years before he first saw her she had developed delusions of a certain kind of persecution—that when she went out on the streets people made remarks about her, trying to annoy her and to injure her. She had these delusions throughout her married life and during her pregnancies. She was a good mother, however, and to most people who knew her she remained a good, kindly woman, about whose mental condition no one had suspicions except her husband, some members of her family, and Dr. Dana. She was probably preserved from a general paranoic state by the fact that she was able to stay in the house and keep away from sources of irritation. He had had under observation also a man, now forty years old, who had been engaged in business all his life. For fifteen or twenty years this patient had had similar delusions of persecution—that the police and detectives were after him and that attempts were being made to watch him. But this undercurrent delusion never broke through except in one little spot in his brain. One or two of his children developed dementia præcox at the age of sixteen. Such

very limited types of paranoia certainly lent themselves to treatment by instruction and by careful selection of environment, which was all essential. As to the general correctness of Dr. Hoch's analysis there could be no question

Dr. Maurice C. Ashley, of Middletown, N. Y., agreed with Dr. Hoch in the main, but he questioned whether the therapeutic talks with paranoiacs would accomplish very much as a curative measure. In his experience there had been no such beneficial results. He recalled one paranoiac who, for ten years, had believed that he had been giving him poison. At first the patient was inclined to retaliate, he threatened, and made definite efforts to take the life of the doctor's children. The man had some somatic symptoms which he himself attributed to the poison which he thought had been given him. He still has the delusions, but no longer attempts to execute his threats. Another patient, a woman, for eight years had believed that he had been turning an electric current upon her for the purpose of annoying her. Every argument had been used to convince her that this was impossible, but without effect. As the disease progresses the reason of such patients becomes enfeebled and less active, and while they continue to have their delusions they become accustomed to them and cease to react much to them.

Dr. William Hirsch thought that in forming a definite opinion concerning the cases analyzed by Dr. Hoch it must first be determined whether one had to deal with genuine paranoia, or with a paranoiacal state of another disease. Genuine paranoia is always a congenital and not an acquired disease, although the true paranoiacal symptoms often do not manifest themselves during the earlier part of life. But there is always a congenital condition, a constellation of mental factors, which not only predisposes to, but which necessarily develops, at some time of life, such a combination as to produce that mental condition known as paranoia. When such a point in any given case would be reached cannot be determined in advance, but we are, in most cases, able to predict the development of a true paranoia. Various conditions, such as environment, worry, etc., might have something to do with it, at least with a premature manifestation of the condition. He did not believe, however, that in any given case anything could be done to prevent the manifestation of the paranoiacal condition, even though it were recognized that the development of such a condition existed. This opinion was not based merely on theory. In his practice he had had children brought to him whose parents realized that they were a little peculiar, nothing more, but whom he recognized as abnormal individuals who in later life would become paranoiacs. In such of these cases as he had been able to follow ten or fifteen years he had found that they developed genuine paranoia in spite of all the precautions which had been taken. He had warned the mother not to let the child have any impressions which would stimulate the imagination or fancy of the child, not to let it read any fiction, to guard it against any undue emotions; all this was carried out with the greatest care. But at some time in life, generally after an unusual emotion, such as falling in love, slight business troubles—something which otherwise would be of no importance—would develop a true paranoia. A normal individual, normal from the start, would never develop paranoia. A normal individual might develop melancholia, or some other acute disease, but never paranoia. When he said one must differentiate between types he meant cases in which there was genuine paranoia and those in which there was a para-

noiacal state. The paranoiacal state might occur in a great many psychoses. He had seen such a case lately. A man of sixty years of age, a good business man, perfectly normal all his life, suddenly developed a paranoiacal condition; he had delusions and hallucinations, imagined there was a conspiracy against him, that his neighbors tried to kill him, etc. After remaining in this condition for nine months he gradually became demented. He is still living, and is suffering from a condition of general arteriosclerosis. The case could be defined as dementia senilis, but not as paranoia.

Dr. P. C. Knapp, of Boston, thought it a mistake always to regard delusions of persecution, with hallucinations of one form or another, as constituting paranoia, and that we should be guarded in speaking of such conditions as paranoiac states. He agreed entirely with Dr. Hirsch's opinion that true paranoia, while not a congenital condition, is dependent upon a congenital condition, is dependent upon a congenital mal-arrangement, so to speak, of the brain Tanzi had taken the same position, viz.: that, whereas other forms of mental disease might be spoken of as true diseases, paranoia was not a disease, but a morbid congenital state which, later in life, under the influence of various factors, might develop into typical paranoia with hallucinations and delusions. He thought that the "under-current" did not always "break through." In this connection he cited the case of a woman who for years had had a limited type of delusion. She had lived a secluded, narrow life in one of the smaller New England cities; for many years she had been active in the care of her household and family and in church work; she had been trained in the old New England habit of keen theological discussion and argument, and for many years she had had the very definite idea that she had been excommunicated from the church. In the main the idea had been suppressed, many of her church associates did not know of it, and those who did kept it secret. The idea existed for many years without going on to any real mental disturbance. Cases were not uncommon in which the delusions occupied a limited field in the consciousness and affected but little the conduct. With a true paranoiac, however, he questioned very much the real importance of any emotional stress, or of any psychical ideas as influencing materially the genesis of the disorder. They might influence the development in so far as changes in modern belief influence the character of delusions. As Dr. Hirsch had suggested, it was impossible to protect these patients from all influences that might give rise to the condition. Not infrequently delusions of persecution developed in normal individuals in connection with hallucinatory conditions having a distinctly physical basis. He had recently seen such a case, a man with well systematized delusions on an alcoholic basis, derived largely from tactile disturbances, which proved to arise from the paresthesias of a very mild alcoholic neuritis.

Dr. L. Pierce Clark was of the opinion that the cases cited by Dr. Hoch might be called paranoid states rather than typical or true paranoia. The therapeutic suggestions outlined would be of undoubted value in these paranoid states. During the past three years he had been treating several cases by analyses and talks and the method had been very advantageous. He thought the method was of little use in true paranoia as the mental state was too fixed; his experience in asylum service had proved this fact to his entire satisfaction.

Dr. Swepson J. Brooks, of Harrison, N. Y., was very glad to know of

the success Dr. Hoch had had with therapeutic talks. He had tried this plan and found it productive of results in many cases, but the patients would relapse into the old condition after being released from institutions. He presumed that Dr. Hoch had reference in his paper to simple paranoid states. The question of paranoia was a hard one to go into, and sometimes one almost concluded that paranoia and paranoid states were the same, only differing in degree. The forcing of patients to do things, as suggested by Dr. Hoch, was often neglected. He had in mind two cases in which it certainly had a very salutary effect. One case was a woman, forty-five years of age, who had delusions of persecution. She was put in a very quiet hall. She complained that she was merely brought to the place to be put in jail, that there were no sick people there, and that she would like to see some sick people. She was allowed to see some sick patients; the next morning she was convinced, and she got well. That was four years ago, and she had remained well since. The other case was of the manic-depressive type. The patient confessed after her recovery that her family physician had had to force her to take medicine, that he would stand her up against the wall and knock her head against it if she did not take the medicine, and that she believed his method did good.

Dr. Smith Ely Jelliffe said that Dr. Hoch's paper had offered glimpses into a large and but partly explored territory. To him four different trends of thought were suggested, all of which were the subjects of much investigation. In the first place, the importance of the study of the mental development of the child was emphasized. The work of Weygandt, on abnormal children; of Koch, on pathological inferiority; of Hall, in his masterly work on adolescence; and of Sommer, on character and personality, were instances in point as to the activity of these lines of investigation. As to the psychogenic origin of certain types of delusions, Dr. Jelliffe was in accord with Dr. Hoch. He spoke of the help that might come from the literary side, as evidenced by the stories of Henry James, "The Turning of the Screw," and the "Two Magics;" Weir Mitchell's "Constance Trescott," and Ansty's "Statement of Stella Maberly." In all these this type of delusion formation is beautifully brought out, with great literary charm, if not with scientific pedantry. Therapeutically, he deemed Dr. Hoch's paper as stimulating, and he himself regarded certain phases of the subject with optimism. Paranoia, he said, was too large a term to use in a general blanket manner. While it is true that little can be accomplished by the most tactful of psychotherapeutic conversations in chronic lunatics who have been in the asylums for years, yet the important factor in the whole problem is to recognize the beginning stages, before the delusional ideas have become too firmly crystallized. Greater success had not been attained because the psychogenic origin of many delusional states had not been sufficiently understood. It required a rare tact to work on these patients, and the outlines given by Dubois, Dejerine and Oppenheim were but the beginnings of a scientific psychotherapy which for some time had been grasped at by pseudo-scientists. Dr. Jelliffe desired to rank himself with those who saw a hopeful outlook for the amelioration, if not cure, of certain cases of dementia præcox, and of the paranoid states, by early and intelligent psychotherapy.

Dr. George H. Kirby had been interested of late in the management of paranoic states along the lines suggested by Dr. Hoch, and thought that much could be accomplished in this way toward the correction of morbid

trends. Dr. Hoch's work was particularly important in regard to the study of delusions in general. Such a method of analysis opened the way to an understanding of certain mechanisms which heretofore had been practically inaccessible.

Dr. Hoch, in closing the discussion, stated once more than what he wished to bring out was the fact that certain paranoic states were produced by purely mental causes; *i. e.*, by conflicts and unhygienic ways of dealing with them, and that they were more or less amenable to treatment early in the course, but that naturally he did not mean to claim that old cases of paranoia could thus be influenced. It was necessary in order to help such cases that one should still get at the root of things and explain to the patient the genesis of his delusions and train him to healthy mental habits. The criticism that his cases were not cases of typical paranoia, he could not quite understand, because he was unable to see where the line could be drawn between cases such as his and cases of so-called typical paranoia. Again, to say that paranoia was caused by heredity was an exceedingly unsatisfactory way of stating the situation because it did not mean enough. He had claimed that some paranoic states were due to an unhealthy dealing with conflicts. Such an unhealthy dealing may be due to tendencies which were more or less inherited, but it was time to make an attempt at determining what these tendencies were, because the mere statement of heredity was absolutely barren, that the same may be said about the statement which has been made that paranoia was due to a congenital mal-arrangement. If Dr. Hirsch said that a normal individual would not develop paranoia, this was doubtless true, if, by normal individual was meant one who had perfectly healthy mental habits.

Periscope

Deutsche Zeitschrift für Nervenheilkunde.

(Band 29. Heft. 1-2.)

1. Experimental Investigations Upon the Anatomy of Traumatic Degeneration and Regeneration of the Spinal Cord. FICKLER.
2. Investigations Upon the Sense of Vibration, and Its Clinical Significance. STERLING.
3. Paralysis of the Abdominal Muscles in Anterior Acute Poliomyelitis in Childhood. IBRAHIM and HERMANN.
4. The Differential Diagnosis Between Tumors of the Cerebellum and Chronic Hydrocephalus, with a Contribution to the Knowledge of Angioma of the Central Nervous System. FINKELNBURG.
5. Intermittent Claudication of One Arm, the Tongue and the Lungs (Dyskinesia intermittens angiosclerotica). DETERMANN.
6. Contribution to the Clinical Manifestations of Tumors of the Corpus Callosum. BREGMAN.

1. *Regeneration Spinal Cord.*—As a result of six experiments, five of which were performed upon cats, and which consisted of exposing the spinal cord and then striking a single blow upon it with sounds whose heads were of various shapes, Fickler classifies the changes as follows: First, contusion, sub-divided into (a) without simultaneous injury of the spinal column; (b) with injury of the spinal column. Second, injury with alteration of the external form; and third, the post-traumatic lesions. In the first form there is disseminated insular degeneration of the nervous parenchyma. This may lead to a spinal paralysis without injury of the spinal column, hemorrhage, or cavity formation. Fickler gives a theoretical explanation for the occurrence of this condition, which depends upon the fact that the wave of the spinal cord is somewhat slower than the wave of the spinal canal, and he believes that experiments which he made upon a decapitated calf confirm this view. The injury is, of course, caused by the violent blow of the wave in the spinal cord against the surrounding bones. The experiments showed that the injuries in these cases were greatest at the position of the contre coup. In a second group, in which direct injury is present, it is found that the changes are greatest in the direction of the blow, although there is a certain degree of reflection from the commissure. The lesion is produced chiefly as a result of compression, and varies of course in proportion to the distance from the seat of injury, and the resistance of the different tissues, the nervous tissue being the least resistant, and the blood vessels the most resistant. Only in case the central canal is torn is there a sufficient extravasation of lymph to cause much destruction. Central hemorrhages never occur unless there is injury of the nervous tissue, and only when the central veins are in direct line with the injury. Thrombosis is of very little significance, and the changes in the blood vessels seem to be capable of producing a late spinal apoplexy. An interesting feature of these experiments was the appearance in some of the spinal cords examined of undoubted evidences of

regeneration. This occurs only in the nerve fibers, never in the ganglion cells. If the ganglion cells controlling the nerve fibers are destroyed by pressure or any other cause, regeneration does not occur. The commonest source from which regeneration proceeds is the spinal ganglion nearest to the focal lesion. The fibers always require a conducting path, usually provided by the blood vessels, particularly their perivascular lymph spaces. Experiments seems to indicate that regeneration only occurs in sensory fibers from the spinal ganglia, and in the fibers uniting various segments of the spinal cord. Consequently, the number of fibers capable of regeneration is not very large. When these regenerated fibers reach the gray substance they may travel upward for a considerable distance, and even come into relation with the dendritic processes of the ganglion cells of the gray substance. The fibers of Clarke's column, however, do not appear to be capable of reaching the central neurones, and the functional result attained by regeneration is probably insignificant.

2. *Sense of Vibration.*—Sterling has made a large number of investigations upon the perception of vibration, according to the method of Gradenigo, which consists of the determination of the point at which the vibrations cease to be felt in any particular part of the body, by means of an ingenious optical arrangement attached to the vibrating forks. In eighteen cases of tabes dorsalis he found, without exception, that this sensation was more disturbed than any other. In twenty-nine cases of other forms of nervous disease he found that, in a group involving the spinal column without affecting the spinal cord, there was no disturbance in the vibratory sense, but that in the cases of paresis with some pain, but without disturbance of touch, pain, or temperature sense, and in cases of distinct paralysis and disturbance of sensation, the perception of vibration was seriously impaired. In seven cases of disease of the peripheral nerves it was found that the disturbance of the vibration sense was limited to the territory controlled by the affected nerve. Sterling then discusses the various theories regarding this form of sensation, and finally states that personally he believes that the perception of vibration is common to all tissues in which the terminations of sensory nerves are found. The physical qualities of tissues have influence in so far that the denser tissues transmit better the vibrations. The skin is not inferior in this perception to the other tissues. He calls attention to the two conditions suggested by Goldscheider; first, disturbance of the sensation of touch in the skin, without loss of the perception of vibration; and second, the preservation of the sense of touch, with loss of the perception of vibration. Goldscheider explains this by the preservation of the vibratory sense in the deeper layers in the first instance, and its loss, in the second, but Sterling quotes some cases in which these conditions obviously did not exist. He does not believe, therefore, that we are capable by this method of determining directly the condition of the deep sensation. Although the subject is at present not clear, he believes firmly in the clinical value of the investigation of the vibratory perceptive phenomena.

3. *Abdominal Paralysis in Anterior Poliomyelitis.*—Ibrahim and Hermann report four cases of undoubted anterio-poliomyelitis. In all of them there was paralysis of the muscles of the abdomen; in two unilateral, and in two bilateral, but unequal. As in similar cases, the muscles of the back were also affected, in addition to muscles of the limbs. In all the children the rectus abdominalis appeared to be practically intact, the oblique muscles

being the ones chiefly affected. The symptoms consist in the bulging of the abdomen at the site of the paralysis. There is no deviation of the umbilicus toward the sound side. The abdominal reflex was usually, but not invariably, lost in all three segments. In two of the cases there were distinct signs of spontaneous improvement. In conclusion they report a case of spina bifida, and meningo-myelocele in which there was also paresis of the muscles.

4. *Tumors of Cerebellum and Hydrocephalus.*—Finkelnburg reports three cases of intracranial disease, the first in a child of fourteen, who had had symptoms for 2¼ years. They commenced with headache, then vertigo, staggering gait, diplopia, difficulty in urination, paresthesia in the back, hallucinations of sensation in the extremities, and finally, impairment of memory, state of excitement, and pains in the back of the head and neck. There were choked disc, left-sided paralysis of the abducens. The reflexes were normal. At the autopsy a moderate hydrocephalus was found. The infundibulum had been forced forward, and pressed upon the trigeminal nerves and the abducens. The fourth ventricle was not distended, but in the floor was found a cavernous angioma. There were other changes of similar character, including chronic inflammation of the choroid plexus. The second case, a child of seven years, had symptoms for seven months. There were vomiting, frontal headache, staggering gait, and loss of vision. There were also choked disc, tenderness over the left posterior portion of the head, ataxia of the right arm and leg, incontinence of urine and feces; the reflexes were normal. The patient finally became blind; was unable to walk or stand: the patellar reflex was diminished; and there was an indication of Babinski. Trephining was of no avail. At the autopsy there was found a small and moderate hydrocephalus, thrusting forward the infundibulum; the meninges and the choroid plexus were normal microscopically and macroscopically. The third case, a woman of thirty-six, began with headache in the parietal region, vomiting, staggering gait, attacks of vertigo and diminution in vision. There was choked disc, particularly on the left side; tenderness to percussion in the parietal region; the tendon reflexes were active; and Schmidt's symptom was positive; that is to say, evidence of increase in the intracranial pressure by change of position. The patient died suddenly, after discharging a considerable amount of fluid from the nose. A diagnosis of cerebellar tumor was made, but at the autopsy a small circumscribed tumor was found in the right corpus striatum, projecting into the third ventricle, where it had produced a moderate degree of hydrocephalus. The essential features of these cases, according to Finkelnburg, are, first, that the cerebellar gait may be present in chronic hydrocephalus, and in tumors of the central ganglion, even in their early stages. Second, that a normal reflex activity, or even a diminished reflex activity, is not against chronic hydrocephalus. Third, that Schmidt's symptom is not characteristic for tumors of the cerebellum, but may also occur in tumors of the cerebrum. Fourth, circumscribed pressure and percussion tenderness of the skull may occur in chronic hydrocephalus, and therefore have only slight value as a local symptom. Fifth, predominant development of the choked disc upon one side is not a certain indication for the homolateral position of the tumor.

5. *Intermittent Claudication.*—A man of fifty-one had had for ten years evidence of passive congestion with pain in the left great toe. This was finally amputated with only partial relief. For some time he had suffered

also from partial intermittent claudication, involving both legs, and a somewhat similar condition in the tongue and right arm. If the patient talked for a long time the tongue became gradually stiffer and stiffer, until further conversation was impossible. From ten to fifteen minutes work with the arm caused it to become impotent, not so much on account of pain, as on account of an uncomfortable feeling of weight. Power in the right arm was less than in the left. The pulse in the dorsal arteries of the foot could not be felt. The pulse in the right arm was slightly weaker than on the left side. The pulse in the lingual arteries was weak on both sides. There was no atheroma of the peripheral arteries, but the second aortic tone was accentuated; the heart was moderately dilated; and on two occasions albumin was found in the urine. Determann discusses the few cases of intermittent claudication of the arm hitherto recorded. In the case he reports there was a family tendency to arteriosclerosis, and the patient smoked cigarettes habitually to excess.

6. *Tumors of Corpus Callosum.*—A man of thirty-eight, developed headache, pain and stiffness in the neck, frequently vomited, and occasionally had convulsions. Finally, there was paralysis of the left arm, and some diminution in vision. When examined the pupils reacted to light; the left lower facial muscle was weak; vision was impaired. All the other cranial nerves were normal. There was marked paresis of the left leg. The patellar reflex was diminished on both sides, but particularly on the left; there was no Babinski. The left upper extremity was almost completely paralysed, and there was distinct ataxia. The tendon reflexes were diminished. The abdominal and cremasteric reflexes were more active on the left. The pulse was 64 and regular. The internal organs were regular. The patient seemed to be exceedingly confused, and was usually apathetic. He died in an epileptic attack. A tumor involving the anterior and middle portions of the corpus callosum and the adjacent portions of the hemisphere, was found, which microscopically proved to be a spindle-celled sarcoma. J. SAILER (Philadelphia).

Book Reviews

THE NERVOUS SYSTEM OF VERTEBRATES. J. B. Johnston, Ph. D., Professor of Zoology in West Virginia University. P. Blakiston's Sons & Co., Philadelphia.

In such a book as the one of Johnston's there is a good deal to be said in way of satisfaction by the reader. It brings to our minds in a clear way the sum of the work which has been done upon the nervous system of the lower forms of vertebrates. The system of the nervous mechanism is shown in its simpler forms of life and the relationship displayed as an organism rises in the scale, which is an aid in the clinical work of the physician. An instance of which is the recent statement that in man there are sensory fibers in the seventh nerve. This observation can be supported by the fact that in a lower vertebrate with gill slits the seventh nerve subserves a considerable sensory function. Also in these lower forms it seems pretty definitely shown that afferent visceral sensory fibers go into the cord by way of the posterior root and end in the region of Clarke's column. This fact is presupposed in human anatomy but not proven, yet to find it so in the low forms is an aid and guide in research.

It is an aid in the proper understanding of the relationship and function of the medullary nuclei and of the sympathetic system with the central nervous system. It is the bringing together of the work on vertebrates in a manner easily referred to that makes the book of value to the medical man.

S. D. LUDLUM.

LA MÉLANCOLIE. ETUDE MÉDICALE ET PSYCHOLOGIQUE. PAR RENÉ MASSELON MÉDICIN-ADJOINT DE L'ASILE DE CLERMONT DE L'OISE. Felix Alcan, Paris.

This is a small monograph of some 284 pages, partly descriptive, partly experimental, which is pleasing, inviting, and possesses a number of features of real value. The author's desire is to elucidate the melancholic syndrome, by which he does not mean the older conception of the term melancholia, nor yet is he prepared to accept the more limited application put upon it by the followers of Kraepelin. For him there is no definite melancholia—there are a number of melancholic states which may be found in different nosological groups. Of these he would select one which is, he believes, fairly clearly outlined—one in which the melancholic state dominates the entire picture, but concerning the etiology and pathology of which we are as yet much in the dark. The analysis of this, to him a clearly delimited syndrome, is the author's task.

This analysis is largely psychological and the author depends in large part for his conclusions upon the study of the mental associations of his patients. He divides his cases into three groups, saying as he does so that it is for purposes of convenience only, and that his simple, delusional and stuporous melancholia groups are not to be interpreted in any sense as disease groups. To this broad basis he adheres throughout. It is impossible to give a complete idea of this work without outlining at considerable length the results of the author's researches. It contains much very excellent material, and is a very commendable and thoughtful modern presentation of a difficult problem.

JELLIFFE.

News and Notes

EIGHTH ANNUAL MEETING OF THE NATIONAL ASSOCIATION FOR THE STUDY OF EPILEPSY.

The eighth annual meeting of the National Association for the Study of Epilepsy will be held at the Jefferson Hotel, Richmond, Va., Oct. 24-25 next.

Addresses will be given at the first session by the Governor of Virginia, the Mayor of Richmond and the President of the Medical Association of the State of Virginia. Responses to these addresses will be made by Dr. Everett Flood, President of the Association, and by Dr. William P. Spratling, of The Craig Colony for Epileptics, Sonyea, N. Y.

Reports from all States in this country which are now caring for epileptics will be made by the Secretary.

Dr. H. M. Weeks, of Skillman, N. J., will read a paper on "The Utilization of Epileptic Labor."

Rev. J. Duncan MacNair, resident Chaplain of The Craig Colony at Sonyea, N. Y., will read a paper on "Colony Life of an Epileptic—Social and Religious."

Dr. James F. Munson, Sonyea, N. Y., "Sewage Disposal—the Construction and Work Done by the Filter Beds at Sonyea, N. Y."

Dr. J. S. De Jarnette, Staunton, Va., "Epilepsy: Its Definition, Treatment, etc."

Dr. Thomas C. FitzSimmons, Wilkesbarre, Pa., "Alcohol as a Primary and Exciting Cause of Epilepsy."

Dr. M. B. Hodskins, Palmer, Mass., "The Etiology of Epilepsy."

Dr. William P. Spratling, Sonyea, N. Y., "The Systematic Treatment of Epilepsy Versus Its Treatment by an Occasional Consultation and Prescription."

Dr. J. Allison Hodges, Richmond, Va., "The Value of Elimination in the Treatment of Epilepsy."

Dr. William T. Shanahan, Sonyea, N. Y., "Pulmonary Edema as a Complication of Epileptic Seizures."

Dr. Matthew Woods, Philadelphia, Pa., "Surgery as a Therapeutic Measure in the Cure of Epilepsy."

Dr. H. H. Levy, Richmond, Va., "Surgical Intervention in the Treatment of Epilepsy."

Dr. A. V. Cooper, Palmer, Mass., "Injuries to Epileptics."

Dr. Edward A. Kennedy, Palmer, Mass., "Myclonus Epilepsy."

Dr. L. Pierce Clark, New York City, "Cranial Nerve Fits."

Dr. D. D. Wilcox, Richmond, Va., "The Relation of Eye Defects to Epilepsy."

Dr. G. Kirby Collier, Sonyea, N. Y., "Some Features of the Epileptic Aura."

Dr. James F. Munson, Sonyea, N. Y., "The Heart's Action Preceding the Seizure."

All persons interested in the study of epilepsy, the care and treatment of epileptics, are cordially invited to attend the meetings of the Association. The day following the meeting in Richmond the Association will hold a session at the "Innside Inn" at Jamestown.

Persons desiring to join the Association should write Dr. James F. Munson, Secretary-Treasurer, Sonyea, N. Y., or Dr. William F. Drewry, Chairman Executive Committee, State Hospital, Petersburg, Va.

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Original Articles

A CASE OF APHASIA, BOTH "MOTOR" AND "SENSORY," WITH INTEGRITY OF THE LEFT THIRD FRONTAL CONVOLUTION: LESION IN THE LENTICULAR ZONE AND INFERIOR LONGITUDINAL FASCICULUS.*

BY F. X. DERCUM, M.D.,

PROFESSOR OF NERVOUS AND MENTAL DISEASE, JEFFERSON MEDICAL COLLEGE; NEUROLOGIST TO THE PHILADELPHIA HOSPITAL.

A new and all-absorbing interest has been given to the subject of aphasia by the views lately advanced by Pierre Marie. As revealed in his recent publications, Marie maintains that aphasia has been fundamentally misinterpreted and that this has resulted from purely theoretical considerations. Many writers indeed have taken as their point of departure complicated diagrams and schemes and have drawn from these long series of deductions. The results are just what might have been anticipated, for the prevailing view of aphasia is a doctrine essentially theoretical and schematic and which has been carried to such extremes as to find itself totally at variance with observed facts. To Marie the problem presented by aphasia is that of a disturbance of the intelligence, a disturbance which constitutes a special defect for the comprehension of language. Marie's views are absolutely opposed to the classical doctrine of aphasia. He denies that there exists in the left hemisphere an auditory verbal center in the foot or posterior half of the left first temporal convolution; he denies that there exists a visual verbal center in the left angular gyrus; he denies that there exists a

*Read at the meeting of the American Neurological Association, May 7, 8 and 9, 1907.

center for writing in the foot of the left second frontal convolution: he denies that there exists in the foot of the left third frontal a motor verbal center. He insists that such an interpretation is merely schematic and theoretical and in no way justified by the facts. The theory of so-called word-deafness which is usually interpreted as a lesion in which auditory images are destroyed and in which the patient is no longer able to receive words in his auditory center and no longer capable of comparing new impressions with the auditory images already amassed and catalogued, he pronounces absolutely erroneous.

He points out that except in the rare cases in which noncomprehension of language is absolute, the patient can comprehend isolated words, short phrases, simple instructions, but in proportion as the phrases become lengthened, as the number of words becomes augmented and as the instructions become complicated, the comprehension of the patient fails. If, however, in testing such a patient we pronounce anew and separately each word, if we dissect the phrase into its elementary parts, if we decompose the instruction into its separate and distinct factors, we discover that the patient is again able to comprehend them. In short, that which disables or confuses the patient the most is the factor of complexity of language. Marie does not consider the zone of Wernicke as an auditory center—a so-called psycho-sensorial center—but as an intellectual center, and he insists upon the inevitable intellectual deficit which ensues when this center is destroyed. At the joint meeting of the Philadelphia and New York Neurological Societies, held in Philadelphia November 24th, 1906, in the discussion upon aphasia, I detailed the results of my studies of fourteen cases based upon the method of Marie. In all of these patients there was present an intellectual deficit. All revealed in a more or less pronounced degree an inability to carry out any but very simple instructions. Some failed absolutely, others complied, provided the instructions did not embrace more than two or possibly three factors. Only one or two could carry out instructions embracing four factors and always failed when this number was exceeded. How profound the intellectual disturbance is can readily be demonstrated when an aphasic is requested to do simple sums in arithmetic. Usually the additions are grossly incorrect and not infre-

quently the patient will begin his additions with the left-hand column, absolutely unconscious of the gross incongruity. Sometimes the intellectual deficit is glaringly revealed when the patient is instructed to perform some act with which he is long familiar and in the performance of which he has at one time been especially skillful. Thus Marie cites the case of one of his aphasics who, though sufficiently intelligent to mingle daily with his fellows and who, though formerly a chef de cuisine, proved to be utterly incapable of preparing so simple a dish as a fried egg. The patient having been taken to the kitchen and placed before the stove, was handed the necessary articles, a pan, an egg, butter, pepper and salt and was told what to do. The man hesitated for a moment and then commenced by breaking the egg very awkwardly. He emptied it into the pan without any precaution to avoid breaking the yolk, then he put some butter on top of the egg, sprinkled it with salt and pepper and then put the whole thing in the oven. Neither this gross blunder, nor the fact that the dish was absolutely unpresentable, disturbed the patient in the least.

As Marie clearly points out, the intellectual deficit in aphasia does not involve the intelligence as a whole. In general intellectual deficit may manifest itself in one of three ways: first, in the form observed in arrested cerebral development; secondly, in the form observed in dementia, as in paresis; and thirdly, in the special form seen in aphasia. Here the deficit instead of being generalized, involves merely one function or related group of functions. In other words, the deficit in aphasia is a special deficit dealing especially as Marie puts it, "with the stock of things acquired by didactic processes." Marie, it will be remembered, holds that aphasia is a unit, that it is not made up of sensory aphasia on the one hand or motor aphasia on the other, but that by lesion of the zone of Wernicke there is established an intellectual deficit for the comprehension of spoken language; that in so-called sensory aphasia or aphasia of Wernicke the lesion involves the zone of Wernicke (i. e., the supramarginal gyrus, the angular gyrus and the posterior portions of the two first temporal convolutions); that in so-called motor aphasia there is in addition to a lesion of this zone of Wernicke, also an involvement of the region of the lenticular nucleus. Lesion

of the lenticular nucleus gives rise to anarthria, therefore in so-called motor aphasia, or Broca's aphasia, we have merely ordinary Wernicke aphasia plus anarthria.

How frequently aphasia is due to softening, the result of embolism or thrombosis, it is not necessary to point out, and Marie calls attention to the difference obtaining between cortical softening on the one hand and deep softening on the other. In cortical softening the extent of territory involved depends upon the point at which the middle cerebral artery has become obstructed. If the obstruction has occurred at or in advance of the point at which the branch or branches supplying the third frontal convolution are given off, the third frontal is of necessity included, but such involvement is by no means necessary to the production of aphasia. Not a small number of cases have been reported in which the third frontal alone has been involved in the softening without the slightest speech disturbance, as note the recent cases reported by Souques, and by Marie and Moutier. Again cases are not wanting in which the third frontal escaped involvement and in which notwithstanding motor aphasia was present. Such a case has only recently been placed on record by Marie and Moutier.

More interesting still are the facts in regard to the aphasia resulting from deep softening in which the cortex escapes and in which the lenticular zone and the white matter alone are involved. The case of aphasia that I here place on record belongs to this group. The case was as follows:

The patient was a man who was admitted to the nervous ward of the Philadelphia Hospital August 20th, 1903. His age was estimated at sixty-five. The diagnosis upon admission was that of aphasia with right hemiplegia. At the time it was learned that he had had an apoplectic seizure in the year 1902, after having previously suffered from attacks of dizziness, and that another attack of dizziness, followed by impairment of speech, had occurred in 1903. Owing to the fact that the patient could neither speak nor understand spoken language and that he could not understand writing, it was subsequently impossible to obtain either a family history and past medical history or a history of the present disease from him. It was noted that the right arm was more affected than the leg. He had great difficulty in articulation and enunciation. He had a very marked anarthria. When

he attempted pantomime with the unparalyzed arm, his movements were confused.

Examined August 22nd, 1903, the diagnosis of right hemiplegia with aphasia was confirmed. The right upper limb was paralyzed and contractured, the forearm was flexed on the arm and the hand on the forearm. The right leg was paralyzed but

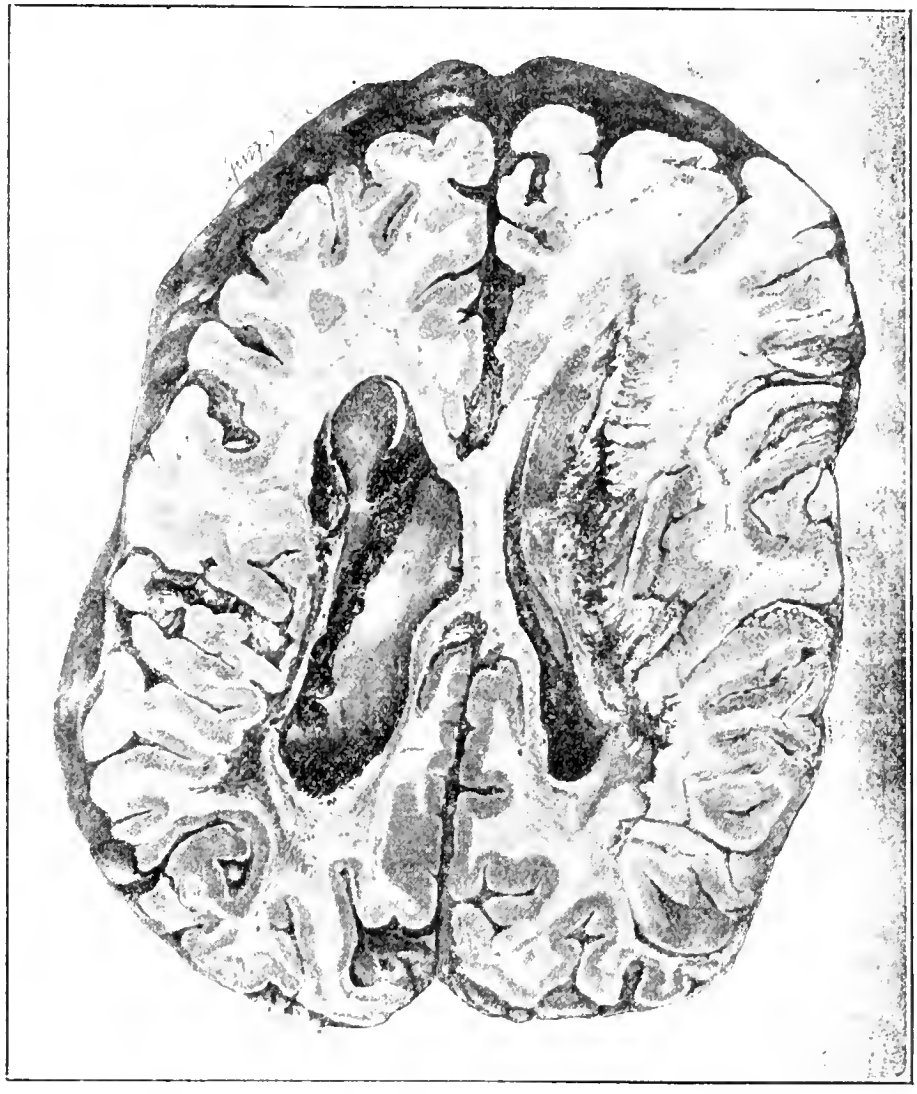

Fig. 1. Showing involvement of left lenticular nucleus and posterior longitudinal fasciculus; freedom of third frontal. Also extensive basal lesion on right side which immediately preceded death.

not completely. The right foot and toes were contractured. There was also present a paresis of the lower half of the right side of the face. The patient could wrinkle his forehead well and equally on both sides, but he could not draw up the right angle of the mouth as well as the left. He closed both eyelids well, but did not close the right eye quite as well as the left. The

biceps, triceps and wrist reflexes were exaggerated upon the right side. They were about normal upon the left. The patellar reflex was exaggerated upon both sides. Ankle clonus was not elicited upon either side; this was also true of the tendo-Achillis jerk. The Babinski reflex was not positive upon either side. No sensory losses could be determined. The pupils were equal and contracted to light. Dr. Spiller, from whose record the

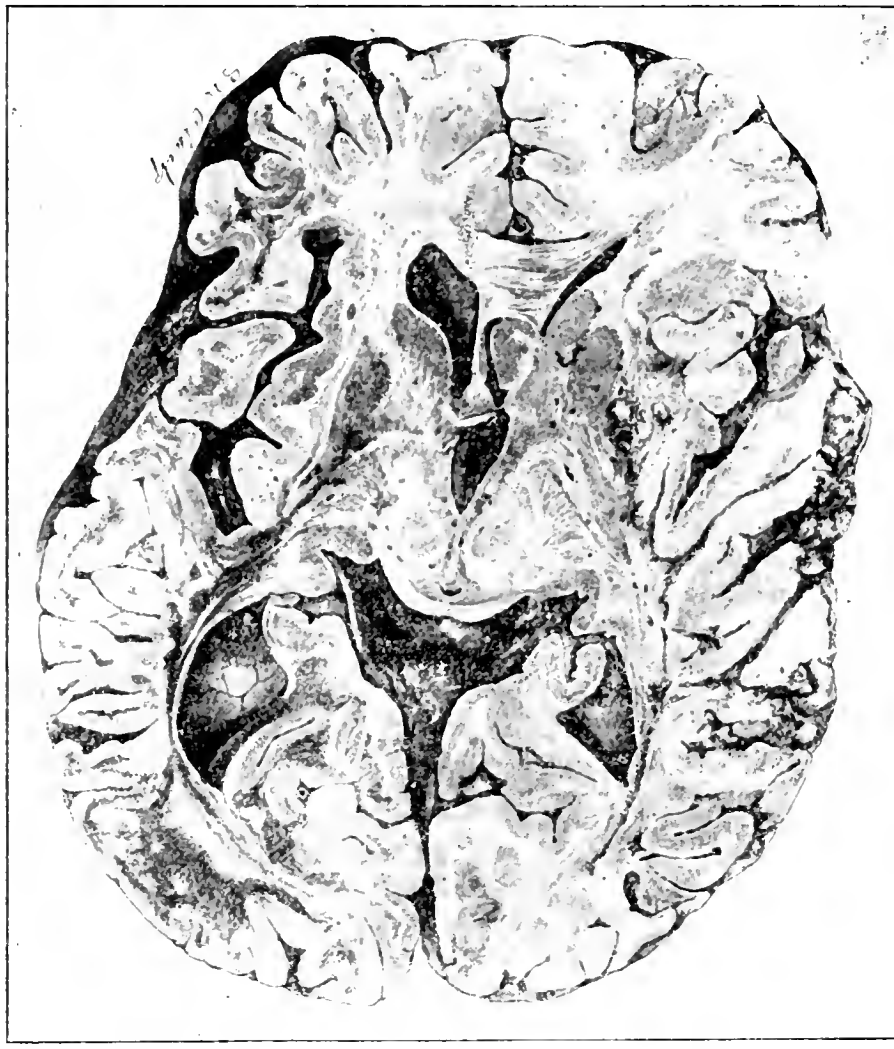

Fig. 2. Showing involvement of left lenticular nucleus and posterior longitudinal fasciculus; freedom of third frontal. Also extensive basal lesion on right side which immediately preceded death.

above facts are abstracted, also noted that the patient had complete motor aphasia and that he was probably at least partially word deaf. He seemed to be able to give merely his name.

The patient remained in the wards enjoying comparative health and remained for some years in about the same condition. Early in November, 1906, he was repeatedly

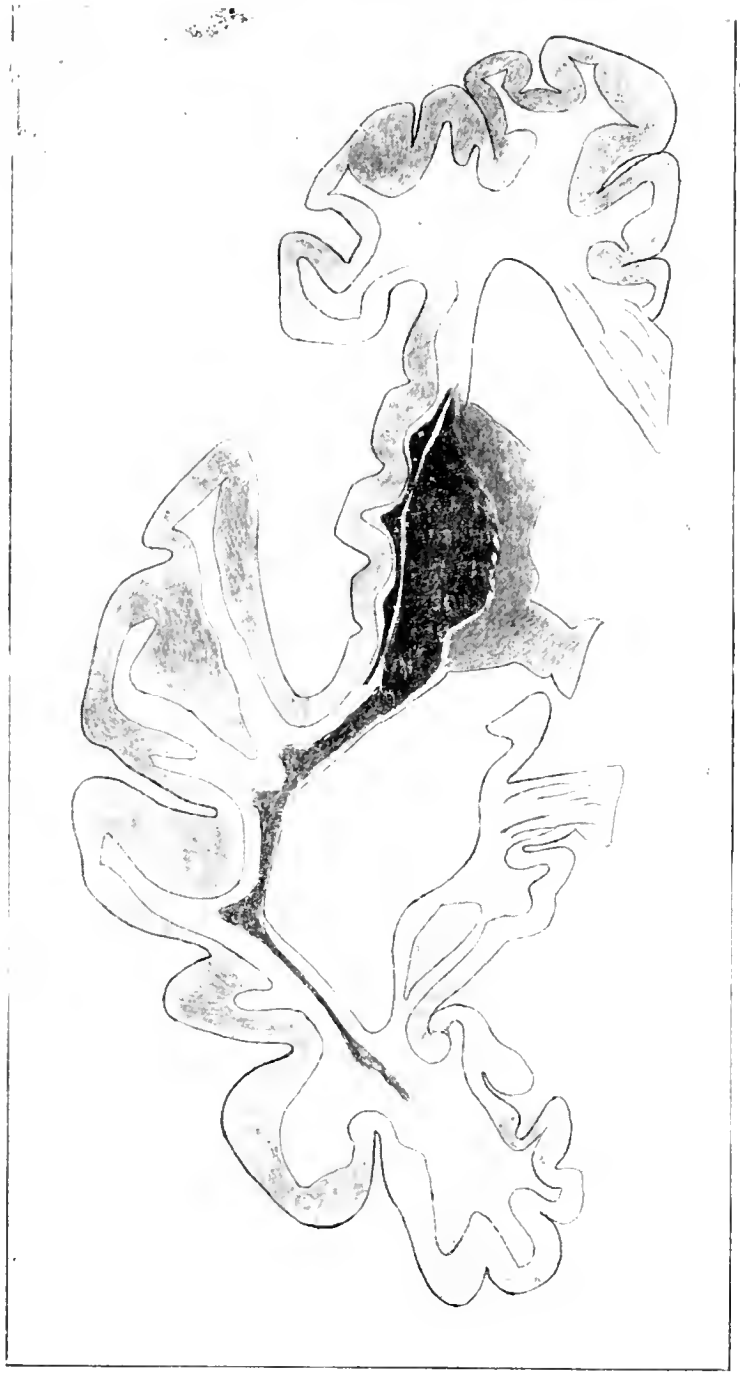

Fig. 3. Parallel Case of Marie.

Fig. 10. Horizontal section of the left hemisphere of Per . . . The softening met with here is of the type of deep softening * * * * * It is represented by the black line which extends from R. to R', it occupies the external capsule and the base of the white substance of the convolutions of the island of Reil. At R' a line of softening bifurcates and is directed forward and inward toward the internal capsule; posteriorly the softening traverses the temporo-parietal isthmus and extends into the white substance of the temporo-occipital lobe R'' where it occupies the neighborhood of the external wall of the posterior horn of the lateral ventricle and follows faithfully its contour; it abandons this wall altogether posteriorly and terminates in the white substance of the occipital lobe. This patient was one of the most beautiful aphasics of Broca which it has been given me to observe. Here the aphasia of Broca has been produced because the lesion R R' has given rise to the anarthria and the lesion R' R'' to the aphasia. (Pierre Marie.)

examined by myself. He presented, of course, as before a right-sided hemiplegia with contractures, together with aphasia. It was clearly demonstrated that he was unable to understand what was said to him, that he was unable to comprehend or to carry out the simplest instruction unless this instruction was accompanied by gestures and pantomime on my part. Without such aid he failed utterly to comprehend what was desired of him. When told "raise your left hand," he failed utterly. If, however, the command were accompanied by my own gesture, he would at once comply. Under such instruction he would raise his hand, place it upon his head or other portions of his body, but in no instance could he perform such an act when instructed verbally. He was with great difficulty able to give his name, though the sounds were barely intelligible. He could also say "yes" and "no," though he substituted a "d" for the "n" in the "no." Upon one occasion during my tests he repeated after me the word "pencil," though very imperfectly. His anarthria was very pronounced. There was present also complete alexia; neither could he write or form letters with his left or unparalyzed hand; nor could he copy letters, though the tests were limited to his own name. There was also present apraxia. Pantomime was imperfectly preserved.

On December 5th, 1906, the patient was in his usual good health, but during the night suffered from a fresh apoplectic seizure, this time affecting the left side. The left arm and left leg became completely paralyzed and flaccid. There was conjugate deviation of the eyes and of the head to the right. The other and usual symptoms of an apoplectic seizure were present; the patient was comatose, presented stertorous breathing and a rapid pulse of rather high tension. There was little change in his condition subsequently. The deviation of the head and eyes to the right became somewhat less pronounced and he became somewhat weaker. Congestion and edema of the lungs gradually made their appearance and the patient died on December 10th at 10 p. m.

Autopsy.—The results of the general pathological examination need not detain us here. They consisted in brief of adherent pericardium, hypertrophy and dilatation of the heart, fibroid myocarditis, congestion of the lungs and interstitial nephritis. Examination of the brain proved to be of great interest. There was no gross lesion of any convolution. A horizontal section passing through the basal ganglia at the level of the foot of the left third frontal convolution revealed two extensive lesions. First an old lesion upon the left side of the brain involving the lenticular zone and the inferior longitudinal fasciculus; secondly, a recent and very extensive softening upon the right side involving the basal ganglia and capsule. The lesion upon the right side was of course the one which immediately preceded death; it is the

old lesion upon the left side which concerns us here. As just stated, the lesion upon the left side involves the lenticular nucleus, the adjacent portion of the internal capsule and can also be traced far back along the inferior longitudinal fasciculus. The third frontal convolution is absolutely intact, as is also its subjacent white matter. This is also true of the convolutions of the regions of Wernicke and of the angular gyrus. This interesting case is almost an exact parallel of the case described by Marie in his article in the *Semaine Médicale*, May 23rd, 1906, and illustrated by Figure 10. This figure is here reproduced. Marie says of his case that the patient presented one of the most beautiful aphasias of Broca—motor aphasia—which it had ever been given him to observe.

The question arises, how can such a lesion as is here described give rise to aphasia—to motor aphasia or the anarthria of Marie upon the one hand and to sensory aphasia or aphasia of Wernicke on the other? The lesion of the lenticular zone readily explains according to Marie's view the anarthria. It would seem further that the degeneration of the posterior longitudinal fasciculus produces sensory or Wernicke aphasia by isolating the zone of Wernicke and secondly the degeneration of this fasciculus also produces alexia because it cuts off all communication between the zone of Wernicke and the visual centers. To me this case has been one of great interest for it is most suggestive. We know very little of the function of the striated body. It is a structure which is persistent throughout all the vertebrate forms and in the lower vertebrates, as is well known, it constitutes all of the cerebrum. Even when we ascend the scale as far as birds we find that in them the pallium,—the part corresponding to our cortex,—is still very rudimentary and that the striated bodies must carry on the cerebral function. In man the striated body has retained so great a size that it cannot possibly be regarded as a rudimentary or vestigial organ. In spite of the enormous development of the pallium, this organ has persisted in the higher vertebrates to such a degree as to necessitate the inference that it has most important functions. That the striated body should be concerned in speech presents nothing inherently improbable. While I do not mean to compare the speech of the parrot with the speech of man, the facts justify the inference that the parrot talks with his striated body; this view has also been expressed by Kalischer. Unfortunately physiologists have thus

far given us but little information as to the function of the striated body: it is possible that the propulsive movements of Majendie, the circus movements of Nothnagel have something to do with movements of co-ordination. However, the facts of the special disturbances claimed by Majendie and by Nothnagel have been questioned and denied and we are of necessity forced to draw our inferences from pathological and clinical evidence.

The function performed by the lenticular nucleus appears to be one of co-ordination of complex muscular movements, and motor speech is pre-eminently a function requiring the co-ordination of complex movements; e. g., the movements of the tongue, palate, lips, larynx and of the muscles concerned in expiration. A derangement of this intricate co-ordination means of necessity anarthria, means of necessity the impossibility of speech enunciation. Just in proportion as the substance of the lenticula is destroyed, so must there be an absence of motor speech: just in proportion as the function of the lenticula is deranged, so must there be present an anarthria.

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DISPENSARY WORK IN NERVOUS AND MENTAL DISEASES.[1] [2]

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A yearly statistical résumé of the work of the Department of Nevous Diseases of the Vanderbilt Clinic presents a number of interesting features. These reports have now been prepared for the years 1902, 1903, 1904, 1905 and 1906, and already the facts brought together are of value in generalization.

The social economic side of disease has yet to be studied. It has been attempted in only a sporadic fashion. In addition to the purely medical aspects of the question, these statistics are of service from the social economic point of view—a standpoint which was emphasized in a previous résumé.

During the year 1906 the patients seeking help in the nervous department were 2,308. Of these 2,141 were examined and their histories recorded in the case books. The number of patients was practically the same as for 1905.

The entire Vanderbilt Clinic population as given me by Mr. J. V. Colgan, in charge, is as follows:

	New patients.	Visits made.
Nervous		
Orthopedic	1,256	7,442
Surgical	4,564	21,886
Medical and applied therapeutics	14,620	44,305
Gynecological	2,813	10,064
Pediatric	4,233	11,362
Ophthalmological	5,018	15,848

[1] Report of Clinic of Nervous and Mental Diseases of Professor M. Allen Starr for the year 1906. Fifth Annual Report. For previous reports see Journal of Nervous and Mental Disease, 1903, 1904, 1905 and 1906.

[2] Members of Clinic Staff, 1906: Richard H. Cunningham, Chief of Clinic; Chas. E. Atwood, B. E. Krystall, Smith Ely Jelliffe, S. P. Goodhardt, E. L. Hunt, L. P. Clark, L. S. Manson, H. R. Humphries, J. M. McEntee, Thos. P. Prout, Geo. W. Todd, J. E. Clark and Chas. D. Cleghorn.

Otological	1,766	6,574
Nose and throat	4,771	13,063
Dermatological	3,959	11,775
Genito-urinary	2,633	10,882
	47,941	163,733

During the past year the visits per person remained, as in times past, about 5.

Of the 2,141 patients whose histories are recorded in the case books, it was found that 134 (72 men and 62 women) were not suffering from any nervous disorder, while for 128 cases (67 men and 61 women) the diagnosis is not recorded, and the facts stated do not permit of the drawing of an inferential diagnosis. The present statistical presentation is limited then to the consideration of 1,879 cases of disorder of the nervous system.

The incidence of disorder of the nervous system when compared with the entire clinic population remains at about 5 per cent., a point which has been fairly constant since 1902.

In view of the fact that mental disturbances are not seen in anything like their true proportion in the population, the figure is undoubtedly below the general average of involvement of the nervous system. Of the 1,879 cases under review, 827 were men and 1,052 women.

Mental Disorders.—The statistics of 1906 bear out the general facts already noted in our previous inquiries. (See particularly Report IV. for 1905, JOURNAL OF NERVOUS AND MENTAL DISEASE, April, 1906.) The number of patients who seek relief is far below the number who need it. We here retain for the time being the subdivisions of our previous reports, and the psychoneuroses in the broad sense as used by Dubois are classified with the nervous disorders. Only 173 cases (9.2 per cent.) are included here under the mental rubric—85 men and 88 women. This is a slightly lower number than in 1905—95 and 99.

As in previous years the defective developmental patients preponderate. Thus, under IDIOCY, were classed 4 boys and 1 girl; under IMBECILITY, 35 boys and 37 girls. The line of demarcation is not sharply drawn in our histories and it were better perhaps to classify them simply as DEFECTIVES. Many more of this class appear in the general clinic statistics as a num-

ber are observed in the pediatric department. This is true particularly for the higher grade imbeciles.

Of the insanities per se, in their milder grades, for only the mild grades come to an out-patient department such as this, there is little to offer. The facilities of our out-patient department are too crowded to permit of a satisfactory detailed examination of these mental cases. This is one reason perhaps why they do not come.

No recognizable cases of TOXIC or EXHAUSTIVE psychotic states were recorded.

THE DEMENTIA PRÆCOX Syndrome was diagnosed in 24 cases, 16 men and 8 women. This number represents about 13.8 per cent. of the mental cases of the clinic. The cases would all fall in the hebephrenic group.

PARANOID STATES, allied to Dementia Præcox, or appearing as post manic-depressive delusional developments, or associated with other psychoses, are reported for 1 man and 3 women.

MANIC-DEPRESSIVE States were diagnosed in 6 men and in 13 women, while SYMPTOMATIC DEPRESSION was noted in 5 women. INVOLUTION MELANCHOLIA was noted in 1 male.

GENERAL PARESIS was diagnosed in 23 instances (13.3 per cent.). Of this number 19 were men and 4 women. There were no special features in any of the patients observed.

Record is made of 12 cases of MENOPAUSE PSYCHONEUROSES. These are probably best considered as mild anxious and depressed conditions, allied, in general, with the involution and presenile melancholic states. Most of them were of comparatively mild grade, and hardly merit being termed psychoses.

SENILE DEMENTIA is recorded once in a man.

Nervous and queer children are recorded in 5 instances, 4 girls and 1 boy.

Nervous Diseases.—Seventeen hundred and six patients, or 91.8 per cent. come under this group. The custom of previous years in the division of our patients will be followed here, although it is well recognized that hard and fast lines of demarcation between the classes are inadmissable. This is particularly true of the groups of Neurasthenia and Hysteria of the present and of previous reports. Although it is believed that the more general term Psychoneuroses is more advisable for this general

group, the arbitrary division of neurasthenia and hysteria is retained.

NEURASTHENIA is represented in the case books of 1906 in 370 instances—200 men and 170 women. The preponderance of the foreign population remains one of the striking features in this annual résumé. As in former years the neurasthenia histories are but fragmentary, as far as the records show. These patients are those who always complain of their little ailments. Inasmuch as they are recruited for the most part from the latest arrivals, particularly from the Jewish race, the influence of underfeeding, bad hygiene and strangeness should not be overlooked. Some of these patients are very ill, and are in need of hospital, rather than ambulatory treatment, but our facilities for taking care of the functional cases are grossly inadequate. The time may soon come when these very cases may receive that which many of them should receive—namely, hospital treatment—and then a large economic saving may be brought about. The present ambulatory treatment of many of this class of patients is, from some points of view, a farce, but it seems to be a necessary one with our specially developed type of institutions.[3]

HYSTERIA was diagnosed in 95 instances (5 per cent.)—12 men and 87 women. A more detailed and systematic study of the hysteria cases is very desirable. True hysteria, we are convinced, is a comparatively rare affection in the clinic population. Hysterical manifestations of various grades are very frequent. The basis of the majority of the hysteria diagnoses is that of one or more hysterical manifestations. The severer grades of hysteria were few in the year's analysis. The crowding incident to the physical construction of the clinic makes it difficult to treat this class of patients. A quiet room is needed where helpful psychotherapy may be carried on. This is impossible in the present state of the clinic's rooms.

EPILEPSY still figures largely in the yearly résumé. There were 87 males and 79 females suffering from this disorder (8.8 per cent.). The general average remains as in previous years. Provision is made for the transfer of many of these patients to Craig Colony.

[3]An excellent analysis of 6,000 cases of neurasthenia from the case records has been made by Dr. Cleghorn. *Medical Record,* March 7, 1907.

SYDENHAM'S COREA was diagnosed for 152 patients, 57 males and 95 females. The marked preponderance of girls is still striking in this, as in the figures of other years. I have been unable thus far to obtain reliable data respecting the infantile rheumatisms and the relation they bear to chorea. The query is one that I believe would prove of value to decide.

Two instances of well defined unilateral chorea were of special interest in the records. The percentage of chorea to the general nervous population for 1906 is 8.2.

TICS of one type or another were recorded as follows: Facial tics, 3 males, 6 females; spasmodic torticollis, 3 males, 1 female: habit spasms, 2 males, 6 females.

PARALYSIS AGITANS was present in 11 men and 9 women, or 20 cases in all.

PARAMYOCLONUS MULTIPLEX was diagnosed in one instance in a male.

THOMSEN'S DISEASE was found in one patient, complicated with an ophthalmoplegic migraine.[4]

...*Peripheral Nerves.*—The peripheral nerve affections were numerous in the statistics of 1906. Combining the peripheral neuralgias, neuritides, and neuroparalyses there were 324 patients—204 men and 104 women. This number coincides exactly with the admissions in 1905. The women are much fewer in this year's summary.

The line of separation between neuralgias and neuritides is difficult to draw. As in previous years, it has been made here in a general way only.

Neuralgias.—These were present in 158 patients, distributed as follows:

	Male.	Female.
General and non-localized	6	6
Cervico-occipital	8	5
Supra orbital	8	8
Trigeminal	23	40
Deltoid-Brachial	0	6
Intercostal	3	0
Lumbar	5	0

[4] *N. Y. Med. Jour.*, July 20, 1907.

Sciatic	32	6
Anterio-crural	1	0
Plantar	0	1

Neuritis in 1906 was diagnosed in 61 instances:

	Male.	Female.
Unknown causation	4	3
Alcoholic	11	1
Anterior Tibial	5	0
Ant. crural	2	0
Brachial	0	2
Lead	10	1
Median	2	0
Musculo-Spiral	0	3
Ulnar	15	0
Pressure	0	2

The PERIPHERAL PALSIES were observed in 112 patients, 6 per cent., 69 men, 43 women. It is highly probable that some of the palsies noted as peripheral were in reality central in origin, but it is at times difficult to establish a differential. As in 1905 the traumatic element causes the marked preponderance in males.

These palsies were distributed as follows:

	Male.	Female.
Erbs (birth palsy)	2	6
Brachial (mostly alcoholic and pressure)	13	9
Circumflex	5	0
Deltoid	1	0
Facial (Bell's)	23	23
Facial (nuclear)	0	1
Median	4	0
Musculo-Spiral	21	2
Ulnar	0	1
Third nerve	0	1

The *central nervous system* was primarily implicated in 235 patients (12.5 per cent.). In these the spinal cord was the chief organ involved in 96 instances. These were distributed as follows: ACUTE ANTERIOR POLIOMYELITIS was present in 30 males and 21 females. CHRONIC ANTERIOR POLIOMYELITIS was diagnoses in 3 males. Bulbar involvements were not observed dur-

ing the year. Amyotrophic Lateral Sclerosis was present in 2 men and 1 woman. Tabes was present in 35 men and in 1 woman. Friedreich's Disease was diagnosed in 1 boy and in 3 girls. Combined Scleroses were found in 1 woman. Multiple Sclerosis was diagnosed in 10 patients[5]—5 males and 5 females.

The Myelitis syndrome was obtained in 14—11 men and 3 women. Of these, syphilitic myelitis was diagnosed in 2 men, traumatic or compression myelitis in 2 men and 2 women. caisson myelitis in 1 man, meningomyelitis in 1 man; 3 cases were of undeterminate character, while in 2 men and 1 woman ataxic paraplegia is recorded, and in 1 male spastic paraplegia without fuller details.

Hematomyelia was present in 2 men. Syringomyelia in 2 men and Spinal Cord Tumor was present in 1 man.

Brain.—This was involved in 139 patients (7.4 per cent.). Epidemic Cerebro-Spinal Meningitis presented itself only in 2 males and 2 females.

Meningo-encephalitis was diagnosed in 1 woman, and Pachymeningitis was thought to be present in 1 woman. Post-otitic Meningitis was diagnosed in 1 male.

Generalized Cerebro-Spinal Syphilis was diagnosed in 12 men.

Meningeal Hemorrhage from trauma was present in 1 woman.

The Hemiplegic Syndrome was present in 63 patients—43 men and 20 women. In 50 instances there was a right hemiplegia, in 13 it was on the left side. The localizing signs were not recorded. Infantile Cerebral Palsy was present in 2 males and 6 females. Cerebral Tumor was diagnosed in 2 males and 5 females. Cerebellar tumor in 2 males. A cerebellar ataxia was diagnosed in 2 females.

Hemianopsia was recorded in 1 male and optic nerve atrophy in 1 female.

Concussion was diagnosed 8 times, Arterio-Sclerosis 23 and Senility once.

Trophoneuroses.—Of the Trophic Disorders the following are

[5]Nos. 2805, 2815, 3017, 3633, 3665 in male and 3742, 3928, 4500, 4828, 5018 in female record book of 1906.

recorded: Erythromelalgia, 1; Muscular Dystrophy, 4; Exophthalmic Goiter, 2 males, 17 females; Arthritis Deformans, 2.

Miscellany.— Acroparesthesia, 6 men, 4 women; Alcoholism, 29 men, 6 women; Flatfoot, 1; Headache, 29 men, 55 women; Hyperidrosis, 7; Fracture Skull, 1; Incontinence of Urine, v; Insolation, 1; Insomnia, 9 men 7 women; Mercurial Poisoning, 1; Night Terrors, 1; Stammerers, 9 men, 9 women; Trauma to Spine, 2; Vertigo, 2 men and 4 women.

TRAUMATIC LESION OF THE PONS AND TEGMENTUM WITH DIRECT AND RETROGRADE DEGENERATION OF THE MEDIAN FILLET AND PYRAMID, AND OF THE HOMOLATERAL OLIVE.*

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of New York.

Director Pathological Institute.

Looking back on my own training in anatomy of the nervous system, I remember keenly the frequent feeling of discouragement over the discrepancy between the schematic presentation and the actual material, which more than the schemes, is the real salvation in case of doubt and the real field for experience and work.

One of the most disastrous consequences of schematic teaching is that the student does not learn how to deal open-mindedly and with equanimity, with the many unanalyzed parts, the unclassified residuum, which after all is material worth the greatest respect as the ground on which the help from discrepancies and uncertainties is bound to come. He ignores it, or fumbles with it, but refuses to meet it calmly for what it is worth.

It is my conviction that a method obviating this difficulty is furnished by a combination of accurate drawings and glass reconstructions, with a system of interpretations by colors. In this manner, that which is plain and simple stands out excellently and yet there will be no need of suppressing that which is the hope of the future. We can give a loud interpretation to the matters which appear safe, and a more tentative one to those which are tentative. The method is one of great advantage to the worker and its results do not have to be modified to become perfectly acceptable to students.

I beg to show the successive stages of presentation with the help of A. T. Thompson's Projection-reflectoscope, in connection with a communication of a rather interesting case of lesion of the pons.

The patient was an Italian who was stabbed in the left side

*Read at the thirty-third annual meeting of the American Neurological Association, May 7, 8 and 9, 1907.

of the neck in March, 1900, and evidently sustained a fracture of the skull. He remained unconscious for weeks, passed through a period with loss of speech, inability to swallow, paralysis of the right arm and leg and partial loss of sensation. He was admitted to Manhattan State Hospital July, 1901, on account of depression and threats of revenge, etc.; the tongue protruded to the right; he could walk unassisted. No accurate status was given. The patient died December 13, 1903, with chronic enteritis, and I owe to the Manhattan State Hospital the opportunity to have the case worked up in the laboratory of the Institute.

The study of the whole brain and of serial sections showed a rather sharply outlined cutting out of nearly the entire left half of the pons. Only the deepest transverse layers are preserved. The pyramid is completely destroyed and about section 421 the tissue-defect involves the mesial fillet which was evidently cut through as well and brought to degeneration, with the exception of about one-half of the mesial fourth and a few bundles near the bend into the lateral fillet. The most posterior actual tissue-defect is seen in section 461, in the center of the left middle cerebellar arm, the most anterior in 190, *i. e.*, in the most anterior part of the pons.

The anatomical interest of the case lies in the secondary degeneration of the left mesial fillet, the so-called retrograde degeneration of the left pyramid, the degeneration of the left olive and its fleece, and the isolation of a number of bundles which are usually covered up. The series shows further a distinct defect of the left T_4 and a marked reduction of the marrow of the left temporal lobe (with remarkable integrity of the callosal radiation) and hardly any reduction of the anterior commissure.

In the glass-models many details are not entered on account of the small enlargements; but in a large model it will be possible to render the individual smaller tracts without danger of confusion. Furthermore, it is desirable, in order to avoid confusion, that a uniform color scheme be agreed upon for the demonstration of fiber tracts.

Society Proceedings

AMERICAN NEUROLOGICAL ASSOCIATION.

Held in Washington, May 7, 8 and 9, 1907.

The President, Dr. Hugh T. Patrick, in the Chair.

(*Continued from page 636.*)

TUMORS OF THE CAUDA EQUINA AND LOWER VERTEBRÆ.

By Dr. William G. Spiller.

The paper is based on the observation of nine cases of tumor of this region, seven of which were with necropsy and three with operation. The differential diagnosis is discussed between tumors of these parts and hysteria, multiple neuritis confined to the lower limbs, intrapelvic tumor, caries of the lumbar vertebræ and sacrum, lesions within the vertebral canal but external to the dura, and lesions of the conus medullaris. The few cases of tumor of the cauda equina with necropsy reported in the literature and the question of operation are considered. Tumors of this region seem to be much more serious for surgical intervention than are those higher in the vertebral column.

Dr. Starr said this paper had interested him very much. He thought these cases were extremely rare. The case Dr. Spiller referred to as under his (Dr. Starr's) care was the only one he had ever seen. It was successfully operated on and the patient recovered entirely, and used to come to the clinic for the purpose of being shown. Dr. Starr said a picture of this case was in his book, one leg being totally paralyzed and the other normal. He thought that in a later report the statement was made that he was entirely cured. Dr. Starr said he felt that one of the most important things in the diagnosis of these conditions is the very careful examination for the areas of anesthesia. When they exist they are really of more importance in the localization of the lesion than almost anything else. With regard to the possibility of the return of power of the bladder and rectum after lesions of the conus, he was rather inclined to think that such a recovery might occur, although it was in his experience exceedingly rare. One case he had on record of distinct history of injury and hemorrhage into the cauda equina with total and distinct anesthesia in the destroyed area up to the level of the second sacral segment and loss of bladder and rectal control; in that case after a time the girl appeared to recover to a considerable extent the control of the bladder and rectum, even though the anesthesia and the slight paralysis in the muscles of the feet, especially the long peronei remained. He said it is of course a very important thing to be able to make this diagnosis early. The pain in the sacrum seems to him to be one of the most valuable of the diagnostic points, referred to the periphery in part, but also occurring in and

around the sacrum, and he thought that without such a sacral pain it would be rather perilous to attempt operation.

Dr. C. L. Dana said with reference to the question of surgical operation in these cases he would like to put on record some rather better experience, perhaps, than Dr. Spiller would lead one to think likely. Last winter Dr. Woolsey operated on two cases of tumor which were not, strictly speaking, of the cauda equina, but of that region. One was a tumor beginning in the root of one of the lower sacral nerves, not originally a conus or cord tumor, although it did eventually press upon it, and it had all the beginning symptomatology of cauda tumor with the exception that there was involvement of the bladder. Dr. Woolsey cut down and removed the tumor, and it had to be dissected out from the lower part of the cord. The patient was an old man, but he made a good recovery so far as life was concerned, and he was relieved of his horrible pain and has continued relieved at the end of eight or ten months. Dr. Dana said they had another case of tumor which seemed to be caudal; it had caudal symptoms, but they were not sure whether it was that or a bony tumor pressing upon the nerve. It turned out at the operation by Dr. Woolsey that it was a sarcoma of the sacrum which had pressed upon the sacral nerves. The patient had a distinct area of anesthesia which was very characteristic and some weakness and atrophy of one leg. In this case operation was successful.

Dr. C. K. Mills said that it was true as Dr. Starr had said that tumors in the cauda equina are comparatively rare, yet he thought he had seen at least eight or ten of these if he included several clearly defined clinical cases with four or five cases in which necropsy or operation or both proved the presence of tumor. The most interesting and the most difficult diagnoses to make are in the very earliest stage from sciatica; and at any stage, except the very late stages, from intrapelvic tumor. Dr. Mills said he has notes of some half dozen cases of intrapelvic tumor which he expects some time to publish. In these for a long time (in one case for fourteen years after the onset of the symptoms) the diagnosis was extremely difficult. In each of these cases the diagnosis of spinal or cauda equinal or other organic disease, or hysteria had been made. The differentiation between these intrapelvic tumors, especially if they are soft tumors, is difficult. The intrapelvic nerves are often robust enough to function through the tumor masses, even if these are of considerable size. It is for this reason that the diagnosis remains difficult and that the symptoms, although the growth involves many nerves, are referable sometimes to one or two. He said he thought the length of time of development of the growth, the commonly (though not always) unilateral character of the symptoms and the dissociation and diffusion, rather than the concentration of the symptoms, are the most helpful points, but of course an accurate statement of the diagnosis cannot be given in a few words.

Dr. J. J. Putnam referred to the apparent tenderness or pain evoked by pressing upon the nerves in one of Dr. Spiller's cases. This he believed to be due to mental association. A patient whom he had seen, with spinal tumor, used to suffer exquisite pain in the back if she turned her head very slightly, or even if she gaped. Such slight movements as these could not be supposed to drag on the ninth thoracic nerve, where the tumor lay, and the only reasonable explanation was that in order to turn the head it was necessary to set slightly the muscles of the back, and that this recalled, by association, the stronger action of these same muscles which

had so often been a real direct cause of motion and of pain. The excitability of the nerve centers related to the affected part may be so great that anything which even suggests a stimulus which had previously caused pain is enough to reproduce it.

Dr. Pierce Bailey said that in speaking of the differential diagnosis of these cases Dr. Spiller had stated that in multiple neuritis the muscles supplied by the peroneal nerves are often most affected. Dr. Bailey said he had seen a case recently with Dr. Jelliffe in which there was an unusual distribution of the paralysis. The man had a history of long and excessive use of alcohol. He had the loss of knee jerks, pains, etc., but the palsy, instead of being most pronounced in the peroneal nerves, was much more pronounced in the muscles around the hip. When the man was taken out of bed to walk he could grasp the floor with his toes, but the muscles above the knee were absolutely powerless. So that in this case there was a great increase in paralysis in the flexors of the thigh and the flexors of the leg with only partial paralysis in the muscles below the knee. If the etiology had been any less distinct in this case Dr. Bailey would have questioned very much the alcoholic neuritis origin.

Dr. Patrick said he had seen two cases of multiple neuritis affecting particularly the pelvo-femoral group of muscles. Both cases presented the characteristic symptoms of neuritis, and both patients recovered in what would ordinarily be considered a proper time for recovery from multiple neuritis.

(*To be continued.*)

JOINT MEETING OF THE

NEW YORK NEUROLOGICAL SOCIETY,

AND THE

PHILADELPHIA NEUROLOGICAL SOCIETY

Held at the Academy of Medicine, New York, March 16, 1907.

The President of the New York Neurological Society, DR. CHARLES L. DANA, in the Chair.

A CASE OF MYOCLONUS.

By Dr. L. Pierce Clark.

The patient was a Jewish boy, 15 years old; the third of a family of five children. His family and personal history was good with the exception of myoclonus in the patient's sister, which occurred when she was sixteen years old. After a severe attack of grippe, eight weeks ago, the patient noticed some fibrillary twitching in the rectus abdominis, and in the course of two or three days he developed a diaphragmatic spasm (inspiratory grunt). Lightning like fibrillary and fascicular muscle spasm was now seen in all trunk and proximal muscles of the extremities. Cold, emotional excitation and depression made the myoclonus worse, and the

same was true when efforts were made to control it. The muscles especially involved were the diaphragm, trapezii, the sternocleidomastoid, the pectorals, the latissimus dorsi, the triceps, the rectus abdominis and the sartorius. The muscle spasm was asynchronous and asymmetrical on the two sides of the body, and was outside the control of the will. Occasionally, the muscles about the angle of the mouth and eye participated in the spasm. The myoclonic movements occurred twenty to thirty times a minute. There were no hysterical stigmata.

Dr. Pearce Bailey said he had expected to show a case of paramyoclonus of the Friedreich type, but the patient was unable to be present. It was the case of a young man, twenty-one years old, who was thrown out of a wagon, sustaining a fracture of one collar bone and of three ribs on the opposite side. As a result of this injury, he developed a traumatic pneumonia and pericarditis, and about six months later, paramyoclonus was first observed. The muscular movements were of two kinds: One, a general fibrillation involving the masseters, the muscles of the upper extremity, the trunk and thigh; the other variety was a contraction of individual bundles of muscles. There was also an increased peripheral reflex excitability. The movements were entirely inco-ordinate, and the picture presented was that of typical paramyoclonus, as described by Friedreich, and apparently the result of the thoracic inflammation which in turn was the direct result of the injury.

In the case shown to the Society, Dr. Bailey said, the picture presented was an entirely different one and was presented as a distinct clinical contrast. The movements, which had followed an attack of grippe some weeks ago, were confined to the muscles of the chest and diaphragm, and from their co-ordinate and distinctly associated character he was inclined to look upon the case as one of multiple tic rather than of paramyoclonus. An interesting feature of this case was that one sister had been similarly affected.

Dr. F. X. Dercum, in discussing Dr. Clark's case, said he certainly did not regard it as a typical one of paramyoclonus, and as Dr. Bailey had suggested, it seemed more closely allied to the multiple tics.

Dr. Wharton Sinkler coincided with Dr. Dercum that the case resembled one of habit spasm or convulsive tic more than one of myoclonus multiplex.

Dr. Alfred Gordon thought the case represented a mixed form of muscular twitchings, and that the picture did not agree entirely with that of paramyoclonus. The patient presented contractions not only of the myoclonic type, but also of electric chorea of the Bergeron-Henoch type; also those of myokymia of Kny-Schultze's type.

TUMOR IN THE ANTERIOR PORTION OF THE BRAIN STIMULATING GROWTH IN THE POSTERIOR FOSSA.

By Dr. William Leszynsky.

The patient was a girl, 19 years old; single; a native of Russia, and a cigarette-roller by occupation, who was admitted to the Lebanon Hospital on April 16, 1906.

She stated that she had enjoyed good health until about six months ago, when she fell down stairs, striking her head in the right occipital region. She remained unconscious about five minutes. Severe frontal headache soon followed, and persisted, with acute exacerbations, for

several months. The headache then extended to both parietal regions, and there was occasional vomiting. Subsequently, she had severe vertigo lasting three days. Vision then began to fail, and at the end of two weeks she was blind.

Six months after the injury she was admitted to the hospital in the following condition. Examination showed a well-nourished, healthy-looking girl, weighing 130 pounds. The pupils were equal, slightly dilated, reacting slowly to light and in attempts at convergence; paresis of the left externus; vision of both eyes equal shadows at eight inches; bilateral choked disks of four diopters; no retinal hemorrhages. Slight asymmetry of the muscles of expression, with weakness on the right side; tongue protruded well, slightly tremulous and coated; speech normal; slight rigidity of the post-cervical muscles, accompanied by pain on movement of the head. Pain and tenderness on pressure and percussion over the temporal and suboccipital regions on both sides. No enlarged lymphatics. The watch was heard at eighteen inches on the left side, and at six inches on the right. Tests for smell and taste proved unreliable. The grasp was equal on both sides, but there was slight weakness of the flexors and extensors in the left upper extremity; also uncertainty in volitional movements of both hands in the finger-to-nose test, but no distinct ataxia nor loss of muscular sense. In either hand she could recognize a pocket-knife and a half-dollar coin, but she could not correctly distinguish smaller coins or keys. There was some uncertainty and unsteadiness in gait, but no titubation. No static ataxia. In both lower extremities the muscular power and resistance were preserved, but there was occasional extensor rigidity on the left side when the limb was manipulated. Both knee jerks were weak and obtained only with re-enforcement. There was spurious ankle clonus on both sides; plantar reflex was feeble, but of normal type. No objective sensory disturbances. Thoracic and abdominal organs presented no evidences of disease. Urine normal; blood, hemaglobin, 80 to 90 per cent.; 9,200 white blood cells. There was no history of syphilis.

The patient soon became totally blind, and during the following month she frequently complained of frontal headache and sharp, shooting pains beginning in the sub-occipital region and radiating through both upper extremities to the finger-tips. The knee jerks became more active, the partial astereognosis had disappeared, and the hearing became normal. Subsequently, there were frequent attacks of intense occipital headache and vomiting, with cervical rigidity and retraction of the head, and pain radiating through the arms. Tenderness and pain on pressure over the suboccipital region persisted; paresis of both external recti muscles and spontaneous horizontal nystagmus developed; the jaw jerk was well marked. Both hands and feet were cold and cyanotic; there was rigidity of the flexors and pronators in both upper extremities, more pronounced on the right side, with slightly exaggerated elbow jerks and increased mechanical and faradic irritability of muscles. Muscular power was unimpaired, and her gait and station remained normal. Passive movement of the lower extremities produced extensor and adductor rigidity. Kernig's sign was present on both sides, and paresis of the right posterior thigh group was observed. The knee jerks were equally exaggerated; right ankle clonus

well marked; spurious clonus and increased Achilles reflex on the left side, and normal plantar reflex.

In August, 1906, the patient's condition was practically unchanged, although the symptoms varied in degree from time to time. The optic neuritis receded, and atrophy developed. The cerebrospinal fluid was found normal, and the withdrawal of 30 c.c. was followed by intensified headache lasting several hours. The patient's condition became gradually worse. The diagnosis was made of brain tumor, probably located in the posterior fossa.

Operation: Bilateral occipital craniectomy was performed, and four days later the dura was opened and the posterior fossa explored, but no tumor formation was discovered. Death from exhaustion occurred on the eighth day, without any indications of infection.

Autopsy: For obvious reasons, the brain was removed through the surgical opening. A glioma about two centimeters in diameter was found in the median line in the anterior portion of the brain, originating in the cerebral substance in front of the anterior horns of the lateral ventricles, and extending posteriorly to the anterior pillars of the fornix, destroying the septum pellucidum and pressing upon the foramen of Munro. It was attached laterally to the caudate nuclei, and inferiorly just in front of the optic chiasm. The lateral ventricles were much distended with serous fluid. There was no growth in the posterior fossa.

In conclusion, Dr. Leszynsky, after discussing the indications and symptoms which led to the diagnosis of a tumor probably located in the posterior fossa, with pressure upon or involvement of the upper cervical nerves, said that in view of the autopsy findings, in all probability the pains in the arms were due to the forcing downward of the cerebellum and medulla into the foramen magnum as a result of prolonged intracranial pressure, as had recently been described by Collier.

Dr. Charles K. Mills said that some years ago he had a patient operated on by Dr. Frazier for a tumor which was supposed to be located in the prefrontal region. No tumor was revealed by the operation, but after death one was found in the cerebellar pontile angle. This patient was not seen until a very late stage of the disease, after terminal conditions had clouded the true diagnosis.

In speaking of the paralysis that occurred in the case reported by Dr. Leszynsky, Dr. Mills said that the explanation offered for this condition by Collier was probably the correct one.

Dr. William G. Spiller said the statement made by Dr. Leszynsky that the posterior horns of the lateral ventricles were very much dilated perhaps offered another explanation for the cerebellar symptoms. With such dilatation there was pressure upon the cerebellum. Dr. Spiller had reported a case in which the symptoms of cerebellar tumor had been caused by dilatation of the posterior horns of the lateral ventricles.

He was much in doubt as to the significance of pain in the upper limbs in cases of brain tumor. He could recall two such cases. In one of them, which came to autopsy, the tumor extended down on the cord several segments. In the other case, the patient was still alive.

Dr. Dercum, in discussing Dr. Leszynsky's case, referred to a case of supposed cerebral tumor in which operation was performed and

nothing was found. At the autopsy, a large tumor was found in the cerebellum.

A CASE OF LANDOUZY-DEJERINE FORM OF MYOPATHY.

By Dr. Charles E. Atwood.

The patient was a boy, five years old, who came under observation in February, 1907. The family history was as follows: The boy's father, a salesman, had rheumatism; one paternal aunt was insane; the maternal grandmother had asthma. The patient was the second birth; the first was a miscarriage, and the third a bright and healthy girl. None of the child's relatives, so far as could be ascertained, suffered from dystrophy. The birth was reported to have been a shoulder presentation. In infancy, he was unable to suckle properly from the beginning, although he had swallowing movements, and nourishment was supplied to him from a bottle in the nipple of which a large hole was cut so that the milk could flow more readily. He had had whooping cough, chicken-pox, summer complaint and tonsillitis; he also had adenoids and was tongue-tied, and operations were performed on the tongue and to remove the tonsils and adenoids.

Dr. Atwood said the nature of the child's disease gave it an appearance of mental deficiency, which was, however, probably apparent only. He was very timid, and it had been rather difficult to examine him. There seemed to be no disturbance of the special senses or of general sensibility, excepting that hearing was somewhat diminished. All the cranial nerves were intact. The muscles of the eyeball were somewhat affected (exophoria, etc.), but not those of mastication or deglutition, nor the laryngeal muscles. The boy began to walk at the age of fourteen months and made the ordinary movements of all the limbs, but the mother had never noticed any movements of the facial muscles excepting lately. There was very little involvement of the tongue. He could make the associated lip movements sufficient to permit "troughing" of the tongue. There was no fibrillation to be found anywhere; also no pain; no vasomotor disturbance; no skin or bone trophic disorder; the sphincters were normal. The mechanical irritability in the affected muscles was less than normal, and their faradic and galvanic excitability, as far as they could be tested, seemed to be diminished, but there appeared to be no R.D. The knee jerks were absent.

The chief facial muscles affected were the zygomatics, as shown by the loss of the labiofacial fold; the risorii, levator menti and orbicularis oris, the patient being unable to whistle, to change expression, as in crying, or to articulate clearly labials and linguals; the buccinators were unable to draw back the corners of the mouth in smiling; the orbicularis palpebrarum closed the eyes incompletely, and the corrugator supercilii and the occipito-frontalis were inactive. There was no movement to puff out the cheeks, but there was some ability to compress them. The appearance of the face was somewhat analogous to that observed in myasthenia, but the muscles in which there was some activity did not become quickly exhausted. Wasting in the facial muscles was not at present demonstrable. The supra and infraspinati on each side were noticeably atrophied, and there appeared to be a slight weakness of the right sterno-cleido-mastoid and omohyoid, and

of most of the shoulder girdle muscles. The left anterior tibial and peroneal muscle groups were slightly atrophied. The left foot was in a moderate degree of varus.

The case was of interest on account of the early development of the symptoms, as shown by the lack of facial expression and inability to suckle, apparently starting as a primary congenital defect.

Dr. E. W. Scripture, who had examined Dr. Atwood's case, described in detail the defects of articulation, which were confined chiefly to the labials and linguals.

Dr. Spiller said he had seen several cases of myopathy of the Landouzy-Dejerine type in Paris, but he had never met with a very pronounced case in this country, or in Germany, or Austria. Where the paralysis is intense, the condition of the face resembles that of double facial palsy, although the differential diagnosis is not difficult to make.

Dr. Gordon in discussing Dr. Atwood's case, said that it did not impress him as being a muscular affection of Landouzy-Dejerine type. The patient also presents some features of the pseudo-hypertrophic variety of myopathy, as can be seen from his gait and his manner of rising from the floor. Moreover, in the facio-scapulo-humeral type the upper part of the face is usually not involved at all, at least to the extent seen in the patient. It appears to be a case of general myopathy with predominance of muscular atrophy in certain portions of the body.

Dr. John K. Mitchell criticised Dr. Gordon's statement that the patient rose from a recumbent posture in a typical manner, and said that he did not "climb up his thighs," but simply touched one hand to one thigh for a moment, after he was nearly erect.

Dr. Mitchell did not think the patient walked in an unusual manner, but only like a person not accustomed to going barefoot.

Dr. J. F. Terriberry, in discussing Dr. Atwood's case of Landouzy-Dejerine myopathy, said the case resembled in some respects a case of congenital myopathy which he had had under observation for some time, and a photograph of which he exhibited. It illustrated the three so-called types of Landouzy-Dejerine, Erb, and the leg or Leyden-Möbius type, and in addition there was weakness of the lower arm muscles and the internal rectus of the left and possibly of the right eye.

The child was born of Austrian parents who were healthy, was of good habits and free from hereditary and other shortcomings. The mother had had three pregnancies and had three living children. The patient was the youngest, the other two being healthy. The child's gestation and birth were normal. It was three years old, and had had no illness other than the present one. The mother stated that the child was very feeble and helpless at birth; it moved its extremities very little, and could not raise its head; its cry was feeble. It was breast-fed. As it grew, it gradually became stronger, and was now able to stand for a short time, but it could not walk nor raise its arms above its head.

Examination showed that the muscles of the shoulder girdle were markedly atrophied, while those of the lower leg were symmetrically hypertrophied; the forearm and thigh muscles seemed enlarged; the face was flattened; the angles of the mouth were raised but little when the child cried; the mouth was open and the lower lip everted. There was external deviation of the left and possibly of the right eye; the

pupils were normal. The knee and ankle jerks were absent. The electrical reactions were reduced, with some qualitative change, particularly in the hypertrophied muscles. There seemed to be no trouble of the special senses, and the child's mentality was apparently normal.

Dr. Terriberry said that these cases probably belong to the category of hereditary muscle disease or weakness which usually did not appear for some time after birth, the profundity of the hereditary shortcoming probably causing the early appearance of the myopathy, this early appearance being undoubtedly exceedingly rare.

A CASE OF HERPETIC INFLAMMATION OF THE GENICULATE GANGLION, WITH FACIAL PALSY AND ACOUSTIC SYMPTOMS.

By Dr. J. Ramsay Hunt.

The speaker said that at a meeting of the American Neurological Association held in Boston, June 4, 1906, he presented a new syndrome dependent upon a herpetic inflammation of the geniculate ganglion of the facial nerve. (Posterior poliomyelitis of Head and Campbell.)

The immediate proximity of this ganglion to the facial and the auditory nerves in the depths of the internal auditory canal, and their common sheath, would explain the frequency of facial and auditory symptoms in this group of cases, the inflammatory process extending to the facial and in some instances to the auditory and its terminations.

The syndrome resolved itself into three clinical groups: 1. Herpes auricularis. 2. Herpes auricularis and facial palsy. 3. Herpes auricularis, facial palsy and auditory symptoms.

The zoster zone for the geniculate was situated in the interior of the auricle (concha, external auditory canal and the tympanum). The geniculate zone was therefore intercalated between the Gasserian zone in front and that of the second and third cervical ganglia behind.

The following case was a typical one of this affection: The patient was a man, 30 years old, an upholsterer. On Saturday evening, February 16, 1907, he was seized with sharp, shooting pains in and behind the left ear. On Sunday the pains were more severe; on Monday and Tuesday the pains were very severe, and so intense at night as to prevent sleep. They were tearing and shooting in character, chiefly in the external auditory canal, but also in the occipital, temporal, and facial distributions. The throat and ear were examined on Tuesday, with negative results. About this time the auricle became somewhat swollen and reddened, and looked as if frost-bitten. It was tender, and he was unable to sleep on that side. He kept the whole left side of the face protected by a dressing, as any draught brought on an attack of pain. On the 19th of February the face was noticed to be paralyzed on the left side, and about the same time his wife noticed a few groups of white vesicles in the interior of the auricle. He had no tinnitus aurium, no vomiting or disturbance of equilibrium, and the diminution of hearing was not observed by the patient until special tests were carried out.

Status Præsens: On February 27, 1907, there was complete palsy of the 7th nerve, in all its branches, with sagging and drooping of the parts, and the patient was unable to close the left eyelid. For a few days after the onset he had an overflow of the tears (epiphoria), but

this had not been present during the past week. Taste was lost over the anterior two-thirds of the tongue on the left side.

Sensation: All sensations were preserved over the left side of the face, the left ear and in the region of the occiput and neck. On comparing the sensations on the two sides, a slight diminution was demonstrable on the left side of the entire cephalic extremity, face, head and neck, including the mucous membranes, sharply demarcated in the middle line. The corneal and conjunctival reflexes were present, but diminished on the left. The aural reflex was diminished on the left. There was no tenderness over the points of exit of the trigeminal or occipital nerves.

Herpetic symptoms: The vesicles had coalesced and formed two plaques, each situated on a reddened base and exuding serum. The smaller of these plaques was situated on the inferior border of the external auditory meatus at its entrance; the other was concealed beneath the fold of the anthelix in its superior portion. The canal and the tympanum were free. The orifice of the canal was still slightly swollen, and smaller than the right. No evidence of herpes was noted elsewhere about the face or neck. The aural examination was made by Dr. McAuliffe. In the left ear, the hearing was diminished one-half, as compared with the right side. The partial deafness was of the central type (nerve deafness). No tinnitus; drum membrane normal. The tear sensation tested on March 8, 1907 (oil of mustard) was normal, and equal on the two sides. The examination of the other cranial nerves, and the neurological examination in general, namely, reflexes, motility, sensation, etc., were negative, as were the internal organs, urine and blood.

Electrical examination, March 7, 1907: The indirect reactions of the left facial showed a slight quantitative increase to both currents. Directly applied, the responses were somewhat diminished. No qualitative changes.

Notes on the course of the disease: On March 15, 1907, the pains had gradually diminished in intensity and had practically disappeared. The most troublesome were those shooting into the external auditory canal. The 7th palsy improved very rapidly and by March 15th only a trace of it remained. (This was one month from the time of onset.) The sense of taste was completely restored. The hearing on March 8 showed only a diminution of one-quarter as compared with the normal right side, and on the 15th, hearing was equal on the two sides. Two small depressed scars marked the seat of the herpetic manifestations.

Remarks: Dr. Hunt said this group of cases was characterized by the severity and intense neuralgic character of the pains. (Pre-herpetic and post-herpetic neuralgia.) Also by the occurrence of the herpes zoster. While the pain in rheumatic facial palsy might endure for a fortnight or more, it never reached the same degree of intensity as in these cases. In many cases the zone was in the trifacial or in the occipito-cervical distribution, and not in the geniculate area.

Dr. Dercum said Dr. Hunt had so clearly analyzed these cases, and had placed the matter in so definite and unmistakable a light, that he thought there was very little to be said on the subject. Dr. Hunt had certainly established a new medical group, and one that was most suggestive.

Dr. Sinkler said the nearest approach he had ever seen to the condition described by Dr. Hunt was a case of facial palsy slightly preceded by a herpetic eruption, or rather a discrete eruption of a vesicular character on the scalp and about the auricle, and with pain in the auricle.

Dr. Gordon expressed his admiration for Dr. Hunt's new syndrome, which he thought might properly be termed "Hunt's disease."

DEMONSTRATION OF GLASS MODELS OF BRAIN LESIONS.

By Dr. Adolf Meyer.

The speaker said that one of the chief aims of a central plant, such as the Pathological Institute of the New York State Hospitals, must be to conduct research work in such a manner as to make it a help and a stimulous to the hospital physicians in their actual work, as much as an achievement in the field of disputed points, or satisfaction along the lines of more personal problems; every problem of investigation was, of course, something like an individual query, but the results would not answer their purpose unless we got them into such form that they pass readily into the fund of practical resources which our hospital physicians need in order to outgrow the routine in which they might too readily lose the best opportunities. This was by no means an easy task in that one of its fields of work which he wished to demonstrate briefly to-night. Brain anatomy has at best a verbal interest with most physicians. It becomes, therefore, especially necessary to devise means of presentation which will make the facts easy of grasp and telling, and an improvement on the ponderous volumes out of which we ourselves had to dig our knowledge of the work of the past.

The glass-models represented an idea which had occurred to many (they are, for instance, used by geologists), but which had not to his knowledge been used in brain anatomy as systematically before. In purely morphological work, with its interest in surfaces, the wax-plates have their special field; but to topographical work with its many interlacing fiber tracts, the glass-model method is so far unrivaled.

The color schemes used were more or less accidental, and determined by what Higgins' inks offered. The choice happened to coincide to quite an extent with Barker's, and with the recent work of Johnston. It was indeed attempted to come to an agreement, and to propose a scheme of colors to facilitate a certain uniformity in the presentation for students.

In the study of the brain-stem of a case of traumatic destruction of one-half of the pons, the segmental efferent or motor nerves are to be found in brick-red; the pyramids in carmine; the segmental afferent or sensory nerves in blue tinges (Higgins' true blue for V, cochlear, IX, X; indigo for the vestibular); the cerebral afferent paths, green for the mesial fillet (destroyed on one side), and violet for the trapezium and lateral fillet. The cerebellar systems appear in yellow, orange and browns; the central tegmental bundle of vermilion, etc.

In the forebrain a kindred color-scheme was used, as was shown in the two cases of reconstruction of occipital lobes, in which the geniculo-calcarine tract appears in blue, the geniculo-temporal in violet; the occipito-temporal afferent paths in vermilion, the callosum in violet, the lenticular complex in indigo, etc.

The original drawings were, of course, hard work; but together with

the series of sections and the models, they were infinitely better than long-winded descriptions for which physicians generally had neither time nor patience. The drawings were open to entries and corrections and the best field of registration of observations. There was, of course, no loop-hole for the haziness with which so many errors pass under the cover of words. One's interpretations show here their natural and indelible color.

Dr. Meyer wished to express his sincere thanks for the increasing care and accuracy with which his colleagues in the State Hospitals furnish both the brain material and the records, and trusted that the return which the Institute was able to give would be a still further stimulus; he also hoped that one or the other of the neurologists present might take an interest in these methods of brain-study and enter into co-operation with the Institute. Well defined, small enough lesions in the Marchi period, i.e., from 3-6 weeks after the onset, were so rare, and the result of their study so dependent on well organized technique, that the material should pass into skilled hands as often and as rapidly as possible, especially since a delay of more than two weeks makes a Marchi reaction with formalin specimens questionable.

DEMONSTRATION OF A NEW METHOD OF BRAIN RECONSTRUCTION.

By Dr. B. Onuf.

The method made it possible to reconstruct fiber tracts and study them in their relation to the surface or other parts of the brain. Particular advantages were offered for studying the Island of Reil, which could be fully demonstrated in its relation to the surface of the brain. It was possible, furthermore, by means of this method, to reconstruct from a series of brain sections of a given direction, say sagittal, for instance, sections in other planes, such as horizontal and vertical ones. Thus from one and the same brain, horizontal, vertical and sagittal sections from any desired level could be obtained. The results of the method could be utilized for publication when desired, which was a distinct advantage of the procedure.

Dr. Alfred Reginald Allen said the beauty and value of Dr. Meyer's work was apparent. The only suggestion he had to make was that if a whiter quality of glass were used, a glass which would not possess quite so much the property of a light filter, these wonderfully instructive preparations could be made the subject of stereoscopic photography, and in that way used as illustrations.

SOME CLINICAL OBSERVATIONS ON THE TREATMENT OF GRAVES' DISEASE.

By Dr. J. Arthur Booth.

The author offered the following conclusions:

1. Although Graves's disease is recognized by the prominence of four symptoms, namely, tachycardia, exophthalmos, goitre and tremor, this symptom-complex may be incomplete in the developmental stage of the disease, and here the other symptoms are sufficient for the diagnosis.

2. At the time of the first examination, the degree of thyroid intoxication should be noted, and the patient then placed, if possible, under one of the three clinical groups.

3. The prognosis depends upon heredity, the social position of the individual, the type of the disease and its early recognition. All these conditions being favorable, with good care and perseverance, there is a fair chance of recovery in those patients classed in the first group. The more advanced cases stand a better chance of recovery by operation, but this must not be delayed too long, lest organic changes have already set in.

4. Recent clinical and experimental data still further emphasizes the general belief that over-activity of the thyroid gland is quite sufficient to explain the appearance of the symptoms of Graves' disease, and their disappearance after operation.

5. Thyroidectomy should be the operation of choice, as giving the best results.

6. The cause of death following operation is shock and the use of a general anesthetic; therefore, this should be placed in only expert hands, and local anesthesia should be employed when possible.

7. The introduction of cytotoxic serum marks an important advance in the treatment of the disease, and perhaps may ultimately prove to be the only rational therapeutic method.

Dr. Gordon said he was astonished to hear the speaker reporting such brilliant results from thyroid treatment. He had treated quite a number of cases of exophthalmic goitre with thyroid extract, and while the patients improved for a time, the final result was disappointing. As to the question of the pathogenesis of the disease, he wished to mention that about two years ago, he had shown at a meeting of the Philadelphia Neurological Society a woman who, while apparently in perfect health, developed ptosis, which was soon followed by an internal strabismus; three or four days later she noticed a bulging of the eyes, tachycardia and enlargement of the thyroid gland. He had in mind also another in which a goitre was associated with paralysis agitans. The literature contains abundant examples of Graves' disease developed in the course of organic nervous affections.

Dr. Gordon said he regarded it as a mistake to hold the thyroid gland responsible for all the symptoms of Graves' disease. As a matter of fact, he looked upon the changes and degeneration in the thyroid as secondary, and that the primary focus of the disease was in the medulla. Dr. Booth, in his paper, made no reference to the work of Jonnesco and others, who operated on a number of these cases, removing the cervical sympathetic ganglia and the latter was followed by total disappearance of Graves' syndrome. The nervous origin of the disease is therefore also evident from the therapeutic results.

Dr. Sinkler said he had had practically no experience with the operative or serum treatment of exophthalmic goitre. He could recall a number of cases in which recovery took place under absolutely no form of therapeutics. It was a fact that a much larger percentage of those patients recovered than was generally supposed.

Dr. Allen said that apropos of the last picture shown on the screen by Dr. Booth he wished to call attention to the fact that three years ago, when he was sent to investigate the Goitre Colony in Indiana

County, Penn., out of seventy odd cases of goitre that he had examined, he had found only one among them that showed any of the usual symptoms of Graves' disease.

Dr. Booth, in closing, said the changes in the medulla to which Dr. Gordon had referred, were doubtless secondary, as are also similar changes found in other tissues of the body. Besides, no such changes are found in the earlier stages of the disease, while on the other hand there has been no death recorded without involvement of the thyroid gland.

The speaker said he had used the anti-thyroid serum in seven cases. One of these was cured, two were improved, and in the others no results had been noticed.

TOBACCO AS A CAUSE OF NERVOUS DISEASE—EXPERIMENTAL OBSERVATIONS.

By Dr. L. Pierce Clark.

1. It is fairly proven that tobacco is primarily a cardio-vascular poison.

2. Its acute toxic effects on the neuro-muscular apparatus are first as an excitant and mild convulsant; second, as a motor nerve depressant; and, finally, a paralyzant of the central and peripheral nerves of the heart and lungs.

3. Its chronic toxic effect on the nervous system (as yet so inaccurately studied), is to induce toxic congestion of the brain, spinal cord and peripheral nerves, inducing finally, in the latter, a mild type of degenerative neuritis.

4. The toxic coefficient, even in animals, under strict test, is so very variable as to vitiate many of the attempts to derive any definite conclusions of its effects on the nervous system in man.

CLINICAL OBSERVATIONS.

By Dr. B. Sachs.

In discussing this phase of the influence of tobacco on the nervous system, the speaker said the chief points which he hoped to establish were that tobacco did exert a most pernicious influence on the heart and the circulatory organs, and that it was a very important factor in the production of functional nervous disease, but that it did not play any sort of rôle in the causation of organic diseases of the nervous system, and that many of the disturbances of the nervous system were due, not to the direct influence of the tobacco upon it, but that the changes and morbid conditions were brought about indirectly through the effect that tobacco had upon the various parts of the circulatory system. Even the question as to which was the toxic element producing the harm done by smoking was not easily settled. Most of us would off-hand claim that it was due to nicotine, the alkaloid contained in the tobacco leaf, but Nessler had shown that Syrian tobacco, which was said to have a strong toxic effect, contained no nicotine whatever. Havana tobacco, the tobacco imported from Porto Rico, contained only 1.6 to 1.2 per cent. of nicotine, whereas some of the German tobacco, which was supposed to be as mild as any, contained 3.36 per cent.

In the causation of purely functional derangement, the excessive

use of tobacco was unquestionably an important factor. In neurasthenia and hypochondriasis its influence was a powerful one, although Dr. Sachs said he believed that even here the deleterious effect of tobacco was brought about in an indirect way. He had not, in his experience, seen a single case of chronic psychosis which he could conscientiously attribute to tobacco and tobacco only. If we allowed that neurasthenia, with its accompanying symptoms of insomnia, tachycardia, gastro-intestinal disturbance and occasional fainting spells may be put down as being due to the excessive use of tobacco, we have brought the heaviest charge against the pernicious use of the weed. In closing, Dr. Sachs referred to the statement that had been made that the excessive smoker not infrequently became impotent.

Dr. Dercum said that almost invariably those who used tobacco were also addicted to the use of alcohol, and he did not think it was possible to differentiate the symptoms due to the use of one from those due to the use of the other. For example, vasomotor changes, which unquestionably resulted from the use of tobacco, also resulted from the use of alcohol. The same was true of amblyopia.

Personally, Dr. Dercum said he did not believe that tobacco did very much harm in the way it was used by a large majority of people; the harm arose when it was used to excess.

Dr. J. K. Mitchell said that he had had occasion to observe a great many employees in snuff factories—persons who lived in a perfect atmosphere of tobacco, much more so than in cigar factories, where there is more or less dampness, and among that class of workmen he never saw any nervous disorders or any form of neuritis that he could fairly attribute to tobacco poisoning.

In regard to the anaphrodisiac properties of tobacco, to which Dr. Sachs had referred, Dr. Mitchell said that there is at least an established belief that it is to that end sailors affect it during their absence from port.

Dr. Spiller said he had seen a number of cases of neurasthenia in young women who were employed in tobacco factories, but he had never been able to prove that they were tobacco smokers. They were, however, in the habit of biting off the ends of the tobacco, and moistening the ends of the leaf with their lips, and occasionally they swallowed pieces of tobacco.

Dr. W. W. Hawke referred to the occurrence of convulsive tic affecting usually the upper face among tobacco smokers. He also mentioned the observation of Professor Millet, of the University of Virginia, that cigarette smoking was harmless unless the smoke was inhaled.

Dr. G. E. Price said that in case of nervous disorders which were commonly attributed to the use of tobacco, the possibility of a neurotic predisposition or heredity should not be lost sight of.

Dr. Gordon said that tobacco, in its effect, apparently had a special predilection for the acoustic nerve, as was seen from the researches of Délie (Congrés Intern. d'Otologie, 1904). Its influence on the sexual function had long been known, and a number of such cases were reported by Ségrais, (*Arch. gén. de Méd.*, 1902). Experimental researches have demonstrated this depressant action of tobacco on the generative organs: atrophy and sclerosis of the testicles and ovaries were observed in chronic intoxication.

Dr. Gordon also referred to the experiments of Ballet and Faure in 1899, who had shown that subcutaneous injections of maceration of tobacco produced epileptiform convulsions, and the speaker said he could recall a case of epilepsy in his own practice where convulsions would follow the use of tobacco. The patient was an inveterate smoker. He was forbidden to chew or smoke: every time he would resume his old habit, the convulsive attacks would increase in frequency and intensity. Rénon, in 1905, reported an important case of intermittent claudication, at the autopsy of which was found a thrombosis of the abdominal aorta. The patient was neither alcoholic nor syphilitic, but used tobacco very excessively. The reporter compares tobacco to adrenalin, which is a vaso-constrictor, and both produce arterial hypertension. In arthritic individuals, who usually smoked excessively, it is not impossible that tobacco plays a role in the production of their arterial sclerosis. Erb observed this influence of tobacco in 38 cases out of 45 of intermittent claudication.

The President, Dr. Dana, referred to the deleterious effects of the inhalation habit among boys who had become addicted to the use of cigarettes.

PHILADELPHIA NEUROLOGICAL SOCIETY.

March 26, 1907.

The President, Dr. Alfred Gordon, in the Chair.

A CASE OF ABSCESS OF THE BRAIN OF TUBERCULAR ORIGIN IN A BABOON.

By Dr. J. H. W. Rhein and Dr. Herbert Fox.

The following is a preliminary report upon a case of tuberculous abscess of the brain in a baboon, whose symptoms were studied three days before death. A future report will be made which will be in the nature of a comparative anatomical study. The brain, in large part, has been cut in serial sections, and, as the study of these sections is not quite complete, only a brief reference to the pathological anatomy will be made. The baboon appeared to be perfectly well until October 18, 1906, when some lameness in the upper and lower extremities on the right side was observed. This gradually increased, and was associated with twitching of the muscles of the left side. On November 30, 1906, Dr. Rhein examined him, and found the following condition. The right upper and lower extremities were weaker than those on the left side. The power in the left upper and lower extremities seemed to be fair. He was able to hold on to an iron bar with the fingers of the upper and lower extremities on the right side, but in withdrawing the bar it was not difficult to overcome his grasp, and the power on this side was distinctly less than that on the left. The movements of the right arm were somewhat ataxic, as observed when he made efforts to grasp the iron bar with which the examination was made. The knee jerks were increased on both sides, and appeared to be equally so. There was no evidence of facial palsy. He moved both sides of the face equally well at those times at which he expressed anger

or fear in the facial expression. The tongue seemed to be retracted equally well on both sides. Tests for hemianopsia were, of course, unsatisfactory, but he seemed to recognize readily the approach of the iron bar from both sides. There was no disturbance of the rectal or bladder functions, although at the autopsy the bladder was full. At the autopsy the brain and spinal cord, with the other organs, were examined. The dura was adherent to the left side of the brain, in the prefontal region, in the upper third, and when the brain was removed it was observed that an area of softening lay beneath this point. A small caseous mass was also observed at the base of the right lung, and beneath the diaphragm on the right side was a large abscess, partly involving the liver. The tubercle bacillus was found in the pus removed from the area of softening beneath the left cortex. A study of the brain revealed the presence of three foci of softening. The largest one was situated in the left hemisphere in the prefontal region, and extended from just beneath the cortex in the upper third of the prefontal region, downward almost to the base of the brain. This area was cylindrical in shape, and measured 2.5 centimeters in its greatest diameter. The area of softening consisted of caseated material and pus, in which the tubercle bacillus was found present. A second area of softening was found on the right side of the brain, much smaller in extent, and measuring about 1.5 centimeters in diameter. There was no pus present in this area, but it consisted of a circumscribed mass of caseous material. The apex of this area of softening was just beneath the cortex in the prefrontal region. On the right side, and in the removal of the brain the cortex was torn just above this area of caseation. This point was .5 of a centimeter in front of the central fissure, and about 1 centimeter below the superior surface of the brain.

A third area of softening was observed posteriorly in the white substance, in the parieto-occipital region, and measured about 6 millimeters in diameter. A study of the sections of the brain shows beautifully the extent of the destruction of the brain tissue. The optic thalamus, the lenticular nucleus, and the posterior limb of the internal capsule on the left side, are destroyed in part. The anterior limb of the internal capsule, in one section, is preserved, notwithstanding the fact that the optic thalamus on the same side has been, in large part, destroyed. And in spite of the fact that the posterior limb of the internal capsule has been destroyed at some levels, it is interesting to note that the degeneration in the pyramidal tract of the pons and medulla, and the crossed pyramidal tract of the spinal cord on the opposite side is not intense—although distinct. It is also very interesting to note that, in view of the severe insult to the posterior limb of the internal capsule on the left side, there was no more paralysis, for it will be remembered that there was considerable power of prehension in the right upper and lower extremities. The smallness of the frontal lobes, and the unusual development of the temporal lobes, should be noted. In looking at the illustration it will be observed that the development of the cerebral convolutions is much better than in the usual picture of the brain of the circopithecidæ, and the accompanying drawing of the ordinary brain of the circopithecidæ will serve to confirm this observation. Dr. Rhein was not prepared to discuss the question of tuberculous abscesses of the brain, either in the baboon or

in man. It is well known that tuberculous infiltrations occur in the brain substance, which may undergo cheesy degeneration. Although he had not had an opportunity, thus far, of studying the literature of the subject, his impression was that tuberculous abscesses of the brain substance itself are comparatively rare in men.

Dr. Herbert Fox said:

In addition, sections made through the brain tissue in the neighborhood of the large abscesses, showed small foci of leucocytic and epithelioid infiltration, in which areas the tubercle bacillus could be demonstrated by stain. The same sections, stained by Gram Weigert method, showed the presence of some bodies at the margins of these small collections, but those could not be identified positively as containing bacteria. This brain came from an adult in the Zoological Gardens, on October 14, 1905, which was kept in the laboratory until March 31, 1906, having been injected twice during that interval with tuberculin, and the last injection which was given on the 10th of March, 1906, was followed by a typical reaction of a healthy monkey. In the Monkey House it was perfectly well until October 18, 1906, when it began to show lameness in the upper and lower extremities on the right side. The animal was returned to the laboratory on November 23rd, and while there showed progressive lameness, with progressive twitchings of the right side, but no convulsive seizures. It died on Dec. 2, 1906.

Dr. D. J. McCarthy thought that tuberculous abscess of the brain of lower animals was a decided rarity. Tuberculosis of the brain, however, apart from tuberculous abscess is not a very exceptional condition. In a good deal of the experimental work that has been done on the lower animals, especially cows and heifers, tuberculous meningitis has not infrequently been met with as a complication. The type of tuberculosis in the lower animals is not as it is in the human being which undergoes liquefaction necrosis; and as a result of the tendency to break down in the human being an abscess of the brain of tuberculous origin is not a very infrequent condition. The tendency is for the development of a distinct granulomatous process or a fibrinous process, the latter much less frequent. In the lower animals there is a general tendency toward a condition of fibrosis. In the monkey there is more frequency of the disease in the spleen and liver than in the brain. Dr. McCarthy came across a tuberculous tumor in a heifer which developed spontaneously, in which the general structure of the tumor at the first glance and in the microscopic section was one which could easily be mistaken for endothelioma. Here again the tendency was for final organization rather than breaking down. Microscopic sections and sections stained for presence of tubercle bacillus made the diagnosis in that particular case. Another very interesting case of tuberculosis which has a distinct bearing upon certain types of tuberculous meningitis, was a case Dr. McCarthy studied a few months ago, the brain of a cow which had received inoculations of tubercle bacillus with the idea of developing antituberculous serum. While under observation she calved, or rather miscarried, and following was septic infection of the uterus and she died of tuberculous meningitis of the septic plastic type seen in the human being. There was infection not only with the tubercle bacillus, but with the staphylococcus and streptococcus. Dr. McCarthy has noticed that in tuberculous proliferation types in the human,

being in the same case where there is miliary tuberculosis but mixed septic plastic type, in those cases they complicate the abscess type of advanced pulmonary tuberculosis and that this condition is due to a mixed infection. He considers this case of Dr. Rhein's so far as the rarity is concerned, not only in the monkey but the lower animals in general, notable.

FAT CRYSTALS IN THE SPINAL CORD.

By Dr. Alfred Reginald Allen.

The spinal cord in question was removed from a young man who had suffered a crushing injury of the spinal column in the neighborhood of the ninth thoracic vertebra. There was evidently an incomplete solution of continuity at the point of injury and the patient lived over a year during which time laminectomy was performed with very poor result. The osmic acid preparations of sections above the injury show numerous crystals stained intensely black. The form is either acicular or irregular plates suggesting a parallelogram in shape. There was not found the feathery variety reported by McCarthy ("Contributions from Wm. Pepper Laboratory of Clinical Medicine," Vol. 6, 1903).

Dr. McCarthy said that as far as these crystals are concerned, they are apparently of the same type of crystals, excepting a little bit larger than those in the case he described. Like Dr. Allen's case the crystals were found only in the posterior columns and the degeneration was secondary to a tuberculous caseating tumor of the lower dorsal region (as far as he can remember). The degeneration of the posterior columns was distinctly yellow in color, or rather uniquely yellow, as far as any gross color changes he has seen in the nervous system; one might have surmised some fatty change by the reaction. There were a large number of acicular crystals superimposed upon them in more or less rosette shapes, giving the feathery appearance. That was due to the fact that the sections were not cut very thin, as in Dr. Allen's case, and Dr. McCarthy thought that would account for the difference in form. The only reference he could find was in Eichhorst, in which it was stated that in the neighborhood of certain areas of focal softening in the nervous system Eichhorst occasionally saw these fat crystals and concluded that they were a combination of fatty acids and magnesium. With the two types the causes were different. Dr. McCarthy's case was a slowly forming degeneration due to a slowly growing tuberculous tumor of the dorsal cord, whereas Dr. Allen's was a rapidly developing case. There must be chemical factors which determine the formation of these fatty crystals. The disappearance of the fat crystals in Dr. Allen's case was probably due to a different chemical combination.

Dr. W. G. Spiller said he had seen Dr. McCarthy's specimens and Dr. Allen's. He thinks it is very singular that these fat crystals should be so rare. He has never found them, though he has been on the lookout for them, and has examined a great many specimens of the spinal cord. The fact that they disappear so readily is suggestive of artefacts, and there may be something unknown at present which produces this crystallization after death.

Dr. McCarthy said he agreed with Dr. Spiller that the crystals probably were not present before death. Both of these specimens were hardened in Müller's solution.

STUDIES ON THE CHOROID PLEXUS.

By Dr. Henry H. Donaldson.

The studies reported were made by Dr. Meek at the University of Chicago, under the immediate direction of Dr. Hatai. The choroid plexus of the lateral ventricles was examined, and attention directed especially to the epithelium covering the vascular leaf, which constitutes the plexus. This epithelium is continuous with the ependyma. The impulse to the present investigation was given by the incidental observation that in the albino rat before birth, the nuclei of the epithelial cells were located near the apex, whereas in the mature plexus, they were found at the middle or towards the base of the cells. It was determined that this shift in position occurred between the second and seventh day after birth. The general morphology of the choroid plexus varies in the animal scale. The plexus is always much folded, but in some forms is also studded with villous like projections. During growth, there is comparatively little change in the size of the already formed cells, although they generally spread so that each cell at maturity covers about twice the surface which it covers at birth. In the rabbit, large globules of fat are often present in these cells. This fat is extruded from the cell, but the process does not appear to destroy the cell itself. Mr. Meek has corroborated the observations of other investigators, which show that drugs which stimulate secretion, modify the shape and structure of the choroid cells. It is inferred from this, as well as from direct observation on the flow of the cerebro-spinal fluid under the action of drugs, that these cells contribute to the formation of the cerebro-spinal fluid.

Dr. F. X. Dercum asked whether the other ependymal cells have been studied in a similar way.

Dr. Spiller stated that Mr. Hutchison, a fourth year student at the University of Pennsylvania, had made a very interesting study of a case of syringomyelia with hydrocephalus, and in the choroid plexus found vacuoles in the cells like those Dr. Donaldson described as occurring in the rabbit. Within the past week Dr. Spiller had an opportunity to study a very curious tumor. It had the structure of the choroid plexus, and was probably an epithelial growth.

Dr. McCarthy stated that two years ago he reported a very extensive tumor of the choroid plexus of a horse. He has sections from nearly two hundred choroid plexuses and has noticed these changes looking like vaculoes in the cells of the choroid plexus, and changes in the shape of choroid plexuses.

Dr. McCarthy thought Dr. Donaldson's study a very interesting and valuable one He has worked with specimens from persons dying of hydrocephalus, or tuberculosis of the choroid plexus, of which he has reported eight or ten cases already. In his own work he has taken not only the plexus of the lateral ventricles but also the plexus of the fourth ventricle. He has seen no difference in structure between those from the fourth and those from the lateral, especially as concerns the ependyma.

Dr. Spiller said Dr. Donaldson had mentioned the well known fact that the structure of the cells lining the choroid plexus is different from that of the cells lining the walls of the ventricles. In the latter the type is cylindrical. Dr. Spiller had found that in tumors growing

from the ependyma, the cells preserve their cylindrical form, whereas in tumors growing from the choroid plexus the cells are more cuboidal.

Dr. Donaldson said he thought the general opinion was that secreting cells are only those which cover the choroid proper. Dr. Donaldson said that the gentlemen had spoken of the work as his, but he wished to emphasize the fact, that it is the work of Dr. Meek, done under Dr. Hatai in Dr. Donaldson's laboratory.

DEVELOPMENT OF THE CORPUS CALLOSUM.

By Dr. G. L. Streeter.

A description of five models reconstructed from human embryos 80, 95 and 150 mm. long, showing the morphology of the corpus callosum, fornix, and anterior callosum. In the youngest stage the corpus callosum and commissure of the fornix form together a round bundle of fibers crossing in the lamina terminalis, dorsal to the fibers of the anterior commissure, as a result of the caudal migration of the fornix commissure, the corpus callosum becomes gradually separated from it and maintains its position so that its center remains dorsal to the anterior commissure. The two originally are nearly equal in bulk, but the fornix commissure early comes to a standstill in growth, while the corpus callosum continues to increase in number of fibers until it reaches the proportion formed in the adult. This transportation occurs in what may be considered as modified lamina terminalis and the so-called fifth ventricle develops as a cavity of the lamina terminalis.

PHILADELPHIA NEUROLOGICAL SOCIETY.

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The President, Dr. Alfred Gordon, in the Chair.

A CASE OF STAB WOUND OF THE NECK.

By Drs. John Mulrenan and Cyrus Fridy.

The patient, J. D. L., a boy of 17, was admitted to the Howard Hospital, in the service of Dr. John H. W. Rhein, on December 2, 1906. He came with a history that he had just been stabbed with a stiletto on the left side of his neck. He had been drinking, but was not intoxicated.

Examination revealed the presence of a small stab-wound on the left side of the neck, just above the clavicle, at about the junction of the outer and middle thirds. Shortly after his admission he was seized with a convulsion, which was limited to the left side. The eyes twitched during the entire convulsion, and there was an internal squint of the left eye. The head was drawn to the left. He remained partially unconscious after the convulsion, until the following day when he could be partially aroused.

Upon examination there seemed to be some weakness of the left arm at this time. He moved both legs freely. The tongue was protruded in a straight line. There was no asymmetry of the face, and the muscles of both sides of the face were moved equally well. He made no effort to grasp the hand of the examiner with his left hand, and did so feebly with the right hand. He was hyperesthetic on the right side of his chest and right arm, but completely analgesic in the left arm. His respirations were 32 to the minute.

On December 5th, 4 days after his admission, the mental condition had improved very much, and he was quite rational. Efforts to raise the left arm were entirely unsuccessful, but the grasp of the left hand was fairly good. There seemed to be no diminution in the power of either leg. There was marked weakness of the pectoral and biceps muscles. There seemed to be an anesthesia of the conjunctiva of the left eye. There was some disturbance of sensation in the left arm as well, but this could not be positively determined on account of the mental condition of the patient.

The eyelids were kept closed, and when forcibly opened by the examiner the eyeballs were found to be turned to the left. The left pupil was slightly larger than the right, and did not respond to light.

The right knee jerk was present, and about normal; the left knee jerk was very slight. The plantar reflexes were active. Babinski's sign was not developed.

During this examination there was a repetition of the convulsive seizures, of which he had had several since his admission. There was an irregular clonic spasm of the muscles of the left arm and leg and of the face. The eyelids were firmly closed and there was a tendency to opisthotonos. He was apparently unconscious during the convulsion. Some of these convulsive seizures, observed later, began with spasmodic twitchings of the left side of the face, and of the muscles of the shoulder girdle on the left side, becoming general. After several of the convulsions the twitchings of the left side of the face and the left shoulder-girdle muscles, persisted off and on for some hours.

On December 15th, 12 days after his admission, he had a general marked convulsion, after the introduction of the catheter, lasting for an hour and three-quarters. This began on the left side of the face, and spread rapidly, becoming general, and was tonic and clonic alternately.

On December 17th, 15 days after his admission, his grasp was equal and good on each side. He extended his left arm with almost as much power as the right. The power in the biceps muscle, however, was feeble, and he had scarcely any power in the left deltoid.

Pronation and supination of the forearm were weak, and there was hypalgesia of the left arm and of the conjunctiva of the left eye. The deltoid, the biceps, the supra and infra-spinati muscles, the pectoral group, and the latissimus dorsi, appeared weaker on the left side than on the right.

Examination of the color fields, form fields, and optic nerves was normal. The ocular movements were normal (Dr. W. C. Posey).

An irregular fever was present for the first 9 days of his sojourn in the hospital.

Lumbar puncture was made on the fourth day, with negative results.

The patient left the hospital in a few days, his condition remaining unchanged.

He was again examined on April 23, 1907, in the Out-Clinic, when he stated that there had been no recurrence of the convulsive seizures, and that he had returned to work some weeks back. On this date it was found that the left biceps muscle was markedly wasted and weak. The deltoid was also weak, but did not show much, if any, wasting. There was distinct wasting of the supra and infra-spinati muscles of the left side. In other respects the muscles of the shoulder-girdle and arm appeared to be normal.

There was a slight hypalgesia over the left deltoid.

The scar was situated just above the clavicle, and four and a quarter inches behind the sternoclavicular articulation, and measured about 13-16 of an inch in length.

An electrical examination at this time showed the presence of reactions of degeneration in the left biceps muscle, and in the infraspinatus muscle. The muscles of the remainder of the arm, shoulder, and chest responded normally.

At first the stuporous state, convulsive seizures, the rise of temperature, and paralysis of the left arm led to the suspicion that the stiletto had entered the vertebral canal, injuring the meninges and possibly the cord. The disappearance of the convulsions, and rapid clearing up of the mental condition, with the paralysis limited to the biceps, deltoid, supra and infraspinati, pectoral and latissimus dorsi muscles on the left side, were all sufficient to change the diagnosis in a few days.

The convulsions were believed to be hysterical in origin, and the cause of the paralysis, an injury to the brachial plexus.

A study of the brachial plexus shows that a single lesion, to produce paralysis of the muscles described in this case, must be at the portion of the fifth cervical nerve just before it joins the brachial plexus.

Paralysis of the upper roots of the brachial plexus arises chiefly from fracture of the clavicle, difficulties at birth, injuries about the shoulder, infantile paralyses, and wounds of the neck and shoulder.

The muscles affected in injuries of the upper roots of the brachial plexus are the deltoid, supra and infraspinatus, biceps, pectoral muscles, brachialis anticus, and the supinator longus, with wasting of the muscles, and reactions of degeneration. There is loss of power at the main joints, with inability to abduct the shoulder, and to flex the forearms. There is likewise a loss of cutaneous sensation in the areas supplied by the circumflex and certain branches of the musculo-spiral nerve.

Penetrating wounds in this region, with injury to the nerve roots forming the brachial plexus, are comparatively rare. One other case, reported by Harris of England, was that of a young girl stabbed in the neck, the blade entering at the fourth cervical spine and taking a course to the posterior border of the sternocleidomastoid muscle. Subsequently, at operation, the root of the fifth cervical nerve was found to be completely severed. There was, in this case, complete paralysis of the muscles abducting the shoulder, and inability to flex the

forearm, with wasting of the muscles, and complete reactions of degeneration.

In this connection it is interesting to read the statistics of Strauch (*Wein. med. Woch.*, Nov. 7, 1903, p. 2131) who classified stab-wounds in the spinal region as follows:

"Those from before backward, in which the weapon may go through —(1) the intervertebral disks; (2) through the body of the vertebræ, destroying them; (3) through the intervertebral foramens; (4) through the transverse foramen of one spine into the intervertebral foramen between this and that of the one below, and finally, into the spinal canal. Stab-wounds from below upward, in which the weapon may pass—(1) through the intercrural spaces; (2) between the spinous processes; (3) through the trigonum arteriæ vertebralis. The size of the wound depends upon the sex, age, and bony structure. The size of the wound between the vertebræ differs in different individuals of the same age, and on the right and left sides of the same individual. It is rare that stab-wounds of the anterior cervical region into the spinal canal are associated with injury to the large vessels of the neck. In these last injuries it is possible—(1) not to injure the membranes of the cord; (2) to involve the membranes; (3) to penetrate the membranes and injure the cord; (4) to injure the membranes, the roots, or root-fibers of the same side, but not to affect the spinal cord; (5) to injure the membranes and the roots of the other side, but not to involve the spinal cord. In neck wounds from before, in the direction of the spinal canal, it is possible for the weapon to glide along the anterior side of the spinal column on the prevertebral fascia, and to injure a nerve on the opposite side. The spinal cord becomes frequently injured in stab-wounds of the back in the thoracic region, in such a way that the external wound, and the spinal cord lesion are on opposite sides. It is possible to demonstrate the direction taken by the weapon from the appearance of the external wound."

Dr. Rhein said that this case had been admitted into the wards of the Howard Hospital in his service, and presented at first some very confusing symptoms. The patient was at first in a semi-conscious condition, and while being examined he developed a convulsion which began with twitching of the left side of the face, extending to the left shoulder muscles, and finally becoming general. During the few days after his admission he had several convulsions of this sort, and also some attacks in which only the left side of the face and the muscles of the left shoulder were involved. He was also anesthetic in the left conjunctiva, in the cervical region, and in the left arm. There was rise of temperature extending over a few days.

At first it occurred to Dr. Rhein that the knife had entered the spinal canal and injured the meninges, and possibly the spinal cord, and that the convulsive seizures were due to irritation of the meninges or cord, but the rapid return to a normal mental state, associated with anesthesia of the conjunctiva of the left eye, of the left arm and shoulder, not limited to any particular nerve distribution, and the irregularity of the convulsive seizures, justified him in the diagnosis of hysteria, added to an injury of the brachial plexus. Dr. Rhein thought that the diagnosis presented by Drs. Mulrenan and Fridy—namely, that the fifth cervical nerve was injured,—was the probable explanation of the symptoms presented by this patient.

PARALYSIS OF THE LOWER EXTREMITIES FOLLOWING A GYNECOLOGICAL OPERATION.

By Dr. J. Hendrie Lloyd.

The patient, a young white woman, had had a pelvic abscess caused by a self-produced abortion. The abscess was behind the uterus and extended up above the true pelvis far enough to produce a mass which could be felt through the abdominal walls. Dr. Salada had operated by a laparotomy, with counter-drainage through the vagina. The patient had been very ill for three months, during part of which time she had had a slight confusional delirium. There was no history of alcoholism. Before the abscess healed the patient became paralyzed. This paralysis was confined almost entirely to the muscles below the knees on both sides. Both extensors and flexors were involved; there was foot drop; lost knee jerks, and wasting of the muscles. There was also a very intense causalgia of both soles, with vaso-motor paresis, causing a contact erythema of the parts. This area was extremely sensitive, the slightest pressure causing the patient to cry out. No anesthesia. The reactions of degeneration were present in the calf and peronei muscles. There was no involvement of the bladder, nor any symptoms in trunk, arms, neck, face or eyes. Dr. Lloyd explained the case as due to a septic infection of the sacral plexus on both sides caused by extension of the inflammation from the abscess to the nerve-sheaths.

Dr. Spiller said he did not know what the difficulties of diagnosis had been, but the case seemed at present to be one of multiple neuritis. The reaction of degeneration, pain on pressure over the soles of the feet, and double foot drop are typical of multiple neuritis.

A CASE OF POST-DIPHTHERITIC MULTIPLE NEURITIS WITH VESICAL INVOLVEMENT.

By Dr. C. D. CAMP.

The speaker said the involvement of the auditory nerve and of the urinary bladder are both very rare in multiple neuritis, and he thanked Dr. W. G. Spiller for the privilege of reporting a case in which these symptoms were both present. The patient is twenty years of age. He had typhoid fever in January, 1907, and when convalescent from this was taken to the Municipal Hospital with diphtheria. At the time of leaving the Municipal Hospital he first noticed that his hearing was impaired, that his voice was changed, and that fluids regurgitated through his nose. About a week later he noticed that his hands and legs felt numb and weak, and at the same time he had incontinence of urine, "could not hold his water," soiling his clothes and his bed. An examination by Dr. Walter Roberts showed deafness of one ear to be of nerve origin. On April 1st, 1907, the voice was still weak but fluids no longer regurgitated through his nose. His gait was uncertain, ataxic, and there was a bilateral foot drop which was worse on the left side. There was a marked Romberg's sign. There was no paralysis of the face, tongue or extra-ocular muscles. There was a hypesthesia in the ulnar distribution of the left hand and the grip of both hands was weak. The knee jerks and Achilles jerks were absent and there was no Babinski reflex. There was tenderness on pressure in the muscles of the

arm and over the peroneal nerves, also on pressing the calf muscles. Examination on April 22nd showed that the deafness due to nerve involvement had remained about the same. His gait had improved slightly and the foot drop was not quite so marked. There was a paralysis of accommodation, ordinary print becoming blurred at a nearer point than twelve inches.

HERPETIC INFLAMMATION OF THE CERVICAL AND THORACIC NERVES.

By Dr. T. H. Weisenburg.

A young man of twenty-six with an excellent family history, played a rather hard game of golf. The following day he felt a little pain in the wrist of the right arm, this pain resembling that of rheumatism. About six days after this the whole hand and arm felt numb and dead, and at the same time there appeared herpetic eruption over the right shoulder and upper arm. Examination about eight days after the exposure showed that power was practically normal with the exception that he could not lift his shoulder as quickly as he should. He had also pain in the back of his head and over the right side of the face and in the right arm. The herpetic eruption extended from the shoulder along the upper border of the whole arm, this including the thumb and the first two fingers, being limited to the distribution of the fifth cervical segment. He had besides a hypesthesia for touch and pain over the whole right side of the face, right ear, the whole right side of the head and neck, this extending to a point about six inches below the angle of the scapula and in front of the chest on the nipple line, then to the middle of the chest and then to the face. The arm was wholly hypesthetic, this being more so in the distribution of the fifth cervical segment. In the course of a week the herpetic eruption disappeared and the area of hypesthesia gradually receded and in about three weeks after the appearance of the symptoms the patient was well. It is evident that the area of first inflammation was in the distribution of the fifth cervical ganglion, and the roots, both above and below, were involved. No herpetic eruptions were present excepting at the point of initial irritation. The case is rather interesting because of the large extent of sensation involved, this area including the fifth cranial nerve, all of the cervical nerves and the first six thoracic nerves, all of these being on the right side. It is also rather interesting to note that the hearing was not in any way diminished.

Dr. Lloyd asked whether Dr. Weisenburg made a puncture.

Dr. Weisenburg replied that the man got well too quickly.

ABNORMAL RESPONSE TO AN IRRITATION OF THE MOTOR AREA OF THE BRAIN.

By Dr. Alfred Gordon.

Abstract: A boy of 16 had been suffering from facial epilepsy affecting left arm, face and neck. An operation was decided upon. An osteoplastic flap uncovered the entire motor area on the right side. A very carefully applied faradic electrode (unipolar method) gave invariably a response on the right side of the body and not for one moment on the left side.

Dr. Lloyd said he thought this was really a very extraordinary observation. He has seen a good deal of faradization of the cortex and he has never seen anything like it. He thought the only explanation was that there was non-decussation of the pyramids. Such a thing is extremely rare. He asked whether there was any possibility of faradization of the membranes by which the current could have been transmitted to the opposite hemisphere. He would also like to know where the indifferent electrode was placed. Dr. Lloyd said he wished to say one word as to the technique. It seems to be the idea that it is better to faradize the ascending parietal convolution first. Dr. Lloyd does not have that idea. He has faradized the human cortex a number of times, and he inclines to the view that according to the theory of Grünbaum and Sherrington, the ascending parietal convolution exercises a reinforcing action, that is to say, if you put a rather strong current on this convolution you may get a little motor response. That is perfectly conceivable, the current may follow through the neurones. He thought under these circumstances it is better to apply the current to the ascending frontal convolution first. If you want to demonstrate that the motor neurones are entirely in front of the Rolandic fissure it is better to faradize them first. Then with an equal strength of current faradize the ascending parietal, when it will be evident that this latter convolution does not give a motor response. But if, at the very first, you use a very strong current, and faradize indiscriminately the various convolutions, you obtain overflow effects, and the results are confusing and valueless.

Dr. Spiller said that complete failure in decussation of the pyramidal tract is so uncommon that great caution should be observed in attributing symptoms to this condition.

Dr. Gordon said he had not mentioned in his talk everything he had written in the paper. He simply wanted to report an unusual response of the motor area to faradization. In answer to Dr. Lloyd's remark about first faradizing the precentral convolution and then the posterior convolution, this is entirely opposite to what Dr. Mills advises. Dr. Gordon compared very carefully the original notes of Dr. Mills and Dr. Lloyd, and Dr. Mills also advises to begin with the posterior convolution, first because he says the precentral convolution is absolutely certain to be motor, but if you start from the anterior convolution and then go to the posterior convolution, you may get some contractions in faradization of the posterior convolution, when no response would have been, perhaps, obtained had the electrode been applied first to this convolution. Dr. Lloyd, however, believes in the opposite. In regard to Dr. Spiller's query, Dr. Gordon said that in his experiments, which embrace two cases, one a normal case and this apparently abnormal case reported, he found certainly very distinct contractions in the opposite side by faradizing the precentral convolution and the sulcus, he also found them present in faradizing the postcentral convolutions, but less marked than in faradization of the precentral convolution. In regard to the possible explanation of the convolutions and the responses on the same side as the faradized side, he is really at sea to explain this occurrence. All possible precautions were taken. In his other case he found normal responses, which means faradization on one side, convulsions on the other side. At no

moment did he observe in his first case even the slightest indication of contraction or twitching on the opposite side. When the area was exposed, Dr. Da Costa found on the side exposed some blood vessels which resembled very much angiomatous veins, but the cortex itself was intact. There was no autopsy in the case, no microscopical examination, no examination of the medulla, but Dr. Gordon did not see how otherwise could be explained the response on the same side than by want of decussation. Dr. Gordon stated that he had put the indifferent pole on the chest and before he transferred the electrode to the brain he tested the strength of the current with his hands, and he obtained a slight tingling sensation and only this current he employed for the experiment. There were extremely slight movements of the fingers. The same strength of current was used in the second case where the patient had normal contractions.

Dr. J. W. McConnell asked if Dr. Gordon had ever taken notice how much less this current is perceived when it is tested on the hand than when it is tested on some more sensitive part, for instance the lip. In many of the University Hospital cases of Dr. Mills they tested on the lip, and a current that was just perceptible to the lip was the current used.

PRESERVATION OF THE SENSATION OF PRESSURE IN THE FACE AFTER DESTRUCTION OF THE TRIGEMINAL NERVE.

By Messrs. R. H. Ivy and L. W. Johnson.

This paper is concerned with the conduction of sensory impulses from the face to the sensorium, and especially the conduction of deep pressure sensibility as contrasted with that of cutaneous sensibility to light touch, temperature, and pain sense. Up to a comparatively recent period it was considered that all sensory impulses of the face were carried by way of the fifth nerve, while the seventh nerve was concerned solely in the motor supply of the muscles to which it runs. Recently, however, the opinion has been gaining ground that while the fifth nerve conveys impulses of light touch, pain, and temperature sensations from the skin of the areas of its distribution, deep or pressure sensibility is conveyed by afferent fibers running in the motor nerve to the muscles, viz., the seventh nerve, except in the case of those muscles supplied with motor fibers by the fifth nerve. To support this view two cases studied by the writers and one case by the writers in association with Dr. Spiller, are reported, in which pressure sensibility was retained in the trigeminal area of the face after lesions completely destroying the Gasserian ganglion. These investigations have an important bearing upon the surgery of the fifth nerve. It has been noticed that after operation on the Gasserian or the sensory root of the fifth nerve, some sensation has been retained in the side of the face supplied by the nerve operated upon, at times giving rise to the assumption that the operation was imperfect. The subject is important also, because after surgical intervention on the fifth nerve a certain form of sensation may be preserved, so that there is not complete anesthesia, even with complete destruction of the fifth nerve.

Conclusions: The fifth nerve conveys sensibility to light touch, pain

and changes in temperature from those parts generally recognized as being within the area of distribution of the said nerve. The seventh nerve contains fibers of deep or pressure sensibility running from the muscles which it supplies with motor fibers. The muscles of mastication which derive their motor supply from the fifth nerve, probably are also furnished with fibers of deep sensation by that nerve.

Dr. Spiller said he thought Drs. Ivy and Johnson deserved much credit for the work they had done. During one of his lectures he had invited the students of the Fourth Year Class to report this case and Drs. Ivy and Johnson had responded.

Dr. Spiller remarked that the deep sensation of the facial nerve may be temporarily interfered with by operation on the trigeminal nerve. Twisting out peripheral branches of the latter causes much swelling of the face, and probably thereby, some impairment of function of the facial nerve. Excision of the Gasserian ganglion causes swelling of the axis cylinders and meduallary sheaths of the peripheral branches of the trigeminal nerve, and if these are intimately associated with branches of the facial nerve, this swelling also may interfere temporarily with the function of the latter. Destruction of the sensory root of the trigeminal nerve by a tumor affords better opportunity for testing the sensation of the face, than does destruction of some part of this nerve by operation, at least in the period immediately following operation.

Periscope

Psychiatrisch-Neurologische Wochenschrift

(March 31, 1906.)

Some Somatic Characteristics of Idiocy. Heinrich Vogt.

The author calls attention to the fact that in general idiots are smaller, weigh less, and die younger than normal persons. They show the signs of senescence earlier and the several organs weigh less, the most important organ to weigh less being, of course, the brain.

(April 7, 1906.)

The Treatment of Alcoholic Delinquents. Otto Juliusbruger.

The author calls attention to the fact that, in general, idiots are smaller. Society has a right to protect itself from this anti-social being, but should subject him to curative rather than correctional measures.

(April 15, 1906.)

Eglfing.—An account of this institution with a picture of the lay out.

(April 22, 1906.)

Course of Medical Psychology in Relation to the Treatment and Education of the Born Defectives. Dannemann.

A short account of some of the work that is being done along these lines as set forth at a meeting of those interested in this work at Giessen, April 2 to 7.

(April 28, 1906.)

The Case of Bremke. H. Schafer.

A detailed account of a case of only local interest.

Proponal. Bresler.

Researches with New Hypnotics. Shecke.

Proponal is a colorless, crystalline solid, melting at 145°, dissolving in 70 parts boiling water and 1,640 parts water at 20°. It has a somewhat bitter taste. The formula is given as:

$$C_2H_7 \quad CO{-}NH$$
$$\qquad C \qquad\qquad CO.$$
$$C_3H_7 \quad CO{-}NH$$

The hypnotics discussed in the second article are veronal, viferral, hypnal, proponal. Nothing new occurs in the article. The author cites an old text that holds that the existence of a great many drugs for a special purpose indicate that none are of much value and counsels trying them all carefully and holding to the best.

(May 12, 1906.)

Oswald Alving. Boege. (Continued.)

(May 19, 1906.)

Ten Years of Family Care in the Province of Saxony. Conrad Alt. Oswald Alving. Boege. (Concluded.)

Family Care.—This article shows that the increase in the number of

patients cared for in families in Saxony has been from two in 1896 to 475 in 1906.

Oswald Alving.—A psychopathological study of the character of Oswald Alving in Ibsen's "Ghosts."

(May 26, 1906.)

The Opening of Roderbirken. ERNST BEYER. (Continued.)

(June 2, 1906.)

The Opening of Roderbirken. ERNST BEYER. (Concluded.)

An account of the new sanitarium for nervous diseases. While its sister sanitaria, Hans Schonow and the Provincial Sanitarium at Hanover, provide for both sexes and classes, this is for women only and for one class. It contains 145 beds.

(June 9, 1906.)

Petition of the Moravian Hospital Physicians for a Bettering of Their Conditions.—Of local interest only.

WHITE.

Revue de Psychiatrie et de Psychologie Expérimentale

(June, 1906.)

1. Conditions of Surgical Intervention in Hypochondria. PICQUÉ.
2. Eroticism in Hysteria. BUVAT.

1. *Surgical Intervention and Hypochondria.*—The author of this article is a surgeon, and while he does not pretend to be able to solve the problem of hypochondria from a psychiatric standpoint he believes that clinically we must recognize that there are two forms of disease, an essential hypochondria which is entirely mental in origin, and a symptomatic which depends upon physical lesions. From the point of view of the indications for surgical intervention, however, he would classify the hypochondriacal syndrome under two captions. In the first class of cases the hypochondria is merely an episode in the course of a well defined psychosis, such as general paralysis, or associated with constitutional psychopathy, toxic-infectious psychoses or neurasthenia. In these cases surgical intervention is useless. In the second class of cases the hypochondria constitutes the entire delirium. It is a veritable morbid entity justifying the name hypochondria. In these cases we find the syndrome with a legitimate somatic basis. Surgical intervention in these cases is not only justifiable, but may produce excellent results. He considers this state under the following headings: (1) The co-existence of a somatic lesion, with the hypochondriacal idea. (2) The conditions in which a lesion can produce the hypochondriacal idea. (3) Consideration of the facts which prove the simultaneous disappearance of the lesion, and the idea.

The Lesion: It is very difficult to tell in a given case whether there is any etiological relation between the pathological condition and the hypochondriacal idea. It is important, however, to make this determination before operating, because it not infrequently happens that operations do no good, but actually aggravate the mental disease. The author in a very few words and superficially gives his method of determining this connection upon the basis of what he calls "the dose of subjective trouble" and the "knowledge of the organic state of the patient." In prolapsus of the uterus, for example, we may ask whether the mental depression is the

result of displacement of the organ, or on the contrary, if it is exaggerated, or invented out of whole cloth, or held as due to no cause. The author has shown that contrary to current opinion some of these patients exaggerate or invent symptoms in order to be subjected to operation, others merely attach their trouble to a physical lesion which they discovered themselves or which their physician called their attention to. The author insists on the necessity of seeing that the cause in the functional disease is proportionate to the lesion. Any disproportion ought at once to engage the attention of the surgeon. When the apparent lesion cannot be considered as the origin of the depression, which is at the base of the hypochondriacal idea, one should look for the existence of latent visceral disturbances which may explain it.

Formation and Evolution of the Idea: For the promotion of the hypochondriacal idea the author states that it is necessary to have a particular psychic constitution, a hypochondriacal temperament, as Roy says. Given this character he believes that with a clearly defined lesion when the pre-occupations of the patient are *en rapport* with the gravity it presents and the degree of its evolution, and when it does not effect the form of obsession with anguish, it imposes itself imperiously on the mind and can no longer be replaced by reason. From the simple condition of hypochondria minor there is a characteristic evolution to hypochondria major in which the patient is incurable and surgical treatment is powerless. In the most marked cases of this condition we find the delirium of negation. In regard to the evolution of the hypochondriacal idea he calls attention to cases where there are some lesions which could not possibly produce the hypochondria, as for example a displacement of the uterus with no disturbance of the adnexa and also cases where the patient believes in a lesion which does not exist in order to explain his mental condition. It would, of course, be a mistake to operate in these cases, and he has reported cases of operation for false appendicitis which in one case occurred as an epidemic, the result of mental contagion.

Results of Surgical Treatment: In certain classes of cases, for instance in metrorrhagia, where the condition is associated with infection and exhaustion, there is no question about the indication for operation. In certain other cases, however, where the operation is plastic or orthopedic we have learned to fear the aggravation of a pre-existing mental state, and operation could only be performed in accordance with the rules previously laid down. The author reports several such occurrences as a result of operation. He appreciates, however, that the fundamental psychopathic state will remain, and that the case may relapse under a new physical and mental stress.

2. *Eroticism in Hysteria.*—The author calls attention to the fact that modern writers on hysteria have contradicted the opinions of the older writers to the effect that hysteria was an erotic neurosis. As a result of a study of seventeen cases, however, which are briefly reported, he says in conclusion that he believes eroticism to be a pathognomonic symptom of the disease.

(July, 1906.)

1. Examination of Some Questions Touching Responsibility. TOULOUSE and CRINON.
2. Some Particular Cases of Mental Disorder of Paranoid and Melancholic Character. SOUKANOFF.

1. *Questions of Responsibility.*—The authors support the thesis that between the normal man and the completely irresponsible there are all sorts of gradations of persons who come under the review of the penal law; that the ill-balanced, the semi-lucid seem to have temporarily partial deliria. The question of these cases should be carefuly considered, not simply from the medico-legal standpoint, but from the point of view of the psychologist and the pathologist.

The author starts in by a short discussion of the question of free will and comes to the general conclusion that there is such a thing as penal responsibility, although there are psycho-physiological antecedents to volition, which in a sense determine it, there does not seem to be any place for the capricious will. He divides acts into *instinctive, spontaneous* and *reflective,* the first two of which belong to Grasset's inferior psychism. as alcohol, only in a more marked degree. The well-known effects of alcohol in retarding the reaction time to sight and sound stimuli is discussed, also the impairment of the muscular sense produced by the same agent. These conclusions do not agree with the subjective sensations, as those under investigation always imagined they were reacting with customary rapidity, or even in excess of their usual speed. From statistical inquiry (to which he gives fair warning regarding their flexibility), he comes to the conclusion that responsibility varies within three degrees. The measure of culpability varies with these three degrees. In the instinctive acts the individual is entirely irresponsible, in the spontaneous acts there is a small degree of culpability, while in the reflective acts there is complete responsibility. Responsibility is based on the clearness of consciousness and the capacity of voluntary effort of the subject which varies much both in the normal and the abnormal. Punishment, which is actually correction, should also, in order to satisfy our desire for justice, have the same gradation. Irresponsibility is not thus always a matter of disease. The question of responsibility is a question to be decided in each individual case by the most careful examination, the ordinary methods of examination by psychiaters being entirely insufficient.

He concludes that the examination of delinquents is a matter of psychology and not one of medical diagnosis simply, and that the preparation of the medical expert should be along lines of psychological laboratory work, and that this is the only preparation. It follows thus that all criminals should be considered side by side, and not be classed in categories of vicious and insane, since the characteristic of irresponsibility is not a question of pathology. Nothing is more artificial than this division. All alleged criminals should be examined by an expert. The prison would become then a hospital where the delinquents could be treated whether they were insane or not. After a period of observation sufficiently long a final disposition of the cases would be urged, the incorrigibles would be put to work and submitted to appropriate discipline, society would thus be protected from them. The corrigibles following treatment would as soon as possible be discharged, and the insane taken care of in special asylums.

2. *Paranoid and Melancholic Cases.*—The author, after citing several cases, reaches the following conclusions: (1) The symptomatological signification of the delirium of obsession by animals (serpents, monkeys, etc.), in its pure form seems more often found with reasoning paranoia (chronic), and the delirium of obsession by the devil is more often found in relation with the grave state of melancholia or with special troubles producing hysteria. (2) Often enough one encounters psychoses having

a paranoid character along with changing delirious ideas and hallucinations of the different sensory organs and delirium of obsession by some animal, in the representation of which (ordinarily among women), the patient associates the ideas with demons. (3) The absurd ideas of possession by some animal or by the devil are observed oftentimes in different states of dementia, for example in the primary dementia of adults and in paresis. (4) In certain complex cases the delirium of obsession by some animal and delirium of obsession by the devil are combined with the delirium of transformation into some definite animal or into a demon. (5) In certain cases of melancholia the emotional state of anguish is associated in a very intimate manner with painful and unpleasant sensations localized in the region which has suffered from a traumatism at the same time of the emotional shock. W. A. White (Washington).

Centralblatt für Nervenheilkunde und Psychiatrie

(No. XX. March 1 and 15, 1907.)

1. Contribution to the Knowledge of Motor Apraxia Upon the Grounds of One Case of One-Sided Apraxia. K. Abrahams.

Abrahams classifies apraxia, according to Heilbronner, in three classes:

1. Cortical: This form is characterized by an impairment of conductivity of the sense-motor paths and by the preponderance of parakinetic manifestations in all movements.

2. Subcortical: By the intactness of the conductivity and the resulting parakinetic complicating voluntary movements.

3. Conducting apraxia: Through well regulated interchange of movements, often in the sense of perseveration, likewise due to the absence of parakenetic manifestations.

He places his case in the third class. He attributes the disorder of apraxia not to receptive faculty, but regards it as purely reactionary. Much importance is assigned by him to the psychical component which enters in the mechanism of apraxia. In Leipman's case of right-sided apraxia the lesion was demonstrated in the superior parietal lobe and supramarginal gyrus. Abrahams does not localize the lesion of apraxia in any particular region, but explains this abnormal phenomenon on the theory of diaschisis, formulated by v. Monakow, "which implies that when a region in the cerebral cortex, the integrity of which is associated with certain functions, is diseased, the specific functions of this area are not the only ones impaired. No region is independent by itself, but each stands with others in union, corresponding to the infinite and manifold relations of the cerebral cortex. A diseased area will, therefore, bring about not only local but also associative disturbances; the process of separation of a diseased region from others is designated by v. Monakow as Diaschisis."

The author's case is exhaustively treated. The following is only a brief résumé of the important features of the clinical picture. The patient was sixty-one years of age, fairly intelligent, and a prosperous business man. From May 27, 1900, till Dec. 5, 1905 (the day of his death), he was subject to many attacks of apoplexy, which at each time were accompanied by peculiar manifestation, the latter soon disappeared with the return of consciousness. After the attacks patient was observant, exhibited interest in the examination, and showed no abnormal irritability.

The disturbance was sensory aphasia which usually lasted for about fourteen days. Then speech remained ungrammatical, patient was able to understand spoken language and had no difficulty in finding words. Only in the later stages of the disease did the amnestic aphasia become prominent and persistent. The capacity for repeating words was not markedly affected. After each apoplectic attack patient showed inability to read and understand what he read, but soon rapid improvement was noticed. At first complete agraphia was present, but he was soon able to write well. Later right-sided hemianopsia was demonstrated which often showed a slow and incomplete remission. After each attack of apoplexy a temporary paresis of the right side of the body was demonstrable. With the improvement of the paralysis a disturbance of purposeful execution of movements of the right upper and lower extremities was observed. For instance, patient made an attempt to put on a sock; after long manipulation he was not able to find the opening. However, he was successful when he used the left hand only. He took a handkerchief in the left hand and wiped his forehead correctly; he was asked to do the same thing with the right hand; he took the handkerchief between the third and fourth fingers, then in the first, but did not wipe the forehead. At last he threw it on the floor and wiped the forehead with the bare hand. He made false movements with the right leg in putting on trousers, etc. Such disturbances were occasionally seen on the left side, but they were only transient and not permanent. Remissions occurred on the right side, but they never completely disappeared. Impressions from external world were correctly conveyed. Stereognostic sense was at first slightly but later considerably affected.

The post-mortem examination of the brain revealed the following: Dura was adherent to the skull; marked cerebral arteriosclerosis, especially of the basilar arteries and arteria fossæ sylvii; the pia over the convexity was more adherent on the left than on the right side; no hemorrhagic areas could be demonstrated; the sulci of the left frontal lobe were widened, and the gyri were much reduced in volume; the left ascending parietal convolution, parietal and occipital lobe underwent great changes; also their convexities were rough and covered with a brownish membrane; the convexity of the left superior parietal lobe was affected and its superior convolution was narrowed; all the convolutions of the left occipital lobe were reduced in volume; the superior temporal gyrus of left temporal lobe was narrow, and also diminished in size and the surface was nodulated. The pathological condition of the encephalon is explained on the basis of arteriosclerosis.

M. J. KARPAS (Ward's Island).

The Journal of Mental Science

(Vol. LII. No. 217. April, 1906.)

1. Alcoholism, Crime and Insanity. W. BEVAN LEWIS.
2. Amentia and Dementia: A Clinico-Pathological Study. JOSEPH SHAW BOLTON.
3. The Morison Lectures for 1906. The Pathology of General Paralysis of the Insane. W. FORD ROBERTSON.
4. The Prognosis in Dementia Paralytica. GEORGE GREENE.

5. Mental (or Asylum-Trained) Nurses; Their Status and Registration. T. Outterson Wood.
6. The Nursing Staff at the Metropolitan Asylum, Leavesden; Notes Upon a Scheme of Promotion. Frank Ashby Elkins.
7. On the Etiology of Asylum Dysentery. W. Bernard Knobel.

1. *Alcohol, Crime and Insanity*—The author prefaces his remarks upon the relation of alcohol to crime and insanity, by a cursory review of the physiological effects of alcohol upon heat formation, deduced from animal experimentation, and the effects of alcohol upon the reaction time and muscular sense. He concludes that thermogenesis is modified in two ways by this agent; at first, there is a decreased thermogenesis due to the stimulating effect of the alcohol on the vaso-motor centers; this is followed by an increased heat production and radiation due to a paresis of these centers. He found, also, that chloral exhibited the same phenomenon to the conclusion that the incidence of alcoholism does not coincide with that of insanity, but does coincide with crime. These conclusions, which are somewhat at variance with other investigators, are based upon the statistics of different counties in England, where the social and racial conditions differ greatly and hence can be used for comparison. However, he does not belittle the influence of alcohol in the causation of insanity, not directly only, but through heredity. A very good exposition of the so-called laws of heredity is given. He doubts that alcoholic tendencies, as such, are inherited, but he is convinced that alcohol, like other toxic agencies in the parents, have a detrimental effect on the germ plasm. He deplores the loose term of "heredity" in cases of alcoholism, as well as in cases of toxemia—such, for example, as syphilis. Here a mother gives birth to a syphilitic child and the congenital syphilis may be the result of a simultaneous infection. That such conditions should be considered as the result of direct transmission rather than a hereditary influence, is a point that is well taken. The author goes even further into the analysis of the effects of these toxic agents on the germ plasm. He reasons from certain biological facts that there is a distinct difference between paternal and maternal forms of transmission to the embryo. The arrest of development, as shown in congenital mental weakness, idiocy and imbecility, he believes is the result of maternal toxemia, while convulsive neuroses, epilepsy, chorea, hysteria and like affections, are the legacy from the paternal side. This fact of alcoholism in the parents, especially at the time of conception, producing detrimental changes in the embryonic structures, has been considered by other authors, and is of considerable importance, and its relation to race hygiene should not be underestimated.

2. *Amentia and Dementia.*—Continued article.

3. *The Pathology of General Paralysis.*—This article is an abstract of the Morison Lectures, for 1906, published elsewhere, and gives the author's recent work upon the bacterial nature of general paralysis, and the relation of the diphtheroid bacillus, or *bacillus paralyticans*, as termed by him and his associates, to this disease. Various criticisms and reviews of the work have appeared, usually unfavorable to the view that general paralysis is caused by the diphtheroid bacillus. The criticism of Ferrier, in the Lumleian Lectures for 1906 (Royal College of Physicians, London), seems to be a fair and just estimate of this work by one of the author's own countrymen. While he considers the views of Ford Robertson interesting, he finds that there are many difficulties in the way of their general acceptance as

a satisfactory pathogeny of tabes, cerebral and spinal. Other investigators (Eyre and Flashman) found this bacillus in the throats of a large number of patients, suffering from different forms of insanity, as well as in the tissues of a considerable number of cases post-mortem, and there was no evidence that this bacillus was more common in the throats of general paralytics than of other insane patients. They were unable to trace any causal relation between diphtheroid bacilli and general paralysis. When competent bacteriologists review this subject, then the weak points of the theory are easily exposed. Dr. Bullock (quoted by Ferrier) states that the diphtheroid organism may be found in the intestines, genito-urinary tract, throat, and in the nasal secretions and elsewhere in perfectly normal individuals, as well as in catarrhal conditions of the respiratory tracts. He also states that the characters given this bacillus by Ford Robertson are not sufficient to differentiate it from other forms of diphtheroid organisms. If diphtheroid bacilli exist in the blood and cerebrospinal fluid of general paralytics, they should be capable of cultivation at once without waiting for the cessation of any supposed bactericidal action of these fluids, for no lysogenic action of the serum for bacilli of this group has been proved to exist. From specimens of cerebrospinal fluid, blood, and urine of several cases of tabes, and general paralysis, absolutely sterile cultures were obtained by competent bacteriologists for Ferrier, and he comes to the conclusion that it is not improbable that the bacilli found by Ford Robertson, so largely diffused throughout the tissues in general paralysis, are the result of a terminal invasion. This view seems perfectly justifiable. He also claims that it has not been shown that symptoms and post-mortem appearances of general paralysis can be produced by toxines prepared from these bacilli. It is difficult to see why it is necessary to have such a severe disease as syphilis in order to cause an impairment of the resistive power of the various tracts and thus allow the *bacillus paralyticans* to gain an entrance to the system; such an impairment could certainly be produced by a large number of conditions, and it seems unnecessary to bring syphilis into the etiology at all.

4. *The Prognosis in Dementia Paralytica.*—The conclusions of the author are based upon the analysis of 500 cases of general paralysis, and are interesting as showing what factors tend to limit or prolong the course of the disease. Among the factors that favor a long course are: *Youth*, from the age of 25 to 35, the duration of the disease steadily diminishes, and from the age of 35 to 40 the acutest form of the disease appears. (2) *Females* live nearly twice as long as males. (3) Recently *acquired alcoholism* has a tendency toward prolonging the course of the disease. He found the early onset of dementia a very unfavorable symptom, and that melancholic subjects rarely survive a year. In another paragraph, however, he states that melancholic types tend to remissions. Maniacal exalted types run a variable course. Systematized and fixed delusions indicate that the course of the disease will be protracted. Pupils inactive to light, sluggish reaction to light, rapidly alternating pupils are unfavorable signs. It is interesting to see that epileptiform convulsions have little or no general bearing on the course of the disease, and that the degenerate types live longer than the more highly developed subjects. The disease in juvenile subjects pursues a long and chronic course, and finally the association of early tabes with early general paralysis is an indication that the course of the latter disease is likely to be lengthened.

5. *Trained Nurses.*—Not suitable for abstracting.

6. *Nursing Staff.*—Not suitable for abstracting.

7. *On the Etiology of Asylum Dysentery.*—The author has endeavored by a thorough investigation to ascertain the cause for the prevalence of this disease in the English asylums. From the reports it would seem that dysentery is much more prevalent in the English asylums than in this country where happily it is rather rare. Scattered cases may occur with us, but never in such numbers as reported by English writers. He comes to the following conclusions: That dysentery in England is mainly confined to insane asylums, and does not occur except rarely in other large institutions, such as prisons and workhouses; that the increased precautions that have been taken during the past few years have made no appreciable difference to the incidence of or the mortality from this disease; that there is strong evidence in favor of the view that not one, but many micro-organisms, either singly or as a mixed infection, can give rise to dysentery. From the fact that dysentery develops after a disturbance of the subsoil in the neighborhood of an asylum, he argues that there must be some relation between this disturbance and the disease. That the evidence deduced from the relation between the inhalation of sewage effluvia and dysentery supports the theory that asylum dysentery can be caused by some microorganism which normally inhabits the colon and becomes pathogenic when the resisting power of the tissue is sufficiently reduced. That the occurrence of dysentery in members of the staff of an asylum is probably due either to infection by a virulent form of some universal organism, or to some normal colon organism becoming pathogenic, owing to the reduction of immunity caused by the frequent breathing of an atmosphere permeated by a fecal odor. That there is strong evidence to support the theory that, in the insane, the vitality and the resisting power of all tissues to infection is reduced, owing to the impairment of their trophic nerve supply. That dysentery is particularly apt to occur in the insane owing to the deterioration of nerve cells affecting the trophic nerve supply to the colon. That it is far less apt to occur in congenital cases of insanity or those in whom the mental disease is stationary. That the statistical evidence is entirely against the view that dysentery is spread by the transfer of recovered cases from ward to ward. He calls especial attention to the fact that it is hardly possible that the disease is communicated from one person to another, thereby disagreeing with Mott, who, in an earlier article on the subject, claimed the spread of dysentery was due to imperfect precautions against infection, but with the best possible means of precaution in Claybury Asylum had failed to lessen the mortality of the disease. H. H. Cotton (Hathorne, Mass.).

Review of Neurology and Psychiatry

(Vol. V. No. 3, 1907.)

1. Tabetic Atrophy of the Auditory Nerve. Gowers.
2. Alcoholic Psychoses: A Study of Their Mechanism and of Their Relation to Other Mental Disorders. C. W. Chapin.
3. Dementia Præcox. J. MacPherson.

1. *Tabetic Atrophy of the Auditory Nerve.*—Gowers suggests that a progressive reduction of hearing from above and below, analogous to

concentric restriction of the fields of vision; and a loss in the centre of the range, analogous to a central scotoma in vision, make the diagnosis probable of labyrinthine deafness due to atrophy of the auditory nerve: and the significance of such deafness occurring in tabes is especially great if the deafness accompany optic nerve atrophy.

2. *Alcoholic Psychoses.*—Chapin (of Ward's Island) mentions the fundamental symptom elements of alcoholic insanity as apprehensiveness and defective grasp.

Alcoholic insanity, he thinks, is analogous in its mechanism to senile psychoses, to the polyneuritic psychosis when due to other causes, and to certain traumatic pychoses, but is fundamentally different from manic-depressive insanity and dementia præcox.

3. *Dementia Præcox.*—MacPherson presents an interpretation of Kraepelin's conception of dementia præcox, quoting Kraepelin's observations on development, physical disturbances, etc., and analyzing the basic symptoms of the disease. He considers that the great importance of Kraepelin's presentation of dementia præcox lies in the essential advance he has attained in the prognosis of a hitherto unclassified group of cases. His explanation of Kraepelin's 8 per cent. of recovery in dementia præcox is that our knowledge is not sufficiently advanced to enable us to distinguish allied types which are not really cases of progressive dementia from those which are. C. E. Atwood (New York).

Miscellany

The Bearing of Philosophy on Psychiatry, with Special Reference to the Treatment of Psychasthenia. James J. Putnam (The British Medical Journal, Oct. 20, 1906).

There are two reasons why psychiatrists should study philosophy: It helps to a better understanding of the origin of mental symptoms, and it helps to dispel the mystery which surrounds the relationship between consciousness and the material world. By philosophy a man is studied in his relations to the community. Insanity in most of its characteristics is a social concept: the general paralytic is usually detected as offending against his social traditions. It is necessary to adequately help our patients, to understand the relation of their consciousness to the material world.

C. D. Camp (Philadelphia).

Gastric Neurasthenia. Hugh MacCallum (The British Medical Journal, Oct. 20, 1906).

"There is no known functional disease of the stomach that cannot have its cause and continuity in neurasthenia." Other organs such as the liver, kidneys, etc., may be similarly affected. Neurasthenia may last for years with dyspepsia as the only symptom. Signs of such condition are: Tiring easily, malnutrition, mental irritability, insomnia, cutaneous flushing and visceroptosis. The author regards as most essential in the treatment of neurasthenia, the training of the body and mind, especially the latter. In the discussion, Dr. William Calwell said that it was important to recognize that neurasthenia might be secondary to chronic stomach affections, especially as it raised the question of operation. If there was a stomach lesion operation would be of benefit.

C. D. Camp (Philadelphia).

THE MICROSCOPICAL CHANGES IN THE NERVOUS SYSTEM IN A CASE OF CHRONIC DOURINE, OR "MAL DE COIT," AND A COMPARISON WITH THOSE FOUND IN SLEEPING SICKNESS. F. W. Mott (The British Medical Journal, Aug. 11, 1906).

Dourine is due to a specific form of trypanosome and affects equines, being transmitted like syphilis by coitus. In the central nervous system of a horse dying of the disease there was found a severe chronic interstitial inflammation of the posterior spinal ganglia, especially severe in the lumbo-sacral region, where there was also atrophy of the posterior roots and their fiber continuations in the spinal cord not unlike the lesions in locomotor ataxia. There was also a subpial and septal proliferation of the neuroglia which was not limited to the posterior columns.

C. D. CAMP (Philadelphia).

CHOREA. From a study and analysis of one hundred and eight cases of chorea treated at the Johns Hopkins Hospital and Dispensary. W. S. Thayer (Journal A. M. A., Oct. 27).

Thayer concludes that there is good reason to think that well-marked febrile symptoms, without rheumatism, occurring in chorea, especially if they are accompanied with undue rapidity or irregularity of the pulse, is at least strongly suggestive evidence of acute endocarditis. It is possible that the fever may be the sign of a deeper lying infection back of the chorea, but there is nothing in his study to settle the question whether chorea represents a secondary infection or a special localization of an infectious agent responsible for essential manifestations of the disease. The study of the circulatory conditions in old patients still remains to be carried out, but Thayer calls attention to the following points of interest thus far developed in his investigation: (1) Of 689 cases of chorea observed at the Johns Hopkins Hospital and Dispensary during one or more attacks, 25.4 per cent. showed evidences of cardiac involvement; such evidence was present in over 50 per cent. of the patients studied in the wards of the hospital. (2) Cardiac involvement occurred with somewhat greater frequency in those cases in which there was a history of acute polyarthritis than where such history was absent. (3) Cardiac involvement was commoner in cases of chorea with frequent recurrences than in those in which there was a history of a single attack. (4) In 110 cases of chorea treated in the wards of the hospital there was fever of a moderate extent in almost every instance. (5) In the large majority of the cases in which high fever was present there was evidence of cardiac involvement. (6) There is good reason to believe that the presence of fever in otherwise uncomplicated chorea is, in a large proportion of cases, associated with a complicating endocarditis.

A CASE OF SUDDEN DEATH POSSIBLY DUE TO VAGUS INHIBITION. E. D. Telford (The British Medical Journal, Aug. 18, 1906).

A girl, aged eleven years, was operated upon under chloroform for the removal of tuberculous glands from the right side of the neck. The operation was tedious, but recovery from the anesthetic was prompt, the only unusual feature being a pulse rate of 120. Two days later she suddenly died. At necropsy it was found that the right vagus nerve disappeared into a mass of tuberculous glands the size of a filbert. The nerve was swollen for one inch before its entry into the mass, while at the point of entry it was constricted. The fibers of the nerve were frayed out in

the interior of the mass. Brouardel quotes a case of sudden death with similar post-mortem findings. C. D. Camp (Philadelphia).

The Relation Between a Cutaneous Naevus and a Segmental Nerve Area. G. Lenthal Cheatle (The British Medical Journal, Aug. 18, 1906).

The author regards as unique in the literature a naevus occupying the complete area of distribution of the third cervical nerve. The article is illustrated by photographs, but further data of the case is not given.

C. D. Camp (Philadelphia).

The Bacillus Paralyticans. F. W. Langdon (Cincinnati Soc. for Medical Research, October, 1906).

The bacillus paralyticans recently announced by Dr. Ford Robertson, of Edinburgh, has been the subject of some research work in the clinical laboratory of the Cincinnati Sanitarium, and pure cultures of the bacillus and also photomicrographs were shown. It belongs to the diphtheroid group, but unlike the Klebs-Loeffler bacillus is non-pathogenic to guinea pigs, although fatal to rats in two or three months. In occurs in rods, singly and with a tendency to groups of threes, and also in a thread form. It has been found in the bronchial, alimentary and genito-urinary mucous membranes, in the cerebrospinal fluid, in the brain, in the walls of the cerebral blood vessels, in the blood, the urine and other secretions and tissues. Robertson believes that it gains access to the system mainly through the respiratory tract and the alimentary canal. Syphilis, alcoholism, etc., are merely contributory factors in breaking down the defenses of the body against bacterial invasion. The invasion of the blood, lymph and tissues by the organism gives rise to the production of toxins, which are responsible for the various trophic, degenerative, convulsive and paralytic phenomena. The polymorphonuclear leucocytes exert a marked lysogenic action upon this bacillus, and to this action is attributed the recession of the bacterial invasion and the remissions so characteristic of paresis. Like the Klebs-Loeffler bacillus, this organism appears remarkable for its polymorphism. It occasionally shows barred as well as solid color forms when stained with methyl blue and carbol fuchsin.

Langdon.

Cerebral Decompression. W. G. Spiller and C. G. Frazier (Journal A. M. A., Sept. 1, 8, 15 and 22).

From a rather extensive review of the literature, these authors concluded that palliative operations in cases of cerebral tumor are justifiable. Headache is the principal symptom calling for relief; Spiller is somewhat skeptical as regards any beneficial effect on Jacksonian convulsions. The possibility that operation may obscure focal symptoms is considered and he advises that palliative operations be performed before general symptoms become intense, and especially before optic neuritis threatening blindness has developed. The general unanimity as regards the effects on choked discs of opening the skull makes the necessity of early operation very evident. Palliative operations are not a substitute for radical measures. The tumor should be removed when possible and when sufficient skill is at command. There should be no attempt to remove a glioma, and Spiller thinks that partial removal is generally inadvisable. Sometimes complete relief may follow simple opening of the

skull and dura, but only one case of actual disappearance of a tumor seems to be on record: that of Horsley. Spiller sums up his views as follows: (1) "Palliative operations should be performed early in every case in which symptoms of brain tumor are pronounced and before optic neuritis has advanced. (2) Partial removal of a tumor, especially of a glioma, is a questionable procedure. (3) Palliative operation does not cause atrophy of a brain tumor, and probably does not arrest its gowth; on the other hand, it probably does not hasten its growth. (4) Palliative operation is not to take the place of a radical operation when the latter can be performed without great risk to the patient. (5) In some cases the symptoms of brain tumor disappear almost entirely for a long time or permanently after a palliative operation. This result is obtained either by relief of intracranial pressure or by removal of some lesion (meningitis serosa, etc.), other than brain tumor, and yet causing symptoms of tumor." The surgical aspects of the subject are discussed by Dr. C. H. Frazier at some length. The question whether a palliative or a radical operation is to be chosen is dependent on whether the tumor is operable or not, and there are two classes of cases, he says, in which a decompressive operation may be required. One is when there is reason to believe that the tumor can not be entirely removed and the other when it can not be localized and yet the symptoms call for relief. This happens more frequently with cerebellar tumors, and in certain cases he has removed from one-quarter to one-third of the cerebellar hemisphere.

Case of Obscure Intracranial Tumor, with Extension to Fourth Ventricle. G. H. Grant Davie (The British Medical Journal, Aug. 11, 1906).

The first symptom noted was a severe occipital neuralgia which was always most severe at about 5 A. M., and prevented further sleep. There was also a choking sensation when lying down. Gait and station were normal, but the patient had a sensation when walking or "running forward on her toes all the time." The pain later increased in severity and extended forward to the left ear and angle of the jaw. A kneeling position with her forehead to the floor gave her the most relief. An eye examination six weeks before her death showed 6-12 vision, but was otherwise negative. At necropsy, a layer of soft gelatinous tissue was found covering to a depth of one-quarter inch the whole of the anterior half of the under surface of the left cerebellar hemisphere and extending as a delicate covering over the hinder half of the floor of the fourth ventricle. The growth was distinct from the adjacent brain and was histologically a round cell sarcoma with almost complete absence of intercellular stroma. The ventricles were enlarged and filled with a clear fluid.

C. D. Camp (Philadelphia).

Book Reviews

DEMIFOUS ET DEMIRESPONSABLES. Par Prof. J. Grasset, Professeur de Clinique, Médicale a l'Universite de Montpellier, Associe National de l'Académie de Médecine Lauréat de Institut. Felix Alcan, Paris.

"Demifous et Demiresponsables," by Dr. J. Grasset, of Montpellier, is a work of more than ordinary interest. No contribution to psychiatry could be more opportune than this, for Dr. Grasset discusses the medico-legal aspect of crime committed by persons who, through genius, heredity, upbringing or trouble, are not wholly insane, and yet are not wholly sane. It is a masterly discussion of the whole problem of the "borderland" cases.

He refutes the theories forced upon the nation by antiquated legal restrictions, that all persons are either crazy or not crazy, responsible or irresponsible. He establishes a middle class, which, he states, has never yet been recognized by society; a class which society must treat, and against which society must protect itself. He says: "The half-insane exist. It is equally erroneous and unjust to class them either with the insane or with the sane. They are different from the sane in that they are psychically afflicted, and from the insane in that they preserve a certain degree of reason."

Prof. Grasset mentions an interesting list of geniuses, including authors, musicians, reformers of his own and other European countries, whom he considers, as did Nordau, not wholly sane. But whereas Nordau unjustly grouped them all with the insane, he more exactly defines their position as Demifous.

One of the most interesting and opportune portions of his book is that dealing with "The Demifous Before the Law," "Semi-Responsibility, Limited Responsibility." "Let us assume," he says, "that a demifous has committed a crime. He has become injurious to society. He is dangerous. Society still owes him assistance and treatment, but it also has a right to defend itself against his misdeeds. For him, one must not choose either a prison or an asylum, he needs both." In view of many examples of the lack of specific legal preparation in dealing with semi-insane criminals, Prof. Grasset's outline of how the law should be moulded to treat such individuals is full of wholesome advice, which the medical profession will rejoice to point out to their legal brethren. Prof. Grasset believes in shortening sentences, special regimen and penitentiaries, special surveillance, and treatment after one sentence, and a special institution, or at least a special section of some institution for the "demi-fous," or half insane.

JELLIFFE.

News and Notes

The Boston Society of Psychiatry and Neurology at its regular meeting Oct. 17, 1907, adopted the following expression of its sentiments with regard to the death of Dr. Charles Follen Folsom: Dr. Folsom was one of the original and most valued members of the Boston Medico-Psychological Society, the parent of the Boston Society of Psychiatry and Neurology. His thoughtful contributions were always listened to with the attention and respect accorded to an acknowledged authority and his continued interest in our meetings was felt by all of us as a stimulus to good work. His public labors were fruitful in many benefits to sufferers from disorders of the mind. Not only was he an admirable secretary of the State Board of Lunacy and Charity, but it was due to the recommendations of the special commission of which he was a member that the present State Board of Insanity was established and the insane removed from the almshouses and placed under the direct protection of the State. His brilliant mind, his fertility of resource in the treatment of disease, his deep sympathy for his patients and untiring devotion to their needs, the far-reaching benefits to the insane of the Commonwealth from his enlightened public work, his warm interest in the labors of his colleagues and loyalty to the claims of friendship so won our regard and admiration that we feel his death as a personal and public misfortune.

Note from Dr. Herman H. Hoppe:

At the request of Prof. H. Oppenheim, of Berlin, I gladly make the following correction: In my article, "Brain Tumor Symptom Complex Terminating in Recovery," I referred to Oppenheim's article in "Serous Meningitis." In this article he speaks of a group of cases simulating cerebellar tumor and expresses the opinion that the symptoms are caused by some, as yet unknown, pathological change, which can disappear completely, or *possibly* by meningitis serosa or internal hydrocephalus. In my reference I quoted Oppenheim as saying that they *are* caused by internal hydrocephalus.

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Original Articles

ON ACROCYANOSIS CHRONICA ANÆSTHETICA WITH GANGRENE; ITS RELATIONS TO OTHER DISEASES, ESPECIALLY TO ERYTHROMELALGIA AND RAYNAUD'S DISEASE.*

BY LEWELLYS F. BARKER, M.D., AND FRANK J. SLADEN, M.D.,
OF BALTIMORE.

Vasomotor disturbances are commanding a steadily growing interest. With the demand for knowledge in regard to neuroses in general has come a special effort on the part of scientific men to make clearer the very indefinite vasomotor and trophic group. Pathological evidence is gradually accumulating, and experimental medicine is slowly coming to its aid.

The group includes erythromelalgia, Raynaud's disease, and a great number of symptom-complexes resembling these two somewhat, but not sufficiently to be classified with either. Scleroderma may be included, but it affects the whole body. In this paper we wish to discuss those types which affect the extremities particularly. Cyanosis of the extremities is seen in many heart and lung conditions, and in Osler's polycythemia. The former are easily explicable; the latter, as Senator[1] points out, may be an affection of the bone marrow.

The idea that the vasomotor tropho-neuroses all belong to one group is being more generally accepted, and this is helping much to clear up the indefiniteness of the group. The most confusing factor has been the number of cases which do not fit any of the known clinical types. Indeed, it is

*Read at the thirty-third annual meeting of the American Neurological Association, May 7, 8 and 9, 1907.

now commonly suggested that these neuroses may pass from one type through intermediate stages into a second type. Kollarits[2] mentions cases of erythromelalgia passing into Raynaud's disease. Simple acrocyanosis (cyanosis of the extremities) is often the first stage of erythromelalgia. Legroux[3] reports a case of chilblains, passing into permanent local asphyxia, and later developing symmetrical gangrene. Sachs[4] states "It is very certain that erythromelalgia, Raynaud's disease, acroparesthesia, and even scleroderma often merge into each other, or are associated with one another."

On analysis the symptoms of these vasomotor trophoneuroses fall into three main groups—vasomotor, sensory, and trophic.

The vasomotor symptoms include (1) hyperemia, (2) syncope, and (3) asphyxia; the sensory, (1) pain, (2) hyperesthesia, (3) anesthesia, and (4) paresthesia; the trophic, (1) ulceration, (2) gangrene, and (3) dystrophies of the skin.

Perhaps the simplest type of vasomotor neurosis is simple acrocyanosis, a symptom of the vasomotor group, which is a prominent feature in many more complex neuroses. Mosse[12] reported two cases of the simple acrocyanosis in mother and daughter. Erythromelalgia is characterized chiefly by two symptoms, hyperemia from the vasomotor group, and pain from the sensory. Still more complex is Raynaud's disease with symptoms from all three of the groups. The vasomotor are hyperemia, syncope and asphyxia; the sensory, pain, anesthesia and paresthesia; the trophic, gangrene. In addition the distribution, and the course of the disease, are of value in the diagnosis. All these diseases affect chiefly the extremities, though Weir Mitchell[5] described erythromelalgia as more common in the feet. Raynaud[6] stated that his type of spontaneous gangrene occurs alike in hands or feet. The course may be acute, paroxysmal, or chronic.

When the great number of combinations of these symptoms possible is realized, it is not surprising that new clinical types are constantly being described. Among the intermediate forms are Schultze's[7] acroparesthesia, Nothnagel's[8] vasomotor neuroses, Cassirer's[9] acrocyanoses. Cassirer's comprehensive monograph[9] gives a complete description of each

type of disturbance, and to him credit is due for development of the idea of unification.

Our interest in the subject has been stimulated by the following case, which came to the Surgical Clinic of the Johns Hopkins Hospital for treatment for his toes, and was referred to the medical side as a probable vasomotor neurosis.

B. J., aet. 44, (General No. 57,972), white, married, walked into the ward with a slight limp, making use of a cane.

Complaint: "Nervousness and loss of ends of toes."

Family History: Father, 78, alive and well. Mother, 65, alive and well. Brothers, four, alive and well.

Negative for rheumatism, tuberculosis, neoplasm, or nervous affections.

Past History: Measles, mumps, chicken-pox and whooping-cough when a child. Pneumonia when 25 years old, confined to bed three weeks, with good recovery. No diphtheria, scarlet fever, malaria, typhoid or rheumatic fever.

Head: Not subject to headache. Eyesight is poor and he uses glasses to read. Cannot read very well, because of eye pain and vision is blurred. No trouble with hearing. No nose or throat trouble. No giddy or fainting spells.

Cardio-Respiratory: No cough. Has shortness of breath sometimes after exertion, as on going up stairs. No pain in chest or palpitation of the heart. No expectoration. No hemoptysis. Had occasional night sweats about two years ago, which were not severe; none during past six months.

Gastro-intestinal: Negative. Appetite always poor. Digestion apparently normal. Bowels usually move once a day.

Genito-urinary: No kidney or bladder trouble. No trouble with urination. Had gonorrhea when 18 years old, which was treated and evidently cured. Denies syphilis. No history of secondary manifestations.

Neuro-muscular: Negative.

Habits: Smokes and chews tobacco, about five cents' worth of each per week. From the age of 18 he has been a heavy alcoholic drinker, up to one month before admission.

Work: Postmaster and station agent, necessitating much standing and tramping about in the wet and cold in the winter months.

Present Illness: Duration about 10 years.

The onset was gradual with the development of "cramp-like pains in the legs, which were very severe, and came and went quick as a flash." There was no sensitiveness of the skin in the affected areas, but in the attacks the muscles of the legs became "hard as rocks," sometimes in one leg and sometimes in both. The attacks were paroxysmal, coming on

at night usually, after a hard day's work, and especially after much tramping about in cold, damp weather. They might occur every night for several days, and then he would be free of them for two or three weeks.

Five years ago they disappeared. At this time he worried a great deal about his condition and became very nervous. He would "tremble all over" and did not feel able to do his work. He had great difficulty in reading and writing telegrams.

He remembers several attacks of slight numbness in his fingers, lasting from five minutes to an hour, but does not think they changed color. Nor was there pain associated with the numbness. What bothered him most was the change in the feelings of his legs. The sensations were "not natural." They gradually became numb and felt as if pins and needles were sticking in them. He had them massaged for a time, until he found the skin was being rubbed off without his realizing it.

Three weeks before admission, while taking a bath, he noticed the skin over the second toe of the right foot was wrinkled. On taking hold of it, the nail and part of the end of the toe came off, absolutely without pain. Several days later he pulled off the tip of the big toe upon the same foot in removing his shoes and stockings. There are sores also upon the third toe of the right foot and big toe of the left. At no time has he experienced any pain. During these three weeks the feet and legs have been swollen, especially the right, and their color has been a diffuse deep red, at times changing to a bluish red. There has been no loss of muscle power, but a slight stiffness of the ankles and knees has bothered him in walking.

Examination on admission: Patient was admitted this noon, walking with a slight limp. He was put to bed and the feet dressed at once. After removing a dressing of cotton and carbolic salve, they were cleaned and put up in balsam Peru. The first two toes of the right foot showed an ulcerating surface with considerable loss of tissue, exposing the bone of the terminal phalanges. The tissue surface was clean and moist. There was no pain or tenderness even on the raw surfaces. An abrasion of the skin of the third toe was covered with a hemorrhagic crust. On the plantar surface of the ball of the left big toe was a cleanly-cut, round ulcer, rather superficial. Both legs were edematous to the knees, the right the more so, and the right was more red and hot. Both feet were cyanotic, shading to a pink color on the legs. A faint pulsation was felt in the dorsalis pedis artery of the right foot—a better one, though still weak, in the post-tibial. A good pulse was felt on the left. Pulsation was strong in both femoral and popliteal arteries. No special enlargement or tenderness of the glands

of the groin. No red lines of lymphangeitis. Patient complains of anesthesia to touch over the lower legs. Rough examination shows it present to some degree below the knees. No pain complained of, even when the fibers of cotton were being picked from the raw surfaces.

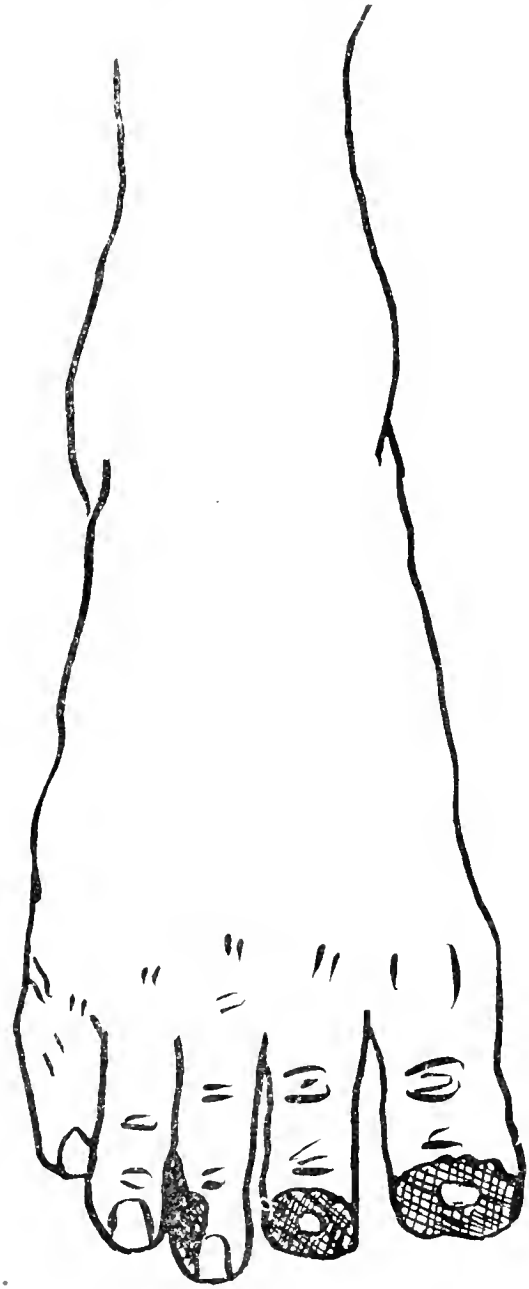

Fig. 1. Sketch of right foot, showing slough of the first two toes and abrasion on the third.

Face flushed. Dilated venules over nose and cheeks. Eyes: suffused; pupils react to light and accommodation; muscular movements good; scleræ clear; conjunctivæ rather pale. Ears: negative for tophi or discharge; no pain on pressure over audit-

ory canal or mastoid. Lips clean. Mucous membranes fair color. Tongue slightly coated with small clean ulcers on edges. Tonsils a little large. No reddening or plugs. Moderate injection of pillars and pharynx. Neck negative. Thyroid gland not palpable. No general glandular enlargement.

Chest: Well formed. Epigastric angle about 90 deg. Expansion fair, equal. Vocal fremitus everywhere present. Lungs clear on percussion and auscultation, except for occasional squeaking rales scattered here and there.

Heart: Point of maximum impulse not visible or palpable. Sounds heard best in fourth interspace about 8 cm. from median sternal line. Relative cardiac dulness to mamillary line to left in fourth interspace, and 3 cm. to right in third interspace and up to upper border of third rib. Sounds clear at apex and base. Aortic second and pulmonic second both loud, pulmonic second slightly the louder.

Pulse: 25 to ¼, regular in force and rhythm; good size; moderate tension; vessel wall just felt.

Abdomen: Looks natural. Respiratory movements free. No tenderness, muscle spasm, or rigidity. Tympany in flanks. Spleen not felt and dulness not increased. Liver dulness from fourth interspace to two fingers' breadth below costal margin in right mamillary line. Edge indistinctly felt.

Genitalia: Negative; no scars, no discharge. Testicles normal.

Reflexes: Knee-jerks active and equal; Achilles reflexes present. Plantar reflexes normal. Biceps, triceps, and periosteal radial reflexes active and equal. Abdominal and cremasteric reflexes present.

Note:—The accompanying chart shows the partial sensory disturbance.

Note, 4th day of admission: Arteries at wrist not palpable. Temporals not increased. Femoral pulse palpable.

Left arteria dorsalis pedis is palpable,—not felt on right. During examination right foot becomes bathed in sweat, left only slightly so. Posterior tibial artery is palpable on both sides. On left foot is a small round ulcer, on ball of big toe, covered with bloody scab. On right foot, tip of big toe is gangrenous, exposing last phalanx; also second toe. On outer side of third toe is a superficial ulceration covered with scab. Right foot the warmer.

Reflexes: Biceps lively on right and left, others not remarkably active. Knee-kicks present. Babinski negative.

If pulse in right arteria dorsalis pedis is not absent, it is greatly diminished.

Note, 14th day of admission: After two weeks in bed, the superficial ulcer on left big toe was healed, as was the abrasion on the third toe of the right foot. But the raw surfaces

of the first and second toes on the right showed no such tendency, until the terminal phalanges were disarticulated by Dr. Sowers. The recovery then was uneventful.

It was at this time, however, that definite hyperesthesia of the soles of both feet developed. Patient could not endure the bed clothes upon them at times. As he explained it, "they were extremely sensitive."

Note, on 28th day after admission: Both upper extremities and lower are moderately cyanosed. The toes look much as they did before operation, except that the exposed ends of the bones are missing. There is a palpable pulsation in each arteria dorsalis pedis. There is very slight anesthesia to pain

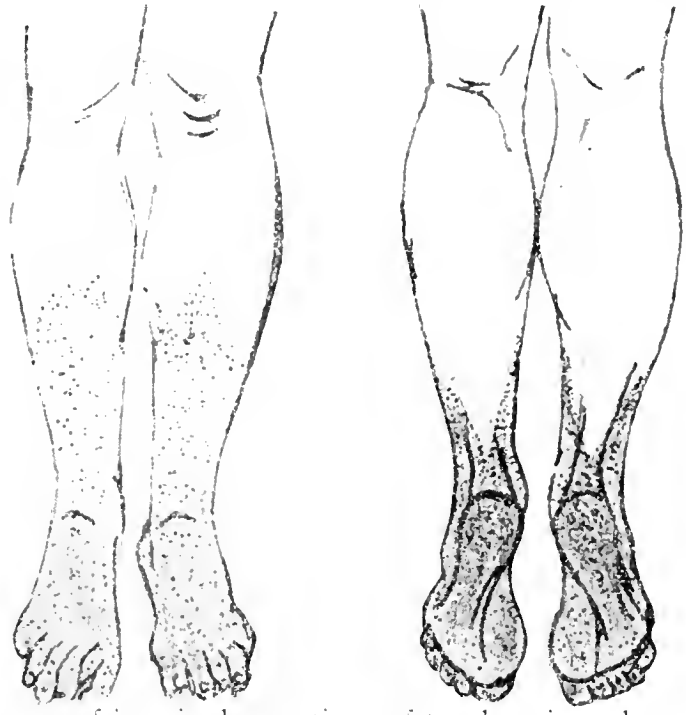

Fig. 2. Areas of impaired sensations of touch, pain and temperature.

in lower limbs. Evidently marked disturbance in temperature sense below knees, especially in lower half of legs. No marked disturbance in kinesthetic sense in both legs (at toes), though a little dulled; sharp at ankles and knees. Hands and feet are now markedly cyanotic; a considerable increase is noted in the cyanosis since the examination began. Bone vibrations (of tuning fork) are well felt in upper extremities and in tibiæ; but are a little dull in feet. No dulling at all in upper extremities distally. No hemianopsia. Cyanosis now at wrist. Marked goose-flesh. It is important to note how the cyanosis has progressed during the 15 to 20 minutes of examination.

Note on 42nd day: Marked cyanosis of right foot. Big toe now looks well. Cyanosis of left foot comes out under examination. Hands cold and cyanotic. Radial pulse good,

also that in the arteria dorsalis pedis—the left pulsates more than the right. Posterior tibial artery beats on both sides. Some pain in the second toe, and some pus can be expressed.

There is no syncope when the fingers are immersed in ice water, nor asphyxia when in warm water.

On discharge: Hands cyanotic to-day. Right foot is normal. Left foot shows slight anesthesia. Knee-jerks are present and active. Plantar reflexes normal. Pulse is better in the left dorsalis pedis artery than in the right. No muscular atrophy.

The blood examination on admission showed: Red blood corpuscles, 4,354,000: hemoglobin, 86 per cent. by the Sahli instrument. It remained practically the same and on discharge there were: 4,644,000 red blood corpuscles, and 85 per cent. hemoglobin (Sahli).

A leucocytosis of 19,460 on admission was associated with a slight bronchitis; it fell gradually to 6,740 on rest in bed and after the amputation and cleansing of the wounds.

The differential count on smears made when the leucocytes were 19,460 was:

Polymorphonuclear neutrophiles	468 or	93.6%
Polymorphonuclear eosinophiles	6 or	1.2%
Large mononuclears	7 or	1.4%
Transitionals		
Lymphocytes	18 or	3.6%
Mast cells	1 or	0.2%
Myelocytes	0 or	0.0%
Total	500 cells	100%

No nucleated red blood corpuscles are found.

The fresh blood at no time showed any evidence of the presence of parasites.

The coagulation time was delayed,—10¼ and 12½ m.,—on two observations by the Boggs instrument.

The blood pressure (maximal) varied from 100 to 120 mm. of mercury by the Riva Rocci instrument.

The temperature rose during a throat infection, and the increase in the rate of the pulse and respirations was parallel. Otherwise pulse and respiration were normal.

Urine: 800 to 1,400 c.c. daily; yellow to orange in color; specific gravity 1,020 on the average; no sugar; an occasional faint trace of albumin and an occasional granular cast; no bile pigments.

Sputum and Stools: negative.

Weight, 131 pounds on admission; 127 pounds in second week; and 133 pounds on discharge.

An analysis of this case with the schema above described kept in mind, will make its nosological localization simple.

The vasomotor symptoms present are (1) cyanosis, and (2) swelling of the soft parts; the sensory are (1) paresthesia, (2) anesthesia, not in the distribution of any nerve, (3) hyperesthesia, and (4) no pain; and finally, the trophic are (1) dystrophy of skin over toes, (2) subsequent loss of tissue, exposing the bones, and (3) a malum perforans.

Localization: the fingers, the toes, feet and legs, but more on the right than on the left.

Course: Chronic, progressive, and not paroxysmal.

This agrees, as it will be seen, with no one of the symptom-complexes above described. In considering the differential diagnosis a number of diseases might be thought of. The case can scarcely be regarded as Raynaud's disease. The absence of syncope and of pain, and the fact that it is not paroxysmal distinguish it. There are no attacks of red, white and blue fingers. There was, however, gangrene.

Erythromelalgia is characterized by a hyperemia rather than a cyanosis; by pain and hyperesthesia, not anesthesia and paresthesia; by the absence of gangrene and such trophic disturbances. The dependent position brings on the attack.

Diabetic gangrene with neuritis and arteriorsclerosis is excluded by the urinary examinations.

Senile gangrene is not warranted by the age of the patient or the condition of the arterial walls.

Tabes dorsalis, despite the anesthesia and malum perforans, can be ruled out. The knee-jerks are active on both sides. The pupils are equal and react to light. The muscle sense is nearly perfect.

Syringomyelia need not be considered, since the lower extremities are chiefly affected, the anesthesia does not present the type of syringomyelic dissociation, and there is no segmental distribution of the anesthesia, and no muscular atrophy.

Peripheral neuritis cannot be excluded. Certainly the peripheral nerves are involved and the alcoholic history and the plantar hyperesthesia are very suggestive. The muscle power is not affected. There is no muscular atrophy or fibrillary twitching. There is no pain. The leprous form (lepra mu-

tilans) is excluded by there being no thickening of the peripheral nerves, especially N. auricularis magnus, nor islands of anesthesia, nor a leontine facies. How large a part the involvement of the nerves plays in the development of the syndrome we cannot be certain.

Finally the symptomatic diagnosis of acrocyanosis or acroasphyxia may be made with certainty. This really is but a symptom, which indeed our case shows prominently. But Cassirer[1] makes this symptom the keystone to many complexes, qualifying it symptomatically by chronica, anesthetica, hypertrophica, and so on as the case may be. He describes a case which he thinks is unique, in a maiden of 19. The trouble followed frost bites. The hands and feet became bluish red and cyanotic, but not syncope occurred. Paresthesia was complained of. Small fissures on the hands were painless. All the sensory modalities were impaired somewhat. There was no muscular atrophy and motility was interfered with only by the stiffness of the joints due to the swelling. This complex he calls "Acrocyanosis chronica anæsthetica," on the basis of symptomatology.

Nothnagel[2] describes the only other case like it which can be found in the literature. Briefly, it was in a kitchen maid of 28, who was accustomed to soaking her hands in water. Numbness and tingling in the finger tips appeared first. They felt dead. Later the hands and feet were involved and cyanosis developed. There was no pain or muscular atrophy, but the sensory disturbances were general.

Our case differs from these only in the addition of gangrene and ulceration. The other symptoms are almost identical. So we might agree with Cassirer that our case is one of Acrocyanosis chronica anæsthetica,—but with gangrene. Considering the group as a whole, our case is one step from Acrocyanosis chronica anæsthetica toward Raynaud's disease.

Cassirer describes many cases which lead to hypertrophy of the soft parts and resemble acromegaly, like Kollarits' case, for instance, but in his complete survey of the literature previous to his time, he found none which led to gangrene.

Legroux[3] reports a case of "permanent local asphyxia" of the hands and feet, following chilblains. The trophic disturbances were marked in the skin and finger nails, and there was

a spontaneous amputation of the terminal phalanx of the right forefinger, absolutely without pain. Muscle power was intact. Cassirer does not mention this case among the acrocyanoses, but discusses it under Raynaud's disease. Of course it is clear that it is almost identical with our case.

In another case of Legroux's, following chilblains, acrocyanosis was followed by gangrene and spontaneous amputation of the terminal phalanges of the third and fourth toes of the left foot and later of the small toes of both feet. But the analogy is incomplete on account of the presence of pain in the second amputation. Pain, of course, allies this case still closer to Raynaud's disease.

Legroux suggests that in these cases the chilblains, the permanent symmetrical asphyxia, and the subsequent gangrene are degrees more and more accentuated of a necropathic dystrophy.

Elsner[10] and Sachs[11] have reported cases of erythromelalgia associated with gangrene, but these cannot be confused with our case. The pains and the intensification of the symptoms when the extremity is in the dependent position make the distinction clear.

In fine, we have found but one case in the literature similar to ours—that of Legroux's. These two we feel are intermediate forms more closely allied to the acrocyanoses than to either erythromelalgia or Raynaud's disease. And we suggest as a rational terminology clearly representing the type "Acrocyanosis chronica anæsthetica cum gangræna."

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POST-APOPLECTIC TREMOR (SYMMETRICAL AREAS OF SOFTENING IN BOTH LENTICULAR NUCLEI AND EXTERNAL CAPSULES.)*

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The features of interest in the case to be reported are, clinically, a tremor of the right arm, resembling post-hemiplegic tremor, and ataxia of the arms and legs, and, pathologically, the presence of symmetrical lesions of the putamen.

The patient, a man of 58, was admitted to the Philadelphia Hospital on April 20, 1903, his chief complaint being difficulty in walking. The family history, as well as the previous history, were unimportant. Owing to the mental condition of the patient, a reliable account of the onset of his disease was not obtainable, his answers being frequently contradictory.

He stated when admitted that his present trouble had begun 3 years previously. He complained of pain in the head, weakness and numbness of the legs, and weakness of the right arm. At this time nothing of moment was noted excepting that the right knee jerk was increased, some ataxia was present in the upper limbs, and the patient had a tendency to lose his balance if he turned suddenly while walking. There was no Babinski reflex, and no apparent weakness in any of the limbs.

He came under the care of one of us (Dr. Potts) in April, 1906. At that time the peculiar movements of the right arm attracted attention. These consisted of alternate flexion and extension at the elbow and wrist, the hand being supinated. The movements were almost constant, only ceasing for a time if his attention was distracted. At this examination he told a somewhat different story than at first, saying that at the beginning of his disease he noticed weakness of the legs, the left being weaker

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than the right, and that these symptoms dated back eleven years, following what he called "a cold." Six years later the right arm became affected, the disorder consisting at first of a difficulty in writing. Examination at this time showed a somewhat ataxic gait, which was more apparent with the shoes off. The right leg was held more stiffly than the left. Station, with the eyes closed, was poor, and there was also marked ataxia of the arms, especially the right. There was apparently no weakness of the arms. The

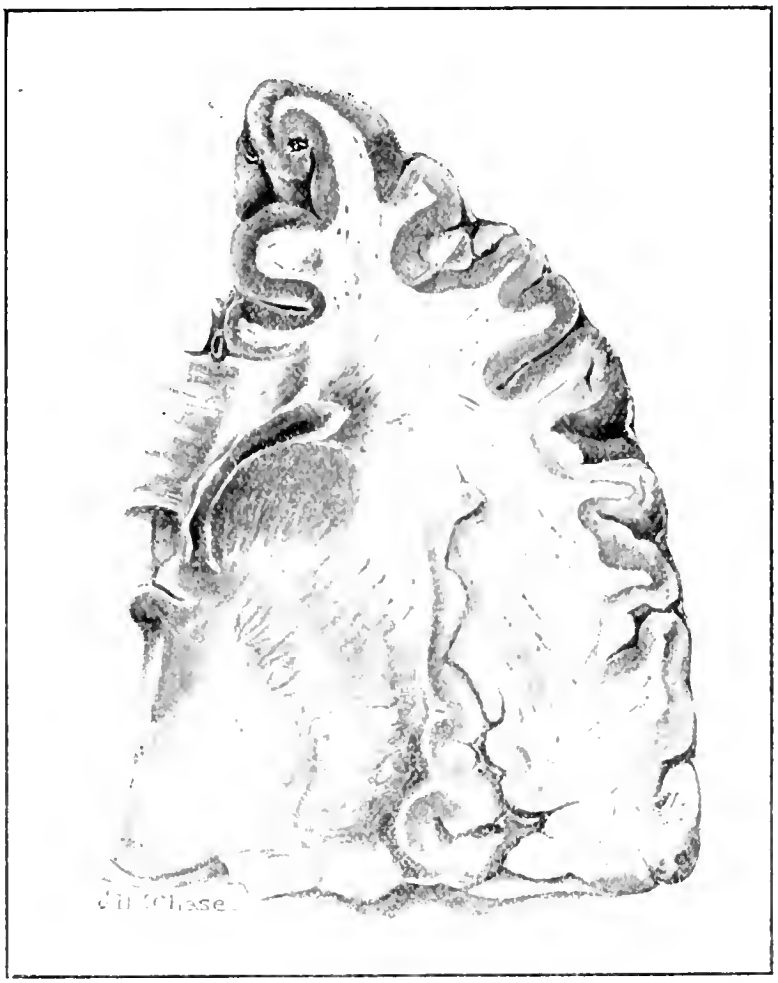

Fig. 1. Linear area of softening in external capsule on right side. Upper limit. (Macroscopical section.)

legs appeared to be somewhat weaker than normal. The biceps, triceps, and wrist jerks were not marked. The knee-jerks were prompt, the left being more so. The plantar reflex gave a normal response. Examination of the eyes showed a sluggish response to light, but otherwise they were normal.

The brain and spinal cord were turned over to Dr. William G. Spiller, who gave them to one of us (Dr. Rhein) for examination. The specimens were hardened in formalin.

Horizontal sections of the brain on the right side revealed the presence of an area of softening which first made its appearance a short distance above the lenticular nucleus, and was situated in the external capsule, *i. e.*, between the claustrum and the internal capsule. This area of softening was linear in shape, and measured at this level 2.5 cm. in length, and about 1 to 2 mm. in width. While it lay directly external to the fibers of the internal capsule, it did not cause any degeneration of these fibers.

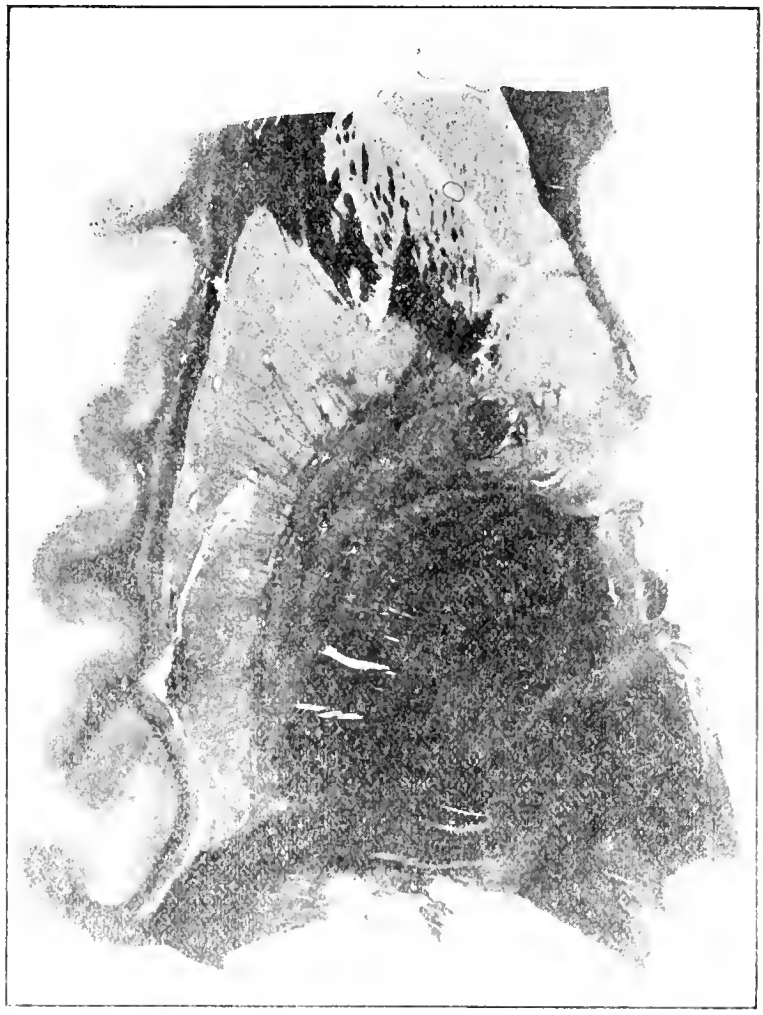

Fig. 2. Area of softening extending into the middle portion of the right putamen (microscopical section, section reversed in photograph).

The focus extended downward in a vertical direction to a position corresponding to the lower limit of the island of Reil. At the lower level of the lenticular nucleus it destroyed in part the external portion of the putamen in its posterior third. At the level at which the fasciculus of Türck is seen ("temporale Brückenbahn"—Obersteiner) these fibers were in part destroyed. The anterior commissure was intact, as were also the fibers of the

internal capsule. The fibers of the external part of the foot of the peduncle, corresponding to the situation of the fasciculus of Türck, stained poorly. The red nucleus appeared to be intact.

On the left side of the brain there was a similar lesion which began at a somewhat lower level than the one on the right side, its upper limit being at about that level where the lenticular nucleus is first seen in horizontal sections. It was situated in the same relative position as that of the cavity on the right side, occupying the region of the external capsule between the claustrum and the putamen, and measured 3.5 cm. in length, and 10 mm. in its widest portion. It extended downward in a vertical

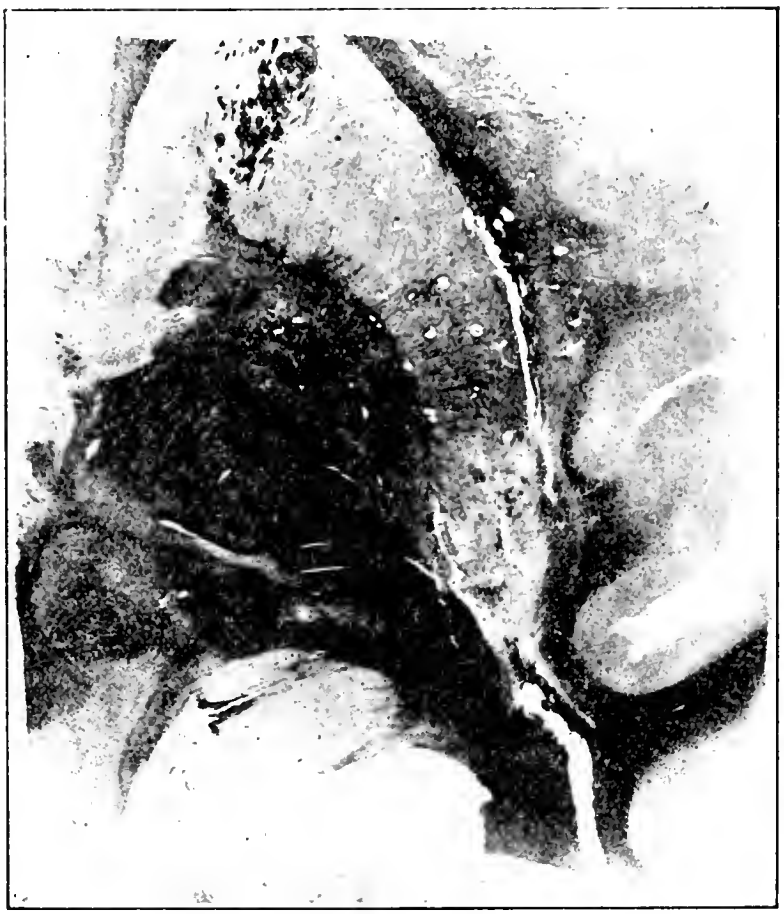

Fig. 3. Area of softening in the posterior portion of the left putamen (microscopical section, section reversed in photograph).

direction, and disappeared a little above the level of the lower border of the island of Reil. The posterior third of the putamen in its lower part was in large measure occupied by this focus of softening. The fibers in the retro-lenticular region were more or less involved.

The fibers in the internal capsule were not degenerated. The red nucleus appeared to be normal.

Certain fibers passing inwardly and apparently through the lenticular nucleus toward the thalamus, stained well on both sides.

In sections from the pons, medulla oblongata and spinal cord the nerve fibers stained normally. There was no evidence of descending degeneration in the pyramidal tracts.

The cells of the anterior horns in the cervical and lumbar regions, stained by hemalum and acid fuchsin, showed no marked change.

Sections from the cerebellum showed an unusual vascularity of the dentate nucleus on both sides. The vessels were numerous and somewhat thickened, and surrounded by a slight round-cell infiltration, and there was also some fresh hemorrhage about some of the vessels.

To summarize the pathological findings in this case, there was an area of softening in each putamen, which caused degeneration in the fasciculus of Türck, on the right side. There was no degeneration in the motor fibers of the internal capsule, the pons, medulla oblongata or spinal cord.

Associated with these pathological findings, the tremor of the right arm and the ataxia of the arms and legs are worthy of some discussion.

In regard to the first of these symptoms, namely, the involuntary movements of the right arm, which resembled a post-hemiplegic tremor, it is interesting to note that lesions of the lenticular nucleus have been thought by some to cause post-hemiplegic chorea. Among the first to describe cases of this nature was Demange,[1] who, in 1883, reported 11 cases with autopsy, in 6 of which the lenticular nucleus was the seat of disease.

Sander,[2] in 14 cases of athetosis collected from the literature, found the corpus striatum involved in 7 cases. In the case described by Grasser and Rauzier[3] the lenticular and caudate nuclei, and the internal capsule were partly destroyed by an area of softening.

The putamen was symmetrically diseased in one case described by Hebold,[4] and in a second case the claustrum was also involved on both sides. He quotes Nothnagel who collected cases of athetosis and hemichorea, in which the lenticular nucleus and the internal capsule were involved. In Anton's[5] case the choreiform and athetoid movements were on the same side as the lesion in the putamen, and in Jacob's[6] case the contralateral lenticular nucleus was the seat of the disease.

Many cases have been cited in which, besides the lenticular nucleus, the thalamus has also been involved. This was true in 50 out of 78 cases collected by Bidou.[7] In about 100 cases studied by v. Monakow,[8] in 70 per cent. to 80 per cent. there was also lesion of the internal capsule.

On the other hand, bilateral lesions of the lenticular nucleus may occur without causing tremor, as in Edinger's[9] case, and in 2 cases of Hebold.

If foci of softening or other lesions of the lenticular nucleus cause tremor, it is hard to reconcile entirely the findings in our case with this statement, as tremor was only present in the right arm, while the putamen was diseased on both sides. Of interest in this connection is the opinion of Hebold who believed that a different explanation for the motor disturbances than injury to the putamen, must be looked for, at least as far as the outer portions are concerned.

It should be remembered, however, that in our case the lesion caused much greater destruction in the left putamen than in the right, involving, in the former, a large part of the posterior third of the putamen, while on the right side the lesion was linear in shape and occupied the external portion, the destruction of the putamen being less extensive. It may be that the lesion on the right side was not sufficiently large to cause disturbances of motion, while the damage to the left putamen, being much more extensive, was sufficient to give rise to a tremor in the right arm. This is of course somewhat theoretical.

At least the findings in our case permit the statement that bilateral lesions of the putamen do not always cause bilateral disturbances of motion.

The pathology of post-hemiplegic disturbance of motion has not been definitely established, and the views held upon the question are far from uniform. On the whole, however, the weight of evidence is in favor of placing the lesion in one of several localities, and preferably in the thalamus, or in the pons, in the neighborhood of the superior cerebellar peduncles and red nucleus. Many observers have, however, described tremors, choreiform movements and athetosis post-hemiplegic in type, in organic disease of the cerebellum, medulla oblongata, cortex and spinal cord.

Lesions of the thalamus have been regarded by many observers as the chief cause of these movements. Since Charcot,[10]

in 1873, reported 3 cases with autopsies, in which the lesion involved the posterior portion of the optic thalamus, caudate nucleus, and corona radiata, a number of cases of this character have been described, namely, those of Stephan,[11] Sander, Gowers,[12] Bidon, Kahler and Pick,[13] Raymond,[14] Bischoff,[15] and Leyden,[16] who found various lesions affecting the thalamus, with or without involvement of the surrounding tissues.

In 100 cases studied by v. Monakow, it is stated that, in the majority of cases the lesion was found principally in the thalamus. In another group of cases the retrolenticular portion of the internal capsule and the thalamus were the seat of disease, while in the remainder of the cases the posterior portion of the corona radiata and the lenticular nucleus were involved.

Raymond, however, on the contrary, noted in 35 cases of hemiplegia, involvement of the thalamus in only 4 cases, and Sander believed that there were only a few cases in which the thalamus alone was involved. Moreover, the thalamus and the internal capsule were affected together in 20 cases in the literature studied by Stephan.

The relation of the post-hemiplegic movements to the cerebellar peduncles, the tegmentum, and the red nucleus, was probably first described by Benedickt[17] who, in 1874, reported 22 cases of hemiparesis and chorea caused by a lesion in the crus cerebri, at the height of the oculomotor nucleus. Halban and Infeld[18] added to these 2 cases of their own, in one of which a tuberculous focus had destroyed the red nucleus in large part, extending almost to the decussation of the superior cerebellar peduncles. Henoch[19] also observed a case with tremor, hemiplegia, and crossed oculomotor palsy, in which there was found a tuberculous lesion of the pons and corpora quadrigemina, and Bergen[20] collected 10 cases in which the cerebellum and superior cerebellar peduncles were destroyed, causing incoördination identical with hemichorea on the same side as the lesion.

The superior cerebellar peduncles, the red nucleus and the tegmentum were diseased in a case reported by Bonhoffer,[21] who believed that choreiform movements and athetosis were due to a lesion of the centripetal fibers extending from the cerebellum to the cerebral cortex, giving rise to a functional disturbance of the motor cells of the cortex. He advanced three reasons for accepting this view, namely, (1) the constant presence of a lesion of

the superior cerebellar peduncles, or its projections into the subcortical ganglia; (2) the hypotonia of the musculature; (3) the disturbance of voluntary motion in chorea. He thought it probable that, in consequence of a more or less complete interruption of the fibers, the centripetal impulses only in part reached the cortex, while others passed along the centrifugal fibers and gave rise to automatic movements the character of which depended upon the nature and extent of the lesion.

Pineles[22] reported 2 cases of athetosis which he considered of cerebellar origin, and cited the case of Oliver, in which there was a glioma of the vermiform process in a case presenting tremors of the arms and legs. He cited also Menzel's case of choreiform movements with atrophy of the cerebellum, a similar case of Meynert's, Ceni's case of atrophy of the right hemisphere of the cerebellum, and right cerebellar peduncle, with a hemorrhagic focus in the left red nucleus, and Sander's[4] case in which there were choreiform movements on the right side, caused by a gliosarcoma in the right cerebellum, destroying the corpus dentatum. Pineles concluded that in many cases choreiform movements and athetosis were caused by disease of the cerebellum, or the cerebellar peduncles. Other cases of a similar nature have been reported by Kirschoff,[23] Bonhoffer, Huppert,[24] Muratoff,[25] and Hammarberg.[26] In Hammarberg's case the movements were pendulum-like.

Lesions in the medulla oblongata, causing hemichorea, were described in Bidon's cases (collected from the literature), in v. Monakow's case and in the cases of Fropier,[27] and Broadbent (in v. Monakow) and Henoch.

Cortical lesions have been described as causing athetoid and allied movements in the 2 cases of Demange, and those of Balfour,[28] Beach,[29] Korella,[30] and Hudovernig.[31] Some of these cases are, however, so meagerly reported as to be of little value.

Opposed to this is the statement of Exner (in Frey) who collected 167 cases of cortical lesion without one instance of post-hemiplegic tremor. In this connection, as bearing upon the irritation of the motor tracts as causing post-hemiplegic movements, must be mentioned the cases in which the internal capsule has been the seat of the lesion, as illustrated by the case of Demange, and those collected by v. Monakow, Bidon (29 cases), and others.

Finally, Eisenlohr[32] and Anton describe cases in which spinal

lesions were mentioned as causing the movements under discussion.

Several theories have been advanced to explain the occurrence of these movements, among the first of which was that of Charcot, who believed that they were due to disturbances of a "chorea-bundle," a view long since abandoned.

Kahler and Pick, who concluded that cysts or areas of softening in the thalamus, with or without involvement of the internal capsule, give rise to hemichorea and other disturbances of motion, believed that these were due to irritation of the pyramidal fibers, a view also held by Sander,[39] Demange, Kolisch,[33] Hudovernig, and Nothnagel.[34] Frey[40] and others, however, object to this theory on the ground that the usual result of irritation of the pyramidal tracts is to cause convulsions and spasms; that hemiplegia is a frequent occurrence, while choreiform movements are rare; that lesions removed from the motor tracts cause these symptoms; that paresis is absent in some cases; that it is improbable that complicated movements should remain circumscribed if thus explained (v. Monakow); that the head is affected in deep-seated lesions, and that these symptoms are infrequent in lesions of the lenticular nucleus (Frey). Furthermore, it is advanced by some authorities that cortical lesions rarely cause post-hemiplegic movements, although the cases of Demange controvert this statement.

Von Monakow does not subscribe to the view held by Kahler and Pick and others, that these movements are the result of irritation of the pyramidal fibers which is conveyed to the cells of the anterior horns of the cord, but in his opinion the tissues surrounding the lesion, as in the subthalamic area, the tegmentum and the pons, become irritated, and that this irritation is transmitted to the motor cortex.

Bonhoffer, in explaining the symptoms, believed that the different choreiform movements are caused by alterations of the impulses which normally pass to the cerebral cortex through the tegmentum. As already stated, he believed that it was plausible that impulses pass centripetally to the cerebral cortex in part only while others pass directly in the centrifugal motor fibers, and give rise to automatic movements.

According to Frey and Stephan, the thalamus is a coördinating center which, when disturbed, gives rise to post-hemiplegic disturbances of motion. Frey believed that the cause of these

movements is to be found in lesions of the thalamus and hypothalamic region.

Anton held that automatic associated movements originated in the posterior part of the optic thalamus and its connections, while the corpus striatum inhibited these movements. Therefore, lesions of these ganglia cause a lowered inhibition, and in consequence an increase of the automatic movements, or chorea.

Halban and Infeld believed that fibers going through the red nucleus have to do with a complicated mechanism, and that disturbance of these fibers gives rise to the automatic movements.

According to Sander, disease of the thalamus was the cause of athetosis in his case, and he expressed the opinion that lesion of the motor tracts causes in part of the fibers incomplete interference with the passing of nerve impulses, and that the accumulation of these impulses centrally from the lesion, *i. e.*, in the cortical cells, becomes so intense from time to time as to overcome the resistance at the point of the lesion, giving rise to movements of a rythmical nature.

Pineles inclined to the belief that while these movements may be associated with irritation of the pyramidal fibers, many of the facts speak for the relation of chorea to the superior cerebellar peduncles. He believed that the choreiform movements were the result of loss of function (an "ausfall" symptom).

One is struck, in studying the cases and the various theories above cited, with the fact that the majority of lesions described affected centers or fibers which are connected directly or indirectly with the cerebellum, and it does not seem improbable that, in some way not perfectly clear, the function of the cerebellum, at least as far as co-ordination is concerned, may be disturbed, and, as a result, give rise to choreiform movements, athetosis and tremor, which, according to some observers at least, may be looked upon as allied disturbances of motion.

The findings in our case may add weight to this view, if we are correct in assuming that the lenticular nucleus is associated with the cerebellum by fibers which pass from the lenticular nucleus on one side, to the contralateral cerebellar hemisphere by way of the ansa lenticularis.

Obersteiner[35] believes that the lenticular nucleus is connected with the lower olive on the same side, and the opposite cerebellar hemisphere in this way, and Mills[36] states that the corpus striatum

is connected with the cerebellum through the red nucleus, the pontine nuclei, and olives, and the other basal ganglionic deposits.

According to Dejerine[37] part of the fibers of the ansa lenticularis penetrate into the antero-internal part of the capsule of the red nucleus, which is composed of fibers from the superior cerebellar peduncles.

These facts justify the assumption, to some extent, that the lenticular nucleus may have some function relating to co-ordination through its connections with the cerebellum which it may have at least indirectly, if not directly, by reason of its relation with the red nucleus. If, then, as a result of a lesion of the lenticular nucleus, the fibers associated directly or indirectly (through the red nucleus or otherwise) with the cerebellum be interrupted, and thus the functions of the cerebellum be impaired, may not disturbances of motion arise expressing themselves in involuntary movements or ataxia?

This brings us to the consideration of the ataxia of the arms and legs which was present in our case, and offers a possible explanation for this symptom, which is not explained in any other way by the pathological findings already described.

The tremor in our case, we believe, was not hysterical, as the patient exhibited no other stigmata of hysteria, although of course tremor may be the only manifestation of hysteria, as in the case described by Mitchell and Spiller.[38]

In Conclusion, we believe:

1. That the lesion in the left lenticular nucleus may have been responsible for the tremor of the right arm in our case, and the association of unilateral tremor with bilateral lesion may be explained by the fact that the lesion in the right putamen was not extensive enough to set up sufficient irritation to cause this symptom on the left side.

2. That post-apoplectic disturbances of motion may be due to lesions of the lenticular nucleus, the optic thalamus, and the pons in the region of the superior cerebellar peduncles, and of the cerebellum, the cause being, in all instances, a disturbance of co-ordination. When the lesion is extra-cerebellar the cause of this disturbance is possibly an indirect result of destruction of fibers related to the cerebellum directly or indirectly through the red nucleus.

3. That the ataxia in our case may have been indirectly of cerebellar origin.

It must not be forgotten, in drawing conclusions from an analysis of the above cited cases, that there are on record instances in which lesions of the posterior portion of the thalamus ("hintere Sehhügelgegend"), red nucleus, superior cerebellar peduncles, dentate nucleus, and cerebellum have occurred without causing athetoid and choreiform movements (v. Monakow).

We gladly express our thanks to Dr. W. G .Spiller for valuable assistance in the study of the specimens.

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Society Proceedings

AMERICAN NEUROLOGICAL ASSOCIATION.

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The President, Dr. Hugh T. Patrick, in the Chair.

(*Continued from page 656.*)

A STUDY IN HEREDITY.

By Dr. James Jackson Putnam.

This paper discusses the results of the study of a large family, most of the members of which, through several generations, have been well known to the writer, many of them personally.

The husband and wife, with whom the history begins, were persons of active minds, but showing symptoms in the former case of neurasthenia, in the latter of hysteria. One object of the inquiry was to ascertain to what extent these disorders had been inherited and whether they gave place in subsequent generations, to more serious affections of the nervous system. Apparently the former of these inquiries could be answered in the affirmative, at least so far as the neurasthenia was concerned, the latter in the negative. More serious conditions of the nervous system and of nutrition have occasionally occurred, but apparently only in a sporadic manner. Other points of interest are brought out.

HEREDITY IN DISEASES OF THE NERVOUS SYSTEM.

By Dr. Philip Coombs Knapp.

The teachings of Weismann have shown that there is grave doubt whether acquired characteristics may be inherited, and have pointed out that one of the chief factors in the inheritance of disease is the pathological change in the germ plasma from various causes, such as intoxication, infection, constitutional disease or local disease of the generative tract.

The teachings of Galton and Mendel have further shown the probability that morbid characteristics tend to die out in later generations. The data as to the inheritance of nervous disease are untrustworthy and unsatisfactory. Simple heredity is rare, and is seen most strikingly in certain rare diseases, such as Friedreich's ataxia, Huntington's chorea, etc. The fact that a number of cases of these rare diseases may exist in a single family shows that hereditary influence is an important factor, but it does not explain the original appearance of the disease, or the occurrence of sporadic cases. De Vries' theory of sudden mutation may possibly explain the original onset of the disease, which leads to such changes in the germ plasma that it is reproduced in the descendants.

In the more common forms of nervous disease similar heredity is rare, but much stress has been laid upon the neuropathic predisposition.

The statistics of heredity in insanity are vitiated by the tendency to

regard insanity as a single disease, and to class all varieties under the one heading. An inquiry was made as to the frequency of neuropathic and similar heredity in epilepsy. The statistics were found to vary from 11 to 87% of neuropathic heredity, and from 1 to 37.2% of similar heredity. The neuropathic heredity in epilepsy, however, is no greater than the amount of neuropathic heredity found in normal individuals, although the similar heredity seems somewhat greater than the percentage of epilepsy in the community at large.

Another fallacy in this method of estimating morbid heredity is that many forms of disease, due to many different causes, are brought forward as indicating inherited weakness of the nervous system, and the etiological factors occurring in the patient's own life are too often disregarded. It seems doubtful whether any acquired mental or nervous disease of the ancestors can be the starting point of a pathologically tainted family, or even of sporadic cases of disease in the descendants, unless there be some general or local cause of injury to the nerve plasma.

The whole question of heredity in nervous disease requires thorough investigation, and the influence of heredity is certainly not as great as at present regarded. Even when morbid heredity exists, the tendency to disappearance of the morbid taint is considerable, and cases of nervous disease in the family, or even in the individual himself, are by no means proof that there is morbid heredity, or that the disease will be transferred to his descendants.

Dr. D. J. McCarthy said he was very much interested in both of these papers, and more particularly in the paper on the rather extensive study of Dr. Putnam's family. He said that anyone who has given this subject of heredity any thought at all and has looked into the subject for scientific data must be impressed with the looseness with which the subject has been investigated by neurologists and by psychiatrists. The great deficiency, so far as he can study out the matter, is the narrow limits to which those studying the feature of heredity in nervous and mental diseases confine themselves. The most that you can get statistics on in the average clinic or in the average study of heredity, is the general nervous condition of the parents. As was called attention to, we limit ourselves entirely practically to the neurological standpoint or to a study of the nervous system. While it is perfectly true that the acquired characteristics, so far as the nervous system is concerned, are probably more often transmitted than personal characteristics (morphological and visceral), we must not lose sight of the fact that this heredity more often depends upon a physical than a nervous basis. Probably the most extensive study in heredity that has been made is in relation to idiocy and similar disorders. Some 28,000 cases have been collected in which, for instance, the most important etiological factor, as far as could be determined, was pulmonary tuberculosis in the ancestors. The question of cancer became a non-important factor, dropping from 3 to 5 per cent. If we are going to study heredity we have to study it not from the standpoint of the nervous system, but we must consider the nervous system not as a system by itself, but as a part of the economy, and the statistics are not valuable unless there is taken into consideration the question of visceral disease, and not only the question of visceral disease in general, but especially the question of visceral disease at the time of the birth of the child. The condition of the mother at the time of the birth of the child is of especial importance. In making a study of this kind the important factor of the neurosis cannot be disregarded.

Dr. F. X. Dercum said that he is entirely in accord with Dr. McCarthy. We must study the organism as a whole, and the occurrence of such a fact as a myxedema is of as much importance as any other incident in the life history of a family. We all of us know that the ductless glands play a rôle in the economy, and that the thyroid gland exerts an influence on the nervous system. And besides, the signs presented by children who have this inherited nervous weakness are essentially those of arrest and feebleness of development, feebleness of resistance, as shown by the history of infectious diseases of childhood with prolonged convalescence and feebleness of resistance in a multitude of other ways. It is the organism as a whole that we should study, with all the incidents of infection, with all the incidents of diseases of various viscera. Doubtless all of these factors play a rôle.

Dr. E. Riggs said that apropos particularly of Dr. Knapp's paper he recalled a conversation with Dr. Savage some years ago, who said: "If I believed the theories of Maudsley I could not practice medicine. For many years I have watched clinically, the manifestations of this so-called heredity. I have seen children who were born, the fathers being insane, children born the mothers being insane, children born both parents being insane, and I have watched these children for years and have never seen any psychopathic developments."

Dr. S. Weir Mitchell said he was tempted to say a few words from quite a different point of view from that taken by his friends. Perhaps too long experience had enabled him to have before him the histories of a great many families, especially in the upper social life of his own city. There would be found among the recorded cases which it had been his habit to note carefully since he was a young man, a multitude of histories running through many generations which enabled him to take on the whole a far more hopeful view of this question of heredity than is usually held. The interesting part of it to him was one which we too rarely hear brought up in any of our meetings and usually miss here, and that was the therapeutics of hope, so to speak, in families where there has appeared in one generation perhaps a succession of cases of some form of distinctly neurotic malady, we will say insanity. He said he was thinking now of a particular family, historically well known, with which he was very well acquainted and knew its history for fifty or sixty years. There appeared, and had appeared for two generations before, in each successive twenty or thirty years, one, two or three cases of insanity, usually what we call melancholia. There had been one or two suicides. Then two generations ago the trouble ceased; some happy marriage or change occurred, and from that time to this there have been many fruitful marriages in the family, there have been absolutely no nervous disorders, and what looked like an exceedingly dangerous probability in regard to those people has resulted in perfectly wholesome young men and young women. The matter he wished to speak of particularly was the question of treatment in the largest sense. There have come to him in his lifetime many cases where people have said, "We have had in our family insanity; there have been many cases. What am I to do to insure the future of my offspring?" That was the question he wished to see dealt with in our discussions. There come other occasions where we are called upon to decide the question of marriage. A woman who has been insane once comes to you with the question, should she marry, saying "there have been such and such cases in my family. Am I justified in getting married?"

Dr. Mitchell related the history of a family, exceedingly well known to him, of extraordinary and almost romantic interest. He was consulted a great many years ago by two maiden ladies who looked like Spanish people. They were people very well to do in the world, but not in the highest social class. They had with them a young woman, a younger sister by many years, the product of the second marriage of their father. These ladies related a history which was written down in a book kept for 150 years, since their migration to America. In that time there had been in the family nine suicides, there had been countless cases of drunkenness, there had been seven persons with epilepsy, and these two sisters themselves had both been in asylums, one of them once and one twice, and felt prepared to go again as they would go to get the services of any ordinary physician. They said: "We have made a compact between ourselves. We are the last of this family, and we have resolved never to marry." They never did. They also said that they had resolved that the young sister should not marry. The result was what might have been anticipated. The young woman formed a very proper love affair with a young German who was quite comfortably off. He was a decided blond, the perfect German type. When this was discovered, to the dismay of the elder women, they asked Dr. Mitchell what they should do. After considering the matter he said they had better take the risk, for the reason that this young woman did not resemble them at all, and probably took her physical and mental characteristics from her mother's family. They accepted his advice, the young people married, and are now fairly old married people and have had a large and happy family with no kind of neuropathic display.

Dr. Mitchell said that before he sat down he wished to express again his great disappointment at not hearing the therapeutic aspect of nervous diseases more often brought up at these meetings.

Dr. Langdon said we are all aware that the shifting line between the normal variation and what we may perhaps, for our purpose, term the pathogenic variation, is an exceedingly important matter. It comes home to us frequently in our relations with the public. He simply wished to direct attention to a line of inquiry bearing upon this subject which he did not remember any one of the speakers referred to; namely, the investigation of what we may term the potentiality, the dynamic potentialities, if you please, of these patients; in plain words, how early they attain the period of usefulness and how long it endures. It is a question which has at least a bearing on the neurasthenic and other types mentioned by Dr. Putnam.

Dr. G. Hammond said that the therapeutic aspect of the question is one which has interested him for many years, and it has been his custom in all cases of children who seem to have inherited a neuropathic constitution to treat them for years with a process of physical culture. He has not taken the children simply of neurotic parents, but has taken those who showed neurotic constitutions by having had one or more convulsions or chorea or migraine or some other nervous affection. He has treated these children for years with systematic exercises, making the physical development paramount to the mental, insisting that they live a most hygienic life, with the greatest amount of sleep, with a simple diet and proper physical outdoor culture, and in many of the cases where the parents have followed this method of education he has seen epileptic children grow up to be healthy, vigorous, strong, useful men and women, having healthy

children of their own; and he believes that this is the one way in which the neurotic constitution can be eradicated, and he believes that if it is consistently carried out by the physician and parents we can rescue many of these neurotic children and make them strong, healthy and vigorous.

Dr. L. P. Clark said that he believes there is a constant law applying to epilepsy, for the transmission of the disease. He said, if statistics do not prove that, as he was bound to admit from the testimony submitted here as well as elsewhere, that they do not, then it is so much the worse for statistics. The specialist in statistics needs to be called to our help. He thought any one having a long experience in treating epilepsies will be very conscious of the fact that there is a definite law which undergoes a wide variation. It seems to him that neurologists could pursue a line of investigation with great profit if they would take general principles which are accepted by a great community of people as fundamental facts, treat it as a fundamental fact and try to discover why it is so, ascribing these views to popular ignorance does not satisfactorily explain them away.

Dr. A. Meyer said that in attempting to plan the study of the question of heredity on a large material he had continuously come across the difficulty of ascertaining even the ontogenetic factors of etiology. The history of the individual brought in is apt to be very faulty, and before one can hope to get very far with heredity problems we must be on a very much better footing concerning the ontogenetic etiology in concrete cases. Any one who tries to formulate the etiology of any patient under observation knows how extremely difficult any such formulation is at the present time. And naturally the sizing up of the heredity factors will be correspondingly difficult. Dr. Meyer said that under all circumstances he would advise not to pay too much attention to any results which are not obtained on specified instances, such as Dr. Weir Mitchell has recorded, and such as Dr. Putnam has brought to our notice. They are the concrete experiences in which something definite has been stated. Of course we come across a great difficulty in that it is practically impossible to induce our co-workers to do that which we ourselves would consider incumbent upon us; namely, to get the pedigrees of all cases used for generalizations.

Dr. C. L. Dana said he had occasion to pay some attention to this subject last winter, and he was very glad that the matter had been brought up at this session. The results of his own studies agree with those given by the speakers to-day. When he got through working over the subject of heredity the most dominant impression left was that we have to study statistics again and get many more facts. He said he wished to state one instance which he had found in his personal experience to be different from that given by ordinary writers; that is the relationship, for example, of alcoholism to insanity. Nearly all text-books say alcoholism is the cause of from 20 to 25 per cent. of the cases of insanity. He took 300 cases of insanity which came under his personal experience, about which he knew very well, both the persons and the families. In these the alcoholism as an hereditary factor was less than 5 per cent. So far as the practical question which Dr. Mitchell has raised comes to one, Dr. Dana said that he tried to formulate certain rules, and it seemed to him as a result of his own personal studies that the only serious reasons against marriage of so-called psychopathics was when there was a direct history of insanity or psychosis or serious neurosis; that even this is not enough to cause a prohibition of marriage if it was on one side alone. If, however, it was

direct on both sides, it should cause us absolutely to advise against marriage. If it was on both sides in indirect heredity it should cause some question, but not, he thought, actual and rigid prohibition.

Dr. Weir Mitchell asked Dr. Dana what he meant by indirect heredity.

Dr. Dana, replying to Dr. Mitchell, said he meant by indirect heredity where the uncle or cousin or grand-uncle, was insane. If there were a great many cases of indirect insanity on both sides the problem of marriage is serious. In other words, if the father had a psychosis alone and the mother is healthy there is a very large chance of the children being pretty nearly as well as others. If both father and mother have serious psychoses he thought marriage should be forbidden.

Dr. L. F. Barker said it seemed to him very difficult in this question of heredity to distinguish what is heredity from what is due to early environment. He said he was sorry he came in too late to hear all the papers in discussion, but he was wondering if much emphasis had been laid upon the influence of early environment. It seemed to him that what we have learned about tuberculosis is also applicable to a certain extent to the doctrine of functional neuroses and psychoses.

Dr. G. L. Walton said the subject had been so thoroughly discussed from every other point of view that he could not help just mentioning one point which had been left out. Dr. Hammond came nearest to it in speaking of the instruction of neuropathic children in the direction of physical development and neglecting the mental development. It is of the greatest importance to train neuropathic children in the direction of cultivating the commonplace ideal, of discouraging their giving way to fussy dislikes, as to odors and sounds, and to indecision about doing a thing for fear it will not be right; in other words, to the New England conscience. Such children should be, for example, taught that it is better to do a thing wrong sometimes than it is to be undecided for half an hour which of two things to take up. The neuropathic children need cultivation in all such directions to establish the commonplace, easy-going ideal and to eliminate the exaggerated ego.

Dr. J. J. Putnam said he was glad this subject had come up in this particular way, as it is certainly a very important one. In closing his part of the discussion he said he would like to say a few words which would bear on Dr. Knapp's very interesting communication. He felt that if the difficulties are so great that we can hardly solve them or solve them satisfactorily in a scientific way, there are certain broad facts which must impress themselves on every one. In studying the histories of large families we see these degenerative signs showing themselves in one and another generation like a bit of paper on a stream which appears here and there, and then disappears, perhaps to be dissolved, or possibly to reappear again a good deal later. It is important to discover the principle on which this sort of thing occurs, but we cannot take the facts themselves as indicating the danger of a racial or community degeneration. If one takes a certain community, closely shut in from the outside world, one can observe that a species of degeneration does go on. If, on the other hand, one takes a large community, one can pretty quickly see that it does not degenerate; that the tendencies which make for progress are greater than those which make for regression. The larger the number of the streams that come in the less is the likelihood that the general stream will suffer in any particular way. Large and active communities tend to improve, and that, too, in the face of the fact that the complexity of life tends to increase. The

complexity of life does not seem to increase the tendency to degeneration. Individuals habituate themselves to poor conditions to an extent that is amazing, and even when living in slums, in bad air and in unhygienic surroundings, in the midst of the clicking telephone, etc., they do not degenerate. A nation does not degenerate as a nation provided the number of its citizens is sufficiently large. Dr. Putnam said he thought there was another large fact which is very important. We see one or another disease, epilepsy or migraine, reappearing through a certain number of generations, and we trace it back to an original case. With that case a certain tendency came in, and we may look upon the first case as a sort of focus. Can we say that each reproduced copy of the first case is to the same extent a new focus? This is doubtful. Tendencies die out. Like the bit of paper on the stream they gradually become softened and disappear. If this was not so we should all of us be epileptic and have migraine. Another point is this: Dr. Woods, of Boston, wrote an interesting research on heredity as traceable among the crowned heads of Europe, where the histories of the different individuals are very well known, and the conclusion he came to there was that education hardly counted for anything, and heredity counted for everything. Dr. Putnam said he thought there was a fallacy in this reasoning, due to the fact that Dr. Woods divides the people of whom he treats, in regard to their various qualities, into only ten divisions. It may be true that a given individual cannot easily raise himself through education from one of these divisions to another, and yet he may be able to make change enough in himself to affect greatly his capacity for usefulness and happiness. The historian might easily overlook an indication of improvement which in the eyes of a neighbor or a co-worker would be of great importance.

As regards the question of marriage which Dr. Mitchell had referred to, it seemed to Dr. Putnam that there is another broad consideration which we ought to bear in mind. There is some truth in the fact that fine qualities go with qualities which are less desirable, as genius with morbid excitability. With the power of organization and co-ordination which makes possible the accomplishment of great results there is undoubtedly a danger which is sometimes very manifest, but it would not do to conclude that we ought to throw over the chance of gain on account of the chance of the loss. He said he had in mind a family like that described by Dr. Mitchell, where drunkenness and psychoses have been prevalent, where nevertheless the benefit brought to a community by one or more members has been so great as to more than counterbalance all the damage done by the others. We ought to consider the community as a whole, and not only the individual case. The community can afford to make great sacrifice for the sake of our great leader. The Jukes family has been much referred to as one showing the baleful influence of heredity; the number of criminals and paupers they have produced has been spoken of many times, but one careful historian of the Jukes family has noted that education, environment and imitation played a very large part in this result.

As regards the hygienic life, Dr. Putnam said that while he agreed with Dr. Hammond, he agreed still more with Dr. Walton. We are not educating people to live on farms. We are educating them to live in the cities and in the midst of people.

Dr. P. C. Knapp said he thought that by careful study of the individual cases, with all the details of the life history in the antecedents and the life

history in the individual, even if we find cases of nervous disease and of mental disease in the antecedents or even at times in the individual himself, we can sometimes hold forth great hope. We can assure that patient that there is no likelihood of an occurrence or recurrence of nervous or mental diseases in himself, or that, if he marries, there is no likelihood of such an occurrence in his descendants. That is often difficult to decide, but in certain cases he thought we could speak with confidence, and the absolute pessimism of the French teaching on heredity leaves out any element of hope. Dr. Knapp said he laid much stress upon the whole life history of the individual, the importance of faulty training, of faulty nutrition and of example and environment in the production of a neurotic, unstable disposition. He believed, however, that the important thing we must consider is the physical status of the ancestors. In spite of what Dr. Clark and Dr. McCarthy have said, it is by no means susceptible of proof that an acquired neurosis in the parent will be followed by a similar neurosis or any other form of functional neurosis in the child, but if the parent has a defective physical condition leading to pathological alterations of the germ plasm, that may produce a weakling physically in every way, and that weakling may develop neurotic disease. Dr. Dercum's reference to myxedema leads to a specific application. If a parent has myxedema, and after the development of that myxedema, if that should be possible, gives birth to a child, that child might very naturally be expected to be a weakling on account of pathological alterations in the germ plasm from the toxic processes involved in the myxedema, but if after the child is born the parent develops myxedema, especially if we could prove it was myxedema resulting from some local injury to the thyroid gland, the question of myxedema as a factor in heredity would be absolutely thrown out of court. We must make a detailed study of the individual and his family, taking into consideration all infections, all diseases, and all the results of education, of nutrition, of feeding, of the whole life and the special forms of disease which develop in the family, before we can come to definite scientific conclusions as to the importance of hereditary factors.

(To be continued.)

NEW YORK NEUROLOGICAL SOCIETY.

April 2, 1907.

The President, Dr. Charles L. Dana, in the Chair.

PRESENTATION OF FOUR CASES OF CONGENITAL CEREBELLAR ATAXIA.

By Dr. Millicent B. Hopkins.

A mother, aged forty-four years, and three children, all girls, made up this group. The father of the children was forty-one years of age and apparently perfectly healthy; his personal and family history were negative, save that a brother died of heart disease. As to the mother's family history, her mother died of cancer of the uterus and her father suffered from rheumatism and was an alcoholic. The mother attributed her condition to the fact that her mother, during her pregnancy, received a severe

blow over the abdomen; otherwise her family history was absolutely negative. Her personal history had no points of interest, excepting that she did not walk until her eighth year. She had had six children and no abortions. One child, a girl, similarly afflicted, died at three years of age of scarlet fever, while one girl of nine was a normal child, and a boy of four years was also entirely healthy. Her deliveries were all easy and normal. The personal history of the children was negative, excepting that they did not walk until very late.

Dr. B. Onuf said the patients shown by Dr. Hopkins, at least the eldest girl, seemed to present some choreiform movements of the face or some ataxia of expression.

Dr. George W. Jacoby, referring to the mother of the children, who was forty-four years old, said he had never seen a case of congenital cerebellar ataxia at such an advanced age.

Dr. Arthur C. Brush said he had one case under his observation at present in a man about forty-six years old. The patient had been in the hospital for ten years, and was employed in one of the wards. The case was a very marked example of cerebellar ataxia.

The President, Dr. Dana, referred to a cast of cerebellar ataxia of a family type observed by him. In that instance, the disease had developed in three generations. One member of the family, in whom the ataxia did not develop until she was about forty, was now nearly seventy years of age. There was no history of any mental defect in these cases. In all cases the ataxia developed late.

SCHLOSSER'S ALCOHOL INJECTION INTO THE FORAMEN OVALE FOR RECURRENT TRIGEMINAL NEURALGIA, AFTER EXTIRPATION OF THE GASSERIAN GANGLION.

By Dr. Otto G. T. Kiliani.

The patient was a man, seventy-three years old, a cook by occupation, and a native of Germany. His family and personal history was negative, with the exception of the fact that he had been suffering from facial neuralgia for the past forty-eight years. He attributed his affection to an injury which he received when he was twenty-five years old. In 1878 his pain became so intense that the second branch of the trigeminus was resected. He submitted to further operations in 1881, 1884 and 1885, and after the latter operation he was free from pain for four years. The pain subsequently recurred, and in 1898 Gasserectomy was performed, which gave him relief from pain for a year. In 1904 another peripheral operation was performed, which relieved him for two months. When he was admitted to the German Hospital, on Dec. 20, 1906, he was having about one hundred attacks a day. After an ineffectual attempt to find the infra-orbital nerve, Dr. Kiliani made an injection of alcohol, according to Schlösser's method, into the third branch, without any result. Accordingly, on Jan. 19, 1907, he made his first injection of two c.c. of alcohol (80%) into the foramen ovale, after which the patient was free from pain for three days. Similar injections were made on Jan. 26, and on Feb. 7 and 14. Since these injections he had remained entirely free from pain.

Dr. Kiliani said that these injections of alcohol into the foramen ovale were done without narcosis, although narcosis had been given the first time, as he was not positive whether he could rely upon the patient to re-

main quiet during the operation. The needle was pushed through the cheek about one inch behind the corner of the mouth, without perforating the mucous membrane. The left index finger was held as a guide behind the last molar of the upper jaw or at its site. One thus felt when the point of the needle struck the external plate of the pterygoid process, along which the point of the needle was pushed upward for about an inch and seven-eighths; there it became arrested, striking the base of the skull. A further lowering of the handle end of the needle permitted the point to travel one-eighth of an inch further upward, where it was definitely arrested. The point of the needle was now pushed backward about seven-sixteenths of an inch, when it entered the foramen ovale. The alcohol was then slowly injected. It was not very painful, but was followed by more or less edema of the eye and the surrounding region. In one instance it was followed by a rise of temperature, which was probably due to some other cause.

After demonstrating the method of this operation on the skull, Dr. Kiliani said that while he did not consider the patient cured without further injections, he was exceedingly gratified with the result. This man had already submitted to one central and repeated peripheral operations without permanent relief, and in such cases, Schlösser's ingenious method of treatment was certainly worthy of a trial.

Dr. Jacoby said the recurrence of the pain after the supposed extirpation of the ganglion in the case shown by Dr. Kiliani indicated that either the ganglion was not removed, or that it had re-formed. The case was a good illustration of the fact that a recurrence might take place after any kind of an operation, and for that reason, a comparatively simple procedure, like the one demonstrated by Dr. Kiliani, was a very welcome addition to our therapeutic resources in dealing with these intractable cases of tic douloureux. While the introduction of this long, straight needle into the foramen ovale was easily carried out, it should be regarded essentially as a surgical procedure, and the promiscuous injection of alcohol or other substances into this or other nerves should be discountenanced. While the ultimate results of the operation were still in doubt, Dr. Jacoby said he thought they would be at least equal to those of other surgical methods.

Dr. Kiliani, in reply to a question as to whether the method had been applied to any other nerve, said that personally his experience with it was limited to the trigeminus. Schlösser, however, with whom the method originated, had also employed it in sciatica, and had never seen any serious accidents result as far as the motor fibers of the nerve were concerned, while he had good results in regard to the sensory fibers. Schlösser had also resorted to it in tic convulsif, injecting the alcohol for that purpose into the facial nerve close to the styloid process, where ready access to it was obtainable. The injections were made into the sheath of the nerve or its immediate neighborhood, and under those conditions it was simply a question of dosage. In dealing with mixed nerves, the method should be employed with the greatest precaution. Professor Schlösser had written a monograph on the subject, which would shortly appear.

Dr. William M. Leszynsky said he had practised the operation on the skull, and was surprised to find how readily one could pass a probe into the foramen ovale, as compared with the foramen rotundum.

Dr. William B. Noyes asked if there was much reaction or inflam-

mation following the injection, and how Dr. Kiliani explained the relief that followed the procedure. Any radical method of affecting a peripheral nerve that was causing repeated attacks of intense pain, either of a spasmodic or a continuous nature, must either cause a degeneration of its nerve fibers, which was distinctly an interstitial change, or cause an accumulation of leucocytes in the neighborhood of the nerve trunk, either as an inflammatory reaction, as in counter irritation, and so reduce any peri-neuritis or neuritis proper.

Dr. Kiliani said he had no explanation to offer as to the effect of these alcohol injections. As a matter of fact, nobody knew what neuralgia was or what produced it, and even careful microscopic examinations had failed to reveal any changes in the involved nerve. It could be safely assumed, however, that the injections of alcohol produced a certain amount of paresis of the nerve, and during the course of the injection the patient described the gradual numbness following the distribution of the nerve. This numbness disappeared in the course of a few days or weeks, but there was no return of the pain. Neuralgia patients were cured by the alcohol injections, inasmuch as the pain disappeared entirely, but a number of cases showed a recurrence after about a year, when a few further injections completed the cure. The operation is so small and connected with so little inconvenience, that the patients are quite willing to undergo the treatment again if necessary.

VOICE RECORDS IN NERVOUS AND MENTAL DISEASES.

By Dr. E. W. Scripture.

A method of recording the voice by a very small capsule was demonstrated, and records of voices in general paresis, hysteria, paralysis agitans, hemiplegia and epilepsy were compared with normal voice records. In these records, the vowels appeared as small waves on a line whose height indicated the rate of expulsion of breath. The regulation of the breath in general paresis was seen to be very irregular, while in hysteria it was erratic.

Curves of occlusives and fricatives were shown. The irregular and uncertain regulation of breath and muscular action were evident here also in general paresis. The faintness of breath action in paralysis agitans was noted. The duration of the sounds was often greatly prolonged in paresis and multiple sclerosis. The melody of speech was obtained by measuring the little waves, each singly. The melody of speech in general paresis showed excessive fluctuation. The peculiar monotony in epileptic voices was so marked that it could be regarded as a regular symptom of the disease. The senile tremolo in melody at the beginning of vowels was shown.

Dr. George H. Kirby said that while attending Professor Kraepelin's clinic at Munich last summer, he had an opportunity to do some work with the methods developed by Dr. Scripture for the study of speech defects and voice alterations. Among the patients upon whom the experiments were made there were some with general paresis in whom one could not find any speech defect by the ordinary tests, yet when the speech curves were studied one could clearly detect an alteration in the vibrations from the vowels, and peculiar fluctuations in the melody not observed in normal persons. These characteristics were quite clear in the records, but could not be detected otherwise, at least not by the untrained ear. The method thus might become really of practical value and

a diagnostic aid. In one patient with pupillary signs and a speech defect the diagnosis was between general paralysis and a hysterical psychosis. The record obtained from this patient showed peculiarities entirely different from those obtained from the general paralytics. The further observation, as well as the anamnesis, made it clear that the case was really one of hysteria.

Dr. L. Pierce Clark said that Dr. Scripture, for a number of years, had maintained that the voice of the epileptic was as characteristic as the facies, or more so, and that he had been induced to undertake with him a careful analysis of the voice in epilepsy. They had found that particularly in the *grand mal* type the voice of the epileptic showed certain alterations which were quite as characteristic as any of the well recognized stigmata of epilepsy. In order to test the accuracy of these observations on the voice in epilepsy, a number of patients at Randall's Island who were subject to that disease were mixed up with others who were suffering from various grades of mental deficiency about on a par with those usually observed in the chronic epileptic, and in nine of these patients out of ten, Dr. Scripture was able to detect merely from the speech whether the case was one of epilepsy or not.

Dr. Clark said that these voice studies seemed to him of immense scientific as well as practical value. For instance, it may be possible in the near future to roughly record in ordinary case-taking the melody, pitch and rhythm curves of speech in various functional nervous disorders, such as those of epilepsy, hysteria, neurasthenia and the like neuroses.

Dr. Onuf asked Dr. Scripture whether in epilepsy he could make the diagnosis from the voice alone, or whether he required the voice record, and what was his interpretation of the latter.

Dr. Scripture replied that while the existence of epilepsy could be recognized, after a little training, from the voice alone, he always preferred to base his opinion on the actual voice records and curves, when possible. When these were once heard and recognized, they were distinct and characteristic. The characteristic voice disappeared after the epilepsy was cured.

Dr. Leszynsky asked whether these characteristic voice curves were present in epilepsy of the ordinary type.

Dr. Dana asked how long after the onset of epilepsy this characteristic voice developed.

Dr. Clark said these questions could not be answered definitely, as the studies were only begun. The cases that had been studied were of the chronic type of epilepsy—patients in whom the disease had existed for periods ranging from two to ten years. The method had not yet been tried at the Craig Colony. He expressed the view that these characteristic voice curves would become as constant and fixed as other symptoms of the disease.

Dr. Scripture said the voice characteristics were apparent in the measurements of the curves long before they could be detected by the ear.

DISCUSSION ON THE ANTI-SYPHILITIC TREATMENT OF TABES AND PARESIS.

Dr. Dana said he had been treating these forms of nervous disease for many years by the use of mercury and iodides in the early stages,

giving the drugs both in mild and heroic doses. Some of the patients had improved under the method while others had grown worse, and whether it was really efficacious was a question that was still open for discussion. In tabes he had seen the treatment applied in the most rigid manner from the very onset without any effect upon the rapid development of the disease.

Dr. George W. Jacoby said that in any discussion of the treatment of tabes and paresis we could not but primarily consider the etiology of these affections, and in so doing it became apparent that their relation to some infection or intoxication was a very definite one.

All clinical observation, as well as other reasons which need not again be adduced, tended to show that the intoxication which was most frequently met with in the evolutionary history of these affections was that which was consecutive to syphilitic infection. This experience must necessarily be followed by the hope that energetic anti-syphilitic treatment might prevent the full development of the disease, or even effect a cure. While theoretical reasoning for or against such an effect would have its place, there could be no doubt that the ultimate decision as to the influence which mercury and the iodides might have upon the tabic or paretic process would have to be arrived at as a result of clinical experience.

Unfortunately, the question was and must remain a very complicated one. The speaker said he had gone over about one hundred of his cases of tabes and one hundred of those of paresis with a view to answering the following questions: 1. Could energetic anti-syphilitic treatment prevent the development of these affections? 2. Could such treatment diminish or remove certain symptoms? 3. Could such treatment cure the disease?

Dr. Jacoby said that as the result of his experience and inquiries the first question should be answered negatively. He knew it could always be said that a cure did not develop because anti-syphilitic treatment had not been inaugurated sufficiently early, yet such negative testimony was of little value in the face of positive cases which proved the contrary. Two such cases stood out prominently among his histories of tabes. One was that of a youth who became infected wtih syphilis. He was treated by a specialist thoroughly and persistently for three years. Ten years after infection he consulted Dr. Jacoby for a hyperesthesia in the territory of one of the branches of the left trigeminus. Careful examination revealed some hyperesthesia in that territory, and an analgesia of the cornea. Beginning tabes was suspected. Anti-syphilitic treatment was again instituted, and for a period of years the patient was more or less constantly under the influence of mercury and iodides, yet in spite of the thoroughness of the treatment he developed pupillary rigidity, pains, loss of foot jerks, loss of knee jerks, ataxia, gastric crisis, and to-day, twenty years after the infection, he presents a fully developed tabes.

The second case gave much the same history, excepting that the diagnosis of probable tabes was made as a result of the occurrence of a transitory ophthalmoplegia of one eyeball. In spite of vigorous antiluetic treatment he had recurrent attacks of ophthalmoplegia of both eyes, together with intense headaches. To-day, fifteen years after infection, he had a fully developed tabes, with permanent ophthalmoplegia, in spite of the most strenuous anti-syphilitic treatment.

Dr. Jacoby said that among his cases of paresis there were many

which had developed in spite of careful treatment with mercury and the iodides from the time of infection. Only a few weeks ago he saw such a case, the paresis coming on six years after infection, in a man who had been under constant treatment by a dermatologist with injections, inunctions and potassium iodide from the very beginning. Such positive cases led him to say that anti-syphilitic treatment, no matter how carefully carried out, no matter how early begun, did not prevent the development of these diseases.

The second question must be answered as follows: Of his one hundred cases of tabes, five showed improvement while undergoing anti-syphilitic treatment, and in those the disease had not progressed since. Yet he was by no means willing to say that this arrest of progress was due to the treatment, for we knew that very many cases showed a diminution of symptoms under any or under no treatment, and we knew that tabes was by no means a necessarily progressive disease. He thought that in about one-third of the cases it was thus progressive, while in the remaining ones the process became arrested, or proceeded so very slowly that the length of life and the economic adaptability of the patient was not materially impaired. All neurologists had seen cases in which the tabic process never progressed beyond the point of diagnostic interest.

The prognosis of tabes certainly was different from formerly, yet he believed that this was due to a variety of factors and to a more intelligent care of these patients, rather than to the effect of anti-syphilitic treatment. He also believed that the differentiation of certain cases of lues spinalis was at certain stages clinically impossible; furthermore, that there certainly existed a combination of syphilitic meningeal affection and real tabes, and in such cases, amelioration or diminution of symptoms might take place as a result of anti-luetic treatment. In not a single one of his cases, however, whether the diagnosis of tabes was made or whether the diagnosis was that of lues spinalis of tabic type was a cure effected. In all of them the diagnosis could be made at all times, after as well as before antileutic treatment.

As regarded the question of the curability or arrest of dementia paralytica, Dr. Jacoby said he had but a few words. The differences of opinion that existed, the fact that the French school had recently claimed so much as a result of mercurial injections in cases of paresis, while the majority of clinicians claimed never to have seen a cured case, could only be understood when we considered the difficulties which existed in establishing an accurate clinical picture of the disease. The more he saw of these cases and the more he studied them, the more did he come to the conclusion that a number of divergent clinical pictures were included under this term. He admitted that there were certain cases of cerebral syphilis or pseudo-paresis which at certain stages he could not differentiate from true paresis. He admitted, furthermore, that he had great difficulty in differentiating certain cases of so-called syphilitic insanity cases with mental excitement and motor restlessness, passing on through stages of mental enfeeblement with muscular insufficiencies into a complete dementia, from cases of genuine paresis. He admitted also that some such cases improved under anti-syphilitic treatment, and ran a very prolonged course. Yet of all his cases of paresis—genuine or pseudo—not one was now living in which the diagnosis was made more than ten years ago, and all of the cases which he had controlled had had

thorough anti-luetic treatment. The speaker said that to him the cure of paresis meant a mistaken diagnosis. He did not believe that this would always be the case, but when cures were effected, it would not be by means of anti-syphilitic treatment, but in consequence of progress in our bacteriological knowledge, and through the discovery of the toxine which caused the disease.

Dr. William B. Noyes thought the whole question resolved itself into a pathological study of syphilis and the toxemias. The pathological changes observed in syphilis—both in its secondary and tertiary stages—were practically embraced by round celled infiltration and gummatous lesions, and in the treatment of those conditions, the ordinary anti-syphilitic remedies could be relied upon; these accumulations of round cells disappeared under iodides, but one could scarcely conceive of mercury or the iodides acting in the same way in dealing with serious degeneration of tissue caused by toxemia. Whatever the cause might be in these cases, whether syphilis or not, the fact remained that we had to deal with a degeneration of the nerve fibers in the cord or the peripheral nerves. The speaker said that in certain cases of tabes, which he had been able to study and follow for years, and where the patients had shown all the typical symptoms of the disease, including the eye and bladder symptoms, and all the classical symptoms of tabes, the autopsy had finally revealed but very slight changes in the posterior columns of the cord, and somewhat more positive changes in the posterior roots and peripheral nerves. The most interesting change that was observed was the development of delicate new connective tissue, supplanting the degeneration in the cord, indicating that regeneration was more in the line of new connective tissue than of new nerve fibers. During every year's time that a tabetic did not grow worse, or improved in his general condition and local symptoms, we could fairly assume that there was such a substitution, to some extent, of new connective tissue for the degenerated fibers. This was practically the condition of the spinal cord in the second or quiescent stage of tabes. It was certainly far better for the patient than a progressive degeneration of the entire posterior columns of the cord.

Dr. Noyes said that in tabes he had seen better results from the use of strychnia and tonics than from the use of anti-syphilitic remedies. In those cases where there was arterial thickening and the symptoms of arterio-sclerosis that were usually present in syphilitics, he thought the administration of small doses of the iodides was allowable, but not with the idea that they would act as an anti-syphilitic, but simply to improve the circulation. No one deliberately treated arterio-sclerosis with large doses of iodides. The speaker said he had seen the ataxia of tabes aggravated by large doses of the iodides, and disappear after their withdrawal. The use of mercury was frequently followed by improvement in various nervous conditions, but this improvement was only temporary unless the disease rested on a syphilitic basis, and even in syphilis the use of mercury was limited to the early stages, and in later stages to occasional use when no careful mercurial treatment seemed to have been carried on. In a tabetic, few physicians would expect to help conditions by the continued use of mercury.

The entire question of the treatment of tabes and paresis rested on the question of diagnosis. In paresis even more than tabes both pathological findings at autopsies as well as careful clinical observations indicated that there was an early sub-acute stage, with the presence of round cells in

the brain tissue, and a chronic stage, marked by degeneration of the ganglion cells of the cortex.

Dr. J. Ramsay Hunt said that his experience with the use of anti-syphilitic remedies in tabes and general paralysis differed but little from that of the previous speakers. Under such treatment, some of the cases improved and some grew worse, so that these remedies should be used with caution in this group of cases. In cases of tabetic optic atrophy, which was regarded as a para-syphilitic manifestation, he had never seen improvement follow the use of anti-syphilitic remedies; on the contrary, he had occasionally observed the condition grow worse under their use. If massive doses of iodide or mercury really influenced favorably the progress of parasyphilitic degenerations of nerve tissue, evidences of this should be observed in this group of cases (the optic atrophies).

Dr. Hunt said it seemed to him that the improvement observed in some cases of tabes and paresis might be ascribed to the disappearance of genuine syphilitic manifestations accompanying the parasyphilis. That these two forms were not infrequently combined was a common observation. It was also possible that the milder meningeal and vascular changes accompanying parasyphilis would be favorably influenced by the use of these remedies.

Dr. Hunt said his personal attitude toward these cases was as follows: During the first and second stage of the parasyphilitic disease he employed anti-syphilitic treatment cautiously from time to time, in the hope of diminishing any meningeal or vascular exudations, and to counteract any genuine syphilitic manifestations. Of the two drugs, iodide and mercury, he gave the preference to the latter.

Dr. William M. Leszynsky said he had seen much harm done in tabes by the use of mercury and the iodides, and he thought the persistent use of these drugs for indefinite periods was not justified. He could recall a number of cases of tabes in which the pupillary symptoms, and especially beginning optic atrophy were apparently aggravated by the use of anti-syphilitic remedies. In any case where he was in doubt between cerebrospinal syphilis and tabes, and the patient had not been subjected to anti-syphilitic treatment, he always gave him the benefit of the doubt by administering these drugs.

Dr. Jacoby, in reply to a question as to how he gave his anti-luetic treatment, said that his method had varied during the past twenty years. In the majority of his cases, the patients had already received thorough anti-syphilitic treatment, some by injections, some by inunctions, etc.

Dr. Kirby said that he had had comparatively little experience in the anti-syphilitic treatment of tabes and general paresis. At the Manhattan State Hospital, at the present time, such treatment was not applied as a routine procedure, but only in certain cases, especially those in which the diagnosis between general paresis and brain syphilis was not clear. Patients suffering from paresis did not come under his observation until the disease was already well established. The anamnesis showed, however, that very few of these patients had been subjected to a thorough anti-syphilitic treatment. The only hospital cases, therefore, in which anti-syphilitic treatment had led to any result were those in which some symptoms of cerebral syphilis accompanied the psychosis.

Dr. Dana said he had in mind certain patients who were affected with syphilis and subsequently developed manifestations of syphilis of the nervous system, such as spinal cord symptoms, and who, after they

had undergone a very thorough and systematic course of anti-syphilitic treatment in Paris or elsewhere, were instructed to repeat the course once or twice yearly as long as they lived in order to avoid tabes or paresis. While he could not speak definitely regarding the efficacy of such a method of treatment, he did know that it was not always effective, as he had seen patients who had developed tabes in spite of it.

Dr. J. F. Terriberry thought the fact had been clearly established that the canonical three-year period or anti-syphilitic treatment was not effective in checking the later manifestations of the disease. For the past five or six years his own plan had been to advise all syphilitics to undergo a two or three months' course of treatment yearly.

With regard to curing tabes or general paresis with anti-syphilitic drugs, Dr. Terriberry thought that was entirely out of the question, at least so far as his experience went. In fact, he did not consider anti-syphilitic remedies indicated in those cases, and he had seen cases of locomotor ataxia improve after the withdrawal of anti-syphilitic medication. He had taken this to indicate that the general welfare of the patient was improved by the withdrawal of those drugs.

Dr. Brush said the worst results he had ever seen in tabes and general paresis were in those patients who had been subjected to energetic anti-syphilitic treatment. He could recall cases of tabes in which the symptoms, after remaining stationary for years, suddenly became aggravated by large doses of mercury or potassium iodide, and the patients went down hill very rapidly. The only good results he had seen from the use of mercury in tabes were when it was given in small tonic doses.

Dr. Dana said that when a patient had once shown any symptoms of nervous syphilis, it was perhaps a wise precaution to tell him that he must take a course of treatment yearly as long as he lives. Such a plan of treatment, carried out in a moderate way, while perhaps not always effective, would be apt to prove beneficial.

THE BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY.

March 21, 1907.

The President, Dr. W. N. Bullard, in the Chair.

A CASE OF PROGRESSIVE MUSCULAR ATROPHY OF THE PERONEAL TYPE.

By Dr. J. J. Thomas.

The boy, H. F., came to the Children's Hospital first on January 27, 1904, when he was two years and two months old. The family was one of Russian Jews, but this child was born in Boston. The parents were healthy. The first child, Solomon, had similar trouble. The second child, at that time a girl of fourteen, and the third, a girl of seven, were healthy.

The patient had had no illness, and there was nothing of importance about the birth.

Four weeks before he was brought in he had had an attack in which he was feverish for four days, and soon after it was noticed that he turned the feet in when walking. At that time it was noted that the

gait was unsteady, and he had double foot drop. A shortening of the tendo-Achillis on the left, so that the foot could only be brought to a right angle. There was wasting of the anterior tibial group of muscles in both legs, but no weakness of the thigh muscles, the child getting up with some difficulty, but not climbing up the legs. The knee-jerks were good.

The following June there was found swelling, redness and tenderness of the left tarsal region, and an X-ray showed only bone atrophy, such as a paralytic would show, and a notch in the lower epiphysis of the fibula, but did not account for the stiffness of the tarsus. This condition was treated by the surgeon, and after a time disappeared. Soon afer this, on June 18, 1904, a general tremor was noticed more evident in the legs and the anterior muscles of the legs showed an absence of reaction to the galvanic current. In January, 1905, the atrophy of the muscles of the lower legs had increased slightly, but it was noted that the strength of the thigh muscles was good. About this time he was given a splint to correct the varus of the left foot, which was evidently due to the shortening of the tendo Achillis. During this time, a little over a year, there had been no extension of the weakness to other muscles, and the paralysis was confined to those of the anterior tibial group, and it was concluded that the case was one of anterior poliomyelitis, from the history of sudden onset after an acute illness and the lack of progress of the disease, and in this in spite of the fact that the presence of muscular dystrophy in the older brother was known.

The child next returned on January 23, 1907, now being five years of age. It was said that there had been no change, and the boy had walked better, but the preceding summer the splints had been left off, as they had become too small and hurt him. Since this was done they noticed the foot turned in more, and for two or three weeks they had noticed the hands being weak.

On examination it was found there was good power of flexion and extension of the legs on the thighs. No power of dorsiflexion of the feet, but some plantar flexion. Marked contracture of the tendo-Achillis on the left and slight on the right. No power to extend the hands in the arms. No weakness of the muscles of the shoulder girdle. Well-marked atrophy of the distal portions of all the limbs, with good preservation of the proximal portions. No pseudo-hypertrophy. Knee-jerks present and fair. In rising from the floor puts hands on knees quite often, but does not climb up the legs, and there is no lordosis.

The atrophied muscles, the tibialis anticus, extensor communis digitorum, peronei, gastrocnemius, all the extensor muscles of the forearm, as well as the vastus externus and internus show no reaction to strong faradic or strong galvanic currents, no reaction of degeneration being present.

Sensation for pin prick was good everywhere and there was no tenderness of the nerve trunks.

The older brother, S. F., now seventeen years of age, was first seen when twelve years old, on June 25, 1902, at the Children's Hospital, but his trouble had been first noticed at the age of three years, when his parents said he began to walk badly and this had increased gradually.

Examination at that time showed marked wasting of forearms, hands and calves. There was a feeble grasp and double wrist drop, but fair flexion, and extension of the forearms and the deltoids were strong. In the lower limbs there was slight power of extension, with flexion stronger, but no movement of the feet or toes. The movements of the thighs on the trunk were good. The knee-jerks, triceps reflex, ankle jerk and plantar reflex were all absent. He could get up from the floor with difficulty and only with support. There was no pseudo-hypertrophy, but the triceps had a slightly lumpy feeling. He walked with balancing from side to side and double foot drop. There was varus on the right, and valgus on the left, and also a marked internal strabismus of the left eye.

Sensation was not tested.

This boy had now become helpless, and the case was probably one of the peroneal type of muscular atrophy, but when first seen showed some of the characteristics of the ordinary type of muscular dystrophy, such as the waddling gait. Later there was marked lordosis also.

The case of the older brother seemed to present some of the characteristics of the transition form lying between the muscular dystrophies and the peroneal type, such as some of the cases reported by Toby Cohn, Hänel and Dänhardt, and these lend strong support to the view that the peroneal type is closely allied to the muscular form. The fact that most clear cases show heredity and that they usually begin in childhood or young adult life, is another argument for the association. Sainton in 52 cases found the onset of the disease 40 times before twenty-two years, with the extremes of two and forty years in the age of onset.

A CASE PRESENTING DELUSIONS CONCERNING THE LIMBS.

By Dr. Courtney.

The case showed certain mental features which constitute its main claim to interest. The patient is an unmarried man of twenty-five years. Before the illness which led to his present trouble he went to school and was intelligent and ambitious. Since his illness he has done nothing. Physically he was of sound habit and given to athletic sports. With regard to his family history there is nothing noteworthy except that his sister, the wife of a physician, has exophthalmic goitre. The patient denied venereal disease, said he used no alcohol and was moderate in the use of tea and coffee. Tobacco he had abused. His bowels have always shown a tendency to looseness. His weight varied from 164 to 172 pounds; his height is 5 feet 8½ inches.

In September, 1903, he went South, where he almost immediately contracted typhoid fever with marked dysenteric symptoms. He entered a hospital and remained there from the middle of September until the following June. He says he was out of his head on three different occasions during this time, but remembers much that transpired. He states that his legs were drawn up by contractures for a long time, and that they were wasted and tender to the touch. The doctors, he says, could obtain no knee-jerks.

On his return home he was completely "devitalized," as his brother-in-law expresses it. He was emaciated to a marked degree, and has

never since fully recovered his weight. What alarmed the family was his mental condition, which will be spoken of later.

Examination of very recent date shows physical findings as follows: Patient tall, pale and cadaverous-looking; gait not characteristic; station with the eyes shut normal. The pupils are somewhat irregular in outline, the right more so than the left. The right is also larger than the left, but both are normal to light and accommodation. The head shows a fine rotatory tremor, and there is distinct nystagmus when the eyes are moved to right or left; none with upward and downward movements. The left brow seems to be less wrinkled than the right; the other facial movements are normal. There is no marked tremor of the tongue, which is protruded straight. The speech is not scanning, but at times seems dysarthric and nasal. There is no intention tremor, and the hand-grasps are powerful—in fact, the general muscular strength is excellent. Sensation is everywhere normal; the deep and superficial reflexes also normal.

The weight is about 140 pounds.

Judged by the quickness and accuracy with which the patient performed certain mental tasks set before him, his intellectual activity seemed unimpaired. His handwriting is normal.

The family state that he cannot be trusted to get the right things at the store, but the patient says his main difficulty is to remember whether it is vinegar or molasses, butter or lard he is after. He states that he always gets the right thing, however.

The striking feature of interest about his mental condition is the peculiar delusion he harbors about his legs. He insists with quiet dignity that the legs he walks about upon are not his legs at all; he knows they are not his, because his had certain marks which these do not possess. He says he thinks somebody else has his legs, but he can't say who. He insists that no amount of argument will convince him of the falsity of his belief.

In coming to a diagnosis in this case the various progressive degenerations were considered but ruled out, partly from the history and partly on the physical findings. The physical signs, such as the nystagmus and the tremor of the head, are most logically accounted for on the basis of unregenerated nerve fibers, while the mental probably came from the general intoxication. The specific delusion about the legs is traced back to sensory perversions which arose during the course of the peripheral neuritic process.

A CASE OF POSTERIOR COLUMN DEGENERATIONS FOLLOWING INJURY TO THE POSTERIOR ROOTS OF THE SEVENTH CERVICAL NERVE.

By Drs. Mitchell and Barrett.

The material for the study came from a patient who died at Danvers Insane Hospital sixteen days following fracture of both arches of the fifth cervical vertebra.

The man was forty-seven years of age. For ten years he had led a vagabond life, frequently confined for drinking habits, and while intoxicated fell and sustained the injury mentioned. For three days he was able to walk about, although there was lessened muscular power. He was admitted to the Danvers Hospital on the sixth day following in-

jury. At that time he had a temperature of 102 degrees, there was almost complete paralysis of muscles of trunk and extremities. Had nephritis, cystitis and a marked impairment of sensations below the level of the second rib, except for the outer surface of both arms. He had been examined by physicians on the second day after the injury, and it was reported that the tendon reflexes were present at that time. All tendon reflexes were absent upon admission to the hospital. Plantar and cremasteric reflexes remained until the day before death. At no time were electrical reactions abnormal.

There was progressive impairment of sensory functions to a point of complete anesthesia over the area mentioned. Patient died of pyelonephritis.

Anatomical findings.—The autopsy showed slight hemorrhagic exudate around the broken ends of bone. The cord was swollen at the sixth and seventh segments so as to fill the canal. There was apparently no bony pressure at any particular point. At the seventh segment the transverse sections of cord studied histologically showed that the injury to the cord was largely confined to the lateral and anterior portions. Many cells in the anterior horns showed axonal reaction, while the cells of the posterior column were normal. Under the Marchi stain the anterior horn was dotted with black, and there were blackened fibers in the anterior commissure, and there were scattered, blackened fibers in the anterior columns and a few in the lateral columns. In the posterior columns the principal change was seen in the entering root fibers. Nearly all the posterior root fibers were blackened. Similar findings were less marked in the sixth cervical segment. The course of the seventh cervical fibers could be traced to the medulla, and the course of the descending fibers of the seventh nerve could be traced downward in the comma tract to the sixth dorsal segment.

The case was interesting clinically as showing loss of deep reflexes following transverse lesion of the cord. It also presented two interesting anatomical points: First, the injury to the posterior columns involving the entering posterior roots of the seventh nerve, and there was no injury to the posterior horn, so that the ascending course of the fibers of the seventh nerve could be traced to the medulla. Second, showed that descending fibers of the seventh nerve were traced in the comma tract to the sixth dorsal segment, lending support to the theory of exogenous origin of the fibers forming this tract.

Dr. Walton said that Dr. Mitchell's case was of especial interest to him on account of the reflexes. The suggestion that the loss of knee-jerk results in a given case from cerebral or upper cord lesion is sometimes met by the objection that there may be coincident lesion in the lumbar region. It is very gratifying that in this case careful examination has eliminated such lesion. The loss of reflex was in this case coincident with the loss of voluntary power resulting from the cervical lesion. The burden of proof rests, therefore, upon those who would deny the dependence of the one upon the other.

The preservation of electrical reactions in such cases shows that the prevailing idea has stood the test of experience, that the electrical reactions have their seat in the spine purely. On the other hand, the long prevailing view that the knee-jerk is purely spinal and merely inhibited by the brain, requires and is receiving readjustment.

THREE CASES OF INVOLUTION-MELANCHOLIA.

By Drs. Southard and Mitchell.

These cases displayed in life feelings of unreality, nihilistic delusions and various other signs which tended to mark them as approximating the features of Cotard's syndrome. The readers did not insist that the characteristic feelings of unreality displayed by these victims of melancholia are never found in patients subject to disease other than melancholia.

The cases discussed were those of a clerk, aged forty-eight; a mason, age seventy-five, and a shoemaker, age sixty-five. The assignable causes were: in the clerk, financial worry; in the mason, senility and domestic worry; in the shoemaker, nothing. All three cases showed more or less arteriosclerosis. The alcoholic and venereal histories were practically negative. The mothers of two patients had suffered at the close of life with continuous depressions. All cases showed ideas of negation, developing in the clerk after slight increase of depression and agitation, with delusions about self and family; in the mason, after gradual senile failure; in the shoemaker, after a slowly developing hypochondria. The senile case made a suicidal attempt.

The most remarkable feature of the autopsies in these cases was the practically normal character of the brains when examined microscopically. The visible arteriosclerosis was confined in all cases to the large branches of the circle of Willis. The senile case showed a small old cyst of softening in the occipital region, the shoemaker showed a mild chronic exudative process.

The brains gave little evidence of general or focal atrophy. No striking alterations in topography or arrangement in layers were detected microscopically. Perivascular pigmentation was found and was attributed by the readers to the results of advancing years rather than of a special factor. Common to all three cases was a neuroglia cell pigmentation in intermediate layers of the cortex. Satellite cell pigmentation was not constantly found. Nerve cell pigmentation was constantly found in the elements of moderate size in all parts of the cortex examined. This pigmentation was strikingly brought out by use of Heidenhain's iron hematoxylin stain. The larger elements failed to show this characteristic pigmentation. The interest of the work, according to the readers, lodged in the somewhat peculiar topographical distribution of the pigment and its absence in the larger elements related with the projection system. The readers promised further work on melancholia.

BRIEF REPORT OF THREE CASES PRESENTING THE FEELING OF UNREALITY (COTARD'S SYNDROME).

By Dr. Harry W. Miller.

Dr. Miller said he wished to offer in as brief a manner as possible a few types illustrating the Cotard Syndrome or a feeling of unreality involving the different fields of consciousness; namely, the consciousness relating to the outside world, the physical and the mental personality, designated respectively by Wernicke as the allopsychic, the somatopsychic and the autopsychic fields.

It was his intention to summarize those cases in which he had observed the feeling of unreality as present in any degree of intensity.

He soon found, however, that time did not permit of any such analysis, so he was forced to offer simply a few typical examples.

He had been much interested in this syndrome since reading Hoch's review of "Some Recent Papers upon the Loss of Feeling of Reality and Kindred Symptoms," and Packard's excellent analysis of his reported case where he offers the hypothesis that the feeling of unreality is due to a disorder of apperception which in turn is due to an associating difficulty of some kind.

These cases are offered not with any intention of attempting to establish an entity, as it is conceded that the syndrome is present in various psychoses which have no other points of similarity, nor did he wish to make any estimate of the prognostic value of this complex of symptoms. Reference was made by Dr. Stedman in his case report at the last meeting of this society of the unfavorable outcome of those cases presenting this syndrome, the contention being held that the presence of the feeling of unreality indicated a dissolution of the personality which was regarded as a bad omen. He could not submit any reliable conclusions on this point from a hasty review of his material. He had found this syndrome most prominent among the cases which we at the present time designate as the involutional type of melancholia. He has also found it in dementia præcox, depressed phase of manic-depressive insanity and in general paralysis.

Case I.—Involutional melancholia, illustrative of the feeling of unreality, involving all three fields of consciousness, the allopsychic, somatopsychic and autopsychic.

L. D. P., fifty-eight years old, on admission to Taunton Insane Hospital in January, 1902; accountant. Dr. Miller read only those parts of the records which illustrate the above symptoms. He would say in passing that there were no disturbances of the organic sensations.

The following quotations amply exemplify the feeling of unreality in the different fields: "This place is sinking beneath the surface of the earth—the engineers are sick, the fires are all out, the water will freeze in the pipes and these things you call men will freeze to death before morning.—I have no stomach and I have no lungs, so I know I will be dead soon.—That grass is not natural grass, it does not look natural.—That man is not a man, he is an invention of the devil made for the occasion. These buildings are not natural—see that sparrow, see how he moves his head. He is not natural, he is made to do that." (Shown cherry blossoms.) "Those are not real flowers. Nothing is real. That squirrel out there is not a real squirrel, it is only an invention, a mockery.—I am going to be squeezed to death by this tremendous pressure. This is not air here. We are under the ground, under the water, and there are too many things you call people. We will all die with the pressure.—These are not rooms, there is only one room. —You call this place a hospital—why, it is only a shed and there is no air, no floors, no wall, nothing but a make-believe shed.—We do not live, we are all dead, we are spirits and some outside thing makes us talk.—I could not help coming aboard this float."

("What do you mean by aboard?") "This is a float under the ground, under water. I am dead, you are dead. This place only represents a room.—You are nothing and I am nothing. It is all an illusion.—I am not talking. It is something in me that talks. I am dead. It must be some kind of a spirit that does the talking. This body has

been given to me lately. It is just a spiritual affair, held together by gravity. I think it was taken from down stairs, stuck together and called me. I have not the least thing to do with this (pointing to himself).—I have never been. It is all a dream and an illusion. You are not alive, nobody is alive, it is all a spiritual representation.—I am losing this support around me, this body was slapped around me that I rest on. See, this whole building is vanishing away, it is only a spiritual building, anyway. It is a building not made with hands.—They talk about women, there are not any such things, it is only a spiritual dream. —We do not eat, we just imagine we eat, it all goes, goes out the window and then vanishes away."

("There is a window, then?") "No, that is just a spiritual representation, it is really not a window. You cannot see, you cannot smell, you have no nose, you have no eyes, you think you see and smell, but you only imagine it."

He was asked to write his sentiments. He wrote: "Life is only spiritual, animal life as we call it is only guess work. We think we have a heart, brain and lungs when we do not have any. We don't breathe at all, it is only a sham. As soon as the spirit leaves us we are lost. I am not P——, they only call me so."

Invariably when he was asked what day it was he would say, "There is no day, there is nothing." When asked how he was, he would respond, "I ain't." On another occasion he said, "I don't understand this world. I wish I did. I don't believe there is anything in it at all. It isn't a world, it is all underground, there is no such thing as a world, this body is only made out of a log, my head is wood, that is why I can't hold it up. What is the sense of trying to shave a wooden face? We are nothing but disembodied spirits. They say they give me injections, how can they give injections to anything that is just screwed together?—I ain't anything. I am only a lot of stuff thrown together. I suppose I am nothing but water and feces.—I am made up out of boards—don't you see I have no eyes, put your finger in there and feel that I have no eyes. Oh, I wish I were dead!" ("You say you are dead.") "Well, I am dead. I died a long time ago. My limbs are loose from my body. I ain't anything at all." (His nose was pinched and he was asked what that was.) "It's nothing." ("Did it hurt?") "Yes, it hurt, but it don't belong to me. See my little neck, there is nothing goes down it."

("You eat?") "I pretend to."

("How old are you?") "Nothing."

("What year is this?") "They say 1903, but they don't know anything. Nobody knows anything. You could pull my leg off and I would not know it. It doesn't belong to me. Oh, I don't know what the matter is with me,—I can't tell anything,—I know these things are not real—everything is unreal—I haven't anything—never did have anything."

At another time he said, "How do you expect nothing to feel? You know I am made up of cinders. When I go to bed and get up in the morning and put on some clothes they fill up with mud. I eat and that makes it all the worse. I ought not to eat a bit. It is not food anyhow, it is only make-believe food." He struck another patient and when asked why he had done it, he said, "It wasn't me, it was the action of this place as it is constantly in motion. Ain't we going down deeper

every day into the ground? You know this is not air we are breathing, it is cinders.—My stomach and intestines are gone and my food just surrounds me. When I breathe I take it in. Just see what a little waist I have got, my wrists are very small. My brain is all gone, don't you see how small my head is? I am blind. My eyes are gone."

("Do you not see me?") "Only apparently."

Case II.—Involutional melancholia; illustrative of the feeling of unreality relating chiefly to the physical personality and in a lesser degree to the mental personality. (Somatopsychic and autopsychic.)

S. W., female, sixty-one; involutional melancholia of the agitated type with affectless depression. Onset at the age of fifty-nine. The feeling of unreality in this patient, as shown from the extracts of her records, related more prominently to the physical personality.

When asked whom she was, she said, "I am not anything." She refused to eat, as she said her throat was filled up and she could not swallow.

("What is the trouble?") "I don't know."

("Does your throat trouble you?") "I haven't got any throat."

("What is this?" pointing to her throat.) "Why it used to be a throat, it is nothing now. You can't have a throat when you can't swallow. You don't see my throat, do you?—I have no stomach and no bowels. My bowels cannot move because I have none. I am nothing."

("What are these?" pointing to hands.) "They are hands, but that is all there is of me."

("How about those feet?") "If they are feet, what good are they?"

("You have a nose?") " I can't have because I can't smell."

("Your eyes are all right.") "No, I don't see anything." She named a number of objects shown her correctly.

("How do you explain that?") "Well, I don't see these things; anything in a room may be seen, but I cannot see them with my eyes."

("Close your eyes.") "I have not any to close."

("You have ears.") "I hear noises in this room, but not with my ears. You see, I am different than you. A great deal different, I am nothing and you are something. I am not living, because I have nothing to live with."

When her throat was pinched she said that she felt it but that it was not her throat.

"I have no lungs or liver or heart, so how can I be alive?"

("Why do you think you have no internal organs?") "Because I can not feel them and I know I cannot have any because if I did I would not feel like this."

Case III.—Dementia præcox, illustrative of the feeling of unreality relating to the mental personality (autopsychic).

Bessie W., twenty-four. This case of the hebephrenic type and together with the feeling of unreality and evidently connected with it was a feeling of passivity which is not uncommon in dementia præcox. Furthermore, she has had many hallucinations of hearing, autochthonous ideas and delusions of control. Her feeling of unreality was shown by the following: "I don't feel like Bessie at all. Somehow I don't feel lifelike. It seems as if I cannot get my brain together. I must have a brain, but it seems as if I cannot use it, yet I think, but it seems somebody else or something else does the thinking for me.—I

think I am hoodooed or a spell is put on me so that I do not feel natural. I know that I must be myself, yet I cannot sense it, that I am anything but a moving object without any control over myself.—I see and hear but somehow I don't feel lifelike, not natural as I used to feel."

Careful examination failed to reveal a feeling of unreality in the other fields of consciousness.

Dr. F. H. Packard said that one of the most striking symptoms of Cotard's syndrome is what he mentions as a delire de negation and which Dr. Packard had spoken of as the feeling of unreality. It is not strange that a symptom which was first described in connection with involution cases and which occurs so often and so obtrusively in those cases should have come to be considered as more or less pathognomonic involution psychoses and should come to share the bad prognosis of such cases. Further observation, however, has shown that the same symptom occurs in manic-depressive insanity, in general paralysis, and in psychopathic states resembling dementia præcox, and it is also shown that cases which show this symptom in its most extreme degree do recover.

Therefore, it seemed to Dr. Packard, that we no longer ought to consider this symptom of such diagnostic and prognostic significance. At the present time he is strongly inclined to believe that these ideas of unreality are conceptual in origin and not due to any change in the organic sensations. It would seem that they may arise in almost any psychosis, provided certain conditions are present, viz., a confusion of thought where complex mental activity is required, with the preservation of a certain amount of clearness and ability to reason to a certain extent.

If this is the case, the symptom at once becomes accidental, as it were, secondary to the above-mentioned conditions, and not fundamental.

The reasons for its being noticed so often in involution cases are (1) that the conditions are right. The characteristic narrowed mental horizon and the preservation of a certain amount of clearness are favorable for its development. (2) Because it is more easily noticed in these cases—the same narrowed mental horizon, the poverty of ideas, allows this symptom to stand out alone more obtrusively with a greater emptiness of background than is the case in manic-depressive insanity, where the more productive patient not only voices these ideas but attempts to explain and qualify them to a certain extent, and even talks about many other things. (3) Dr. Packard is quite sure that the diagnosis of involution psychosis is sometimes made on this one symptom and (4) because the long duration sometimes obscures the prognosis.

In manic-depressive insanity it occurs, as indicated before, where the confusion is slight and is limited to complex mental activity and is not seen in cases where the confusion is deep and of quick onset, and from which the patients emerge quickly.

It also occurs when the conditions are right in those psychopathic make-ups described by Kraepelin in his chapter on original diseased states. Many of these cases with acute outbreaks of excitement or delirium with more or less suspicion and absurd ideas closely resemble dementia præcox, but Dr. Packard has not yet seen it in the classical cases of dementia præcox. It may occur, but rarely. The reasons are clear—

the conditions are not right. Whenever an association disorder of the necessary kind is present in these cases there almost always accompanies it too great a mental apathy to allow the development of any ideas in particular, and too great an emotional indifference to bring out such ideas even if they were present. And finally it occurs in general paralysis. Text-books mention it, and he has occasionally seen it, but has not observed it carefully in those cases.

From this Dr. Packard would conclude that it is not a fundamental symptom, not a pathognomonic symptom, and that the prognosis of cases showing it is not necessarily bad, but depends upon the more fundamental symptoms. Since it occurs in various mental conditions, at various ages, and varies in its outcome, Dr. Packard would be rather inclined to doubt its relation to any pathological findings.

Dr. Mitchell said that he had no intention to argue that Cotard's syndrome was a distinct clinical entity, but he felt that the feelings of unreality deepening into nihilistic ideas were not necessarily Cotard's syndrome, because these delusions of negation might be seen in many different psychoses.

He felt that Cotard's syndrome in its entirety would be seen only in cases of anxious, agitated melancholia where depressive ideas might deepen and become transformed into ideas of negation and nihilistic beliefs. He would agree with others that delusions of negation when seen in the case of manic-depressive insanity would have no special prognostic significance, but believes that the group of symptoms described by Cotard carries with it a bad prognosis. Many of the patients die during the height of their psychical disturbance, and he has seen no case of complete mental restoration following this condition.

Periscope

Miscellany

THE GRADUAL CURE OF HYSTERICAL PARALYSIS. H. T. Pershing (Journal A. M. A., May 11).

The sudden cure by suggestion or otherwise, of hysterical paralyses, however practicable and tempting in cases in which one can be sure of the diagnosis and of his power to impress the patient, is not the one to be generally chosen. Failure may compromise future success, and too prompt success may have its disadvantages in depriving the patient of the discipline and enlightenment that are necessary to a permanent cure. The safer way is more gradually to arouse and exercise the dormant kinesthetic centers, overwhelmed by inhibitory impulses from other centers, due to the perverted emotional conditions and ideas, by first raising the emotional tone, encouraging the patients to believe they will recover, and if such cheering is opposed or resented, appealing to their *amour propre* by judicious suggestions. The physical measures—rest, food, tonics and sedatives—while of great importance, are sometimes overvalued, he thinks, but both mental and physical treatment should be employed in these cases in harmonious combination. While doing this, the dormant kinesthetic centers can be acted on specifically by sensory stimulation. Hysterical anesthesia is not absolute and strong faradic currents, applied to the skin and muscles, followed by vigorous and even rude, passive motion of the affected limb, have a powerful tendency to restore the lacking sensations and ideas of motion. The patient's close attention should be directed to the anesthetic part and every slight beginning of sensation noted. This will tend to bring the cortical sensory centers again into relation with the other centers in the cortex. After awakening some degree of normal sensibility and overcoming inhibitory fears, the cure can be completed by exercise in voluntary motion. If the paralysis is nearly total it is as well to let the first attempts be favored by gravity, which will assure some motion and encourage the patient to increase it. A beginning once made, the attempts can be gradually increased, every encouragement being given, till at length some normal control is obtained, and the patient is able to practice some assigned exercises in the physician's absence. The length of time taken in accomplishing the cure may be made an advantage by using it to carry on a re-education in composure and self-reliance, without which permanent recovery is impossible. Two cases of typical hysterical paralysis are briefly described in which this method had been employed with complete success.

SYRINGOMYELIA, EXTENDING FROM THE SACRAL REGION OF THE SPINAL CORD THROUGH THE MEDULLA OBLONGATA, RIGHT SIDE OF THE PONS AND RIGHT CEREBRAL PEDUNCLE TO THE UPPER PART OF THE RIGHT INTERNAL CAPSULE (SYRINGOBULBIA). William G. Spiller, M.D. (British Medical Journal, Oct. 20, 1906).

The interesting features in this case are found in the pathological study.

The extent of the cavity is greater than any heretofore described, and in fact has even been regarded as impossible. The patient's first symptom was a weakness in the left foot developing when he was seventeen years old, which progressed slowly until, in six years, the whole left side was paralyzed. The right side then became gradually weaker, and four years later he was completely helpless. Examination showed atrophy of the right side of the tongue with a marked impairment of the sense of taste, especially on that side. There was great impairment of motion in the arms, especially in the right. There was considerable wasting of the arms more marked in the distal portions. The right leg also was more affected than the left, and was 2½ inches shorter. Sensation for touch was felt normally throughout the body, but for pain and thermal sensation the disturbance was pronounced. The patellar jerk was exaggerated on the right side and diminished on the left. Death was due to bronchopneumonia. The cavity in the spinal cord began in the lower sacral region in the left posterior horn to which it was still confined in the upper sacral and lumbar region. From the midthoracic region upward the spinal cord was distorted and flattened. In the medulla there were distinct cavities in each anterior pyramid; the one on the right side being larger and cutting off the fibers of the hypoglossal nerve on that side. In the upper part of the medulla the cavity in the left anterior pyramid had disappeared. In the pons; the cavity in the right side extended nearly to the raphe. The right adducens fibers were degenerated. In the right cerebral peduncle the cavity was a narrow slit in the substantia nigra and extended into the upper part of the crusta near the median side of the peduncle. In the lower part of the internal capsule the cavity divided; one part soon terminated, but the other extended upward to within two or three millimetres of the lower surface of the lateral ventricle, but did not open into the ventricle. Schlesinger, in his monograph, says that in syringobulbia the cavity does not extend above the fifth nerve and involvement of the first four cranial nerves indicates a complicating lesion. This case shows that the third, fourth, or fifth cranial nerves might be involved by a cavity and that symptoms in the distribution of these nerves need not necessarily imply a complicating lesion. The atrophy of the right side of the tongue is explained by the complete degeneration of the hypoglossal nerve and its nucleus. C. D. Camp (Philadelphia).

Dislocation of Vertebræ in Lower Cervical Region. William C. Krauss Annal. of Surg., November, 1906).

In connection with a review of the literature on this subject, Dr. Krauss reports an interesting case of dislocation of vertebræ in the lower cervical region, followed by symptoms of complete severance of the spinal cord. Laminectomy was performed with a result of partial restoration of function later. The report is in brief: Male, 22, healthy, dived into shallow water, became semi-conscious and devoid of power in arms and legs. On admission to hospital there existed total paralysis of legs; arms and hands partially paralyzed. Speech, eyes, pupils and facial muscles not affected. Head slightly retracted, but moves in all directions with some pain. Tenderness over spinous process of the fifth cervical vertebra, and indefinite pain about third thoracic spine. Pulse slow, full, and good tension. Heart normal, respiration diaphragmatic, no temperature. Patellar, ankle and plantar reflexes absent. Marked priapism. Patient able to flex arms, but not to extend them. Movements of fingers and

thumb impossible. Extension and flexion of forearm greatly weakened. Adduction of arm, pronation and supination of forearm very weak. Considerable weakness of deltoids, triceps, and chest muscles, also muscles about scapula and paralysis of serratus magnus. Triceps and biceps tendon reflexes absent. Zone of anesthesia corresponds to the level of the second intercostal space, is symmetrically limited to ulnar side of the arm and forearm, including the middle, ring and little fingers, and is for both temperature and tactile senses. Abdomen tense, reflex lost; likewise the cremasteric. Incontinence of feces. Condition diagnosed as complete severance of cord at level of sixth cervical. Placed on water-bed and traction to head. In few days bed sores developed and mild cystitis appeared. Eighteen days later laminectomy of fifth and sixth cervical performed by Dr. Roswell Park. Sixth cervical apparently abnormally loose. Dura appeared normal, but contained large amount of cerebrospinal fluid. Remains of old clot found. Cord seemed flattened and shrunken. No constriction above or below. Wound closed with buried and superficial drainage. On leaving hospital four weeks later the condition was: Can move head freely without pain. Area of anesthesia not materially changed. Can raise arm over head and flex forearms, but flexors of hand are weak; likewise pronation and supination. Can draw right leg as far as left knee; extension and flexion of right foot quite strong. Left leg can be drawn up to middle of right leg; extension and flexion of foot very weak. Patellar and plantar and Babinski's reflexes exaggerated; ankle and patellar clonus present. Abdominal and cremasteric reflexes still absent. Priapism disappeared and can tell when bladder and bowels are going to act, though can't control sphincters. Involuntary contraction of leg muscles and spasmodic flexion of legs and feet an annoying symptom. Eight months later, patient inclines slightly forward; Romberg present; flexes right leg upon thigh and pelvis, but can lift left leg only three inches from floor. Can walk with support. Tendency to fall backward. Gait is spastic; drags toe of left foot. All motion of arms and shoulders free. Pronation and supination of forearms good; likewise extension of wrist. Closes right hand fairly well, writes easily and uses telegraph key. Can open and close left hand, but without much power. Tendon reflexes of triceps, biceps and forearm muscles exaggerated, muscle reflexes heightened. Abdominal and cremasteric reflexes absent. There is still some difference of sensation at level of original zone. Temperature and tactile sense of left leg normal, but in right leg cannot distinguish between hot and cold. Breathing still diaphragmatic. This case is of interest since it shows a marked regeneration and recovery of function following a late operation.

H. C. Cowles (New York).

The Blood Pressure in Paresis. G. L. Walton (Journal A. M. A., Oct. 27).

In order to test the correctness of the general belief that the blood pressure in paresis is low, the author has examined 108 male patients with this disease in the Massachusetts hospitals for the insane, with special reference to this point. He used the Riva-Rocci instrument and employed the ordinary precautions in making the observations. The average of all the observations indicated a high rather than a low pressure, but to eliminate the influence of renal and arterial conditions, he separately tabulates the results in forty-four cases, without record of renal, cardiac or arterial disease, which show a tendency to hypotension rather than to hypertension.

The difference in this respect is not considered of very great value for diagnosis, the less so since variability rather than high pressure is the characteristic of the psychoneurotic. The author gives his conclusions as follows: (1) The average blood pressure in paresis, taken as a whole, is high. (2) This is doubtless due to the prevalence of atheroma with its cardiac and renal accompaniments. (3) The average blood pressure in cases of paresis without atheroma, cardiac enlargement or renal disorder, is probably somewhat lower than that of health, but the variations are so great that it can not be said to be uniformly low. (4) The test is not likely to prove of great practical value in the differentiation of paresis from other nervous disorders, though here, as elsewhere, it is of great value in estimating the circulatory condition of the individual. (5) These observations are too few to establish a rule with regard to the blood pressure in varying emotional states. So far as they go, however, they tend to show that: (a) The excited states of paresis are as likely to be accompanied by high as by low pressure: (b) mental depression is accompanied by high oftener than low pressure, but that it is not incompatible with low pressure; (c) while the average pressure in euphoria is perhaps somewhat lower than in the other mental states of the general paralytic, it is not inconsistent with high pressure or with pronounced atheroma with its cardiac and renal accompaniments.

PROGNOSIS OF MULTIPLE SCLEROSIS. O. Maas (Berl. klin. Woch., 44, 7, 1907).

The author makes an interesting communication based on the history of a patient from Oppenheim's Poliklinik. A 58-year-old woman had had for eighteen years a typical case of multiple sclerosis, which developed slowly and progressively. The process then came to a standstill. She became very much better, and although showing unmistakable signs of disturbance of the nervous system she has been in this improved condition for thirteen years. JELLIFFE.

THE PRESENT STATUS OF BRAIN SURGERY. M. A. Starr (Journal A. M. A., Sept. 22).

The author considers that sufficient time has elapsed to enable us to estimate the value of brain surgery for the relief of tumors, epilepsy and abscess with considerable accuracy. It is only in localized Jacksonian epilepsy (about 2 per cent. of all cases), that operation is indicated and in only about 20 per cent. of these is it successful. Trephining for epilepsy, therefore, is of very limited application and is only to be recommended in a few selected cases which present the necessary guide to both physician and surgeon. In abscess of the brain, early operation as soon as the condition is diagnosed is imperative, and in cases of skull fracture or concussion followed within two or three weeks by symptoms suggestive of abscess, even if there are no localizing symptoms, trephining is imperative. There are many regions of the brain, injuries of which are associated with no localizing signs. In abscess due to chronic otitis, operation is demanded as soon as the diagnosis is made. While statistics show the percentage of recoveries after operation for cerebral abscess at present is only about 60 per cent., there is every reason to believe that it will be much greater when early diagnosis and immediate operation is the rule. In brain tumor with positive localizing symptoms, operative interference may be warranted, but in the far greater number, without localizing symptoms,

operation promises nothing. Post-mortem statistics indicate that about 10 per cent. of brain tumors are open to surgical treatment, and that the best results may be expected when the growth is located near the Rolandic or Sylvian fissures, and the highest mortality when it is in the cerebellum. The proposition to afford relief in inaccessible tumors by making a considerable opening in the skull to relieve pressure, may be of value in some cases. Starr mentions one of his own observations in which this procedure was of benefit and two others in which it failed. In cases of extradural hemorrhage from traumatism, with symptoms of intracranial pressure, slow pulse, steady rise in blood pressure, deepening coma, Cheyne-Stokes respiration, and increasing hemiplegia, all appearing within six hours of the injury, trephining is sufficiently clearly indicated. The hemorrhage is usually from the middle meningeal artery, hence a large trephine opening or a large, bony flap should be made in the area just above the ear. In apoplexy Cushing has applied successfully, in hospital cases, the test of the condition of the blood tension in determining the need of surgical intervention to save life. When the blood pressure rises steadily to 250 mm., measured by the Riva-Rocci or the Janeway apparatus, in a case of apoplexy, and when coincidentally with this there is a slow pulse falling to 50 a minute it may be said that the case will be fatal unless pressure is relieved by a considerable opening in the skull, without regard to the finding or removal of the clot. The best place for this is over the motor area of the side opposite the paralysis, as the clot may be there. Cushing's cases show that this operation may sometimes save life in an otherwise hopeless condition. Cushing has also treated surgically with success newborn infants who, after a difficult labor, have suffered an extradural or intradural hemorrhage. Such infants usually die, or if they survive are defective, hemiplegic, idiotic, etc., and any measure for their relief is justifiable. It is easy in these cases to relieve intracranial pressure by opening the sutures of the parietal bone with scissors, and his success warrants urging obstetricians to consider this operation in the case of asphyxiated infants of the class described above. Obstetricians see these cases, and if they are convinced that delays are dangerous the percentage, Starr says, of idiocy and hemiplegic epilepsy will certainly be reduced. The last class of cases of cerebral hemorrhage suitable for trephining is that in which hemiplegia or hemianopsia develops slowly after an injury and does not come to its height for three or four days. In these there is probably a surface hemorrhage from a vein in the pia mater and lumbar puncture will probably reveal blood in the cerebrospinal fluid. The symptoms may progress and threaten life, or come to a standstill, leaving the patient permanently incapacitated. In either case surgery is indicated. Starr refers here to a case of this kind in which a clot was removed from the lower third of the Rolandic fissure with good results, and remarks that many other similar cases, equally successful, could be cited. In conclusion he refers to the methods that have been recommended and employed to cure microcephalic idiocy by relieving pressure on the brain and permitting its expansion. Experience has shown the uselessness of such surgery, and it is no longer recommended.

Book Reviews

PSYCHOTHERAPEUTISCHE BRIEFE. Von Prof. Dr. H. Oppenheim, Berlin. S. Karger, Berlin.

LETTERS ON PSYCHOTHERAPEUTICS. By Professor H. Oppenheim, of Berlin University. Translated by Alexander Bruce, M.D. Otto Schulze & Company, Edinburgh. G. E. Stechert, New York.

So closely following the appearance of Professor Oppenheim's little pamphlet on psychotherapeutic letters has Dr. Bruce's translation of the same appeared that we are tempted to commend first the letters themselves, and second their excellent translation.

Psychotherapy is the prevalent term to express an old form of therapeutics, but further, however, it is a hopeful sign of the times to find men of such signal ability as Oppenheim taking so decided an interest in the functional disorders of the nervous system, and devoting their energy to the building up of a tactful scientific mode of therapy for patients suffering from such disorders.

These letters are excellent, not better than many an intelligent and tactful woman might write under similar circumstances, not as good, in our opinion, as many of George Eliot's or Robert Louis Stevenson's letters, but few practitioners can hope to have the genius of these letter-writers. They emphasize, however, many of the little things that are of so much importance in the treatment of neurasthenic, psychasthenic or other functional mental states, and afford to those of us not so gifted a clue as to the proper method of approaching, conversing or writing to these peculiarly difficult patients.

The translation is admirable. It closely retains the author's helpful, buoyant style and common sense throughout. JELLIFFE.

THE INTEGRATIVE ACTION OF THE NERVOUS SYSTEM. By Charles Sherrington, Holt Professor of Physiology in the University of Liverpool. Charles Scribner's Sons, New York.

This monumental work consists of a series of ten lectures delivered by the author at Yale University as the Mrs. Hepsa Ely Silliman Memorial Lectures. It is the second lecture course given under the new foundation, and is a work of some four hundred pages of compressed and valuable physiological facts.

The chief work done in neurological science has been the mapping of anatomical tracts and boundaries, but the more exact and detailed study of functions has lagged somewhat behind. This is natural. In the present work, however, which is too technical and close to permit a short review, Sherrington has covered the entire ground work of the more essential and fundamental conception of neural physiology.

In his early chapters he discusses the unit of the nervous mechanism; namely, the reflex arc. The reflex is the primary integrative unit. What the functions are of this elemental reflex and the conditions, electrical and physical, surrounding it, is the work of his first three lectures. The receptor is first considered, then the conductor. Variations in susceptibility, in selective excitability, by a lowering of the threshold for certain stimuli, are the conditions which are discussed as affecting the functions of the receptor. In discussing the functions of the conductor, Sherrington

introduces a new term—the synapse, or the surface contact space, between the neurones. There is a point where resistance is met with, and which takes time to overcome. This "synaptic phase" he has carefully measured. Special consideration is given to the study of variable stimuli in regard to conductivity.

Inhibition is regarded by Sherrington not as an independent phenomenon, but as a part of the function of excitation, and an essential part. Reciprocal innervation brings about suppression, and an internuncial mechanism is posited as necessary for the production of the inhibitory process.

Sherrington then takes up the problem of the compounding and interactions of reflexes and follows by an analysis of the physiological position of and reasons for the dominance of the brain.

The interesting position, which will probably lead to much discussion, is taken that a physiological basis for psychical fusion is not present, nor is it required.

The work is one of great significance, and though difficult to follow for the student who has not been saturated with the modern experimental physiology of nerve trunks and fiber tracts, its reading will prove stimulating. JELLIFFE.

GEHIRN UND SEELE. Vorlesungen von Prof. Dr. med. Paul Schultz; Herausgegeben von Dr. Hermann Beyer. Johann Ambrosius Barth, Leipsig.

This is a series of semi-popular lectures on philosophical subjects given by Prof. Schultz during the years 1899 to 1904 in the University of Berlin. They differ from most lectures of the kind in the wideness of their scope, general philosophical considerations being paramount in the earlier pages, while later the author discusses at length the question of the reflexes, the activities of earth worms, heredity, psychical functions of ants and bees, embryology of the human brain, critique of Gall's phrenology, Broca's speech centers, Hitzig-Flechsig's theory of association centers, hallucinations, sleep, hypnosis, etc.

There is no attempt at completeness in the present presentation. It is a readable philosophical treatise, couched in popular language. JELLIFFE.

GESAMMELTE HIRNANATOMISCHE ABHANDLUNGEN; MIT EINEM AUFSATZ UEBER DIE AUFGABEN DER NEUROBIOLOGIE. Von Dr. August Forel, ehemals Professor der Psychiatrie und Direktor der Irrenanstalt in Zürich, in Yvorne (Schweiz). Illustrated with 12 plates (engravings). Ernst Reinhardt, München, 1907.

To find Forel's contributions to brain anatomy all published in one volume, will give great pleasure to his many admirers, and will give to those less familiar with his work opportunity of appreciating his merit as one of the pioneers of present-day brain anatomy.

The undersigned, who was so fortunate to be one of the author's assistants when he was Director of the Insane Hospital, Burghoelzli, and Professor of the Psychiatric Clinic of Zurich, deems it a particular favor to have the opportunity of reviewing his work in brain anatomy as embodied in the volume before us.

Those who have worked under Forel cannot but have been deeply impressed by his powerful personality and intellect, and have carried away with them rich experience serving as intellectual food for many

years to come. Under the stimulating influence of his vivacious conversation, always interesting and many-sided, but often deeply philosophical, the minds of those who listened to him could not help being broadened. To hear him discuss one day Haeckel's biogenetic law and another time Kant and Herbert Spencer, or listen to a discourse on color blindness, or to his views regarding the interpretation of the marvellous sense of locality in birds, or again to hear him make a harangue of the French school with humoristic allusions to the grand diseases issuing from Paris, such as the grand mal, grand hysterie, grand hypnotisme, etc., was full of inspiration and of lasting benefit to the listener.

To the reviewer's knowledge Forel's studies in brain anatomy were in the main initiated under Meynert, and the second* paper of the collection before us was worked out under Meynert, and to some extent bears the stamp of his influence. So, for instance, he made use in it of Meynert's "Abfaserungsmethode," which consists in following fiber tracts by picking off the adjoining matter by means of a forceps and thus isolating the tracts.

But even in this paper he was already aware of some of the defects of Meynert's methods in reaching results and conclusions, and kept himself to a considerable degree free from them. He later gave expression to this stand (in his monograph on the tegmental region) in the following words: "Finally I wish to state that when five years ago, under the guidance of Professor Meynert, I published the work just mentioned, I was still entangled in many prejudices and in particular considered most of the statements of Meynert as well established, which explains many divergences from my present results. The circumstance as well as the fact that it was my first attempt in brain anatomy, and that I worked with a less complete material, may serve as an excuse. That on many points I already then had my doubts, every careful reader will perceive."

The paper referred to bears the title, "Beiträge zur Kenntnis des Thalamus opticus und der ihn umgebenden Gebilde bei den Säugetieren." It constitutes a very valuable objective, comparative-anatomical contribution to the knowledge of the optic thalamus and the structures surrounding it. Of the points brought out in it, be it briefly mentioned, that the importance is shown of the development of the pulvinar in defining the location of the geniculate bodies, which in man with a well developed pulvinar are crowded outward, but move upward and inward—particularly the external geniculate body—the lower one descends in the scale of mammalians. To enter on other details of this work the space allotted for this review does not allow, except to mention that Forel's contentions are accompanied by numerous measurements of the structures in question.

The contribution next in order, entitled "Untersuchungen über die Haubenregion und ihre oberen Verknüpfungen im Gehirne des Menschen und einiger Säugertiere; mit Beiträgen zu den Methodon der Gehirnuntersuchung," was issued from Professor Gudden's laboratory. In this monograph F. has altogether emancipated himself from Meynert's influence, and put forth his guiding principle in the following words: "My leading thought in the following lines as well as in the figures was always the strict avoidance of all diagrams and of going beyond well established facts, since I consider these two faults to be the most assailable points of brain anatomy. May I have been successful." That he indeed was

*The first paper will be discussed at the end of this review.

successful in carrying his point certainly must impress itself on the careful reader of these investigations.

His appreciation of Meynert's merits is expressed in the following words, taken from the same monograph: "The only author who on the basis of histological researches of the present has dared to attempt to clear up the mysterious obscurity of the cerebrum and basal ganglia, is Meynert. This was an extremely meritorious and stimulating attempt." On the other hand, F.'s perception of Meynert's failings, while aware of his greatness, is embodied in the following statement: "It must soon become clear that notwithstanding his perspicacity, his foresight, which unquestionably bore the stamp of genius, and in spite of his great knowledge of the brain, the certain and uncertain hypothesis and fact are in his statements interwoven with each other in a manner to often make their separation or distinction impossible."

The above mentioned investigations begin with a subject of historical interest, one might truly say; *i. e.*, a careful detailed description of the Gudden microtome, the instrument by means of which so much splendid work has been done in brain anatomy, by Gudden's school, v. Monakow, Dejerine and many others. That instrument had then only recently been invented by Professor Gudden, and its minute description by Forel, with pointing out of all its advantages and disadvantages and the avoidance of the latter, came àpropos.

This is followed by a description of the hardening and coloring technique used by him; with a critical discussion of the methods then in use. Next the methods of examination of the brain in general and their delimitations are considered, which again forms an historically interesting chapter. The "Abfaserungsmethode" of Meynert which has already been mentioned, is here strongly condemned, owing to its absolute unreliability.

Then comes the actual anatomical research which fully deserves to be designated as a classical treatment of the subject. It gives a masterly description of the structures forming the subject of study, in which, true to the principle laid down by him in the introduction, facts are strictly kept apart from probabilities, and these again from conjectures, giving each of them their true value. So important are these investigations for the knowledge, even purely topographically, of the regions described, that one can hardly take up a proper study of these regions without reference to Forel's work. This has particular reference to the subthalamic region of which Forel gives a complete map; and, as already pointed out, his statements are made with such objectivity, free from speculation, that even as our knowledge of the anatomical connection and physiological function of the different structures encompassed in these regions advances, yet the land-marks laid down by him will continue their value as topographical guides for years and years. This value is greatly enhanced by the numerous fine drawings accompanying the monograph, and made by the investigator's own skilful hand.

The investigations on the tegmental region are followed by some smaller communications and discussions, in which Forel points out the clearness of the results obtained with Gudden's method, as compared with the purely anatomical method. Gudden's method "consists in removing from the new-born animal (rabbit, dog) certain parts of the central or peripheral nervous system. The animal is then allowed to grow up and one studies both the loss of functions and the secondary

inactivity-atrophy which the structures functionally dependent of the extirpated part undergo."

He summarizes the results obtained by Gudden and his pupils, Mayser, Ganser and Forel himself, as applied to motor nerves, sensory nerves, nerves of special sense, cortical motor centres and other structures.

A brief report of some extremely interesting findings in general paresis and senile dementia then follows, which shows the author's diagnostic powers. In one case a hemiplegia was clinically diagnosed and confirmed post-mortem to be not focal, but the outcome of the diffuse anatomical process underlying general paresis, predominating in one hemisphere. In the second case of paresis, the almost complete lack of physical signs with marked involvement of the psyche led to the diagnosis of a paresis with affection mainly of the frontal lobes, leaving the motor centres almost intact; and the autopsy fully bore out that diagnosis. In the third case, which was a senile dementia, an atrophy particularly of the temporal lobe was found as the substratum of word-deafness.

Three preliminary communications on the origin of the auditory nerve, one of them in conjunction with the undersigned, then follow. The interesting results embodied therein were obtained by Forel by means of Gudden's method, which in this special application required a particular skill.

The contribution next in order, entitled "Einige hirnanatomische Betrachtungen und Ergebnisse," reveals some extremely important facts. We note therein not only the author's early appreciation of the value of Golgi's work, as achieved with the method of metallic impregnation, but also the utilization of Golgi's results and of the results obtained with Gudden's method, for building up the neurone theory. That this theory indeed is distinctly formulated here already, built up on a substantial basis, must become clear to every careful reader. Of great interest is the fact that while His, in an article appearing four months earlier than that of Forel, had come to a view identical with that of Forel and, therefore, could justly claim priority for the formulation of the neurone doctrine, nevertheless, Forel could prove that the manuscript of his article was already in the hands of the publishers at the time when his paper appeared in print; so that there could be no question that both had independently reached the same result. (See page 210.)

Another important issue in this paper is the thorough study of the laws underlying the results of Gudden's method, this study being based on experiments on new-born and adult animals, with variation of the points of attack in the nerves experimented on.

The paper is full of other important deductions and suggestions which lack of space forbids to discuss here.

Then follow two communications, one relating to the origin and connections of the auditory nerve (Zur Acusticusfrage), the other on the connections of the elements of the nervous system.

In two further short communications the results of counts of nerve fibers in the oculo-motor nerve of the new-born and adult cat respectively are given. The work was done by M. H. Schiller but inspired by Forel, and reveals that the number of fibers of said nerve is virtually the same in the adult as in the new-born. This observation, if confirmed in other instances would allow important conclusions as to the manner of growth of the nervous structures, indicating growth in size rather than by increase in the number of elements.

The paper succeeding these two communications and entitled "Ueber das Verhältniss der experimentellen Atrophie und Degenerations methode zur Anatomie und Histologie des Centralnervensystems," deals again to a great extent with the method of Gudden and its merits. Here the writer shows how early he appreciated the general principle underlying the method of Gudden. As early as 1868 the latter author, having torn out the facial nerve from the Fallopian canal in new-born rabbits had thus, contrary to former observations, produced an atrophy of the facial nucleus, "which he, in his lectures in Zürich demonstrated to us (*i. e.*, to Forel and other students), macroscopically as a depression of the basis of the oblongata in the region of the nucleus on the side of operation." Later this atrophy was verified microscopically by Dr. Mayser, who cut the brain in serial sections. "Strangely enough," Forel goes on to say, "von Gudden remained in the belief that this was a peculiarity of certain nerves only, while I repeatedly expressed to him my conviction that it must be a general law for all motor nerves; that their central cells of origin perish, when in the new-born they are separated from the basis of the brain (or spinal cord respectively)."

Another point deserving quotation here is the broad view he takes as to the methods which should be pursued in studying the morphology and functions of the central nervous system: "After the experience of many years, and basing on my own investigations in the domain of the morphology and of the functions of the central nervous system, I wish to put up the following dictum: That all methods of investigation must be employed hand in hand and compared with each other in their results; that each brain anatomist and physiologist should know and duly appreciate them, and that in involved conditions as a rule only those results and theorems should go as established anatomical truths, which have successfully stood the test and confirmation by all methods." He then enumerates these methods as follows: First, gross structural anatomy. Second, study of continuous series of sections. Third, final histology of the elements. Fourth, comparative anatomy and histology of the central nervous system. Fifth, embryology. Sixth, the experimental method of secondary atrophies and degenerations. Seventh, the study of abnormalities of the central nervous system (especially agenesies of the same), and of pathological cases in general. Eighth, the method of studying fiber systems by means of the development of the medullary sheaths at different periods of time. Ninth, the physiological experiment itself.

In this paper Forel also explains the reasons why the work of Gudden and his school has not received the appreciation and notice which it deserves, giving, at the same time, the result of researches of Mayser and Ganser on the origin of the tenth, eleventh and twelfth nerves, and of the connections of the optic nerve respectively.

The contribution following, published in conjunction with Wl. Onufrowicz, deals with the defects of the corpus callosum and is accompanied by numerous illustrations made by Forel hmself. This work, because of its interesting conclusions, has given rise to considerable discussion.

The book winds up with a brief communication regarding the nuclei of the glossopharyngeal and trigeminal nerves and two discussions on the neurone doctrine. In the latter one of these, Apathy's and Bethe's work are critically reviewed and a reconciliation of their results with the neurone theory attempted.

The reviewer has left to the end the paper entitled "The Problems of Neuro-biology," which appears as the first one in the book. This was done for the reason that it is the latest one chronologically. It gives a critical review of our present knowledge of neuro-biology and indicates the lines along which further knowledge should be gained. From a man who has enriched this field as much as he by original research and critical discussion of the work of others, such a review must be welcomed as very valuable and suggestive. Very modestly he expresses himself on this point in the introduction of the book, as follows: "I have nevertheless always followed the further development of brain morphology with interest and endeavored to further its connection with the other branches of brain science and with neuro-biology in general. May I, therefore, not be accused of presumption if I precede these old contributions by a brief study on "The problems of neuro-biology."

The publisher, M. Reinhardt, Muenchen, is to be congratulated and commended for the manner in which the book is gotten up, and particularly for the fine reproduction of the numerous illustrations, for which the original drawings were almost all made by Forel himself.

B. ONUF (Onufrowicz).

ORGANIC AND FUNCTIONAL NERVOUS DISEASES. By M. Allen Starr. Second Edition. pp. 816. Lea Brothers & Co., New York and Philadelphia. 1907.

This is the second and revised edition of Dr. Starr's book and contains in addition a section devoted to functional nervous diseases. It has been so well received that it has been found necessary to put forth a second edition within a year, and it is so far the most complete book on nervous diseases attempted by a single American author containing a treatise on all organic and functional nervous diseases. Its distinguishing feature is that it is a presentation of the personal experience of over twenty-five years of neurological practice.

Not much space is given to the anatomy of the nervous system, and the author does not enter into discussion of the various neurone theories. Eight chapters, this including 122 pages, are devoted to a consideration of the nerves and their diseases, their injuries, neuralgia and multiple neuritis. Without question this discussion is about as complete and well written as is to be found in any book upon nervous diseases. Dr. Starr's experience in the treatment of neuralgia of the fifth nerve is interesting. He states that aconitine is the best remedy, and he gives it in pill form in doses of 1-500 of a grain until constitutional effects are obtained. In the discussion of the symptoms of sciatica he fails to mention the usual absence of the Achilles jerk, and in speaking of the symptoms produced by carbon bisulphite poisoning the author states that he has not found any published cases in this country, evidently not being acquainted with the cases published by Jump and Cruice, and he also does not mention the hysterical symptoms so common in this disease. He justly says that multiple neuritis is probably not caused by syphilis.

The diagnosis and localization of spinal cord diseases is next taken up and well discussed, and the original diagrams used to illustrate the course of the motor and the various sensory tracts, are excellent and so far the best that have appeared in any book. He rightly speaks of chronic anterior poliomyelitis and progressive muscular atrophy as identical diseases, although in the discussion of the symptoms he mentions three types: That

beginning in the muscles of the peroneal group; second, in those of the back and trunk; and third, the usual type beginning in the muscles of the hand. His description and classification of the muscular dystrophies is that usually adopted. When speaking of the pathology of tabes dorsalis, Dr. Starr accepts without reservation that the primary lesion lies in the posterior spinal ganglia and in the ganglia of the cranial nerves. With this opinion most neurologists will disagree, for there is more evidence to show that the disease starts in the posterior roots and that the degeneration of the ganglia is secondary. He believes that while syphilis is a predisposing cause of tabes, we cannot consider it a syphilitic infection, and he adopts the most reasonable hypothesis that syphilis leaves a toxin in the system which weakens the sensory elements of the nervous system.

The author next takes up the diagnosis and localization of brain diseases. His well known and long experience in this particular department of neurology renders his opinions on these matters authoritative. He inclines to the old and classical views of aphasia, the early publication of this book probably hindering him from entering into the more recent discussion of this subject which was excited by Marie and Dejerine. He accepts the view that lesions of the angular gyrus cause word blindness, although in the next page he gives illustrations of lesions causing this symptom, several of which show involvement of the occipital lobe and no disease whatsoever of the angular gyrus. He ascribes the hemichorea and hemiathetosis which occasionally remain after lesions of the thalamus as symptoms of irritation of the motor and sensory tracts passing near it, but fails to specify the more recent views that these movements are probably the result of lesions of the superior cerebellar peduncle. When discussing the symptoms of lesions in the pons he does not mention paralysis of the associated ocular movements, and he does not devote enough space to the symptoms of cerebellar lesions. Omissions, however, are only exceptional, for the general description of the diagnosis and location of brain diseases is excellent. His discussion of apoplexy is very good. He next takes up tumors of the brain, and the part of the book devoted to this is without question very complete with the exception that he does not devote as much space to the discussion of cerebellar tumors as would seem to be necessary. His advice as to the treatment of tumors of the brain is sound.

He next takes up bulbar palsy, but he does not lay any special stress upon the diagnosis between this disease and pseudobulbar paralysis. The discussion of the cranial nerves and their diseases is complete.

The second part of the book is devoted to functional diseases, and included in this are chorea, ties, myoclonus, myotonias, epilepsy, paralysis agitans, neurasthenia, hysteria and migraine. Considering Dr. Starr's well known views that epilepsy is an organic disease, it is rather surprising to find it among the functional diseases. The discussion of neurasthenia and hysteria are very brief and not in proportion to the rest of the book. What there is of this, however, is very good.

The book as a whole is an excellent presentation of the clinical phenomena found in organic nervous diseases. The author's opinions are presented in a clear, forcible and logical manner, and little doubt is left in the reader's mind as to what is the author's opinion on any subject. The book is well balanced and is a distinctly valuable contribution to neurological literature.

T. H. WEISENBURG.

INDEX TO SUBJECTS.

Figures with asterisk * indicate original articles, and are accompanied with title. Figures unaccentuated, accompanied with title, indicate abstracts.

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