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THE
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Original Articles.

COMPOSITE PORTRAITS OF GENERAL PARESIS AND OF MELANCHOLIA.

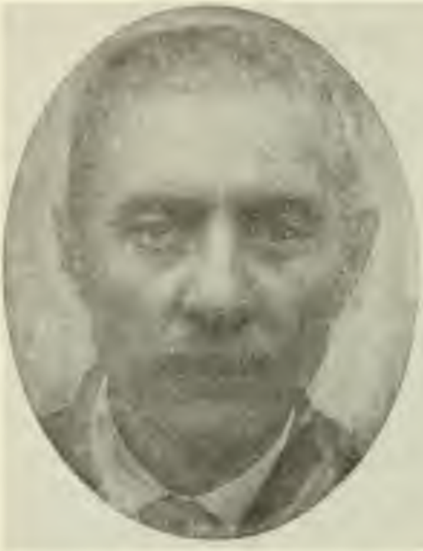
By WILLIAM NOYES, M. D.,

ASSISTANT PHYSICIAN, BLOOMINGDALE ASYLUM, NEW YORK.

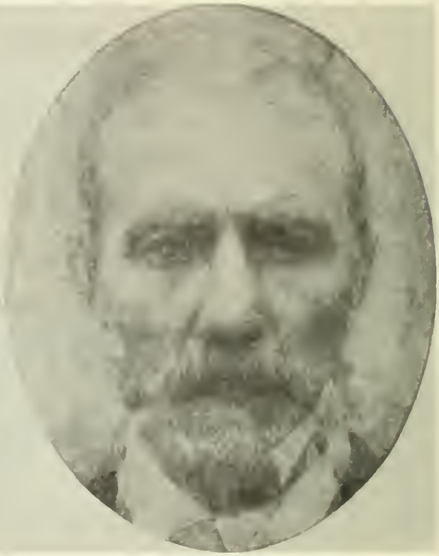
THE accompanying composite photograph of general paresis is made from the portraits of eight patients, three females and five males. The composite negative was made by THE NOTMAN PHOTOGRAPHIC COMPANY, of Boston, from negatives taken by the writer. The percentage of females is higher than in the natural ratio of the two sexes in the disease. The cases are all in the second stage of the disease, and their individual portraits show the marked characteristics of general paresis. One of the women and three of the men have had apoplectiform seizures. The average duration of the disease, at the time of photographing, was, in the women, two and one-third years, and in the men one and three-fourth years. With the exception of one woman, all were in good general physical condition, and able to go out walking and join in the usual round of asylum life ; the one woman who was the exception was still able to go out walking on pleasant days, but was not so vigorous as the others.

The composite of general paresis seems fairly to represent the physiognomy of general paresis ; the eyes have the typical inexpressive and staring look ; and the facial lines of expression have been gradually obliterated and smoothed out, giving the well-known appearance of easy-going complacency. It is to be hoped that further work in this line may give a more just conception of the typical expression in the different forms of mental disease than has hitherto been obtained from portraits of individual cases.

The composite portrait of melancholia is made up of eight subjects,—all men.



General Paresis.



Melancholia.

The composite of melancholia is perhaps somewhat more clear and distinct than that of paresis. A comparison of the eyes of the two portraits shows much more expression in the portrait of melancholia, and the facial lines in this are all more clearly defined than in the other. The well-known staring look of paretics is entirely absent in the composite of melancholia.

PAIN IN THE FEET.

By CHARLES K. MILLS, M.D.

PROFESSOR OF DISEASES OF THE MIND AND NERVOUS SYSTEM IN THE PHILADELPHIA POLYCLINIC;
NEUROLIGIST TO THE PHILADELPHIA HOSPITAL, ETC.

A Clinical Lecture delivered at the Philadelphia Hospital, Nov. 12, 1887.

Reported by WILLIAM H. MORRISON, M.D.

THE first patient I bring before you this morning is suffering not from an affection of the head, with which specialists in nervous and mental diseases are supposed to be more particularly interested, but from a disease of the feet. It is my purpose to show you this and some other patients, and then make some remarks upon the subject of pain in the feet, certainly a very practical topic, and one that interests us as neurologists, as surgeons, and as general practitioners of medicine.

I will first give a partial history of this patient, and then examine her in your presence.

A. K., 38 years old, born in Philadelphia, white, a seamstress, weighs 189 pounds Both father and mother died of phthisis ; one brother is an epileptic, and another died in the insane asylum. She has been a drinking woman, and for several years has been subject to epileptic seizures.

About ten months ago, during the winter, she began to have pain in the left foot, chiefly in the heel and along the outer border of the foot. This pain was accompanied by a "pins and needles" sensation. It was dull and aching in character. In a few weeks she began to have a similar but worse pain in the right foot, chiefly also on its outer border and in the heel. Many remedies and methods of treatment were tried without affording any continuing relief. They included severe counter-irritation, temporary rest, galvanism; and internally, gaultheria, salicylate of sodium, iodide of potassium, colchicum, etc. June 11, 1887, Dr. J. D. Deaver, of the surgical staff of the Hospital, stretched the posterior tibial nerve, making an oblique incision about two and one-half inches long between the internal malleolus and the tuberosity of the os calcis.

The right posterior tibial was the one operated upon, for at the time of the operation the pain in the right foot was more severe than in the left. The woman was kept in bed from six to seven weeks after the operation. Strange to say, the left foot got well, while the right, although much improved, is not yet entirely free of pain. This fact led some of those observing the case to think that possibly it was a hysterical case, for you may have a hysterical pain in the feet, as hysterical pain may have its seat anywhere. I think, however, that a more rational explanation is to be given of the recovery of the left foot: the woman was put to bed and kept absolutely quiet for six to seven weeks, and under the influence of rest the trouble subsided. The rest instituted before the operation was neither as absolute nor as complete as that after the nerve-stretching. This is an interesting therapeutic observation, and shows the great value of complete and long-continued rest in such cases.

Let us next determine the condition of this woman's feet at the present time. The proper way to examine a foot which is the seat of pain, is first to study it in a general way, eliciting the statements of the patient, and then to manipulate it by certain definite methods. First manipulate it with reference to the ankle and other joints. In this case there never was, nor is there now, any distinct pain in the ankle-joints. The metatarso-phalangeal joints should be examined in detail. At present I can elicit no pain in the left foot either by pressure or handling. The foot in the course of the examination should be squeezed laterally, as you see me do. By so doing, you will often not only bring out a particular sort of pain, but also perhaps the exact location of the lesion that causes pain. In the left foot this compression causes no pain. Examining now the right foot, I find no pain in the ankle-joint. Squeezing the foot in the line of the metatarso-phalangeal articulations causes severe pain. Pressing each metatarso-phalangeal articulation vertically, I find severe pain in and about this joint of the fourth toe, and also in the corresponding joint of the great toe ; there is very little in the joints of the second and third toe, and none in the joint of the fifth toe.

As I have already said, it was supposed by some that this was possibly what, for want of a better name, we call hysterical pain. Against such a diagnosis are the facts that pain is present in different locations, and that it is increased by special manipulation ; that the pain in one foot gets well under a certain kind of treatment, while in the other foot, although abated, it continues. This woman has symptoms which indicate something real, and, temporarily at least, organic. Without doubt she is suffering from a local but somewhat diffused neuritis. Her weight, and the more or less depraved condition of her blood, have probably acted as causative factors.

A professional friend, a man weighing between two and three hundred pounds, consulted me informally with reference to a severe pain in his heels. The pain continued, and to a certain extent spread so as to involve a portion of both feet, although it was most severe in the heels. In view of this lecture, I wrote to him a few days ago, and he replied that, after trying various methods of treatment, the pain had disappeared since he had adopted shoes of a certain pattern. In this letter he states that he has had several patients who have complained of pains in the heel or in the feet, particularly across the instep and ball of the foot, and that these cases occurred especially in young men, who either wore shoes that were too tight or bad fitting, or who followed occupations which required them to be on their feet for a long time.

Pain in the feet is of great interest not only from the standpoint of a particular case like this, with a local cause and seat, but also because frequently it is the evidence of the beginning of some general or constitutional trouble. Such pain may be due to a number of conditions, as to a local neuritis, periostitis, or arthritis, which is either rheumatic or gouty, and this is the conclusion jumped to in many cases; sometimes it is a correct conclusion, even cases in which acute redness or swelling are not present. In these cases, treatment will help decide the diagnosis.

I now have a patient under my care who has suffered from gout, and has also had acute rheumatism within a few years. A few months ago, he was taken with a pain at the junction of the tendo-Achilles with the bone on each side. Slight evidences of thickening were also present. The pain was so severe as to seriously interfere with his walking. Under the use of lithium, iodide of potassium, and arsenic, with galvanism and massage, the case is getting well with moderate rapidity.

The late Prof. S. D. Gross paid especial attention to this subject of pains in the feet, and as far back as 1864 published a paper on Pododynia, describing cases. In his Surgery a brief allusion to this subject will be found. He speaks of the fact that a large number of his cases of pains in the feet occurred in tailors, and he had begun to think that it was a tailor's disease; but further experience showed him that others exposed to similar causes developed the same trouble. Those among tailors most likely to be affected were cutters, who had to stand at their work for hours. His view was that in all probability it was due to periosteal or aponeurotic inflammation. "The soreness in pododynia," says Prof. Gross,* "is generally most severe in the sole of the foot, over the calcaneum and the ball of the great toe, or in the line of the metatarsal-phalangeal joints, parts which are particularly subject to pressure during the erect position. The hollow of the foot, however, occasionally participates in the suffering. The pain and tenderness are deep-seated, and are always aggravated by the pressure of the finger, and by walking and standing, which the patient is often obliged to forego in consequence. Instead of pain there is often a disagreeable tingling sensation in the parts. Little swelling attends the disease, and there is seldom any marked discoloration of the skin, except in the more severe forms of the affection, when the surface occasionally exhibits a mottled or purplish appearance, evidently dependent upon a congested condition of the capillary vessels. A sense of coldness often pervades the entire foot ; and in some cases the disease extends to a considerable distance up the leg. Both feet often suffer simultaneously. The general health is seldom materially, if indeed at all, affected.

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* System of Surgery.

" What the pathology of pododynia is, I have never been able to determine, as no opportunity has been afforded in dissecting the parts. The probability is that it is a form of inflammation, chiefly of the periosteum and plantar aponeurosis, attended with disordered condition of the vaso-motor nerves and an inordinate determination of blood. In the cases which have fallen under my observation, it has not been in my power to trace any connection between the disease and gout, or between it and rheumatism."

Dr. Sayre* treats at length of a certain class of cases of pain and weakness of the foot or feet, with flattening of the arch, of which I have seen many examples.

" When the arch of the foot," he says, " is properly supported by a healthy tibialis-anticus muscle, the articulating facets of the bones composing it press upon each other, so as to sustain the weight of the body without producing pain. These articular cartilages having no blood vessels or nerves of their own, are insensible to pressure ; but, when the arch of the foot loses its proper support in consequence of a complete or partial paralysis affecting the tibialis-anticus muscle, these articulating facets no longer press upon each other equally, but are made to tilt a little, and the pressure is brought to bear upon the edges of the articular surfaces, where the supply of blood vessels and nerves is most abundant, which gives rise to indescribable pain and suffering with every step that is taken. The pathology of these cases is, first, paralysis of the anterior tibial muscle ; second, settling of the arch of the foot ; third, abnormal pressure upon the edges of the cuneiform and scaphoid bones. The pressure in this abnormal position produces periosteal, it may be osteal, or synovial inflammation, and then it is that the case is so often regarded as one dependent upon constitutional disease."

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* Orthopædic Surgery and Diseases of the Joints, by Lewis A. Sayre, M.D.

Dr. Sayre's treatment is the division of tendons in some cases, and the use of a shoe with a steel sole, which is a well-known treatment for valgus. My own experience with some cases of a milder type is that it is not always necessary to use a steel sole, and that the trouble can be remedied by having a shoe with a leather sole made in a peculiar way. The heel should be low rather than high, and should run from half an inch to one inch further under the foot than is usual. In order to take away from the clumsy appearance of such a shoe, the heel can be cut away a little from behind. A thickness of stiff leather should be put in the position of the hollow of the foot, and another should extend up the inner side of the shoe.

PAIN IN THE FOOT DUE TO LOCAL NEURITIS AND BONE PRESSURE—DR. THOMAS G. MORTON'S OPERATION. NERVE-STRETCHING AND NERVE-SECTION.

While upon this subject, I wish to give you an experience from my private practice which illustrates the value of another method of treatment, which some time ago I was inclined to try in the present case, but did not because of the diffuseness of the pain. You will occasionally meet with cases in which for months, or it may be years, pain will be localized in one foot and perhaps in a certain spot. Sometimes the pain is absolutely excruciating. On a winter's day even, the patient will stop in the street and will remove his shoe, or will sit down anywhere for a time, in order to get relief. This pain is present more or less all the time. I have certainly had from five to ten such cases during a few years. If they are examined closely, it will be found that in some of them the pain is localized to some one part of the foot. One point is the spot which in our first patient is the most painful—about the metatarso-phalangeal articulation of the fourth toe. Dr. Thomas G. Morton, the distinguished Philadelphia surgeon, has paid great attention to the subject of pain in the feet from the surgical standpoint. About ten years ago he described several cases of this kind, and he has devised an operation for their relief. I think that his view as to the causation is correct, at least in a certain number of cases. As I have already pointed out, one of the spots at which pain is most likely to be felt is about the metatarso-phalangeal articulation of the fourth toe. Dr. Morton gives an anatomical reason for this location of pain. So important is the matter and so ingenious the explanation, that I think it is best to quote his exact words :

"The occurrence of the neuralgia," says Dr. Morton,* "may be understood by a reference to the anatomy of the parts. The metatarso-phalangeal joints of the first, second, and third toes are found on almost a direct line with each other, while the head of the fourth metatarsal bone is from one-eighth to one-fourth of an inch behind the head of the third, and the head of the fifth is from three-eighths to half an inch behind the head of the fourth ; the joint of the third, therefore, is slightly in advance of the joint of the fourth, and the joint of the fifth is considerably behind the joint of the fourth.

"The fifth metatarsal joint is so much posterior to the fourth that the base of the first phalanx of the little toe is brought on a line with the head and neck of the fourth metatarsal, the head of the fifth metatarsal being opposed to the neck of the fourth.

"On account of the character of the peculiar tarsal articulation, there is very slight lateral motion in the first three metatarsal bones. The fourth has greater mobility, and the fifth still more than the fourth, and in this respect it resembles the fifth metacarpal. Lateral pressure brings the head of the fifth metatarsal and the phalanx of the little toe into direct contact with the head and neck of the fourth metatarsal, and to some extent the extremity of the fifth metatarsal rolls above and under the the fourth metatarsal.

"The mechanism of the affection now becomes apparent when we consider the nerve supply of the parts. The branches of the external plantar nerve are fully distributed to the little toe and to the outer side of the fourth ; there are also numerous branches of this nerve deeply lodged in between these toes, and they are liable not only to be unduly compressed, but pinched by a sudden twist of the anterior part of the foot. Any foot-movement which suddenly may displace the toes, when confined in a shoe, may induce an attack of this neuralgia. In some cases no abnormality or other specific cause for the disease has been detected."

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* Philadelphia Medical Times, October 2, 1886.

Dr. Morton's operation consists in making an incision usually about two inches in length along the outer edge of the tendon of the toe most affected ; then opening the articulation and removing the adjoining portions of the metatarsal bone and phalanx. Under antiseptic precautions the operation is comparatively trifling. It has been performed from the sole of the foot, but the operation from above is preferable as the bones are thus more easily reached.

Let me now recur to the illustration from my private practice of a case successfully treated by this operation. The patient kindly wrote out for me the history of her case, and from this and my private note-book I have prepared the following account :

In July, 1877, in jumping from one stone to another three or four feet lower, her right foot slipped and she came, with all her weight upon her left foot, upon a sharp point of rock, twisting the ankle. The sole of the shoe was cut through, but the foot was not bruised at all, and it was for only about a week that she could not walk straight. During the next two years, at intervals of from two to eight weeks, she would have a peculiar pain in the foot, which would only last about two or three days.

In 1879 she hurt her foot again in the same way, and then pain was seldom absent longer than about a week, and each time would be more severe than the last. In 1881 the pain became constant, never being absent longer than an hour. For two weeks it would be so intense that she would be nearly frantic, then for about a week or ten days it would be less severe.

The pain was a dull, heavy, sickening ache, from the foot to the hip, and with a sharp, hard pain through the foot. When easier the ache would only be in the foot, but the sharp pain was there constantly.

Rising in the morning, the patient could not put her weight upon her foot, until she had taken hold of the foot suddenly from the top and pressed it hard together, and held it in both hands with all her strength for several minutes.

Many plans of treatment were tried in this case—gouty and rheumatic remedies were used, and many forms of external application, without success. Painting with iodine, anodyne ointments and liniments, galvanism, and temporary rest, were alike unsuccessful. Persistent blistering gave the most relief. The patient was kept in a sitting or recumbent position for several weeks, while successive blisters were applied to the sole of the foot. She was benefitted by this treatment probably as much from the rest as from the blistering, but in a short time the pain returned with all its former severity.

I now concluded it would be best to have an operation performed for the relief of the condition which was scarcely endurable. Dr. D. Hayes Agnew was called in consultation, and, after etherization, excised the end of the third metatarsal bone and the approximating portion of the phalanx. The patient had some little trouble in the after treatment of the wound, but in about three months everything was completely healed and the pain much better. About a year after the operation the foot was roughly trodden on; inflammation ensued, and an abscess formed and discharged, but healed without special difficulty. The patient writes to me this week that she has not had the slightest trouble for a year and a half, and has walked as much as twelve miles at a stretch without producing any trouble in the foot.

Another private patient, a lady of about sixty years of age, tells me that forty-two years ago, after stepping on a stone, she was suddenly seized with a pain near and in the line of the fourth toe. The pain was extreme, and was accompanied by some swelling and redness. She was compelled to keep off her feet from three to four weeks, and suffered considerably for some weeks afterwards. Ever since, at times, averaging at least once a year, she is seized suddenly with pain, sometimes very severe in the same locality. The pain usually disappears with rest and care in using the foot.

Let me say a word or two more about operative procedure in such cases. As you will remember, the posterior tibial nerve, after supplying the muscular, osseous, articular, and cutaneous tissues of the back of the leg and ankle, divides upon the body of the calcaneum into the internal and external plantar nerves, which supply the sole of the foot and adjoining tissues. The internal plantar nerve has a distribution resembling that of the median nerve in the hand, the external that of the ulnar nerve in the hand. I here show a diagram illustrating the distribution of the two plantar nerves. The internal planter nerve divides into two branches called the internal and the external. The internal branch is also called the median branch and first digital nerve ; the external, also called the lateral branch, divides on about a line with the tarso-metatarsal joint into the second, third, and fourth digital nerves. The first, second, third, and fourth digital nerves supply the first, second, and third toes, and the internal aspect of the fourth toe. The external plantar nerve divides into internal and external branches, which supply the little toe and outer aspect of the fourth toe. This is simply, in general terms, the distribution of these nerves. Numerous small branches are lodged in various positions.

I think when the trouble is limited to the branches of the external plantar nerve going to the fourth and fifth toes, it may sometimes not be necessary to perform an operation so radical as the excision of the articulating faces of the bones. The nerve itself might be resected or stretched. This was Dr. Deaver's idea in connection with our first case. The posterior tibial nerve was stretched in the hope of avoiding excision of the joint. For some reason, however, this operation in all cases does not succeed so well as excision. Nerve stretching is not so good an operation for the relief of pain due to neuritis as for true neuralgia, or where certain central troubles are the cause of pain. I think that section is better than stretching if you are sure that you can locate the particular branch of the particular nerve going to the particular spot affected. Of course, certain objections to the operation of nerve section are apparent. It may produce a paralytic condition of the foot which may interfere with its usefulness, but it is nevertheless sometimes a desirable procedure.

In 1882, a young lady was under my care at the University Hospital. She suffered with pain, burning and tingling sensations, marked and very distressing, chiefly along the outer side of the top of the foot. Many measures of treatment were tried without success, including counter-irritation, galvanism, rest, and rheumatic and gouty remedies. Eventually, two operations were performed by Dr. H. R. Wharton. Early in July, he cut down and stretched the musculo-cutaneous and external saphenous nerves, an operation which was followed by a slight temporary diminution of pain. August 18, 1882, he excised one and a half inches of the external saphenous nerve, and peroneal communicating nerve, behind the external maleolus. This operation was followed by marked diminution of pain, and after a long time the patient recovered.

PLANTER NEURITIS (OR NEURALGIA).

Dr. C. H. Hughes, of St. Louis, under the name of plantar neuritis, or neuralgia, has described a painful affection of the foot, which may be due to a variety of causes. We had in this Hospital, not long ago, a patient with caisson disease. This man, while working in a caisson, during the building of a bridge across the Schuylkill River, was taken down with symptoms of this affection. He suffered terrible pains in the legs, had various head symptoms, was paralyzed, and went through the whole series of phenomena of caisson disease. He was paralysed for months, but finally recovered and left the Hospital. Among other symptoms, he had at one time excruciating pains in the feet.

Dr. Hughes speaks as follows of this affection :

"It comes on as a sequel, usually of a low form of blood depraving fever, like typhoid or protracted malarial, with typhoid-like depression, or in the latter stages of phthisis ; but it may be the sequel of an exhausting, long-continued rheumatism, or possibly of a badly managed or neglected and chronic gonorhœa, as Ross asserts, though I have never seen this as a result of that disease. It appeared as a conjoint symptom in some cases of caisson disease at the time of the building of the St. Louis bridge, and I have seen it follow upon a residence in the high altitudes of Colorado and an attack of the so-called mountain fever of that region. It comes upon a nervous organization, shattered and tremulous and choreic, and the painful paroxysms are agonizing. The patient cries out with pain, and often cannot rest at night, even after prolonged wakefulness, without powerful anodynes. The slightest touch, such as the application of local anodynes with the hair pencil, to the painful parts, often cannot comfortably be borne. A peculiar burning sensation, without themometric evidence, accompanies the pain. The pain is usually localized in the balls and the tips of the three toes supplied by the internal plantar nerve, and in the heal and plantar arch of the foot, but sometimes implicates also the two smaller toes, which are supplied from the external branch of the plantar nerve, the fifth toe being supplied exclusively by the external plantar, while a filament from the internal joins with the external in giving the fourth its æsthesiodic supply."*

The following case, evidently one of neuritis occurring in the course of typhoid fever, is interesting in connection with these remarks of Dr. Hughes on plantar neuritis as a sequel of low fevers. Such cases indeed are not rare, but are often not understood.

The patient, a young man, had typhoid fever in July, 1884. The attack was a severe one, confining him to the bed for six weeks entirely, and partially for two weeks longer. His convalescence was slow. During the first two weeks of the fever he had marked brain symptoms and was unconscious of his surroundings. On coming to himself at the end of this time, he began to suffer severely with burning, tingling, and numbness of the feet, which were also hyperæsthetic, so that he could scarcely bear the weight of the bed clothes upon them. The condition was most marked across the toes and back of the feet near the toes. The pain and uncomfortable sensations were temporarily relieved by friction.

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* Western Medical Reporter, April, 1887, and Alienist and Neurologist, April, 1887.

On getting out of bed, he found that he was weak in both legs, and for a long time had a paretic or ataxic gait. He was troubled with numbness and tingling from the upper part of the thighs to the feet. Both feet and legs were tender to pressure. The feet showed a tendency at times to swell, and on exposure became white and mottled; they also showed a marked tendency to sweating. The soles of the feet were exceedingly tender.

As the patient grew stronger, the condition of his legs and feet gradually improved, but for many months he suffered greatly. The pressure of the feet upon the floor when standing caused him intolerable discomfort. He went about, however, suffering more or less all the time. He had shoes of half a dozen different patterns made, in order, if possible, to procure some relief. Squeezing the foot, either vertically or laterally, caused great pain. The numbness and tingling disappeared slowly from above downwards, a marked girdling or encasing sensation being ususlly present at its upper line. Numerous corns formed upon his feet, he not having been subject to them before his sickness. His feet felt as if they were cramping or contracting downwards, and at times assumed a condition of slight plantar flexion.

Although more than three years have elapsed, this gentleman still has some numbness in both feet, particularly across the dorsum of both great toes and the adjoining sides of the second toes. If he walks much, he has a contracting sensation in the balls of both feet.

Possibly a more prompt recovery would have taken place in this case if the patient had been taken off his feet, and had had gaultheria, salicylate of sodium, or small doses of mercury with tonics, internally, with counter-irritation and galvanism locally.

ERYTHROMELAGIA.

A disease, of which I have seen five or six cases, is included by Dr. Hughes under the head of plantar neuritis, although I do not think properly. It was described a few years ago by Dr. S. Weir Mitchell,* under the name of erythromelalgia, which term indicates pain and redness. This is a most remarkable effection, and differs somewhat from those already described. These cases begin in much the same way, with pain in the heel, or in the sole, or in a toe, and then the pain will increase and multiply. It augments both in extent and intensity. Later, another phenomenon is added, that is, a distinct flushing, or even a reddish or purplish appearance of the feet. The moment the feet are allowed to assume a dependent position, a horrible pain starts in them, and almost at once there appears a slight flushing, then a diffused redness, and finally a mottled, purplish appearance spreads over the feet. One of the patients described in Dr. Mitchell's paper had been under my own observation. This man was perfectly comfortable so long as the feet were kept elevated. I remember very well the first time that I saw him. He was lying on a lounge, with the head and feet about in the same place, and was suffering no pain. In order to examine his feet, the leg was allowed to hang over the side of the lounge. In a moment it begun to change color, and instantly the patient was in agony. As long as the horizontal position was maintained he was free from pain and discomfort. This is not always the case in neuritis. Erythromelalgia, in some cases at least, is, I believe, disease of spinal origin. Sometimes it may be due to a neuritis, or even possibly to some disease of the veins. In the following case of multiple neuritis, or poliomyelitis, it was present as a symptom.

S. H., aged 40, white, born in Ireland, is a dressmaker. She has had syphilis, and drinks. She was always well until about five years ago, when she had an attack, which she described as similar to the present, but from which she recovered in a few months. Six years before this she had had still another similar attack.

About four weeks before admission to the Hospital, she began to have pains, of a sharp shooting character, in the feet and legs, with burning and flushing; these were followed by a feeling as if the legs were asleep. The pains were worse at night. Just before admittance, she began to have contractures in both legs, and both were atrophied.

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* American Journal of the Medical Sciences, 1878.

The knee and muscle jerk are abolished. She has always had control over the bowels and bladder. Sensation is good, and she has no hyperæsthesia. She has contractures of the fingers of the left hand. Her legs feel numb, and she has severe pain in them occasionally. She has pain on pressure along the trunks of the posterior and anterior tibial nerves at their upper parts. They are somewhat sensitive towards the foot, though not as much so as nearer their origin.

In one case of multiple neuritis or general poliomyelitis, a woman who was confined to her bed for fourteen months, when gradually recovering, the moment she attempted to put the legs in a hanging position, exactly the same thing happened as we see in erythromelalgia. The feet would begin to pain, turn red, and then purple. This tendency continued, with gradually decreasing severity, for two or three months, when it entirely disappeared.

FEET PAINS IN POSTERIOR SPINAL SCLEROSIS, AND IN SOME OTHER FORMS OF CHRONIC SPINAL DISEASE.

It is well known that one of the very earliest symptoms of posterior spinal sclerosis is the occurrence of shooting, lancinating, or lightning-like pains, usually in the lower extremities. The foot is not the commonest seat of the initial occurrence of these pains, but it is by no means an infrequent locality. Pain in the heel is somewhat common. Spitzka, in the American System of Practical Medicine, speaks of one patient, who, after experiencing initial symptoms for over a year, woke up at night with a fulminating pain in the heels, which recurred with the intensity of a hot spear thrust and with the rapidity of a flash every seven minutes. A private patient, a lady 46 years old, who came to me first a few weeks ago, has been suffering, off and on, for four years with cutting pains in the right heel, calf and thigh, and sometimes in the first and fourth toes. These pains preceded other symptoms, which are now tolerably well developed, such as double vision, difficulty in holding her urine, diminished knee jerk, and slight ataxia.

For the sake of actual illustration, I show you here an ataxic patient from the Women's Nervous Wards. She is 42 years old. About five years ago, she was kicked in the left side, as the result of which she was confined to her bed for a week. Soon after she had a severe uterine hæmorrhage. After this she did not menstruate for two years, then she menstruated regularly till September, 1886, then her menses ceased entirely. Soon after she received the kick, she commenced to have sharp pains in her limbs and feet, and later in the trunk and fingers. Gradually she developed the typical symptoms of posterior spinal sclerosis—ataxia, failing sight, Argyle-Robertson pupils, anæsthesia, and paresthesia, abolished knee-jerk, etc. She sometimes has sharp pains in her feet; a few days ago, for instance, she had a severe pain in the outer side of her left foot just in front of her heel, as if a knife had been suddenly thrust into the spot and then suddenly withdrawn. This pain sometimes runs along the outer side of her foot ending in the ball of the fifth toe.

Of course, I could furnish from my note-books many similar illustrations of pains in the feet in ataxic patients, but it would be a tiresome and unnecessary repetition. My only purpose in calling attention to these pains in this lecture, is to emphasize the fact that such heel or feet pains are sometimes very early, and even initial, symptoms of this affection, and therefore the physician should be on his guard and not make an improper diagnosis. Such pains are often supposed to be rheumatic or gouty, or to be evidences simply of a local neuritis from an unknown cause.

Posterior spinal sclerosis is not the only form of chronic spinal disease in which feet aches or pains are among the early or initial symptoms, but in disorders other than locomotor ataxia, the pains are usually not simple in character, but are associated with other conditions, as of spasm or vaso-motor and trophic conditions.

About three years ago a lady, 60 years of age, consulted me about a burning pain or sensation along the outer border of the left foot from the toe to the ankle. This was accompanied by a peculiar jerking upwards of the left great toe, and sometimes of the second toe. Her fingers also, sometimes, felt stiff, and she was nervous and apprehensive, but had no other positive symptoms. During the last three years, however, the case has become one of well marked disseminated sclerosis.

In 1884, I was consulted by a gentleman from Illinois, 52 years old. Three years before coming under observation, a slight burning sensation was felt under the nail of the second toe of the right foot. This sensation increased, and soon became very painful. One by one, all the other toes of the same foot were affected in a similar way, and at the time he came to see me the painful disease had extended until it had involved the second, third, and fourth toes of the left foot. The affected toes were of a bluish red color, and the feet were mottled to a short distance above the roots of the toes. So great was his suffering, that in hope of relief he had the first toe affected amputated in January, 1883. The patient did not remain under my care, but I was satisfied as the result of my examination, that he was the victim of a chronic nervous disease, probably of the spinal trophic centres.

REFLEX OR TRANSFERRED PAINS IN THE FEET.

In order to round out my subject, let me say, in conclusion, that occasional pains in the feet are reflex or transferred. Of these Dana* speaks as follows: "Studies of the cause of reflex pains in the feet show that they may be referred in almost all cases to irritation of the genito-urinary tract, and occur more often in the male ("Med. Record," July 25, 1885). The pains of uterine disorder when reflected down, appear rarely to go below the knee; in other words, they affect the lower branches of the lumbar plexus, and not the sacral nerves. It has been stated that pain in the heels may be caused by ovarian abscess. In my experience, such pains are due to lithæmic and neurasthenic conditions, and will be relieved by remedies addressed to such states. It may be said in general, then, that pelvic irritations are felt most frequently in the upper and short branches of the lumbar plexus, next perhaps in the intercostal nerves and upper cervical nerves, then in the trigeminus, and last in the hands and feet."

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* New York Med. Journal, July 30, 1887.

REPORT OF A CASE OF ANENCEPHALY, WITH A MICROSCOPICAL STUDY BEARING ON ITS RELATION TO THE SENSORY AND MOTOR TRACTS.

By CHARLES L. DANA, A. M., M.D., New York

PROFESSOR OF NERVOUS AND MENTAL DISEASES, NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL.

CASES in which infants are born at full term with no fore-brain are rare. Syme describes a case, *Edinb. Med. and Surg. Journal*, vol. xxiv, p. 295, in which the infant lived six months. After death only the cerebellum, pons, and parts below were found. Panizza, of Paris, reports a similar case that lived eighteen hours (Gintrac, Maladies de l'appareil nerv., Paris, 1867, v. i., p. 51). Ollivier reports one that lived twenty hours (Maladies de la moelle epiniere, Paris, 1837, v. i., p. 179). The two cases of Rohon and of Starr have been reported recently, and are familiar.

This form of monstrosity, so far as I can judge, would be called by Geoffry St. Hillaire "pseudo-encephaly."

The interest in such cases previous to the discovery of the modern methods of studying nerve anatomy has been purely clinical and teratological.

We know now, however, that all such cases of congenital anomalies of the nervous system constitute natural experiments in the atrophy method, and that one of the chief interests in monsters lies in what they tell us post-mortem regarding nerve tracts and centres.

CLINICAL HISTORY.—The case is that of a male infant born at full term. The mother was unmarried, but was healthy, denied syphilis or anything unusual in the mode of sexual intercourse or incidents of pregnancy. She had had no other child. The father, she said, was healthy.

The child was large for its age, must have weighed eight or nine pounds, was very plump and healthy-looking. It showed no deformity except that the head was peculiarly shaped. It was large proportionately, the forehead narrow, and the whole head very narrow and long in the occipital-frontal diameter. The sutures were not united, and the bones were freely movable. The eyes were generally closed, but occasionally opened. The child nursed a little, but had difficulty in swallowing, and after feeding would have attacks of cyanosis.

It was noticed that it cried very little, and only in a feeble way. It had natural movements of the bowels and bladder. It had had no convulsions or rigidity or paralysis. On pinching or pushing it, the infant cried. It lived two and one-half days.

AUTOPSY.—On puncturing the membrane between the cranial sutures, a yellowish liquid spurted out. Cutting a way the calvarium, the whole cranial cavity above the tentorium was found perfectly empty and smooth. The anterior and middle fossæ contained nothing but the foldings of the membranes. In the posterior fossa the tentorium was bare, but the cerebellum could be felt beneath it.

The cerebellum was fully developed and of good size, measuring 6½ ctm. in width and 3½ ctm. anterio-posteriorly. The roots of all the cranial nerves were present except the first. The optic nerves were small, and the chiasm scarcely to be made out. The optic tracts showed barely a trace. The third nerves were well developed. The fifth nerves were all well developed, and both motor and sensory roots could be seen. They arose far out on the lateral surface of the pons in the sulcus between it and the cerebellum. The other cranial nerves could be made out in the sulcus external to the olives, the hypoglossal apparently arising from the lower outer border of the olives.

The vertebral arteries joined to form the basilar, which at the cephalic end of the pons abruptly terminated in four small arteries. The two anterior of these passed forward to the optic thalami, the two posterior to the cerebellum.

The pons varolii was very small. The medulla appeared to be made up mostly of the two large olivary bodies, which almost met in the median line. The corpora quadrigemina seemed fairly well developed. Anterior and external to these were two lobes, evidently parts of the optic thalamus, and the sole representation of the first cerebral vesicle. The left was much the larger, and measured, in a direction from outside and posterior up and in, 3 ctm. The spinal cord appeared normal, but small.

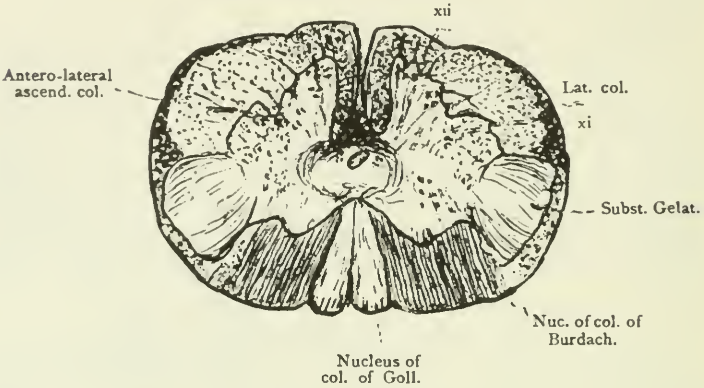
The specimen was hardened in Muller's fluid and then in alcohol. Sections were made at the level of the third to fourth lumbar nerves, eighth to tenth and fourth to sixth dorsal, fourth to sixth and first cervical

Sections were also made, at different levels, through the medulla and pons. These sections were stained in Weigert's and ordinary hæmatoxylon, carmine, and aniline blue.

The most interest attaches to the appearances in the pons and medulla, and these will be described first. The cerebral hemispheres and corpora striata being entirely absent, as already stated, we should expect to find absent all those uninterrupted tracts depending for their nutrition upon the integrity of the parts mentioned. The afferent tracts to the brain should be present.

MICROSCOPICAL APPEARANCES.—Section I. Beginning from below, I find, in sections at the level of the motor decussation, absence almost entirely of this decussion and of the lateral pyramidal tracts. In place of this, one sees only a decussation of sensory fibres starting from the nuclei of the post-median and post-external columns. The fibres cross over at the bottom of the anterior median fissure, part (1) turn up directly, and part (2) pass anteriorly along the median surface of the anterior fissure to go towards the olivary body. The first set (1) form the ascending fibres of the inter-olivary tract.

Posterior to the sensory decussation is a decussation of a few fibres connecting the cells of the anterior horns.



The post-median and post-external nuclei are well developed, as are also the corresponding columns at a lower level.

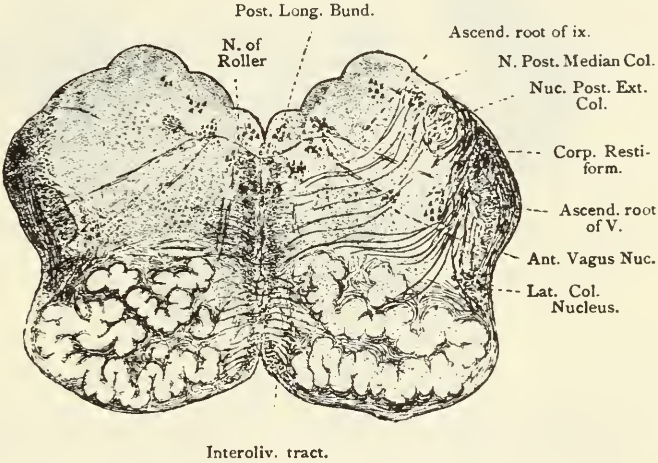
The gray matter is normal in arrangement and in its cells, though the anterior horns are a little smaller than normal. The place of the crossed pyramidal tracts is occupied only by a few nerve fibres.

The anterior columns are abnormally narrow. The direct cerebellar tracts are well and distinctly marked. There is apparently only a partial development of the antero lateral ascending column.

The anterior and posterior nerve roots are well developed. The fibres of origin of the ascending root of the fifth are visible. The nuclei and fibres of origin of the eleventh and twelfth nerves can be seen. Fibres can also be seen to pass from the posterior columns into the lateral column and lateral part of intermediate gray, then turning up to enter at a higher level, the formatio-reticularis.

SECTION II.—In the next series of sections, at the level of the lower part of the olive, the pyramids are seen to be entirely absent, so that the olives form the ventral edge of the section.

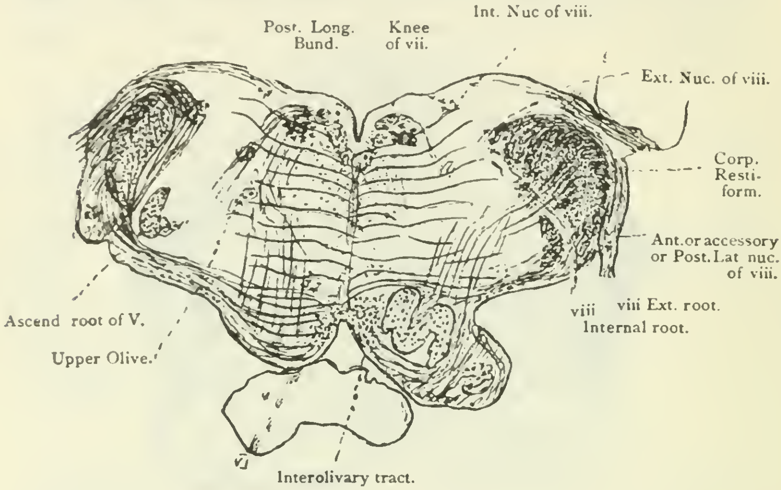
The interolivary tract is present, but is very poorly developed. The part best seen is the ventral portion lying almost at the bottom of the fissure between the olives. This ventral portion of the interolivary tract is seen to be made up from internal and anterior arcuati fibres from the post-median and post-external column nuclei. Posteriorly, the longitudinal bundle is seen normal in size. The nucleus of Roller is present. The formatio-reticularis is of nearly normal appearance, but not quite so rich in fibres as it should be ; the internal and external olives are present.


Post. Long. Bund.
N. of Roller
Ascend. root of ix.
N. Post. Median Col.
Nuc. Post. Ext. Col.
Corp. Restiform.
Ascend. root of V.
Ant. Vagus Nuc.
Lat. Col. Nucleus.
Interoliv. tract.

The respiratory bundle is normal. The anterio-posterior fibres of the raphe are numerous, but seem to end abruptly at the bottom of the anterior median fissure between the olives. The raphe fibres in general are much more numerous in the interolivary portion, as is the case in normal cords.

The lateral column fibres and nucleus are very distinct. The gray matter and cranial nuclei on the floor of the ventricle are normal.

SECTION III.—The next section is made at the upper border of the olives and lower edge of the pons. The inter-olivary tract is quite distinct in the ventral portion between the olives. Its middle and dorsal portions are fairly represented, while the posterior longitudinal bundle is very distinct. The anterior arcuate fibres pass in a distinct strand from the corpus restiforme to the ventral part of the raphe. Here some cross and some enter the lemniscus. The fibres of the raphe are fully developed dorsally.

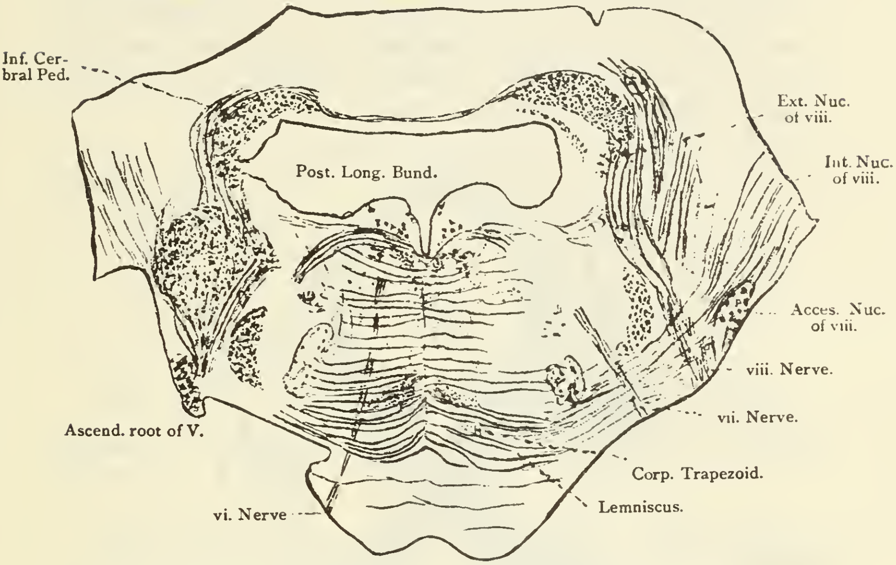

Int. Nuc of viii.
Post. Long. Bund.
Knee of vii.
Ext. Nuc. of viii.
Corp. Resti-form.
Ant. or accessory or Post. Lat nuc. of viii.
viii
viii Ext. root.
Internal root.
Ascend root of V.
Upper Olive.
vi
Interolivary tract.

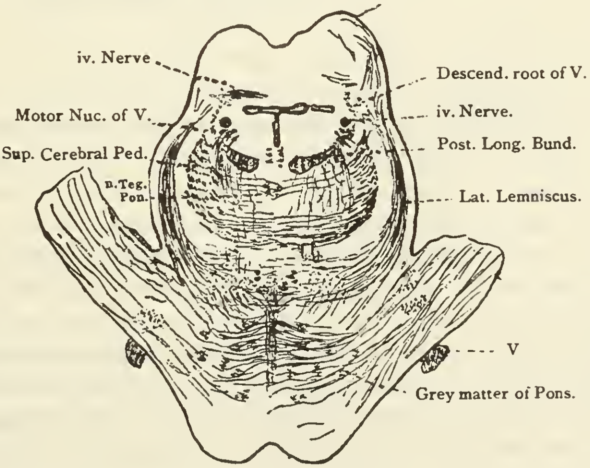
The nuclei of Roller, which is supposed by Bechterew to be the end station of the fibres of the lateral fundamental column, are here very distinct. The formatio-reticularis is rather poor in fibres. What corresponds to the caudal edge of the pons is an an irregular mass of undifferentiated tissue through which the sixth cranial nerves run, and in which can be seen a few fully developed transverse nerve fibres.

The dorso-ventral or linear fibres of the raphe are moderately developed, and are most numerous ventrally. Here they come *via* the anterior arcuate fibres from the corpus restiforme ; they pass up, and having crossed, enter the lemniscus.

It thus appears that the fibres of the raphe serve to connect the cranial nerve nuclei with the pyramids, and also with the lemniscus or corpus restiforme.

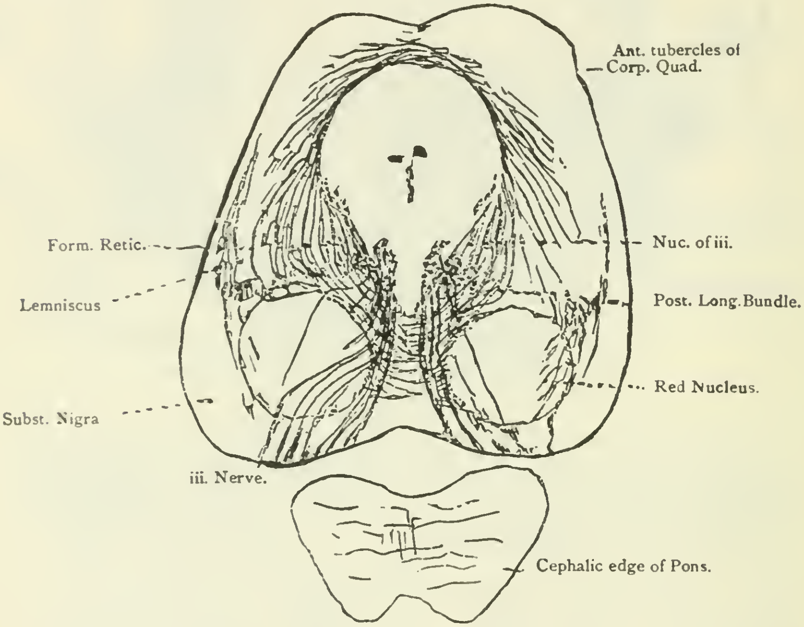
SECTION IV.—At a higher level of the pons, but still below the middle and at about the level of the nucleus of the sixth, the lemniscus is seen in its altered position lying horizontally upon the fibres of the corpus trapezoid, which are here very well marked. It is here small. The fibres of the raphe are very few. The formatio-reticularis is but imperfectly developed here.


Inf. Cerbral Ped.
Post. Long. Bund.
Ext. Nuc. of viii.
Int. Nuc. of viii.
Acces. Nuc. of viii.
viii. Nerve.
vii. Nerve.
Ascend. root of V.
Corp. Trapezoid.
Lemniscus.
vi. Nerve


Post. tubercles.
iv. Nerve
Descend. root of V.
Motor Nuc. of V.
iv. Nerve.
Sup. Cerebral Ped.
Post. Long. Bund.
n.Teg. Pon.
Lat. Lemniscus.
V
Grey matter of Pons.

SECTION V.—In a section still higher, through the upper part of the pons and the posterior tubercles of the corpora quadrigemina, there is a very distinct development of the lateral or lower lemniscus, while the median or upper lemniscus is small.

SECTION VI.—At a still higher level, just through the red nuclei and touching the cephalic edge of the pons, one notes the entire absence of cerebral peduncles. The red nuclei are apparently well developed.


Ant. tubercles of Corp. Quad.
Form. Retic.
Nuc. of iii.
Lemniscus
Post. Long. Bundle.
Red Nucleus.
Subst. Nigra
iii. Nerve.
Cephalic edge of Pons.

The median lemniscus is now plainly visible, also the nuclei and fibres of the third nerve. The lateral lemniscus has ended in the posterior tubercles, and is not seen. Fibres can be seen coming from the posterior commissure and passing down to enter the posterior longitudinal bundle. There is no lenticular loop. The corpora quadrigemina seem small and almost structureless, save for the fibres of the posterior commissure which run through its lower part. There is still some evidence of the formatio-reticularis. The substantia nigra is present, but imperfectly developed.

At this level, the dorsal part of the raphe contains no fibres, the lower or ventral part is very distinct, The fibre bundle from the inner part of crusta is absent. The fibre bundle from the posterior commissure is absent. The offshoot bundle of Wernicke is absent. The nucleus reticularis-tegmenti-pontis sends down fibres through the raphe. This nucleus, according to Bechterew, is the end station of fibres of anterior fundamental column.

The aqueduct of Sylvius is imperfect. The posterior longitudinal bundle is well marked, as are also the fibres from the anterior cerebellar peduncle. The descending root of the fifth is here visible. The nuclei of the pons are present.

Sections through the optic thalami showed a nearly structureless mass containing but a few nerve fibres and cells.

SUMMARY: The pyramidal tracts are absent, and in consequence the shape and relations of the parts are changed.

The formatio reticularis is apparently normal.

The cranial nerves and nuclei, the respiratory bundle, posterior longitudinal bundle, olives and supplementary olives, and in inner and outer nuclei of Roller, are present and normal.

The sensory decussation and interolivary tract and lemiscus are present, but the median portion is about half the normal size.

In the lower sections the ventral portion of the interolivary tract alone is normal in size, the parts dorsal to it being barely visible; at higher levels (upper olive), the tract in question lies as it should in the fibres of the corpus trapezoid, but is small.

At still higher levels (crossing of the anterior cerebellum peduncles) the lateral or lower lemniscus is very clearly defined, the median very scanty in fibres. At the level of the red nuclei the median part is quite distinct.

The following, therefore, is the condition of the lemniscal tracts:

The innermost bundle to the crusta is not present.

The median lemniscus is about half the normal size, but is traceable as far as the anterior tubercles of the corpora quadrigemina, which it appears to enter. This (the median) is the lemniscal tract which goes partly to the anterior tubercles, partly to the optic thalamus or Luys' body, partly to the lenticular nucleus and parietal cortex (*Hauptschleife*, Bechterew, *Rindenschleife* of Monakow, etc., upper lemniscus.

In experimental atrophies the degeneration has usually been downwards, but in Meyer's case it was upwards *in toto*, and it evidently consists, in large part at least, of afferent fibres.

The lateral lemniscus (lower), which begins chiefly in the upper olive, by which it is connected with the posterior branch of the acoustic nerve (Bechterew, Flechsig, Obersteiner), is present, and apparently ends in the posterior tubercles. This tract is doubtless, therefore, afferent.

The tegmental part only of the crura cerebri is present: The substantia nigra is present, but is structurally but little developed.

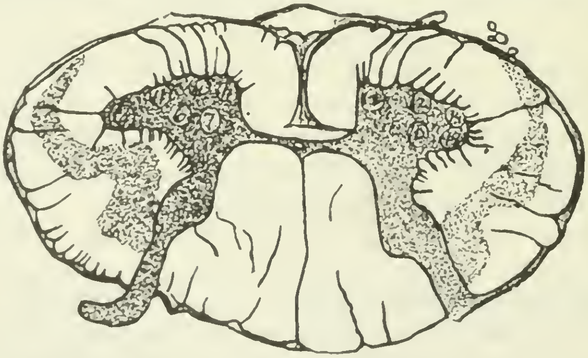
The gray nuclei of the pons are present.

The Spinal Cord.—As a whole the cord is smaller than normal.

The diminished size is due to the small anterior and lateral columns. In comparison, the posterior columns look unusually large.

The nerve roots are normal. The anterior columns are much narrower than they should be, but show nothing abnormal otherwise.

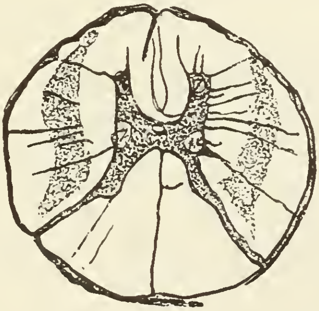
The lateral columns reveal a tract of connective or undifferentiated tissue extending throughout the length of the cord. The position of this undeveloped tract in the upper cervical region is peculiar. As shown in the cut, it appears to extend laterally and ventrally to the surface, occupying, in a measure, the region of the anterior ascending tract. A few nerve fibres are present in this region; on transverse



sections of the cord they are cut off obliquely and appear as if running forwards and inwards. The direct cerebellar tract, Lissauer's bundle, which may be called the posterior root column, the postero-external and postero-median columns are normal.

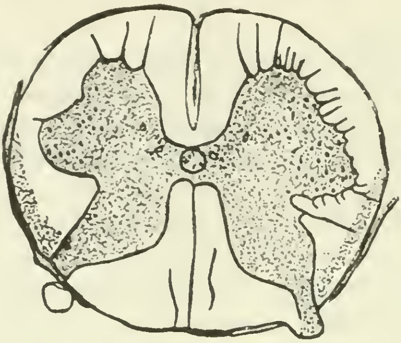
The longitudinal sections were made at the level of the fourth to sixth dorsal roots, the plane of the sections being directed anteriorly, the appearance of the anterior commissure was studied and compared with similar sections in a normal cord.

In the *anencephalic cord the commissure was poorer in fibres, and these crossed diretly* from one side to the other at a very acute angle. In the normal cord some fibres crossed in this way, but others ran along the edge of the fissure for a short distance and then cross at a more obtuse angle. This *must represent the mode of crossing* of the direct *pyramidal tract fibres.*



The posterior roots can be seen to enter in two bundles of fibres; one of fine fibres lies external, appears to connect directly with the posterior nerve and Lissauer's bundle. The other divides into fibres which pass through (?) and around inner side of subs. gel. to posterior cornual cells and Clark's cells. These fibres can be traced to the anterior cornu of same side and through the anterior commissure to the anterior cornu of the other side.

The anterior commissure is divided into two parts, one lying anterior to the other, and it is through the posterior one that the posterior cornual fibres pass.



The gray matter of the cord and its cells are fairly developed.

ON OIL OF GAULTHERIA AND SALOL IN RHEUMATISM OF NERVES AND MUSCLES.

By F. X. DERCUM, M.D.

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Read before the Philadelphia Neurological Society.

IT will hardly be necessary to-night to speak in favor of a drug, the usefulness of which has become so well established as that of the oil of gaultheria. I bring it before you merely to provoke discussion on such points as the proper indication for its use, its dosage, and its value compared with that of other salicylates.

It appears to be the fate of all new drugs to be ushered into the world with acclamations and applause, and the oil of gaultheria has been no exception to the general rule. At the same time, it must with justice be maintained that no other drug has suffered less from the ever-increasing test to which it has and is being subjected. While many of you will not accept, without modification at least, the conclusions of Kinnicutt,* one of the earliest writers upon the subject, I think that you will all agree that the drug has acquired a fixed and definite position among our therapeutic resources. I myself believe that Kinnicutt's conclusions are in the main correct, and yet it is very evident that such assertions as that the administration of the drug is unaccompanied by occasional toxic effects, and is unattended by frequent gastric disturbance, need qualification. Kinnicutt speaks especially of its application in acute forms of rheumatism. He believes it to be at least equally efficient with the sodium salicylate in reducing both temperature and pain. On the other hand, Dr. Squibb,[*] in a recent article, claims that it does not relieve pain or reduce temperature as promptly as sodium salicylate or salicylic acid, and that it disturbs sight and hearing less promptly and to an inferior degree. On the whole, he concludes that it is best adapted to the milder cases of gouty rheumatism or rheumatic gout.

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* Francis P. Kinnicutt, New York Medical Record, Vol. xii., No. 19, p. 505.

My own experience with the drug is limited almost entirely to rheumatic affections of the nerves and muscles. I have tested it in a large number of neuralgias and in almost every form of muscular rheumatism. In trigeminal neuralgias, in neuralgias of the nerves of the extremities, especially sciatica, I have had the happiest results. The failures have been the exception, and indeed they have been so few that I have difficulty in calling them to mind. In those cases in which I have found the drug to act most promptly, there was almost always, I should state, more or less distinct tenderness of the nerve trunk or tenderness over the foramina of exit.

I should also state that I have not been able to elicit a history or other evidence of rheumatism in every case of successful application of the drug, and it is perhaps unfair to conclude, that because a nerve or muscle pain is benefitted by a salicylate, that it is necessarily due to rheumatism. In fact it is not only possible, but extremely probable, that the salicylates are beneficial in pains other than rheumatic. This suggestion I have before thrown out, and I may be perhaps pardoned for quoting briefly from a former paper.[†] "That salicylic acid acts directly on the nervous system cannot, I think, be doubted. To me the single fact of the ringing in the ears is an evidence of such action, and the further fact that increased doses result in deafness, a confirmation of this idea. Now, if salicylic acid produces ringing in the ears and even deafness, it is probably by a direct action of the drug in what we may call the acoustic protoplasm; that is, the acoustic protoplasm is *molecularly impressed;* it has the rates and directions of its molecules so affected as to give rise first to abnormal sensations, and finally it has the molecules so much arrested or inhibited as to give rise to an entire loss of function. May it not be that the action of the salicylate in nerve pains is sometimes to be explained by its modifying impress on the abnormal movements going on in the molecules of the diseased nerve, and is it not possible that it deadens hyperæsthesia and annuls pain by virtue of the same quality by which it produces deafness?

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[*] Ed. A. Squibb, Ephemeris, October, 1887, p. 950.

[†] "A case of tic douloureux of twelve years standing treated by large doses of salicylates with marked success." By F. X. Dercum, Phila. Med. Times, Vol. xvii., p. 471.

It is hardly necessary to refer to the physical properties of the oil of gaultheria in order to call attention to the practical advantages it possesses over the salicylate of sodium. In the vast majority of cases it is preferred by patients to the latter drug. It is free from the soapy, nauseating taste of the latter. In therapeutic doses, it is well born by the stomach, provided its administration be not too long continued. However, like the sodium salt, though to a less extent, it is a gastric irritant. If it be exhibited for many days in succession, disturbance of the stomach, though not marked at first, is very apt to follow, besides the strong and pronounced flavor of the drug makes its continued administration in some cases practically inadmissible. In some instances, however, as in a case now under my care, it is well borne for a period of many months.

Regarding the dose of the drug, I have been in the habit of prescribing ten or twenty minims of the oil to be taken at intervals of three or four hours. This has, on the average, proved sufficient to produce marked impression within twenty-four or forty-eight hours, as evidenced by the subsidence of pain and ringing in the ears. Occasionally I have given it in larger doses and at shorter intervals. In one instance, a case of muscular rheumatism, in which the pain was obdurate and excruciating, I gave a half drachm every two hours, with the result of a rapid, absolute, and permanent relief. In this patient, five or six doses of the drug were taken before marked symptoms of gastric disturbance became evident. The fifth or sixth dose—I do not remember exactly which—was rejected by the stomach. The patient's pulse became rapid, the tongue coated, and his nausea was extreme, and, to judge by the description given by himself, "cinchonism" was excessive.* He described the ringing in the ears as frightful. However, he reacted quickly, and although he had been sickened by the oil, he was cured of his pain. On the following day, in fear that his pain might return, he of his own accord resumed the drug in smaller doses.

Under circumstances in which the patient ceases to be tolerant of the drug, it has been my habit of late to fall back on salol.

Salol, the salicylate of phenyl, is also a powerful salicylate. Like so many other new drugs, it has been heralded to the world in a most extravagant manner. It would be difficult indeed, judging from the paper of Herman Sahli,† to discover the therapeutic virtues that the drug does not possess. It is good, he tells us, all the way from rheumatism and typhoid fever to gonorrhœa and the initial lesion of syphilis, and a great deal more besides.

In my own experience, salol has special virtues of special application. It is an almost tasteless, fatty, insoluble powder, which is as bland and unirritating to the mucous membrane of the stomach as so much powdered parafine would be. It is apparently not acted upon by the gastric juice, but depends for its digestion upon the pancreas. At least it is decomposed in the small intestine into salicylic and carbolic acids. This change likewise takes place when salol is mixed with pancreatic secretion or with pancreatic tissue outside of the body.

As far then as the stomach is concerned, it is innocuous and inert, and it frequently proves a grateful relief to that viscus, especially when the oil of gaultheria or the salicylate of sodium have been given for some time. Judging from my own experience, it is slower in producing its physiological action than the other salicylates. The effect is not as pronounced and much less prompt. That it is, however, efficient in the same class of cases as the oil of gaultheria there can be no doubt. I generally prescribe fifteen or twenty grains to be taken every three or four hours. The effect is gradually produced, and is manifested in large doses by more or less "cinchonism." Curiously enough, I have noticed that the ringing in the ears is less, but the deafness more marked, than from the gaultheria. Occasionally patients will mention the darkening of the urine due to the presence of carbolic acid.

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* See also Wood & Hare, Therapeutic Gazette, 1886, p. 73.

† Herman Sahli, Ueber die therapeutische Anwendung des Salols. Correspondenz-Blatt für Schweizer Aerzte, 1886, xvi., p. 321 and 350.

Now and then patients object to the drug on account of its greasy feel and greasy taste, and sometimes of their own accord ask to be placed back on the gaultheria Occasionally, of course, it is well to alternate the gaultheria, instead of with salol, with the sodium salicylate.

For the various affections, then, which we as neurologists are called upon to prescribe salicylic acid preparations, I for one much prefer the oil of gaultheria. Secondly, I fall back upon salol now and then in order to give the stomach a rest ; and, thirdly, I regard salol as inferior in efficiency. Occasionally I use the sodium salt, but only when the patient objects to the decided flavor of the oil.

NOTE ON NITRO-GLYCERINE IN EPILEPSY.

By WILLIAM OSLER, M.D.,

PROFESSOR OF CLINICAL MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA; PHYSICIAN TO THE INFIRMARY FOR NERVOUS DISEASES, PHILADELPHIA.

NATURALLY enough, when the remarkable action of the nitrites on unstriped muscle was discovered, their use was suggested in epilepsy, a disease believed to be due to arterial spasm in the hemispheres. Nitrite of amyl, nitro-glycerine, and nitrite of sodium have been carefully tried by many observers with very varied results. Dr. Wier Mitchell and Dr. Hammond were, I believe, the first to employ nitro-glycerine in this disease, and they have reported beneficial effects in suitable cases. I was induced to try it by the very favorable results reported by Dr. F. W. Campbell, of Montreal. I have used it in nineteen cases. It may be administered in solution, one per cent., or in pilules of 1-100 of a grain; and I find the latter, as prepared by reliable chemists, very satisfactory. I begin with two, three times a day. As individuals appear to differ in their susceptibility to this drug, each case must be tested before the proper dosage can be determined. I doubt if any good follows unless the physiological effect is obtained. Sensations of flushing of the face, fullness of the head, and a pleasant glow over the body, indicate that the proper dose has been reached. In some patients these symptoms are produced by one or two pilules, but in others not until six or eight have been taken. Headache and dizziness were the only unpleasant symptoms complained of, and on this account, in two instances, the medicine had to be stopped. I have notes of nineteen cases in which the nitro-glycerine was tried for periods ranging from six weeks to six months. In thirteen of these cases there were severe epileptic seizures, six were instances of *petit mal* with occasional convulsions. Briefly stated, in nine cases there was improvement, as shown in the reduction of the frequency of the attacks. Of these, six were cases of major epilepsy ; and three, instances of petit mal. The benefit was usually manifested within a week or ten days. Thus case 16, a man aged 27, had had fits for ten years, and when seen, April 5th, had as many as two or three a day. He had taken potassium bromide largely, and at one time with great benefit. Antifebrin was given in gr. viii, two or three times a day, but seemed to be without any influence. On June 1st, nitro-glycerine was given, ♏v of the one per cent. solution, three times a day. Within a week the attacks were greatly lessened, and in the second week after beginning he had only two attacks. He continued to take it all through the summer, getting up to ♏viii doses, t. i. d. He does not think that anything he has ever taken reduced the fits so much. On November 11th, he stated that he had stopped it for a month ; the attacks have recurred less frequently, and he had been able to be at work.

In some of the cases in which the betterment was most striking at first, the remedy seemed to lose its influence, and after a month or two had to be abandoned. I cannot say that in any one of the nine cases the improvement has been more than temporary. In two of the cases of petit mal the attacks were greatly reduced, and one patient remained free for two months, but I learn by letter that the attacks have returned. Altogether, my experience has not been very encouraging. We may say that, in a limited number of cases, when the bromides have failed or are beginning to lose efficacy, nitro-glycerine may be used with advantage. I have also used the nitrite of sodium in a few cases with indifferent success.

NOTE ON ANTIPYRIN AS AN ANALGESIC.

By J. C. WILSON, M.D.

PHILADELPHIA.

ANTIPYRIN—dimethyloxy chinizin, $C_{11} H_{12} N_2 O$—may share with antifebrin, thallin, and other antipyretic drugs the property of reducing fever. All antipyretic drugs may, in view of the dangers which attend their use, and the passing duration of their effects, yield to the employment of external antipyretics in the treatment of grave febrile states. But for the relief of certain forms of pain, antipyrin seems destined to occupy a place in therapeutics second only to that of the derivatives of opium.

The prediction of Germain Sée, made July, 1886, that this drug would largely take the place of morphine, has already been realized. While its employment was yet restricted to the management of fever, careful observers were impressed with its calmative action upon the nervous system, an effect often manifested when the doses have failed to bring about any considerable fall of temperature. It is evident that the therapeutic activity of antipyrin is exerted in a two-fold manner, first, against fever as a symptom ; second, against pain. These effects are produced independently of each other. The general experience of the profession indicates pretty clearly that the antipyretic action of the drug is without influence upon the pathological conditions underlying the fever, and is therefore not curative, its beneficial influence being limited to the lowering of abnormally high temperatures and the prevention of the secondary damage which intense pyrexia inflicts.

It appears to me that the action of antipyrin in painful affections, and especially in those in which its more beneficial effects are shown, namely, painful affections of the nervous system, in contradistinction to those due to lesions of other structures, as traumatism, inflammations, pressure, and so on, is somewhat analogous ; that is to say, antipyrin relieves pain without especially influencing the pathological underlying condition. Where pain constitutes the chief or only symptom of the malady, as in neuralgia, myalgia, angina, and the like, to relieve it is to cure. When such a malady is in itself transient, to anticipate its more intense manifestations, or to ckeck them in the beginning, is likewise to cure. A study of antipyrin from this point of view may serve to indicate at once its sphere of usefulness, and to approximately fix the limitations of that usefulness.

At a time time when the journals teem with observations in regard to this drug I would hesitate to occupy your attention with the results of my own experience were it not for the hope of eliciting useful discussion.

I have used antyprin in a great variety of painful affections during the past year, both in private and in hospital practice. The time allotted me does not permit any detailed review of the cases, and a mere statistical enumeration of them would serve no useful purpose. It has been administered by the mouth, by the rectum, and subcutaneously.

With regard to dosage, my experience leads me to believe that, as a very general rule, full analgesic effects may be obtained by amounts much smaller than usually advised. I have seen the best results, not at all inferior in promptness and degree to those of morphine injections, follow ten-grain doses by the mouth. My present practice is to order five, or seven and a half, or ten grain powders, according to circumstances, and to direct that one, two, or three to be taken in cold water at intervals of an hour. So soon as pain ceases the medicine is discontinued, to be resumed at once upon the recurrence of pain. In many instances, one or two doses only have been required.

The dose by enema is about double that by the mouth.

Antipyrin is extremely soluble, and, as a rule, unirritating Its use by subcutaneous injection is convenient. A sense of tension and moderate stinging pain are experienced at the place of injection. These phenomena rapidly subside. The dose for subcutaneous injection is from three to seven and a half grains.

The administration of antipyrin in those doses to patients free from fever, is not followed under ordinary circumstances by marked manifestations beyond the abatement of pain and a certain transient sense of *bien aise*. The temperature does not fall below the normal; sweating is sometimes copious, oftener only of moderate degree, still more often scanty or absent altogether. The pulse is at first slightly accelerated, but soon resumes its previous traits. In a few instances, I have observed transient and insignificant cardiac depression; once only, and that after a five-grain dose by the mouth, in the case of a very fat, middle-aged woman with feeble heart, alarming syncope with cyanosis and urgent dyspnœa.

Patients taking antipyrin for the relief of pain are not allowed to go about, but are in all cases advised to keep the recumbent posture.

In two or three instances there has appeared the transient scarlatinous eruption which occasionally follows the use of the drug. The nature of the cases in which antipyrin has proved of service has been, in the order of the degree of benefit: neuralgias—trigeminal, occipital, sciatic, intercostal; gastralgia and enteralgia, migraine, and the nervous headache of fatigue; myalgias—especially wry neck, lumbago, pleurodynia, the pains of neurasthenic subjects, and the myalgia of the abdominal muscles caused by persistent cough, as in measles; the precordial pain of advanced disease of the heart, both fibroid degeneration and the dilatation of late valvular diseases, pseudo-angina pectoris—I have had no opportunity to test it in true angina pectoris nor in tabes—and finally the paroxysmal pains of gout and rheumatoid arthritis.

I have found antipyrin much inferior to the salicylates, both in relieving the pains and in hastening the termination of rheumatic fever, and no longer employ it in the treatment of that malady.

It has yielded very satisfactory results in the paroxysms of asthma, in the asthmatic form of hay fever, in whooping-cough, and in certain forms of dysmenorrhœa. It has failed me in cases of pleurisy, internal malignant disease, thoracic and abdominal aneurism, and in painful inflammatory diseases generally.

Germain See has experimentally established the following facts :

1. "A very noted diminution in the general sensibility and a true analgesia in the members which receive the injection of the remedy ; sometimes also in the opposite limb.

2. "The electrical excitation in the sciatic nerve, in the animal under antipyrin, produces only a feeble reflex contraction, indicating enfeeblement of the sensory perceptivity and reflex activity of the spinal cord." (*Therapeutic Gazette*, Oct. 15th, 1887.) Its physiological antagonism to strychnine has been shown by the experiments of Chouffe—*ibid.*

Antipyrin is a true analgesic, acting through the central nervous system. Its role in therapeutics is the relief of certain kinds of pain. To this end it should be used as we have been in the habit of using morphine for the same purpose ; that is, from time to time as pain recurs. It is useless to administer it to patients suffering from recurrent neuralgias or other paroxysmal affections in the intervals of the attack, in the hope of affecting a cure. On the contrary, the very action which renders it useful during the paroxysm must, when prolonged in certain cases, increase the underlying defect in the nervous system. Furthermore, the danger of establishing such a degree of tolerance as would render ordinary doses inoperative, is to be borne in mind.

Finally, among the advantages of antipyrin as an analgesic, must be included these : that it is in ordinary doses well borne by the stomach ; that it causes neither loss of appetite nor constipation ; and that, so far as is yet known, its use is not, as with morphine and chloral, attended by the danger of the formation of a vicious habit.

THEINE IN PAIN.

BY THOMAS J. MAYS, M.D.

THEINE is the active principle of Chinese tea. It is an alkaloid, and was first found by Oudry in 1827, and subsequently confirmed by Mülder and Jokt in 1838. It was believed to be identical in composition and in action with caffeine and guaranine. Chemically, it is known as tremethylxanthine, and occurs in snow-white, needle-like crystals, is almost tasteless, and is soluble in fifty parts of cold, but more soluble in warm, water.

Our first experiments were made with this agent about two years ago; and on a frog we found that its action is as follows:

(1). It has a special affinity for the nerves of sensation. (2). It produces anæsthesia when administered subcutaneously. (3). Its anæsthetic action is confined below the seat of injection. On man its physiological action may be summed up as follows: When tasted, it produces a slight tingling on the end of the tongue, which is immediately followed by local anæsthesia. Subcutaneously, in the arm —dose from one-fifth to half a grain—numbness of arm and hand below seat of injection, a feeling of coldness and a slight disturbance of temperature, and a reduction in the pulse rate, but no intoxication of the brain.

Since theine is of precisely the same chemical composition as caffeine, it was supposed that these two substances must be identical in their physiological action, although Léven, as far back as 1868, demonstrated that theine produced convulsions in frogs while caffeine did not, and that caffeine was more poisonons than theine. The principal reason why Léven obtained such different results from previous investigators was that he used the genuine alkaloids extracted from tea and coffee separately, which does not appear to have been the case with the other experimentors. This procedure is absolutely necessary, for until very recently theine and caffeine were made from tea, coffee, guarana, kola nut, etc., from whichever source it was least expensive, and both alkaloids were dispensed out of the same bottle and labeled to suit the demands of the purchaser. Hence it is useless to employ the commercial theine as contained in the market at the present time, with the exception of that manufactured by Merch, who has informed us that, since our second series of experiments, he manufactures these alkaloids separately. We may say that we have examined specimens of his manufacture, and that they gave rise to the characteristic distinctive test. Hence, so far as we know, only that which comes from Merch and is labeled theine is reliable, and can be expected to give the reputed clinical results.

When the action of theine is compared with that of morphine, we find that there is quite a marked difference in their action. Morphine produces its analgesia by primarily influencing the cerebral centers, although there can be no doubt that it causes a certain degree of local anæsthesia at the seat of injection. But, specifically speaking, its action is central and not local, while theine, as we have seen, has a local action independent of the central nervous system. Its action is confined altogether below the seat of its injection, and never, according to our own experience, is the brain intoxicated. This localized influence demonstrates one of the great therapeutic advantages which theine possesses over morphine, and over all other agents of this class. By acting only on the peripheral portion of the trunk of a nerve and of its ramifications, it leaves the higher nerve centres uninfluenced, and therefore does not develop the undesirable central intoxication which so frequently follows a dose of morphine. In order to obtain the best practical results of these agents, we must give morphine when the pain is acute and when, in order to relieve it, it is necessary to narcotize the central seat of sensation, while theine is most beneficial in chronic affections of the sensory nerves.

Administered to a properly adapted case, theine is surprisingly prompt in its analgesic action. We have on numerous occasions observed patients with lumbago, who were too stiff to bend their bodies, or had almost too much pain in their backs to rise from a chair or sit down after they were up, comfortably straighten their bodies in less than five minutes after its introduction. When not so prompt, it is probably due to the small size of the dose, and where one-half of a grain does not produce the desired results, it should be increased to one, or even two grains.

When injected, it gives rise at first to some burning, but it does not produce any prolonged irritation or inflammation. The burning is soon replaced by an area of anæsthesia. Since its solubility in water is very low, we prepare it with benzoate of soda, according to the following formula :

℞	Theinæ, - - - -	
	Sodæ benzoa, - - - -	āā ʒ i.
	Sodæ chloridi, - - - -	gr. viii.
	Aquæ, - - - - -	fl ℥ i.

M. Sig.—For hypodermatic use. Six minims equal one-half grain of theine.

We have used theine as an analgesic for more than a year, but we will not inflict on you a relation of the histories of cases which we have treated, and will simply say that we have had very good results from it in myalgia, intercostal neuralgia, brachialgia, etc., and in addition to these will relate a few cases of sciatica in which it was employed. It is but fair to state that, in connection with the theine, we also prescribed iron, quinine, salicylate of soda, iodide of potassium, etc.

SCIATICA.

Case I.—Mrs. A., aged 49 years, came under my care May 2d, 1885, when she had constant pain in right leg from hip to foot ever since the preceding Christmas. The pain followed the course of the nerves of the leg, and, of course, was very much aggravated by walking. The leg was considerably atrophied, and was weak and uncertain in its gait. Her appetite was poor and bowels irregular, and she passed whole nights without sleep on account of pain. She was anæmic, and had been treated with iron, quinine, ammonia, salicylate of sodium, iodide of potassium, atropine, morphine, poultices, blister, etc., without avail, until the following 18th of July, when I injected one-fifth grain of theine into the calf of her leg. The pain ceased in less than five minutes, and never returned in its original force. In half an hour her heel and foot began to feel numb and insensible, which lasted for about twelve hours ; but her mind was perfectly free from its influence. She experienced no headache or drowsiness.

July 20th.—Pain in whole leg better since last injection, but has not entirely disappeared from the thigh, so I introduced one-fifth grain into the latter region.

July 28th.—Leg altogether free from pain, but still complains of some in foot ; otherwise she is improving. Injected one-fifth grain over instep.

September 6th,—Feels better ; slight pain around ankle-joint. Injected one-tenth grain at this point. This was the last injection she received, and she made an uninterrupted recovery.

Case II.—B., aged 50, carpenter, was first seen April 3d, 1886, when he complained of a severe pain for nearly two years in lumbar region radiating down both legs. He is stiff in both legs and in the back, and rises from a chair with great difficulty. He had malaria, but gives no specific or rheumatic history. Injected half-grain doses of theine on each side of the sacrum, and two doses of similar strength between the trochanter on both sides. He said it relieved the pain and stiffness at once, and that he was as able as ever to get up from a chair.

April 6th.—Says he is better, but still has some pain. Repeated the injections, and internally gave him the quinia and ammonia mixture.

April 10th.—Pain nearly all out of back ; legs feel somewhat stiff. Two injections between trochanters on both sides. Same internal treatment. This patient continued to improve, and was entirely well by end of same month.

Case III.—L. M., aged 60, came to me April 22d, 1886, complaining of severe pain in sacral region, which radiated down both extremities as far as the knees. Injected half a grain on each side of sacrum, and she expressed herself much relieved immediately after the injection. Internally she received the quinia and ammonia mixture.

April 24th.—Better ; pain in sacral region almost gone, and she could walk better than before. Injected half a grain in the fleshy part of each leg.

April 27th.—No pain in back. Did a heavy day's washing without producing any pain in back, since her last visit. This she had not been able to do for a long time. The only pain she complains of now is in her left leg from knee to ankle-joint. Injected half a grain above the knee. The pain disappeared at once. With the same internal treatment and an occasional injection, she continued to do well.

Case IV.—E., aged 35, came under observation June 20th, 1887, when he gave the following history : Pain along both sciatic nerves during the last three years. The attacks of pain come on simultaneously in both nerves, although the left leg is the worst. Some loss of motor power in left leg. Has had malaria and rheumatism. Some burning in soles of feet. Injected half a grain of theine in each buttock over the course of the sciatic nerves. Internally gave him the quinia and ammonia and soda salicylic mixture in alternation every three hours.

June 21st.—Pain better last night than it has been for a long time. Slept well, which he had not done for several nights. Gave him two more injections in same regions, and continued same internal treatment. The pain at no time became bad again, and in less than a month of the treatment outlined above he was entirely free from pain, could sleep well, had regained the want of power which existed in his left leg, and he was able to resume his vocation.

REVIEWS.

INSANITY—ITS CLASSIFICATION, DIAGNOSIS, AND TREATMENT. *A Manual for Students and Practitioners of Medicine.* By E. C. Spitzka, M.D. Second edition. F. B. Treat, publisher, 771 Broadway.

The rapid exhaustion of the first edition of this work is a demonstration of the profession's recognition of the ability of the author and its desire to become acquainted with his view, which, it had every reason to suspect, would be to a great extent original. Nor can this expectation be said to be not realized by a perusal of this work, since the author's personality pervades almost every page.

In reviewing a second edition, it becomes the duty of the critic to compare it with the first. By such a comparison we cannot say that any great changes present themselves in this manual, excepting the correction of a few typographical errors; the modification of the tone of the foot-notes, which in the first edition, although they may have expressed the author's true sentiments, ought to have been nevertheless foreign to a scientific work and to have found no place therein. Still we can very well understand the position of the author in this regard, for it is our opinion that the patience of Job himself would have been sorely tried to have men who claim to be expert alienists, denying *in toto* some of the most fundamental truths of psychiatrical science. Hence, we suppose, the author seized the only opportunity which presented itself to show the deficient education of such men and thus established the fact that no weight should be attached to their teachings. That the author saw his mistake and corrected it is a matter for congratulation.

Other modifications consist in the adoption of the term "paranoia" for monomania, and the addition of an appendix which is composed of a series of additions to the original text.

That the change of term was a wise one will be accepted, we think, by all, excepting a few controversialists who still cling to the belief that "monomania" and "paranoia" are not expressive of any definite mental aberration. But the fact that Mendel, of Berlin, adopted this substitution for the German "Verrucktheit" has led the author, as he says in the preface, to make the same adoption and for the same reasons.

The additions comprising the appendix either supply deficiencies in the original text or further elucidate the author's statements therein. The strongest and best timed of these is a very just criticism on oophorectomy as a means of curative procedure in mental disorders. It his high time, we think, that indiscriminate oophorectomies should cease and that gynæcologists should view the fact that, though they be good operators, they may not be good alienists; and should hence hesitate to operate, especially where such a great difference of opinion exists among alienists as to the curative effect of this operation on hysteria and insanity. In fact the great weight of opinion is against operation, for in the largest number of cases operated upon for these troubles no change occurred in the mental condition. The author well says "certainly those forms of insanity which, like primary paranoia and moral imbecility, are due to a neurotic taint and defect, should be regarded as a *noli me tangere* by the oophorectomist."

It is not often that we are called upon to pass criticism upon the *publisher's* portion of a work. In fact, works of small scientific value are often gotten up regardless of expense. We might expect that when a book of such intrinsic worth as the one under consideration was published, that the publisher would prepare and issue the typographical portion in uniformity with the value of the contents. But here our expectation was not realized. Indeed, not only the general appearance of the book is shabby, but the paper and character of the type seem to exhibit a most parsimonious and impecunious spirit on the part of the publisher. We would hence suggest to the author to seek another publisher when he issues his larger work on insanity which he promises, and to thus prevent a physician's library being disgraced by such binding which so demeans the present volume.

N. E. B.

FUNCTIONAL NERVOUS DISEASES, their causes and their treatment. Memoir for the concourse of 1881-1883. Academie Royale de Medecine de Belgique. With a supplement on the Anomalies of Refraction and Accommodation of the Eye and of the Ocular Muscles. By George T. Stevens, M.D., Ph.D., Member of the American Medical Association, etc., etc. New York: D. Appleton & Co., 1887, pp. 217.

The work before us is the memoir which received, from the Academie Royale de Medicine of Belgium, the highest honors awarded, 1881-1883. The author's views have long been known to neurologists and ophthalmologists, and have been considered by them as heterodox, especially his views on the cure of chorea and epilepsy by the treatment of ocular muscles. The first eighty pages treat of "Functional Nervous Diseases," such as neuralgia, migraine, etc., by the correction of errors of refraction, especially astigmatism. In this part of the work there is nothing new, as all have known that errors of refraction are a common cause of headache, and that as soon as the correction was given the pains in the head and neck would disappear. On the other hand, the treatment of muscular inco-ordination, as practiced by the author, is new and of his own devising. There has no doubt much been learned during the past few years in regard to muscular asthenopia, and both Landolt and Stevens have done much to this end.

The nomenclature suggested and used by the author for the different ocular insufficiencies is an improvement on the former modes of expression, as the term *exophoria* is much more convenient than *insufficiency of the internal rectus.*

As to accepting the belief of the author, that ocular muscles out of equilibrium are the cause of epilepsy, chorea, and such affections, and that by partially dividing them relief is obtained, the reviewer is not in accord. In fact he has yet to see any case of chorea or epilepsy permanently relieved by the partial or even thorough division of the ocular muscles. That cases of epilepsy are occasionally for a short time improved by an operation on the ocular muscles, is doubtless true, and it is as equally true that any operation, the patient knowing it, no matter how little the pain inflicted, will have the same benefit, owing to the mental effect produced. In closing this review, we cannot advise the practice of its teachings. The book is well written and well published.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Meeting held December 6th, 1887.

THE PRESIDENT, DR. C. L. DANA, IN THE CHAIR.

DR. WAITZFELDER presented a case of

PROFESSIONAL CRAMP.

The patient was a man 38 years of age, and had been a cigarmaker for twenty years. He was first seen by the speaker six months ago. Six months before this he had commenced to notice a stiffness and awkwardness in forming the head of the cigar. This required a rotary motion of the thumb and index finger. At first he was able to substitute his middle finger in the manœuvre, but later the stiffness extended to this and finally to the little finger. When first seen there was some anæsthesia of the skin and tonic spasm of the flexors of the fingers. These contractures were partially relieved by rest, although they never entirely disappeared. They were aggravated by any effort to work at his trade, and by changes in temperature either to heat or cold. Exhibition causes relaxation of the spasm, while associated movements increase it. A short time ago the patient had tried varnishing, work which requires only coarse motions of the hand, but was obliged to give it up. The patient had been under treatment for six months with galvanism, faradism, strychnine, etc., without improvement. Regarding the lesion as central, the speaker suggested stretching the radial nerve, with the object of inducing atrophy in the central cells, which might be considered the source of irritation. As Dr. Seguin had seen the case, his opinion was requested.

Dr. Seguin saw nothing which would distinguish the case from one of aggravated professional spasm. He regarded such dyskineses of central origin, and would favor nerve stretching. His experience had, in fact, been discouraging in any treatment for professional cramp. A case of waiter's cramp had come under his observation. He mentioned it because rare. The man had been a waiter for fourteen years, carrying plates in the manner of his class, until reaching a stage when he broke dishes and spilled their contents over the guests.

Dr. E. C. Seguin read a paper upon

THREE CASES OF HEMIANOPSIA,

of peripheral or neural origin, with a study of the symptom hemiopic pupillary inaction, its diagnostic value, etc.

All of the cases were supposed to have a neural peripheral lesion. All presented the pupillary reaction hinted at by Wm. Graefe, and described by Wernicke as the hemiopic pupillary reaction, a symptom never yet observed or described in this country.

Case I. was first seen October 26th, 1887, referred from the Eye Department of the Manhattan Hospital by Dr. Webster. Six years previously the patient had commenced to suffer from headache, mostly frontal, also nausea and vomiting. Two years previously he had suffered from more or less constant pain. For the last few months the headache had been less, but he had lost vision in the left eye. No history of syphilis was obtained. On examination, the right visual field showed temporal hemianopsia outside the point of fixation. The visual field of the left eye was completely blind. Vision was at this time, R. E. $\frac{20}{200}$, L. E. 0. Light thrown into the left eye caused no reaction in either pupil. If thrown into the right eye in the optical axis, good reaction in both pupils occurred. If the light was moved nasalward, reaction was obtained in both pupils, also if moved temporalward within an angle of sixty degrees. If thrown further toward the temporal or blind side, no reaction in either pupil occurred. The centripetal portion of the arc controlling pupillary reaction was complete only in the right eye, while the centrifugal portions were perfect in both eyes.

Nov. 21st the same reactions were obtained. If light were thrown at an angle of ninety degrees, from the nasal side at any angle, or from the temporal side up to an angle of seventy or sixty degrees, reaction occurred in both pupils ; but if the light entered the eye from the temporal side at an angle of sixty to forty degrees from the horizontal, no reaction was obtained. Vision was now found as follows : L. E. 0, R. E. $\frac{60}{100}$, showing progressive loss during the month.

The lesion in this case is probably one of the chiasm, destroying both fasciculi in the left eye, and the fasciculus cruciatus for the right eye.

Case II. was one of typical temporal hemianopsia, with darkness also of the lower nasal quadrant upon the left side. The patient, 41 years of age, was referred to the author by Dr. David Webster.

In 1886 the patient had noticed occasional headaches, with increasing failure of the left eye. There was diplopia at that time. If two objects were present, the patient saw three ; if four, five, etc. Light attacks of vertigo were complained of. There was no history of syphilis in the case. The patient was first seen by the author, Nov. 11th. The right eye showed the vertical line of separation passing through the point of fixation. This was the first case in the author's experience of either central or peripheral origin of which this had been true. There was right hemiopic pupillary inaction. If light were thrown from the temporal side at an angle more oblique than sixty degrees, no reaction occurred. If thrown from the nasal side or directly into the eye, reaction of both pupils was obtained. In the left eye, slight reaction occurred only to central illumination. Vision, R. E. $\frac{8}{200}$, L. E. $\frac{20}{50}$. The latter was an interesting finding, as the left eye, with the better vision, had a smaller field than the right eye, showing that the vision is not measured by the geometrical extent of the field, but rather by the general state of nutrition of the optic nerve.

The lesion in this case involved both fasciculi cruciati and the ventral or inferior half of the fasciculus lateralis of the left eye. A test made Dec. 16th showed that color perception was normal in the preserved half and quarter fields of vision.

Case III. was 25 years of age, also referred to the author by Dr. Webster from the Manhattan Eye Department. The case was first seen Oct. 10th, 1887. The patient had commenced to suffer from loss of vision two years previously. At the date of examination the right eye was completely blind. There was general loss of strength, but no ætiology of injury, and no history of syphilis. There was no paralysis, no loss of equilibrium. The dynamometer showed twenty-three degrees in the right hand, twenty in the left. At a subsequent examination, the left eye showed typical temporal hemianopsia. Left vision was good; can read No. 1 Jaeger at seven inches. The right eye gave a peculiar field, viz., all dark except two sectors in upper quadrants, separated by a dark median strip. The lesion in this case involves the fasciculus cruciatus for the left eye, and, irregularly, both fasciculi for the right eye.

The author then referred especially to the symptom common to the three cases, called by Wernicke hemiopic pupillary reaction, but which he would designate as hemiopic pupillary inaction; and its diagnostic value in hemianopsia of central or peripheral origin. The optic arc consists of the retina, especially the macula, the optic nerves and tracts for its peripheral portion, the anterior group of the corpora quadrigemina with the nuclei of the motor oculi nerves as centers, the motor oculi, the ciliary nerves, and the iris for its centrifugal portion. Contraction of the whole iris follows stimulation, because the termination of the ciliary nerves is plexiform in arrangement. The path may be broken by lesion of the centripetal portion, by lesion of the reflex center (or optic lobes), or by lesion of the centrifugal paths. The termination of this—the iris—may be immobile from iritis. In the cases reported, the symptom was observed with temporal hemianopsia. If a pencil of light were directed in the optic axis, or moved nasalward so as to strike upon the temporal or normal half of the retina, good reaction was obtained; also if moved temporalward up to an obliquity of sixty or seventy degrees; but if it entered at an angle of forty to sixty degrees from the horizontal, no reaction was obtained. The light experiment was not exact, because it is impossible to focus exactly the pencil of light. By focusing nearly in the optical axis, crossing of the rays and diffusion upon the temporal or sensitive portion of the retina occurs. Without this diffusion, inaction of the pupil would be noted as soon as the rays of light struck the nasal side of the field, and the experiment would be as exact as the perimetric test.

In making the examination, the patient should be in a dark room, with the gasjet or lamp behind him in the usual position for ophthalmic examination. The patient should be directed to look toward the farther side of the room, and a faint light from a plane or large concave mirror held out of focus thrown into the eye. Relaxation of the iris is thus obtained. A beam focused by an ophthalmological mirror is then thrown into the pupil from the optic center and from various angles nasalward and temporalward, and reactions noted. It is especially important that the patient should look at a distant object ; the same was true for pupillary examination in tabes, etc.

The author then added a number of rules for determining the location of the lesion in hemianopsia.[*]

DR. GRAEME HAMMOND then presented

A CASE OF BITEMPORAL HEMIANOPSIA

for Dr. C. H. Brown.

The patient was a woman, 37 years of age. Aug. 10th, 1887, was suddenly seized with pain in the right eye, the sight of which was lost. The pain spread to the left eye, and in three minutes she was totally blind. The pain continued spreading over the head to the base of the skull. The patient was in bed six weeks. Nov. 15th she commenced to walk a little, and under the iodide treatment, vision returned, so that she could see an object if within two feet of the eye. She can now read newspaper headings, but effort to do more than this gives pain. Examination showed obscuration of the nasal half of the field covering the point of fixation in both eyes.

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[*] For the complete paper with diagnosis, *vide*, JOURNAL OF NERVOUS AND MENTAL DISEASES, Dec. 188 .

Dr. Dana had seen the fields drawn by Dr. Francis Valk, and the obscuration was very marked.

Dr. Seguin was able to give a satisfactory demonstration of hemiopic pupillary inaction in this patient.

Dr. Brill proposed three divisions for cases of hemianopsia; namely, into those of prosencephalic, mesencephalic and peripheral origin. Where the pupillary reaction was retained the two latter would be thrown out and the lesion would pertain to the cortex or subcortical expanse.

Dr. Starr suggested that the Society owed its thanks to Dr. Seguin for a series of three papers upon so interesting a subject. To make accurate diagnosis possible by the working out of a single symptom was an enviable result. The symptom had been known and its lesion tabulated, especially by Wernicke in his Gehirnkrankheiten, but not before in the English tongue. The symptom has to be searched for carefully. Unless the physician uses the correct method, it will not be found. In many published cases it was undoubtedly present, but was not discovered. The speaker referred to the fact that Ferrier, in Brain, Vol. VI., makes the escape of the macula a diagnostic point, stating that where the lesion is located in the chiasm and tract, central vision is impaired; that where it is located in the cortex or between the thalamus and the cortex central vision is not impaired. He had formed his new diagram in the second edition of his Functions of the Brain upon this theory, according to which central vision should have been impaired in all of the reported cases.

The speaker did not agree with Dr. Brill as to the practicability of classification according to the fœtal development of the brain. The optic nerve is derived from the thalamencephalon and the retina from the mesencephalon; thus two portions of the neural tube are concerned in the peripheral apparatus.

Dr. Leszynsky had had the pleasure of examining two of the cases referred to in Dr. Seguin's paper. The examination was not difficult, though it required care. He considered the head mirror of assistance in the manœuvre. The term inaction of the pupil he thought misleading.

Dr. Birdsall thought the classification of Dr. Seguin practicable and likely to be of service.

Dr. Brill referred to the fact that von Gudden had obtained reaction of the pupil by the use of intense sunlight after division of the optic nerve, showing ganglionic elements in the iris itself, which are able to function independently.

Dr. Seguin closed the discussion. He found clinical records opposed to Ferrier's classification, as the dividing line hardly ever passes through the point of fixation in any form of hemianopsia, and he failed to find light on the escape of the macula by any theory of decussation. He was rather inclined to think it dependent upon the structure of the macula which was somewhat ganglionic in character and markedly different from that of the remainder of the retina. Before von Gudden, Brown Sequard had demonstrated the existence of a local mechanism for iris contraction in lower animals, but there was no evidence that such a mechanism was active in the human eye.

Dr. Noyes then presented specimens of

COMPOSITE PORTRAITURE IN THE INSANE.

A composite, taken from eight general paretics, was exhibited. This showed in a marked degree the washing out of the lines of the face. Incidentally, Dr. Noyes also exhibited the college composites recently published.

Dr. Seguin suggested that the Vassar composite had a more womanly character than that of the Harvard Annex.

Dr. Birdsall agreed in this opinion. The Harvard composite figured an intellectual rather than a womanly being.

The meeting adjourned.

THE PHILADELPHIA NEUROLOGICAL SOCIETY.

A regular meeting of the Philadelphia Neurological Society was held November 28th, 1887, Vice-President Dr. **Charles K. Mills** in the chair.

Dr. J. C. Wright read a note on

ANTIPYRIN AS AN ANALGESIC (see p. 40).

DISCUSSION.

Dr. William Osler.—I have used antipyrin in a few cases at the Infirmary for Nervous Diseases, and I regret to say that, on the whole, my experience does not accord with that of the reader of the paper. Last Wednesday, four cases that had been ordered antipyrin one or two weeks previously, returned, and the uniform statement was that no benefit had been obtained. Two were cases of migraine and two were cases of sciatica. I am well aware that in some cases of sciatica, antipyrin is of undoubted benefit, but other cases appear to resist it. I have used it with success in one case of neuralgia, and in two cases of rheumatism it has had no influence upon the pain. In one case of gastralgia it was entirely inert. Altogether, my experience, which is however quite limited, is not favorable to the use of the drug in painful affections.

Dr. Wharton Sinkler.—I have given antipyrin in two cases of migraine during the paroxysm with marked benefit. I have also met with disappointment in its use. My experience is however not extensive.

Dr. Charles K. Mills.—I have used antipyrin, but not to any great extent. I have tried it in epilepsy, and, in general terms, I may say, without success. I have used the drug in two cases of sciatica. In one case it did not succeed, but in that case all other remedies failed, and I suspect that there is some intra-pelvic trouble. In another case, the patient was benefitted by its use. I recall one case of trigeminal neuralgia in which it was of benefit. I used the drug in one case of recurring headaches, sometimes taking the form of migraine, at other times appearing to be purely neuralgic. In this case, antipyrin was of no service. It seems to be of some use in purely neuralgic affections.

Dr. James C. Wilson.—I believe that the statement which I have made in the paper is perfectly true, that is, that antipyrin is destined to take a place in therapeutics second only to the derivitives of opium, as a pure analgesic. I have carefully studied the subject clinically, but the time has not permitted the naration of cases in detail. I should, however, like to refer to one illustration of the effect of the drug. Two days ago, I saw a case of migraine in a child eight or nine years of age. He had been living in the South, and had suffered with severe malarial outbreaks from time to time. The spleen was enlarged and the general health was considerably undermined. The child was suddenly seized with well-marked symptoms of migraine, and these had continued six or eight hours when I saw him. I ordered four grains of antipyrin, to be repeated in an hour if necessary. The first dose caused decided relief, and the second was followed by complete cessation of the pain. The child fell asleep, and in a few hours awoke in his usual condition. Other cases similar to this have presented themselves to my attention.

While the drug relieves the paroxysm, it is not to be depended upon to cure the underlying condition to which the pain is due. The true use of a drug of this kind is to be found in its restricted application.

Dr. Wm. Osler next presented a note on

NITRO-GLYCERINE IN EPILEPSY (see p. 38).

A paper was read by Dr. F. X. Dercum on

OIL OF GAULTHERIA AND SALOL IN RHEUMATISM OF NERVES AND MUSCLES.

(See p. 33.)

Dr. J. H. Musser.—While not bearing directly on the subject of the paper, I would say that I have found salol of extreme benefit in rheumatic arthritis. I have now in mind a case which has been under observation for several months, and therefore the effect of diet, rest, and general tonic treatment can be eliminated. This patient has been taking salol for four weeks, and has been greatly benefited. The pains have almost entirely disappeared, and, in addition, the joints have become limbered and the thickening and infiltration has diminished. In a second case there has also been improvement, although the remedy has not been continued so long. The dose in the first has been five grains every three hours. This has caused no disturbance of the stomach and no marked physiological effects. In the second case this dose produced marked symptoms, and had to be reduced to five grains three times a day.

Dr. Charles K. Mills.—I have used the oil of gaultheria quite extensively during the past year in the class of cases referred to by Dr. Dercum ; that is, where the affection is not directly neuralgic, but rather a form of neuritis, usually rheumatic neuritis. I have also used the oil of gaultheria in combination with salicylate of sodium. This has been usually efficacious in these cases. My experience is that it is not so useful in chronic cases as in the acute or subacute. I have had two or three remarkable successes with this drug. A gentleman applied to me with a severe pain in the neck, which had continued ten days, with also slight toticollis. Ten minims of oil of gaultheria, with five grains of salicylate of sodium, dissipated the pain entirely. He took a few more doses, and has had no return of the trouble. In another case where, with pain over the brow, undoubtedly due to supra-orbital neuritis, three doses entirely relieved the trouble.

Dr. James Hendrie Lloyd.—I should like to ask, in connection with this discussion on neuro therapeutics, if any of the members have observed benefit follow the use of these drugs—antipyrin, oil of gaultheria, and salol—in cases of neuritis of traumatic origin.

Dr. William Osler.—With reference to the oil of gaultheria, I have been more and more impressed with its efficacy in rheumatism the more I have used it. It has rarely failed me. I have one case under treatment at the present time, in which it has proved inert. In this case, salol and salicylate of sodium have also failed. The only difficulty is in its administration. Patients turn against the drug, and it is only with great difficulty that they can be induced to continue it more than a week or ten days.

Dr. James C. Wilson.—I have found oil of gaultheria of decided benefit in the sub-acute and lingering forms of rheumatism, much more so than in acute attacks. I have, however, not been able to administer such large doses as Dr. Dercum has mentioned. This has been more on account of the disagreeable symptoms produced than on the account of the disturbance of the stomach. I have given the remedy in capsules containing five minims of the oil. Of these I have ordered two or three, three times a day. In a case recently under observation, I have been able to give the drug only twice a day. This patient takes three capsules after breakfast, and five or six capsules just before retiring. The disagreeable effects of the drug usually subside before morning.

I used salol last winter quite extensively. I had no trouble as regards the stomach, but the doses required were so large and the cost was so great, that patients objected to it. I have usually employed the remedy in pill form, but occasionally have used emulsion. While it has not a strong and penetrating taste, the flavor is disagreeable.

With reference to Dr. Lloyd's question, I would say that in a case of traumatic neuritis, I tried antipyrin without any benefit whatever.

Dr. Judson Daland.—I would mention the fact that the disagreeable taste of the salicylate of sodium can be greatly lessened by dissolving it in the compound tincture of cinchona. One drachm of the latter will dissolve ten grains of the former.

Dr H. A. Hare.—It has been proven by recent physiological experiments that the effects produced upon the special nerves, such as deafness and amaurosis, the salicylates and quinine are partially due to the action of the drugs upon the peripheral ends of the nerves. It has not been proved that the peripheral ends of the nerves of other parts of the body are similarly affected. But such an action might explain the results which have been reported.

The fact that salol acts so much more slowly than the other drugs may be because it is absorbed by the small intestine. Ewald has taken advantage of this in studying cases of so-called motor palsy of the stomach. By giving salol he could determine the exact time at which the food left the stomach; for as soon as the drug reached the duodenum, it was absorbed and appeared in the urine.

In reply to Dr. Dercum, Dr. Hare stated that he believed salicylic acid and its compounds circulated in the blood in the form of salicylates, more especially of soda, and a very large part of it was eliminated as salicyluric acid. In some experiment performed with Dr. Wood, it was found that where oil of gaultheria was given, even in large amounts, no unchanged oil escapes from the kidneys.

Dr. Wm. Osler presented

MICROSCOPICAL SECTIONS FROM A CASE OF GLIOMA OF THE MEDULLA OBLONGATA.

Dr. J. K. Mills, for Dr. F. X. Dercum, presented a brain containing a hæmorrhagic cyst limited to the lenticular nucleus and the adjoining border of the internal capsule.

Dr. J. H. Musser exhibited sections from a sarcoma of the brain, which had produced no symptoms.

Adjourned.

Meeting of December 19, 1887.

VICE-PRESIDENT, DR. CHARLES K. MILLS, IN THE CHAIR.

Dr. Thomas J. Mays read a paper on

THEINE IN PAIN (see p. 44).

DISCUSSION.

Dr. Charles K. Mills.—A few months ago, I began the use of theine, ordering it in almost every case of neuralgia, superficial neuritis or lumbago that came to the polyclinic service, and also using it at the Philadelphia hospital. I have used the drug probably in about fifteen cases, but I have not had time to prepare notes of them for this meeting. I recall three cases of sciatica, two of which were of long standing. These cases were all improved, but none of them were cured by the use of theine alone. It was applied hypodermically according to the formula of Dr. Mays. The pain was usually much relieved by the use of the theine, but it would return after a shorter or a longer time. In connection with the theine, I used galvanism to the nerve and muscles, and internally Donovan's solution, or iodide of potassium. Two of the cases of sciatica were cured under this conjoint treatment.

In a case of facial neuralgia or neuritis, I used a hypodermic of theine in the face. Following this the patient became pale, sick at the stomach, and seemed to be in a slightly dazed condition for a time. Whether this was due to the drug or simply to the slight operation I cannot say. The injection entirely relieved the pain for three days. The patient then returned and another injection was given without bad effect. In another facial case the patient was certainly relieved by theine, and so far as I know remained well. I have used it in the back with great benefit in so-called myalgia, including under that term muscular rheumatism, and possibly true lumbar neuralgia. I believe that theine is an analgesic; that it relieves pain in cases of recent and superficial neuritis or neuralgia. In chronic, painful nerve troubles, particularly where the nerves are deeply situated, it seems to be simply a helping remedy, and sometimes fails.

DR. THOMAS J. MAYS.—I merely wish to say that the proper province of theine is the relief of pain. I think that it is expecting too much of it to look to it to cure the cause or diathesis upon which the pain depends. The physiological action of the drug is to deaden sensibility. I have always found this its characteristic action when I have employed it clinically.

A paper was read by DR. A. P. BRUBAKER on

DENTAL IRRITATION AS A FACTOR IN THE CAUSATION OF EPILEPSY.

Remarks were made on severe neuralgic headache as the initial system of typhoid fever.

DR. WILLIAM OSLER.—I should like to make a brief reference to the occurrence of severe neuralgic headache in the initial stage of typhoid fever. In some cases, typhoid fever comes on with such severe headache that at first the true nature of the disease may not be recognized. I have had three instances of this kind come under my observation. In two the patients were women, one plethoric and full-blooded, and the other pale, nervous, and somewhat emaciated. In the latter case the disease was not recognized for over a week. The disease was considered one of neuralgic headache, although it was thought remarkable that the temperature should so persistently remain above normal.

In the other case the patient suffered more severely from headache than any that I have seen. The pain resisted all remedies. At the end of four or five days, it was suspected that some constitutional disease was developing, and the case proved to be one of well-marked typhoid fever.

A few weeks ago I saw a gentleman who had been ill for ten days, and whose most marked symptom was severe headache, chiefly over the brow, but extending to the vertex. The whole scalp was exquisitely tender. The patient was heavy and dull, and typhoid fever was suspected. The pain resisted chloral, the bromides, urethran, and other remedies. He was then ordered eight leeches, and that night he had the first quiet sleep that he had obtained for ten days. He subsequently developed well-marked typhoid fever.

We of course all know that headache is an early symptom of typhoid fever, but the occurrence of this severe form of headache is not as well known as it should be. Indeed, in one case the diagnosis was not made until fatal perforation occurred early in the disease.

DR. CHARLES K. MILLS.—It might be interesting to know whether or not these patients who suffer from this severe headache are liable to neuralgic headaches when in ordinary health. A case of typhoid fever occurred in my own practice, and I had myself a severe attack of the disease within a short time of the occurrence of this case. I did not suffer particularly with headache, whereas the other patient suffered severely with headache. He was, however, subject to severe headaches when in ordinary health.

DR. WILLIAM OSLER.—Two of the cases to which I have referred were not especially subject to headache. I cannot speak so positively in regard to the third case.

DR. WHARTON SINKLER.—About one year ago, I saw a young man aged about 23 years. He came home one evening with a violent headache. This continued thirty-six or forty-eight hours, and marked the beginning of a long attack of typhoid fever. The headache did not recur during the course of the disease.

Reading Notices.

CONGRESS OF AMERICAN PHYSICIANS AND SURGEONS.

Dr. Græme M. Hammond,
Secretary of the American Neurological Society,
New York City.

Dear Doctor:

As supplementing the information given in the circular recently published respecting the Congress of American Physicians and Surgeons to be held in Washington, D. C., in 1888, I now beg to communicate the following arrangements, viz.:

The sessions of the Congress will occur on the evenings of Tuesday, Wednesday, and Thursday, September 18th, 19th, and 20th.

On Tuesday evening, the subject of "Interstitial Obstruction in its medical and surgical relations" will be presented and discussed.

On Wednesday evening, the subject of "Cerebral Localization in its practical relations" will be presented and discussed.

On Thursday evening, the Address of the President will be delivered, and afterwards there will be a General Reception in the Museum Building.

No entertainment will be provided in connection with the Congress.

No invitations will be extended by the Congress to physicians from abroad; but all such as may be present as guests of any of the special societies participating will be considered members of the Congress, and shall be entitled to participate in the proceedings.

The sessions of the Congress will be open to the profession at large.

It will thus be seen that the morning and afternoon of each day are left free for the sessions of special societies participating.

It would seem desirable that the Secretary (of the Council) of each participating society should communicate to the Secretary of the Congress, as soon as convenient, any information about the arrangement of sessions and the programme of scientific work. All such information shall be promptly communicated to the officers of the other participating societies, so as to afford opportunity for any adaptation of sessions and subjects of discussion that may be desired.

It is understood that the American Association of Physicians proposes to have morning and afternoon sessions, on Tuesday, Wednesday, and Thursday, and that the American Surgical Association will probably have its meetings at the same dates.

Respectfully yours,
W. H. CARMALT,
Secretary of the Congress.

January 30th, 1888.

Dr. Græme M. Hammond,
Secretary of the American Neurological Association.

Dear Doctor :

I beg leave to forward to you a copy of the preliminary programme of the Association of American Physicians for their Third Annual Meeting to be held in Washington, D. C., on the mornings and afternoons of September 18th, 19th, and 20th, 1888, in conjunction with the Congress of American Physicians and Surgeons.

Very respectfully yours,
W. H CARMALT.

PAPERS.

"Cardiac Changes in Chronic Bright's Disease,"	Dr. Alfred L. Loomis, New York.
"Relation between Chronic Intestinal Nephritis and Angina Pectoris,"	Dr. Samuel C. Chew, Baltimore.
"Disturbance of the Heart Rythm, with reference to Causation and their Value in Diagnosis,"	Dr. Gustavus Baumgarten, St. Louis.
"Fatty Heart,"	Dr. Fredrick Forcheimer, Cincinnati.
"Cardiac Lesions producing Presystolic Murmur,"	Dr. Frank Donaldson, Baltimore.
"Treatment of Valvular Affections of the Heart,"	Dr. Jacob Da Costa, Philadelphia.
"Clinical Investigations in the Treatment of Cardiac Disease,"	Dr. James K. Thacher, New Haven.
"Causal Therapeutics in the Infectious Diseases,"	Dr. James C. Wilson, Philadelphia.
"Management of the Stage of Convalescence in Typhoid Fever,"	Dr. James H. Hutchinson, Philadelphia.
"The Geographical Diffuseness in Typhoid Fever in the United States,"	Dr. W. W. Johnston, Washington.
"The Pathology of the Thymus Gland,"	Dr. Abraham Jacobi, New York.
"The New Cæsarean Section,"	Dr. William T. Lusk, New York.
"Gastric Neurasthenia,"	Dr. George M. Garland, Boston.
"Neuritis,"	Dr. Francis S. Miles, Baltimore.
Subject not announced,	Dr. George Ross, Montreal.
" "	Dr. Samuel C. Busey, Washington.

DISCUSSION.

"The Relation between Trophic Lesions and Diseases of the Nervous System."
Referee: Dr. Edward C. Seguin, New York.
Co-referee: Dr. Wm. S. Councilman, Baltimore.

"The Absolute and Relative Value of the Presence of Albumen and Casts and of Renal Inadequacy in the Diagnosis and Prognosis of Diseases of the Kidneys."
Referee: Dr. Robert T. Edes, Washington.
Co-referee: Dr. Edward G. Janeway, New York.

"Demonstrations in Pathological Anatomy,"
Dr. T. Mitchell Prudden, New York.

PAPERS TO BE READ BY TITLE

"Creasote as a Remedy in Pulmonary Phthisis,"
Dr. Beverly Robinson, New York.
"Malarial Modifications of Typhoid Fever,"
Dr. Edward Whittier, Boston.
"Therapeutics of Phthisis,"
Dr. Edward S. Solley, Colorado Springs.
"Antiseptic Medication," Dr. Fredrick P. Henry, Philadelphia.

Furnished by

Dr. HENRY HUN,
Secretary of the Association of American Physicians,
33 Elk Street,
Albany, New York.

Dr. James Hendrie Lloyd has been elected Neurologist to the Philadelphia Hospital in place of Dr. Roberts Bartholow, resigned.

The Georgia Medical Society,
Office of Corresponding Secretary
Savannah, Ga., January, 1888.

To the Editor of Journal of Nervous and Mental Diseases:

Dear Sir:—At the annual meeting of the Georgia Medical Society, held January 3d, 1888, the following resolution was unanimously carried:

Resolved, That the Corresponding Secretary enter into correspondence with the medical journals of the country in order to enlist their influence in support of the movement to remove the import duties from all medical and surgical instruments and appliances, including those used in the diagnosis as well as treatment of disease, so that they may be furnished to those needing them at the lowest possible price.

In compliance with the above resolution, I wish to solicit your earnest attention and a notice in your publication, which will claim the attention of your readers, hoping that your country readers, especially, will appreciate the truth and importance of our proceedings.

Perhaps the statement of a few facts will assist the reader in realizing the extent of the grievance and the justice of the plea for which we ask co-operation.

1st. Physicians are at the mercy of instrument makers in regard to price, make and quality of finish, because of the lack of sufficient competition.

2d. The price of instruments made in this country is out of proportion to that paid for similar instruments on the Continent of Europe.

3d. Surgical instruments and appliances are so costly that but few doctors entering the profession can provide themselves with an outfit adequate to carry on a general practice. At present prices it is impossible for a country physician's income to sustain his investing in costly instruments, and, as a result, many simple cases, such as retention of urine, foreign bodies in nose and throat, deep-seated abscesses, etc., all of which could be relieved at once with the proper instruments, must either die from the immediate cause or from the effects of time lost in seeking skillful manipulation, or else they are frequently crippled and disfigured because the most intelligent help, though patiently given, is itself crippled for want of proper instruments.

4th. The cheaper grades of instruments are either antiquated or so poorly made that they may prove a cause of failure in operations, sapping, as it were, the natural inclinations to surgery in its inception.

5th. European instruments are from 25 to 75 per cent. cheaper than ours, and their introduction into the market will enable the mass of doctors to buy those of prime necessity, will bring down the price of home-made appliances, and oblige the makers to use good material and put a better finish to their work.

6th. The removal of import duties on surgical and other instruments used by the profession and on medicines in general, will produce rhe same results as we all know it did on the article of quinine.

Respectfully,
J. C. LeHARDY,
Corresponding Secretary Georgia Medical Society,
113 Congress Street,
Savannah, Ga.

PERISCOPE.

By Drs. G. W. JACOBY, N. E. BRILL, and LOUISE FISKE-BRYSON.

ANATOMY OF THE NERVOUS SYSTEM.

The Brain Weight in the Insane. Dr. Bartels, of Hildesheim (*Algemeine Zeitschrift für Pschiatrie, etc*).

The author, after giving the literature on the subject, tabulates the findings of the brain weights of males and females in ten different psychoses. Accepting the average normal weight of a healthy brain to be 1,460 g. in males and 1,320 g. in females (Henle and Krause), the following deviations in the various psychoses present themselves :

	Males.	Females.	Average Weight in Grammes.
1. Mania	27		1423
		32	1288
2. Melancholia	74		1437
		95	1284
3. Periodical Insanity	23		1446
		15	1255
4. Paranoia	115		1416
		62	1263
5. General Paresis	276		1353
		44	1185
6. Acute Delirium	5		1385
		2	1325
7. Epileptic Insanity	70		1421
		32	1231
8. Idiocy and Imbecility	27		1335
		18	1194
9. Secondary Dementia	181		1408
		124	1263
10. Senile Dementia	12		1359
		6	1200

Too much weight ought not to be placed on these deviations ; for, as the author well says, in certain groups the number of cases is not sufficient to deduce a safe estimate. In paranoia and secondary dementia, which affect the individual in a large proportion of cases late in life, in which the brain itself would naturally begin to diminish in weight and in which the disease itself may have been of long standing, no reliable estimate could be attained. He concludes as follows :

1. All psychoses necessarily diminish the weight of the brain.

2. This diminution depends (*a*) on the age of the patient, (*b*) on the duration of the disease, (*c*) on the intensity of the disease.

   a.—The diminution in weight is smallest in both sexes between twenty and thirty years of age, largest in males of seventy years of age, and in women of sixty years of age.

   b.—The shorter the average duration of the disease the smaller in general is the loss in brain weight and *vice versa.*

   c.—The deeper the disease affects the mental life of the individual, and in the one who shows the smallest ability for mental work, the greater is the loss of brain weight, and *vice versa.*

3. The diminution in females is larger than that in males by from ⅛ to 1.6 per cent.

N. E. B.

CONTRIBUTION TO THE MORPHOLOGY AND MORPHOGENESIS OF THE CRUS CEREBRI, by G. Jelgersura (*Centralblatt für Nervenheilkunde, etc., September 15th and October 15th.*)

This embraces a short resume of the author's investigations of the brains of five idiots, in which the cortex was very much atrophied as the result of various pathological processes. Only one of the hemispheres of two of these cases showed atrophic changes, the other hemisphere in each being almost entirely normal. There were almost equal changes in both hemispheres in each of the other cases, and almost the entire cerebral cortex was destroyed by encephalitis or by meningitis. The latter cases were extreme idiots, the former two only half idiots with the somatic signs of cerebral hemiatrophy, and one of which was epileptic. All died of some intercurrent affection, between the ages of twenty and forty years. These cases hence afforded to the author a good opportunity of utilizing a pathological imitation of the atrophy method of anatomical research.

"The pons and arciform nuclei were changed in all cases either on one or both sides, according to whether the pathological process involved one or both hemispheres and involved the cells as well as the fibres. The connecting paths between the ganglion cells of the pons and the cortex cerebri were also atrophied, and were best demonstrated in sections at the level of the crus, which was much reduced in volume even in the mesal as well as in the lateral thirds. The pyramid tract in the mesal third was constantly atrophic. In none of the cases did the atrophy of the pyramids involve any of the nuclei in the medulla and spinal cord which belong to the reflex systems. All motor nuclei presented an entirely normal appearance ; and, in the cases of cerebral hemiatrophy, no difference between the motor nuclei of either side belonging to the reflex arcs could be demonstrated. Only the nerve fibres were involved in the atrophy."

"In the pons those transverse fibres were atrophied which go through the raphe and pontis-brachium to the opposite side of the cerebellum." In fact these cases corroberated the statements made by other authors regarding the course of the fibres.

The nuclei arciformes with their connections were changed in unison with the pons, and the author regards them as distal prolongations of the ganglion cell group of the pons.

The author corroborates the findings of Bechterew, who described a group of cells in the pons under the name of "nucleus reticularis pontis," and which lay in the raphe and presented two large wing-shaped extensions between the lemniscus fibres, and which were involved in the atrophic process. This atrophy was traced to the mesal third of the pes pedunculi, and corresponded in course with the course of Meynert's "bundle from the pes to the tegmentum."

"The changes in the olive were numerous. In two cases the ganglion cells were atrophied, on both sides in one case, and very extensively, so that very little of the olive remained; in another, the cells of the olive on the atrophic side were remarkably smaller than on the sound side. In the other cases, also a cell atrophy could be unequivocally demonstrated."

In four cases there was atrophy of the tract, which the author described, although ignorant of the description of Flechsig and Bechterew of the same tract connecting the large olive with the cerebrum (nucleus lentiformis), and which they called the central tegmental tract (*centrale Haubenbahn*).

In one of the cases the connecting tract between the olive and the cerebellum, by way of the raphe and the restiform body of the opposite side, was atrophic.

The hemispheres of the cerebellum were plainly atrophic in four cases. The dentated body of one side was larger than that of the other. In all five cases the tegmenti-brachium was atrophic, as well as the red nucleus of the tegmentum.

The thalamus was also involved, and there was also a diminution in volume of the body of Luys and the body of the substantia nigra.

"Although the cerebral atrophy existed for years and a secondary atrophy of the pyramid tract resulted thereby, the primary nuclei in the medulla and the anterior cornua of the spinal cord remained intact."

The author then considers the question whether there is not an anatomical basis to account for the fact that some of these tracts follow the law of degeneration of Waller, while other tracts do not, and refers to the well-known investigations of Golgi on the structure of the nerve cells and their connections with nerve fibres, and to the similar investigations of Forel, as an explanation. N. E. B.

NUCLEAR ORIGIN OF THE OCULAR FACIAL—Mendel, Berlin Medical Society, Nov. 9th, 1887.

Investigations on rabbits and guinea-pigs resulted in the discovery that the upper facial branch takes its origin in the posterior oculi-motor nucleus. In reference to this relation in man, there are not sufficient anatomical and pathological data to establish it.

The way the upper branch reaches the peripheral facial from the oculi-motor nucleus is through the posterior longitudinal fasciculus and the facial genu. Analagous relations in the division and origin of nerves exist in the spinal cord, and the orbicularis palpebrarum and the levator palpebræ superior are functionally combined. N. E. B.

PHYSIOLOGY OF THE NERVOUS SYSTEM.

ANÆSTHETICS AS A FACTOR IN INSANITY.

Any cause giving rise to delirium may set up a more chronic form of mental disorder quite apart from any febrile disturbance. (*a*) The most common form in such cases is of the type of acute delirious mania; (*b*) though such mental disorder is usually temporary in character, it may pass into chronic weak-mindedness, or into (*c*) progressive dementia which cannot be distinguished from general paresis of the insane.

Alcohol is the most common example of a cause producing permanent disorder of the mind. Symptoms of mental disorder may follow delirium tremens, but instead of the delirious stage disappearing, it becomes established more firmly. A young man of poor nerve inheritance took, within a few days, a large amount of spirit to tide him over some business worry. The symptoms that made the illness appear to be delirium tremens passed off, and left the maniacal excitement persistent. Exhaustion and mental exhaustion followed, with ultimate recovery. Delirium accompanying fevers may have similar results. A seventeen-year-old girl, of neuropathic antecedents, bright, intelligent, and active, became very delirious during the early stages of scarlet fever, and, after several days of sleepless delirium, passed into a condition of mania, at once alarming and revolting. In an asylum she slowly recovered and has remained well ever since. Another of similar age and circumstance developed acute delirious mania after measles, and died in a few days. Pneumonia has also given rise to such states. Belladonna has proved efficient in starting the insane process, as when taken by mistake in the form of liniment. A girl of neurotic stock took belladonna liniment instead of cough mixture, and passed through a sharp attack of mania, though the bodily illness was not extreme, and recovery took place. All the usual anæsthetics are known to produce like mental disorders. A young officer in the British army, of uncompromisingly bad heredity—neuropathic, rheumatic, and phthisical—had violent and destructive acute mania after alcoholic excesses. It was difficult to keep him clad, or out of harm's way. An injury to his hand seemed to clear up his wits. Chloroform was given for a better surgical examination, when the old maniacal ideas at once returned, the same delusions and antipathies. He recovered, but with a mental relapse when the hand healed. An elderly patient was operated upon for cancer about the rectum. Upon regaining consciousness, it was apparent that his mind was affected. Admitted shortly after to Bethlem, he had the aspect of a man half-drunk or half-awakened from some anæsthetic. He was restless, incoherent, repeating meaningless expressions, and of very defective memory. Discharged later as a harmless dement, he remained some weeks at home in the same condition, when he suddenly recovered, and performed his clerical duties with all his past zeal and intelligence. A young woman, the mother of a child ten years old, at whose birth transfusion and large quantities of stimulants were necessities, had occasional hysterical attacks, afterwards known to be due to alcohol. She never had delirium tremens, although a chronic drinker. After the administration of nitrous oxide for the purpose of having teeth extracted painlessly, this patient had an attack of delirious mania which settled into dementia, she never after regaining her senses or recognizing her friends, but remains to-day silly and fat. The points in this case are the acquired nervous irritability, the acute delirious mania, with its consecutive dementia, following in a few hours the use of nitrous oxide. Insanity has followed ovariotomy and parturition when chloroform has been given.

Any toxic agent, more especially those which directly affect the nutrition of the nervous system, as alcohol, lead, belladonna, etc., will cause temporary disorder of intellect in the nervously unstable, and this temporary disorder may assume the form of true insanity ; and this insanity generally, though not always, is acute delirious mania. Shock also may produce similar mental disorder, and must be considered as an element in surgical procedures. In the cases recorded, it had the least possible force. Should neurotic inheritance or neurosis in the individual "give us pause" in considering the expediency of operations not essential to life ? This is a practical question for the surgeon and the neurologist. George H. Savage, M.D., F.R.S., in the *British Medical Journal*, Dec. 3d, 1887. L. F. B.

A CASE OF LINGUAL MONO-HEMIPLEGIA WITH CORTICAL LOCALIZATION—*Bernheim* (Nancy). French Association for Advancement of Science, sixteenth session, 1887. *Gazette des Hôpitaux*, p. 1,016, 1887.

It is known, through the experiments upon animals by Ferrier, and from clinical observations by Charcot and Pitre, that the lower third of the precentral convolution presides over the movements of the opposite side of the face and tongue, and that destruction of that territory produces facial and lingual hemiplegia. Therefore this center corresponds to the lower facial and hypoglossus. It has however not been possible thus far to dissociate these two centers. In all cases of glossoplegia due to cortical lesion of the precentral convolution, facial paralysis has coexisted. In Bernheim's case we have an isolated lingual hemiplegia. The patient was a girl 23 years of age, affected with multiple sarcomatous tumors, the first of which having been observed in 1886. Suddenly (Jan. 8th, 1887), a decided deviation of the tongue supervened, the tip pointing to the right. She swallowed easily, articulated fairly well, but could not whistle. No other paralysis was present, but the pressure shown by a dynamometer was four degrees less with the right hand than with the left (fifteen degrees with the left, eleven with the right); three weeks later, ten with the left, seven with the right. The patient died on the 2d of February, lingual paralysis persisting. The autopsy revealed, in addition to the generalized sarcomata, a cortical lesion consisting of an excavation of five to six mm. in depth and in diameter, and caused by a sarcomatous hemorrhagic clot. This lesion was situated at the lower border of the inferior end of the precentral convolution. The conclusion from this case therefore is, that at the lowermost extremity of the precentral convolution there is a special center, the cortical center of the hypoglossus. G. W. J.

URINE OF TABETICS.—At the same session of the above society, Charles Livon and Henry Alezais (Marseilles) read a paper upon the Urine of Tabetics. From a series of experiments they arrive at the following conclusions:

1. There is in tabetics a tendency to a diminution in the amount of urea excreted in twenty-four hours.
2. Diminution of the total amount of phosphoric acid eliminated with a proportional increase of the phosphates.
3. Considerable variation in the amount of chlorides eliminated, with a tendency to an increase.
4. Intravenous injections of tabetic urine seem to be fairly toxic, twelve to forty-four cubic centimetres for each kilo. of animal, were required to produce death in dogs by this means. G. W. J.

THE DISEASE OF THE TICS CONVULSIFS.—*Burot* (de Rochefort), *Gazette des Hôpitaux*, p. 1,042, 1887.

At the sixteenth session of the French Association for Advancement of Science held at Toulouse, Burot read a paper under the above title, which had first been employed by Charcot, and which affection Gilles de la Tourette has described as a nervous affection characterized by motor inco-ordination, with cholalia and coprolalia, the American Jumpers, the *Tatah* of Malasia, and the *Majirachik* of Siberia probably belonging to the same class. Burot's patient was a girl, 19 years of age, very intelligent, and belonging to a family of the better class.

The convulsive twitchings occur in the face and upper extremities, and are accompanied by the sudden emission of inarticulate cries and of obscene and filthy words. At the age of six years, choreiform movements commenced in the eyes, face, neck, and arms. At twelve years, indistinct gutteral noises, as ouh, ouah! were emitted. At fourteen, obscene and sacriligious words. She repeated all noises which attracted her attention, barking when she hears or even speaks of a dog. Gestures are sometimes imitated. She has various caprices. She not only does and says that which she does not want to, but cannot always do that which she does want to. Wishing to carress a cat, she calls it, but an impulse forces her to push it from her. There is no inco-ordination, but a true impulse. In this affection the reflexes are unduly excitable, whereas the voluntary nervous system is weakened. There is no inhibition of voluntary acts. All in all, it is an impulsive mania.

The author believes that the affection is curable, and his plan of treatment is to lessen the reflex excitement and to strengthen the will-power. This was attempted by moral influence (faith cure ?), and with beneficial result.

In the discussion, Dulony (de Rochefort) cites a case which shows that the affection may occur in persons of very strong will-power, and believes that it is closely allied to hysteria. Bezy (de Toulouse) also cites a similar case.

G. W. J.

THE LESIONS IN MORPHINOMANIA AND THE PRESENCE OF MORPHINE IN THE VISCERA.—Prof. Ball, *Gazette de Hôpitaux*, p. 1,053, 1887.

At the meeting of the French Academy of Medicine of October, 1887, Prof. Ball read a communication, as above. Thus far all investigations of morphinomania have been made upon the living organism, to the neglect of anatomical investigations. This may be ascribed to the rarity of autopsies in such cases. All that has been noticed anatomically is fatty degeneration of the heart, morphinic phthisis, various other acute or chronic pulmonary lesions, gangrene of the extremities, deep abscesses, cerebral œdema, etc, but no one has until now noticed the presence of morphine in the viscera. Ball has observed this feature in a case which occurred at the Asile St. Anne of Paris upon an hysterical morphinomaniac, who took one gramme of morphine daily. An attempt was made at sudden suppression, in consequence of which severe collapse ensued. A hypodermic of morphine, however, restored her. Then gradual suppression was attempted ; but as soon as the morphine was entirely discontinued, severe collapse occurred and death ensued. The autopsy showed fatty degeneration of the heart. Microscopical investigation showed no changes in the nervous system. Kidneys and liver normal. Chemical examination revealed the presence of morphine in nearly all the organs, in the nervous centers, in the spleen, the kidneys, and, above all, in the liver. Ball, in view of this case, warns emphatically against the sudden withdrawal of morphine. G. W. J.

TWO PECULIAR FORMS OF SPASMS. *Report of the 60th Congress of German naturalists and physicians at Wiesbaden, Sept. 18th to 24th, 1887.*

Benedict, of Vienna, before the section of internal medicine, read a paper which described two peculiar forms of convulsive or spastic attacks. The first relates to patients convalescing from myelitic paraplegia, who can walk but are unable to stand. In these cases there is a propulsive and retropulsive convulsive movement on standing. As soon as these patients attempted to stand, they were driven a few steps forward and then just as many steps backward from their original position. In a case of convalescence from subacute myelitis, this symptom developed ; and from the fact that it conformed to a sort of static spasm, the author desired to call it forward "pendulum spasm." The patients oscillate forwards on attempting to stand.

The second form developed in the convalescence from hemiplegia. The affected foot is not put forward in regular tempo, but the patient makes a number of forcibly interrupted attempts; each interruption forwards follows a shorter backward one, and may be compared to some forms of tremor. This condition, the author remarks, belongs to the group of post-hemiplegic chorea and athetosis. Benedict calls it the "trill spasm." (*Triller-Krampf*—Centralblatt für Nervenheilkunde, Psychiatrie, etc., Oct. 15th, 1887, No. 20). N. E. B.

ON THE RELATIONS OF BODY-WEIGHT IN THE PERIODICAL PSYCHOSES. Dr. W. Stark, of Illman—*Allgemeine Zeitschrift für Psychiatrie, etc.*

The author, by constructing a series of co-ordinates, the space between whose vertical lines represented the mouth, and between whose horizontal lines the weight in an ascending scale, was enabled to present a graphic account of the changes of the body weight resulting in the various emotional conditions of exaltation and depression, and in the interval occurring in twelve cases of periodical insanity, six of which were suffering from the circular form of alienation. These patients, all females, were under observation for a period extending between three and five years, and varied in age from 18 to 59 years, in height from 1.54 to 1.69 m., and in average weight from 51 to 89 k. He derives the following general conclusions therefrom, a few of which are here given:

1. The more severe and protracted the paroxysm, whether maniacal or depressive, the quicker and deeper was the descent of the curve.

2. The longer and uncomplicated the interval, the higher and quicker the curve arose.

3. Descent and ascent occurred the most rapidly in the beginning of the paroxysms and of the intervals.

4. When two paroxysms of different characters follow each other, the negative deviation of the curve remains more or less unmoved.

5. Repeated paroxysms force the entire curve *niveau* sharply downwards.

6. Short attacks and small intervals do not materially influence the nerve movements of the curve resulting therefrom.

7. Developmental periods of the individual, as puberty and the climactesic are represented in the excursive magnitude of the curve. N. E. B.

EARLY SIGNS OF LOCOMOTOR ATAXIA.

Observations made on one hundred and seventeen cases of locomotor ataxia by Dr. Max Karger, of Berlin, give the following results concerning the early symptoms of this disease, at the stage when treatment may be rational and satisfactory. In fifty-three per cent. of the cases there was a history of syphilis. There were symptoms affecting sensation, lacinating pains, numbness, especially of the lower extremities, cord-like sensations about the waist, retardation of the rate of conduction of sensations, and Romberg's symptom, which the observer does not consider due to the ataxia, but thinks it an abnormal condition of sensation. Dr. Karger finds at the commencement of the disease diminution in the acuteness of vision, and a concentric contraction of the field, amblyopia and amaurosis, due in thirty-five per cent. of the cases to optic nerve atrophy. Slight and transitory paralyses of eye-muscles were also present. Insensibility of the pupil to light was found in sixty-six per cent. of the cases, due sometimes to a paralyzed condition of the sphincter, and sometimes to disturbances of reflex action. Patellæ reflex was present in eight of the one hundred and seventeen cases examined. Bladder reflex was often diminished, suggesting tabes as the cause of chronic vesical disease of unknown origin. Impotence was more common than any sexual reflex ; and gastric and cephalic "crises," and joint affections rarely noted. L. F. B.

THOUGHTS ON INSANITY.

The elements of continuance and decay are identical. Conditions determine evolution or dissolution, integration or disintegration, development or degeneration. Function is the essence of vital existence. But function works destruction of its organic basis, even as it works its evolution.

Structure originates function, but reaction of function develops or destroys structure. Insanity, as function, determines its own evolution and evolution of its structural basis; but the very forces of insane function can be turned to work dissolution and destruction of insane function and organic substrate. Here lies the therapeutic power of metaphysical diversion. Vital existence contains in itself the elements of its continuance. Morbid ideas contain in themselves potencies to continue their nutrition. Excitation and flushing of cerebral processes with blood supplies the physical nutrition; introspection and external metaphysical agencies supply the spiritual pabulum.

Whether there is a special set of idio-volitional nerves, originating in the brain, and distributed to the periphery of sense, or whether perceptional or hallucinational insanities have a central origin and reference to spacial distance; or whether it is through local vaso-motor peripheral excitation, we can hardly determine. Several cases that have come under my observation would lead me to believe that the fault was peripheral. An excited imagination, directed to the morbid sense, is the cause of the evolution of hallucinational insanities, for this state of things continues nutrition of the perceptional illusion. Imagination unlocks the channels of vaso-motor activity, and flushes the structural basis with blood; but the metaphysical function, that is, the illusion, shapes the substrate to functionate the illusion.—Frank W. Vance, M.D., in *Medical Bulletin.*

L. F. B.

THERAPEUTICS OF THE NERVOUS SYSTEM.

ANTITHERMICS AS SEDATIVES OF THE NERVOUS SYSTEM.

A certain number of antithermics act, not by lowering febrile combustions, but by direct and special influence upon the thermic centres of the spinal cord.

Salicylic acid has taken its place among analgesics, having been successfully employed in neuralgias and tabes dorsalis. Acetanilide (C_8H_9NO), or antifebrin, is almost the peer of salicylic acid in the treatment of acute rheumatism. Acetanilide, when employed as a medicine, should be perfectly pure. Whatever impurities the aniline of commerce may contain ought especially to be eliminated. Only slightly soluble in water, it may be given in wine or some preparation of alcohol. The following are the characters (Yvon) which should belong to medicinal acetanilide:

1. It should possess no odor.
2. It should be perfectly white.
3. Heated on a platinum foil, it should give a colorless liquid.
4. When thus heated, it should be entirely volatilized, leaving no residue.
5. It ought not to give with hypobromite of sodium an orange-yellow precipitate.

Doses may vary from twenty-five centigrammes to three grammes in twenty-four hours. Whatever the daily quantity, it must not be given in one full dose, but in several small doses of fifty centigrammes or more, at regular intervals. Administration of the entire amount at one dose makes collapse a possibility.

Experiments seem to show that acetanilide is not by itself a true poison, but that it acts toxically by robbing the blood little by little of certain principles indispensable to calorification, causing thus a progressive refrigeration incompatible to life. Oxyhæmoglobin is markedly diminished in the blood and methæmoglobin appears. Acetanilide is not eliminated in substance. No trace of it is found in urine. It is almost devoid of antiseptic properties. It has an antithermic value of moderate energy, depressing temperature by acting on the nervous system and the respiratory power of the blood. The antithermic action is unequal. In small doses it produces and sometimes fails to produce any considerable thermic fall. Acetanilide frequently causes cyanosis. It is inferior to antipyrin as an antithermic and decidedly resembles phenic acid in its physiological action; and like phenic acid, should be discarded as an antipyretic. As a nervine medicament it is a precious acquisition to therapeutics. I have combatted by acetanilide three orders of phenomena,—the element of pain in general, the special pains of locomotor ataxia, and lastly epilepsy.

In facial neuralgia, this remedy is inferior to aconite. Yet when the pains in the head are linked to nerve-alterations, as in certain cases of neuritis of the orbital nerves, acetanilide has been found of more value than anything else. In rheumatic, muscular, neuralgic, and even articular pains, it seems superior to salicylic acid; and will cure when aconite, bromide, and iodide of potassium fail. It also has marked somniferous qualities.

In the lightning-like pains of locomotor ataxia, acetanilide renders us special service. The painful crises are completely removed. Sometimes the effect is lasting; but in most cases the amelioration is but transient. After a fortnight or so the remedy may fail utterly to relieve. Doses of one-half gramme three times a day have broken up attacks of epilepsy, though here acetanilide is also uncertain.

Antipyrin is of value in migraine and in angina pectoris accompanying certain diseases of the heart, especially those of the aorta and coronary arteries. These thoracic and cardiac pains may disappear like magic after the administration of a few gramme doses of antipyrin. It is an analgesic, whether introduced by the mouth or by the hypodermic syringe, and has none of the disadvantages of opium.

Salo' is chemically produced by the combination of salicylic acid and phenic acid. Like all its cogeners, it has antithermic properties. Experiments fail to reveal any poisonous qualities. It allays nervous irritability in acute articular rheumatism, and renders real service in the pains of tabes dorsalis, thus bringing about rest and sleep.—Dujardin-Baumetz, Paris, France, in *Therapeutic Gazette.* L. F. B.

HYPNOTISM IN THERAPEUTICS.

In the discussion at the Medical Society of Berlin, on Oct. 26th, 1887, the use of this measure was severely critized by Mendel, Moeli, and others. The tendency of the remarks of these gentlemen went to establish its employment as a dangerous remedy. Mendel considered it not only not advisable, but almost useless; for his experience taught that it produced nervousness in the healthy and increased the disease from which the sick were suffering. (Centralblatt für Nervenheilkunde, Psych., und gericht. Psychopath., Nov. 15th, 1887, No. 22, s. 681.)

N. E. B.

THERAPEUTICS OF THE URIC ACID DIATHESIS.

The subject was introduced for discussion in an address recently delivered by Dr. Burney Yeo before the Section of Pharmacology and Therapeutics at the Dublin meeting of the British Medical Association. The pathology of the condition in which uric acid is present in excess in the organism is still doubtful. Murchison regards the liver primarily at fault, and with this view Latham is disposed to concur. According to this theory, the essential condition present is the non-metabolism of glycosin into urea. Garrod thinks the kidney is the active producer of uric acid, while Ebstein places its production in the muscles and marrow of bones. Frerichs holds that the essential point is the perverted metabolism of albuminoid substances into urea. Bouchard denies that the presence of uric acid in excess is the chief feature of the morbid condition in question. One thing appears certain—the uric acid diathesis has its foundation in the imperfect metabolism of food, especially albuminoids. Dr. Yeo would define it as "mainly a disturbed retrograde metamorphosis." Three things must be taken into account, as in all therapeutic questions: 1, the pathogenic factor; 2, the constitutional factor; 3, the remedial factor. Next to heredity, errors in eating and drinking are the most potent causes in this diathesis. A tendency to obesity has been regarded a potent factor in the production of this condition. A minimum of fats has been advised, and the yolk of eggs prohibited. Malt liquors and bad wines are to be carefully avoided. The *quality* rather than the *kind* of wine is really the important point. Wines having a diuretic action are generally the best. Exercise in moderation is important as tending to improve the general health. A warm, dry, equable climate is useful. All climatic conditions that interfere with the action of the skin are hurtful. The regular use of large quantities of water, preferably hot, is highly beneficial. Garrod, Sir Thomas Watson, and Graves bear witness to the value of colchicum, which has its chief action upon the liver (Yeo), and is also sometimes a diuretic and diaphoretic. Benzoate and salicylate of sodium, guaicum, and iodide of potassium, with lithia, magnesia, and lime, have all been highly praised as remedial agents.

L. F. B.

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Original Articles.

THE HEAT CENTRES OF THE CORTEX CEREBRI AND PONS VAROLII.

By ISAAC OTT, M.D.

IN my experiments upon rabbits I found when a puncture was made just in front of the ear into the cortex there ensued a fugitive rise of temperature. This observation lead me to try in cats the effects of removal of areas of the cortex in this and other regions. The method was as follows : the animal was etherized in a bag, the skin in front of the ear divided, the muscles separated, the bone bared, and the trephine of Pasteur applied, which rapidly makes an opening through the skull. With a small hook-shaped knife the dura mater was divided, and the cortex cerebri beneath broken up to the depth of a sixteenth of an inch. The wound was then washed with a carbolized solution and the integuments brought together by sutures. The weight and rectal temperature were taken previous to the operation and in some cases the animal was placed in d, Arsonval's calorimeter, and heat production and heat dissipation noted for an hour. After the operation the animal was placed in a box of straw and allowed to sleep off the ether, after which he was set in the calorimeter at similar dates for several days. All observations with the calorimeter were made about the same time each day for a student of mine, Mr. W. S. Carter, has found that heat production and dissipation have periods of rise and fall, being lowest from 6 to 9 A. M., and highest from 12 to 2 P. M. They were fed, but partook sparingly of food. A point at the juncture of the supra Sylvian and post-Sylvian fissures was found to have the highest thermic value. S in Fig. 1 shows the position of this centre and from this area it extends downward to the fissura postica. In my descriptions I shall follow the nomenclature of the fissures as given by Prof. Wilder. Other parts of the brain, with the exception of the cruciate centres, had but small effect upon the temperature. In the experiments will be found the details of observations upon other regions of the brain. The rise of temperature after injury to the Sylvian centres is from three to four degrees, and continues till the death of the animal, which is usually about the sixth day. The calorimetric investigations show that either immediately or at the end of twenty-four hours, the heat production and heat dis-

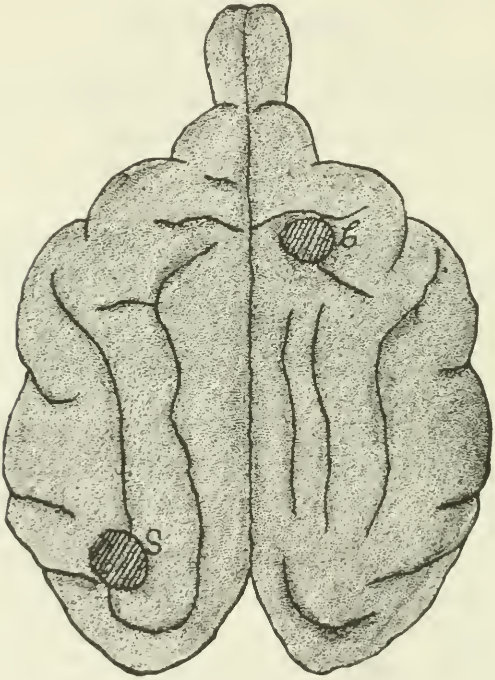
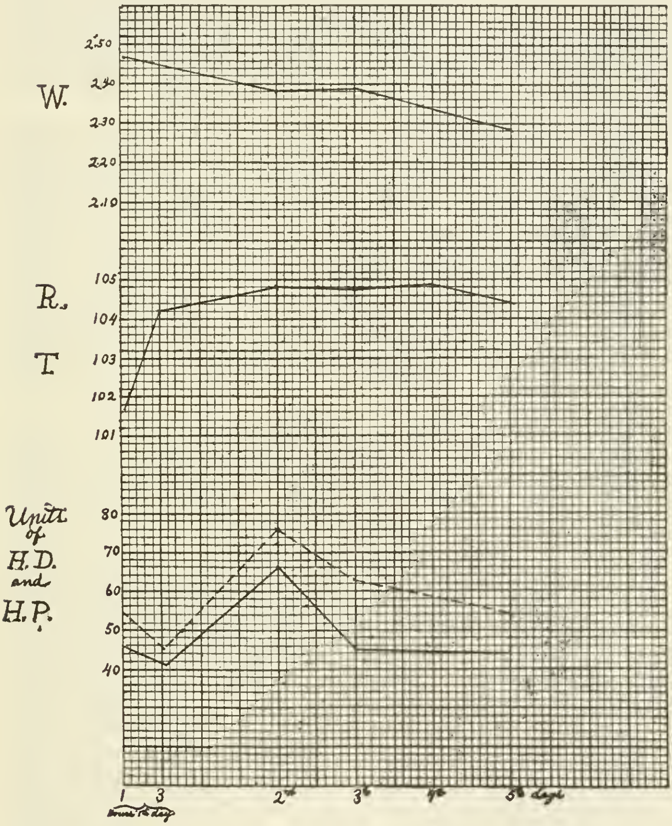


Fig. 1. Twice the natural size. After Wilder.

sipation are increased, after that they usually fall below normal, although the temperature remains elevated, with a weight decreasing daily. In Fig. 2 are given the curves of an experiment, the highest line is that of weight ; the second



line, the temperature curve ; the dotted line that of heat-dissipation ; the continuous line that of heat production. The numbers at the bottom of the figure are the days during which the observations were made. That this increase of heat-production is not due to circulatory changes is seen in the experiment where the pulse and pressure were noted after an operation on the Sylvian centre. They both rise for a short period and then fall to a certain extent below normal, although the temperature is afterwards rising. No data that I know of would justify any one in assuming at the end of twenty-four hours, an increased production of heat is due to a depressed circulation. It is true that after section of a nerve there is an increased temperature in the parts, but it by no means follows that there is an increased production of heat in the part due to increase of blood, for the section itself may remove the inhibition of thermogenesis in the parts supplied by the divided nerve,

Prof. R. Meade Smith,* in a series of observations on the thermic phenomena of muscles, arrived at the conclusion that with a large supply of blood the cool skin, even though exposed to excessive and rapid loss of heat, will become warmer, while on the other hand, the warmer muscle will become cooler. Consequently he states the conception must be erroneous which is generally held as to the temperature changes in muscle from alterations in the blood-supply after section of the nerves.

That the increased heat production does not continue beyond the first twenty-four or forty-eight hours is in part due to diminished ingestion of food. I have observed a similar increase for the first twenty-four hours when experimenting upon the four cerebral heat-centres. In examining the curve of temperature in Fig. 2, it will be found to be similar to that seen after injuries about the corpus striatum, and dissimilar to those seen after the lesions of the thalamic or extra-thalamic centres. It was found that usually the temperature of the trunk and the extremities was elevated, the extremities opposite the side of lesion being slightly warmer than those on the side of injury. Whilst exploring the cortex I found that the cruciate centres (C. Fig. 1.) of Eulenberg and Landois† exhibited upon injury the phenomena described by them.

I shall denominate their centre the cruciate to distinguish it from the Sylvian. I should like to state here that after injuries to the Sylvian I saw no evidences that the cruciate was involved by an meningo-encephalitis, in the production of the phenomena attributed to the Sylvian.

°Archives of Medicine, 1884.

†Therapeutic Gazette, Sept., 1887. Vircho wArchiv. B.d. 68, 1876.

The cruciate is bounded anteriorly by the cruciate sulcus, and embraces entirely a part of the fourth primitive convolution and particularly the posterior and lateral convolutions of the "Häckenformigen" gyrus, which corresponds in man and apes to the antero-central convolution, and appears to be the post-frontal gyrus of Owen. Destruction of the parts was accomplished in dogs by means of the cautery, which caused a considerable rise of temperature in the opposite extremities. The increase of temperature often takes place before the animal recovers from the chloroform. The difference between the temperature of the extremities is from 1.5° C. and 13° C. They also used chloride of sodium, which after a stage of excitation acted like the cautery on the centres, destroying them. The temperature of the ear on the side of lesion was higher ; destruction of the gyrus praefrontalis, the super sylvian and others is without effect on the temperature. They seem to think that when a part of the brain near the cruciate is destroyed, that the lesion in a few hours or a day, may cause a meningitis or encephalitis, invading the cruciate centres and thus elevating the temperatures. If this is true, areas in the neighborhood may have thermic value falsely attributed to them. The temperature after injury to the cruciate centres, gradually increases and remains elevated from three days to three months. Irritation of these centres causes a cooling of the opposite extremities. Prof. Wood has also noted phenomena similarly to those stated above, and he found that the increase of temperature was attended with an augmented production of heat. He did not ascertain how long this increase of heat-production continues. I have made experiments upon this point and determined the weight, temperature, heat-production and heat dissipation for several days. The animals used were cats, and the same method was followed as in experiments upon the Sylvian centres. It will be noted that at the end of twenty-four hours the heat production is elevated above normal, and then returns to its original level, to again rise on subsequent days. Heat dissipation closely follows heat-production. I have also sought to determine the effect of these centres, as well as the Sylvian on the rectal temperature, and found that when irritated they depressed it. When by puncture of the corpus striatum the temperature is elevated, then irritation of the cortical centre by the faradic current still depresses the hyperthermic condition generated by lesion of the heat-centres about the corpus striatum. Experiments upon the circulation when the cruciate centres are mechanically destroyed, produce the same results as after Sylvian destruction.

Pons Varolii: The discovery of the four cerebral heat centres by the method of puncture at the base of the brain, lead me to use it in the pons varolii. When the lateral columns of the spinal cord are divided, and the temperature of the ambient air is about that of the animal, the temperature of the animal rises because there is increased production of heat. If the pons is transversly separated from the medulla a similar increment of heat-production ensues, causing an elevation of temperature. In the experiments upon the pons I have used rabbits, and the puncture was made through the occiptal bone, and cerebellum into the pons varolii. Notwithstanding a large number of punctures, the increase of temperature was only a few degrees and not permanent. These results convinced me that it was not necessary to make any calorimetric investigations, for it is usually, when the temperature is excessive and transient or when it is moderate and continuous for some days, that any notable increment of heat-production ensues. The rise that takes place after an injury to the pons varolii from a transverse section, is due to removal of an inhibiting influence upon it and the spinal cord. In section of the lateral columns of the spinal cord, a similar cause is at work. The idea that a dominant heat centre exists in the pons varolii is not supported by these experiments or by any others when they are held up to the light of recent discoveries.

The query now arises what is the nature of these six centres in the brain? I have already referred to the three divisions of the phenomena of fever by Dr. Donald MacAlister into thermotaxic, thermogenetic, and the thermolytic. The fact of the cruciate and Sylvian centres, upon their destruction, causing an elevation of temperature for days, their irritation a slight refrigeration of the body, and, when striate hyperthermia is produced, irritation of the cruciate centre still reduces it, causes me to believe the Sylvian and cruciate to be thermotaxic. The striate, extra-striate and thalamic centres, with the one about Schriff's crying centre, constituting the four heat centres at the base of the brain, may be regarded, according to the different kind of impulses sent into them by different irritants, as either thermotaxic or thermogenetic.

In another paper* I have spoken of the reasons which lead me to so regard them. The effect of section of the lateral columns of the spinal cord and of the great spinal stimulant, atropine,† in causing increased temperature, induces me to hold that the spinal cord is the main seat of the thermogenetic centres. The increase of heat production after injury to the Sylvian and cruciate centres, the fall to normal, and the subsequent rise in some cases (Exp. 12), indicates that there is a play between these centres and those beneath for mastery, a state of things seen in the temperature of fever patients. If these experiments are examined, they afford excellent ground for the belief that in certain feverish states the normal of heat production and heat dissipation is reset at a higher rate, but at the end of twenty-four hours slides down below normal, partly from want of food. Experiments upon the four basal heat centres also show a similar state of affairs during the first twenty-four hours, heat production and heat dissipation are increased, but afterwards fall, although the fever continues. It is probable that after injury to the cortical heat centre, the basal and spinal thermogenetic centres are temporarily permitted to obtain the upper hand, but that shortly the other cortical heat centres bring the thermogenetic centres into subjection, and thus reduce heat production. In the case of lesion of the basal and spinal thermogenetic centres, they primarily overcome the cortical centres for a short period, but finally succumb to the domination of the thermotaxic centres of the cortex. In other words, the Sylvian and cruciate centres constantly antagonize the basal and spinal thermogenetic centres. It is also probable that under certain impulses the cortex and basal centres combine together to antagonize the spinal thermogenetic centres. It would seem that any injury to the thermotaxic or thermogenetic apparatus sets up a fever which is primarily accompanied by increased production and dissipation; but but they soon fall below normal, whilst the fever continues till the lesion is repaired. This would lead to the belief that in continued fever the generation of a ptomaine is continuously carried on for some time, and thus keeps up the fever. These experiments show how delicate and complex is that most important mechanism of the body temperature-regulation; each of these six cerebral heat centres has its own laws. Some are slow, others are rapid in their elevation of temperature; some continue their activity for days, others for a short time; some affect one part of the body more than another; others do not.

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* Therapeutic Gazette, 1887.

† Therapeutic Gazette, 1887.

The crossed action of the cruciate and Sylvian centres, the former being stronger in crossed activity than the latter, is another fact in support of a view already advanced* as to the decussation of thermotaxic fibres.

The mechanism of temperature production is as follows:

Thermotaxic Centres :	Cruciate (Eulenberg and Landois) and Sylvian.
Thermotaxic and Thermogenetic :	The centre about Schiff's crying centre and the extra-striate, striate (Sachs and Aronsohn), and the thalamic centres.
Thermogenetic Centres :	Spinal centres.

Appended are the experiments upon which the preceding statements are mainly based:

R. T. means rectal temperature.
C. T. " calorimeter "
A. T. " air "
W. is weight in pounds.
H. D. is heat dissipation.
H. P. is heat production.

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* Journal of Nervous and Mental Diseases, July, 1887.

EXP. I.—Cat; weight, 3.36 lbs.

	A. T.	C. T.	R. T.
12.50 P. M.	80.	79.95	102.5
1.50 "	83.55	80.7	100.5
		+.75	—2.0

H. D.=31.29 H. P.=25.42

2.10 P. M., Sylvian centre destroyed.

Second Day.

	A. T.	C. T.	R. T.
12.06 P. M.	73.4	71.45	105.2
1.06 "	75.3	72.7	100.4
		1.25	—4.8

H. D.=52.15 H. P.=49.57

Third Day.

1 P. M. 106.5

Fourth Day.

1 P. M. 105.4

EXP. II.—Cat; weight, 7.6 lbs.

	A. T.	C. T.	R. T.
5.06 P. M.	76.4	70.98	102.4
6.06 "	73.0	72.5	100.3
		+1.55	—2.1

6.30 P. M.—Sylvian centre destroyed on right side.

H. D.=63.41 H. P.=51.00

8.15 P. M. 102.2

Second Day.

8 A. M. 105.9

Left posterior extremity	105.2
Right " "	104.8
Trunk	104.8
Left anterior extremity	104.7
Right " "	104.3

	A. T.	C. T.	R. T.	Weight, 7 lbs.
6.57 P. M.	76.4	76.3	104.3	
7.57 "	76.7	75.2	101.1	
		1.6	—3.2	

H. D.=66.75 H. P.=48.16

Third Day.

	A. T.	C. T.	R. T:	W., 7.38 lbs
10.50 A. M.	72.	71.75	106.1	
11.50 "	74.	73.6	101.9	
		+1.85	—4.2	

H. D.=77.18 H. P.=52.18

Fourth Day.

11.30 A. M. 104.3

Fifth Day.

2 P. M. 105.4

EXP. III.—Cat.

R. T.

7.45 A. M. 101.4

8.00 " Left cortex, injury between the median line of brain and f. lateralis at the posterior part.

Second Day.

10.30 A. M. 101.3

12.50 P. M. 102.4

1.00 " Right cortex injured at the posterior edge of f. ansata towards the median line.

Third Day.

2.05 " 104.2

This injury involved the area of the cruciate centre.

EXP. IV.—Cat; weight, 2.64 lbs.

	A. T.	C. T.	R. T
11.51 A. M.	62.	61.9	102.1
12.51 P. M.	64.5	62.95	99.3
		+1.05	—2.8

H. D.=43.80 H. P.=38.50

2.00 P. M.—Sylvian centre destroyed on left side.

2.30 " 97.3

3.25 " 101.1

5.00 " 102.5

Second Day.

8.00 A. M. 101.9

12.00 M. 103.1

2.15 P. M.—Sylvian centre destroyed on right side.

	A. T.	C. T.	R. T.	Weight, 2.60 lbs.
4.38 P. M.	63.	62.75	103.8	
5.38 "	66.	63.95	100.1	
		+1.20	—3.7	

H. D.=50.06 H. P.=40.97

Third Day.

	A. T.	C. T.	R. T.	Weight, 2.50 lbs.
1.12 P. M.	65.5	61.05	103.4	
2 12 "	63.8	62.0	100.6	
		+1.05	—2.8	

H. D.=43.80 H. P.=37.99

Fourth Day.

12.00 M. ....104.5.

Fifth Day.

	A. T.	C. T.	R. T.	Weight, 2.34 lbs.
1.46 P. M.	65.5	63.2	102.4	
2.46 "	65.0	64.0	100.3	
		.8	—2.1	

H. D.=33.37 H. P.=25.59

EXP. V.—Cat.

R. T.

7.55 A. M. 100.9

8.00 " Right side, injury of cortex at anterior end of super-Sylvian fissure

2.30 P. M. 98.3

Second Day.

12.50 " Left side, injury of cortex just back of anterio end of supra-Sylvian fissure near f. ansata.

Third Day.

2.05 " 96.3

EXP. VI.—Cat; weight, 2.98 lbs.

	A. T.	C. T.	R. T.
11.26 A. M.	61.	60.75	101.7
12.26 "	61.8	61.95	100.6
		+1.20	—1.1

H. D.=50.64 H. P.=47.34

12.30 P. M.—Sylvian centre on right side destroyed.

2.15 " 103.1

	A. T.	C. T.	R. T.
3.32 P. M.	65.4	62.8	104.2
4.32 "	64.8	63.9	102.4
		+1.1	—1.8

H. D.=45.89 H. P.=41.44

Second Day.

8 A. M. 104.5

	A. T.	C. T.	R. T.	Weight, 2.76 lbs.
12.12 P. M.	64.2	61.8	103.9	
1.12 "	64.6	63.38	100.3	
		+1.58	—3.6	

H. D.=75.91 H. P.=67.67

Third Day.

	A. T.	C. T.	R. T.	Weight, 2.78 lbs.
11.54 A. M.	60.	59.75	104.8	
12.54 P. M.	66.4	61.05	101.6	
		+1.30	—3.2	

H. D.=54.33 H. P.=46.93

Fourth Day.

12.00 M. . . . Left post. extremity. 104.9
Right " " 109.0
Trunk. 104.5
Left anterior extremity. . . . 104.5
Right " " 104.5

Fifth Day.

	A. T.	C. T.	R. T.	Weight, 2.36 lbs.
12.46 P. M.	67.5	61.95	104.3	
1.46 "	67.6	63.20	100.1	
		+1.25	—4.2	

H. D.=52.15 H. P.=43.86

EXP. VII.—Cat.

2.30 P. M. 101.5
2.35 " Sylvian centre broken up on left side.
4.49 " 102.3

Second Day.

8.00 A. M. 103.6
5.00 P. M. 104.1

EXP. VIII.—Cat.

2.30 P. M. 100.8
2.35 " Sylvian centre on left side broken up.
4.50 " 100.9

Second Day.

8.00 A. M. 103.2
5.00 P. M. 104.4
Left posterior extremity. . . 103.4
Right " " 103.6

Third Day.

8.00 A. M	103.1
4.00 P. M	104.3

Fourth Day.

8.30 A. M	102.1

EXP. IX.—Cat.

		R. T.
2.15 P. M		102.1
2.22 "	Sylvian centre exposed and faradaised for two minutes, Du Bois coil at 8.	
2.25 "		101.5
2.32 "		101.1
2.42 "		101.1

EXP. X.—Cat.

		PULSE.	PRESSURE.
11.29 A. M.		76	124
11.30 "	Sylvian centre destroyed.		
11.30.15 A. M		83	140
11.30.45 "		70	144
11.40.00 "		72	118
12.18.00 P. M		70	118

EXP. XI.—Cat.

		R. S.
3.50 P. M		99.7
	Excision of cortex on both sides of brain just back of the sulci cruciati—considerable hæmorrhage.	
4.25 "		93.9
6.00 "		96.9
8.35 "		100.7
9.22 "		101.2

Second Day.

8.00 A. M	102.2
12.15 P. M	103.8
3.00 "	104.3
6.30 "	102.9

Third Day.

8.00 A. M	102.9
3.45 P. M	103.

EXP. XII.—Cat; weight, 4.78 lbs.

	A. T.	C. T.	R. T.
11.55 A. M.	62.6	62.55	101.9
12.55 P. M.	63.4	63.7	99.7
		1.15	2.2

1.20 P. M.—Left post-cruciate centre ablated under ether.

H. D.=47.97 H. P.=39.25

	A. T.	C. T.	R. T.	Weight, 4.78 lbs.
4.43 P. M.	71.9	65.6	101.0	
5.43 "	71.6	66.85	99.0	
		1.25	2.0	

H. D.=52.15 H. P.=44.22

9.30 P. M. 102.5

Second Day.

9.00 A. M. 102.5
6.00 P. M. 102.3

Third Day.

11.30 A. M. 103.8

Fourth Day.

11.30 A. M. 104.4

Fifth Day.

11.30 A. M. 103.2

Sixth Day.

Right side, posterior extremity...... 103.0
Left " " " 102.6

Seventh Day.

2.35 A. M. 104.1

Eighth Day.

	A. T.	C. T.	R. T.	Weight, 4.36 lbs.
12.36 A. M.	62.	61.45	102.0	
1.36 "	65.4	62.7	99.5	
		1.25	2.5	

H. D. =52.15 H. P.=42.07

Ninth Day.

11.55 A. M. 100.9

Tenth Day.

1.20 P. M. 98.7
1.35 " Puncture into corpus striatum.
3.15 " 103.2
3.55 " 103.2
4.00 " Irritation for fifteen seconds; Du Bois' coil at 10 of post-cruciate centre of right side.
4.02 " 102.1

EXP XIII.—Cat; weight, 4.98 lbs.

	A. T.	C. T.	R. T.
11.30 A. M.	67.8	67.7	101.9
12.30 "	68.6	68.45	101.6
		·75	·3

H. D.=31.29 H. P.=30.06

1.40 P. M.—Etherized, left post-cruciate centre broken up with galvano-cautery.

1.41 "	98.7
2.00 "	101·2
3.42 "	102.1
5.00 "	102.0
8.20 "	102.7

Second Day.

	A. T.	C. T.	R. T.	Weight, 5 lbs.
12.00 M.	68.	67.5	103.2	
1.00 P. M.	69.5	68.65	100.6	
		1.15	2.6	

H. D.=47.97 H. P.=37.18

EXP. XIV.—Cat; weight, 4.80 lbs.

	A. T.	C. T.	R. T.
11.30 A. M.	65.3	62.45	102.9
12.30 "	65.4	63.55	101.2
		1.10	—1.7

H. D.=47.97 H. P.=41.20

12.50 P. M. Under ether has left post cruciate centre destroyed.

1.00 "	98.7
1.20 "	101.6
3.50 "	102.7
5.50 "	103.0
9.00 "	102.9

Second Day. Weight, 4.62 lbs.

9.00 A. M.	101.9

	A. T.	C. T.	R. T.
12.05 P. M.	64	63.6	103.9
1.05 "	64	64.5	103.1
		+.9	—.8

H. D.=39.63 H. P.=37.00

Third Day.

	A. T.	C. T.	R. T.	Weight, 2.22 lbs.
11.00 A. M.	66.	65.6	103.9	
12.00 M.	66.8	66.5	101.2	
		.95	2.7	

H. D.=39.63 H. P.=34.66

Fourth Day.

	A. T.	C. T.	R. T.	Weight, 2.22 lbs.
2.10 P. M.	66.0	65.2	105.2	
3.10 "	67.2	66.35	101.7	
		1.15	3.5	

H. D.=47.97 H. P.=41.53

Fifth Day.

9.00 A. M. 104.9

Fifth Day.

11.00 A. M. 105.1
Right posterior extremity............ 104.6
Left " " 102.6

EXP. XV.—Cat; weight, 2.78 lbs.

	A. T.	C. T.	R. T.
3.43 P. M.	64.2	61.45	102.5
4.43 "	64.	62.18	102.0
		73.	.5

5.00 P. M.—Left Sylvian centre destroyed.

H. D.=30.45 H. P.=25.47

6.45 P. M. 102.6

	A. T.	C. T.	R. T.
8.37 P. M.	63.7	62.3	102.9
9.37 "	63.6	63.28	102.0
		.98	.9

H. D.=40.88 H. P.=38.81

Second Day.

	A. T.	C. T.	R. T.	Weight, 2.26 lbs.
4.35 A. M.	66.5	65.3	103.8	
5.35 "	67.0	65.95	102.8	
		.65	1.0	

H. D.=27.11 H. P.=25.24

EXP. XVI.—Cat; weight, 3 lbs.

	A. T.	C. T.	R. T.
11.00 A. M.	62.55	62.55	102.0
12.00 M.	66.0	63.9	100.0
		1.25	2.0

12.15 P. M.—Left post-cruciate centre destroyed.

H. D.=56.32 H. P.=51.34

	A. T.	C. T.	R. T.
3.26 P. M.	64.5	63.8	104.5
4.26 "	66.0	65.3	100.9
		1.5	3.6

H. D.=62.58 H. P.=53.62

Second Day.

	A. T.	C. T.	R. T.
1.28 P. M.	66.5	66.35	106.1
2.28 "	68.5	67.45	103.6
		1.10	2.5

H. D.=45.89 H. P.=40.29

EXP. XVII.—Cat.

1.00 P. M.—A puncture was made into the left corpus striatum.

Second Day.

	R. T.
11.50 A. M.	103.5
11.51 " Right post-cruciate centre was irritated with Du Bois' coil at 10 for two minutes.	
11.55 "	102.1
12.00 "	101.5
12.05 P. M.	101.8

EXP. XVIII.—Cat.

	PULSE.	PRESSURE.
2. 1. 0 P. M.	70	74
Destruction of the right cruciate centre.		
2. 1.15 "	68	74
1. 1.45 "	92	100
2. 2. 0 "	—	74
2.52. 0 "	64	—

EXP. XIX.—Rabbit.

		R. T.
2.35 P. M		102.6
2.36 "	Puncture in median line at the junction of the pons and medulla oblongata.	
2.45 "		101.7
3.35 "		99.5
4.00 "		98.9
6.25 "		98.5
	Second Day.	
9.12 A. M		89.7

EXP. XX.—Rabbit.

		R. T.
12.20 P. M		102.0
12.21 "	First puncture into right side of pons varolii.	
12.21 "		102.3
1.15 "		100.9
7.31 "		101.3
	Second Day.	
2.10 P. M.	Puncture into right side of pons.	
3.40 "		101.4
6.30 "		103.0
8.05 "		103.9
9.40 "		103.6
	Third Day.	
	Puncture into right testes.	
7.40 A. M		101.9
8.00 "		101.8
11.15 "		102.3
12.37 P. M		103.5

EXP. XXI.—Rabbit.

		R. T.
12.00 M.		102.7
12.01 "	Puncture into median part of pons varolii.	
12.30 "		102.0
5.30 "		100.5

EXP. XXII.—Rabbit.

		R. T.
12.04 P. M		103.2
12.05 "	Puncture in median line at junction of medulla and pons varolii, runs forward in violent manner at times.	
12.30 "		103.3
1.40 "		104.8
2.05 "		103.6

EXP. XXIII.—Rabbit.

		R. T.
12.09	P. M.	102.9
12.10	" Puncture into right side of pons varolii.	
12.30	"	102.7
2.15	"	102.2
3.25	"	103.3
5.30	"	101.3

Second Day.

12.52	" Puncture into right testes.	
2.22	"	104.0
3.05	"	104.3
6.30	"	103.5

Third Day.

1.30	P. M.	99.8

EXP. XXIV.—Rabbit.

		R. T.
2.24	P. M.	103.0
2.25	" Puncture about the middle of the pons varolii.	
3.15	"	102.9
4.05	"	103.3
5.25	"	102.9

EXP. XXV.—Rabbit.

		R. T.
12.06	P. M.	102.6
12.07	" Puncture at end of pons varolii, where it joins the crura cerebri.	
12.25	"	101.6
2.10	"	101.1
6.35	"	101.7

EXP. XXVI.—Rabbit.

		R. T.
2.54	P. M.	101,7
2.55	" Puncture into pons varolii.	
3.30	"	101.0
6.15	"	102.3
9.30	"	102.5

Second Day.

8.15	A. M.	101.3

EXP. XXVII.—Rabbit.

		PULSE.	PRESSURE.
3.28 P. M		60	64
3.28.30 "	Puncture into upper part of pons varolii.		
3.29 "		64	76
3.29.45 "		52	76
3.38 "		58	78
3.41 "		—	78
4.07 "		—	66

EXP. XXVIII.—Rabbit.

		R. T.
2.14 P. M		103.2
2.15 "	Puncture into median part of pons varolii.	
2.30 "		103.6
3.10 "		102.9
6.00 "		103.7

EXP. XXIX.—Rabbit.

		R. T.
2.14 P. M		102.9
2.15 "	Puncture into the right upper end of pons varolii.	
2.30 "		102.9
3.20 "		100.2
6.00 P. M		99.2

IS ATROPHY OF THE CONDUCTING APPARATUS OF THE EAR IDENTICAL WITH PROGRESSIVE ARTHRITIS DEFORMANS?

By S. O. RICHEY, M.D.,

OF WASHINGTON, D. C.

Read in the Section of Otology of the Ninth International Medical Congress, Washington, D. C., September, 1887.

THE name *General Atrophy of the Conducting Apparatus* may not be better than the numerous other names by which the affection is designated, but it has the merit of describing the result of the process as we see it, instead of indicating it by some particularity of its course. Some attempt will be made to show its probable neurotic origin in the spinal system by its similarity to a more general affection, which has been supposed to find its source there.

Atrophic degeneration of the conducting apparatus of the ear may not be, to any great extent, inflammatory in any part of its course, "is not pre-eminently local in its character," *is* influenced by constitutional dyscrasia, probably begins at the cervico-spinal nervous centres, and is propagated through the sympathetic nervous system, or the sensory spinal nerves, interfering with local trophic action.

Many pathological changes have been observed in the cavity of the middle ear at its examination after death, but we are not assured thereby that any given structural variation has been a result of this affection alone, as we are denied the opportunity of observing it during the progress of the disease. For many reasons, we must think it has a broader pathogenesis than that generally accredited to it, in the exposition of which we may be aided by the processes of analogy and induction.

Garrod says of "Progressive Arthritis Deformans:* "It is much easier to prove what rheumatoid arthritis is not, than to give the slightest clue to what it is. . . . It appears to result from a peculiar form of malnutrition of the joint textures, an inflammatory action with defective power. . . . It usually occurs in weakened subjects, and exposure to cold is, in many cases, the exciting cause of its development." Weber,† considers it of neurotic origin.

In its entire history, except in the functional peculiarities of the locality attacked, it is almost a complete analogue of atrophy of the middle ear: in causation, symptoms, progress, and therapeutics. It will be an advantage to be able to watch the process, as we cannot in the ear.

Herewith is a parallel of the two affections.

Progressive Arthritis Deformans.	*Atrophy of the Conducting Apparatus.*
(1). It is seldom fatal.	(1). We do not know it ever to be fatal.
(2). At an early stage, swelling and the appearances of ordinary inflammation are prominent.	(2). At an early stage, this may be the cause of the symptoms of inflammation.
(3). When the effusion into the joint is absorbed, the capsule is commonly found thickened, the cartilages are sometimes absorbed, and the ligaments so much lengthened as to allow unusual mobility and dislocation.	(3). When this happens it would be liable to cause tinnitus or impaired hearing, or both: a flapping mt. and disarticulation of the ossicula auditus
(4). At the commencement of the process slow absorption of the cartilages takes place, often followed by fatty degeneration and the formation of ligamentous bands.	(4). The result of this change has been seen in anchylosis of the ossicles, especially of the stapes: retraction of the mt., and bands of adhesion in the cavity.
(5). Heredity does not seem to influence the affection, for one member of a family may be affected and the rest be free.	(5). Complete correspondence.
(6). Is frequent among women and rare among men.	(6). Is more frequent among women than among men.

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* Reynold's System of Medicine, vol. i., p. 555.

† Journal of Nervous Mental Disease, vol. viii., p. 630.

(7). It occurs at any age, and individuals of weak frames whose extremities are cold are most liable to the disease.	(7). The symptoms manifest themselves at middle age or just before, and at any later period. Cold extremities are common.
(8). Everything debilitating, as uterine hæmorrhages, prolonged grief, persistent mental distress, loss of rest and dissipation, damp dwellings, poor food, and all rheumatic influences are supposed active causes.	(8). *Idem.*
(9). By test, no uric acid or urate of soda, thus removing rheumatism and gout from consideration as causes. Reduction of phosphoric acid in the urine.	(9). No tests have been made, so far as I know.
(10). The disease is slowly but steadily progressive. It may be stationary for a time, but exacerbations are sure to follow (Weber). There is slight remission, but no intermission during the rest of the patient's life (Haygarth).	(10). There are long intermissions in progress, judged by the impairment of function.
(11). It usually begins as a subacute disease.	(11). An open question.
(12). It is very intractable. When the disease is not advanced, *the affected joints few in number*, and progress slow, the prospect is more hopeful, especially if there is no disease to keep up the impairment of the general health.	(12). *Idem.*
(13). The treatment must be sustaining. *Local* treatment by blisters, *iodine paint* and croton oil in the beginning. Later, use counter-irritation ; later still, friction and slight motion. Living in a moderete climate in the winter, nutritious food, warm clothing, etc.	(13). This general line of treatment is the best with which we are yet acquainted.
(14). There is generally aching in the affected joints, prophetic of an increase of pressure in the atmosphere.	(14). Any existing impairment of hearing or tinnitus is increased under the same circumstances.
(15). Frequent mental depression without a known sufficient cause.	(15). *Idem.*
(16). It does not lead to suppuration, but to atrophy and more or less deformity (Weber).	(16). *Idem.*

Progressive arthritis deformans begins in the *smaller* joints of the body, is symmetrical in appearance and progress, with lesions of the tissues surrounding the joints, atrophy of the muscular tissue, and, in old cases, a state of fatty and connective tissue degeneration (Weber). The Lilliputian joints of the ossicula auditüs are peculiarly exposed to atmospheric changes by their location, and are in one of the extremities of the body, for which reasons they would seem to be more liable to an attack of this affection than even the joints of the hand or foot. Rheumatoid arthritis beginning in the small joints of the extremities advances to the larger joints of the body. This may explain the pressure and pain about the head, and the diminution of intellectual apprehension, so common in cases of profound deafness in advanced aural atrophy. It may furnish a better demonstration of the deafness of boiler-makers and that of locomotive engineers.

Taking its symmetrical onset and advance as a point in evidence of its neurotic origin, it may also explain the change in voice so commonly met with among the profoundly deaf, who have become so by slow and progressive stages, for the recurrent laryngeal nerve makes the connection between the cerebro-spinal nerve centres and the vocal cords very intimate. The recurrent laryngeal is supposed to get its motor power from the pneumogastric, and irritation of the pneumogastric in the upper part of the neck has been proven by experiment to cause heat and tingling of the ear. Jewell* "looks upon articular rheumatism, as well as certain painful affections of the joints simulating rheumatism as produced . . . by disease of the nerve trunks or nerve centres, leading to decided local irritation at the peripheral termination of certain nerves ;" and Brown-Sequard has shown that nerve fibres going to the blood vessels of the various parts of the head come out chiefly from the spinal cord by the roots of the last cervical and the first dorsal nerves.

Leloir and Dejerine† observe that, in a case of chronic rheumatism with considerable muscular atrophy and rapid eschars, they found the cutaneous nerves adjacent to the eschars affected with atrophic parenchymatous neuritis which seemed to have been existent anterior to the eschars. Acute atrophy of the muscles has occurred without *lesion* of the cord. Those suffering with arthritis deformans appear to be emaciated and neurasthenic as a rule, complaining of pains over a great part of the body, associated with periodical failure of control or power in the muscles and tendons.

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* Jour. Nerv. and Mental Dis., 1874. 426.

† Progrès Médicale, April 2d, 1881.

The pain in the joints, weakness in the muscles and tendons, and some emaciation, often precedes the manifest changes in the size and form of the joints.

Women, according to Rosenthal, are more subject to prosopalgia in early life than men: neuralgia is most frequent between the thirtieth and fiftieth years of life; is sometimes accompanied by *inability to fix the mind* on any *subject or attend to business*, and this effect is not due to pain. Arthritis deformans is often introduced by hemicrania, and lean persons have a more decided predisposition than the stout, as in neuralgia of the fifth. The temporo-maxillary and the upper cervical vertebræ are joints particularly likely to be affected. No one questions the character of neuralgias, and arthritis appears to have a similar neuropathic origin and similar favoring causes.

Weber-Liel* has seen thirteen persons affected by progressive deafness presenting the symptoms of spontaneous nervous pain over the tracts of the cervical and brachial plexus, associated with pain in the ears and disagreeable tinnitus, varying from that habitual to the case. Otalgia is often met with in the later stages of atrophy, generally uncomplicated with neuralgia elsewhere, but among individuals of the neurasthenic type. Arthritis deformans, nervous exhaustion, and aural atrophy (progressive deafness) very greatly resemble each other. Each follows causes exhaustive in character; does not terminate fatally; most of the symptoms are subjective and functional, and often without apparent structural variation. In each there is intermittent and periodical hopelessness and discouragement. In nervous exhaustion and the ear affection there is diminished ability to fix thought on any subject (lack of mental control), and change in the voice; and Garrod claims that the *irregular* form of arthritis sometimes attacks the internal (middle?) ear and the larynx, and causes hoarseness and a peculiar dry cough.

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* Monats f. Ohrenheilk., August, 1874.

Mr. R. W. Parker reports* a case of rheumatoid arthritis; the girl, aged 15 years, whose father died of phthisis and whose mother died of chalky rheumatism, had, in six months, become almost completely deaf. She had double keratitis and enlarged joints. No examination of the physical condition of the ears appears to have been made, unfortunately; only the reference above to the disturbed function.

Burnett† mentions a woman, aged 26, well-nourished, who six years before had an attack of probable rheumatic facial paralysis. Two or three years later she noticed singing in her ears and impaired hearing. Lustre of Mtt. good: ett. pervious. When excited or fatigued there was flush of the cheeks and neck and increased tinnitus.

In boiler-maker's deafness, undisturbed control of equilibrium and the absence of vertigo argue against the theory of labyrinthine trouble. Buck‡ thinks the peculiarities of these cases due to rigidity of the ligament at the base of the stapes, or to some change in the membrana secondaria, which to my mind is the most natural explanation. The fact that individuals who have had acute or subacute catarrhal inflammation of the middle ear present the feature of hearing better in a din of some kind, does not invalidate Buck's theory, as, in even acute suppuration, the membrana secondaria may undergo changes calculated to produce this effect: persistent thickening calcareous deposits, adhesive secretions, etc., for which reason some of these cases can be comparatively promptly improved. "Pathological alterations take place in the stapedio-vestibular articulation in the course of chronic inflammation of the middle ear *sometimes also with a perfectly normal state of the lining membrane*." §

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* Trans. Internat. Med. Congress, 1881, Vol. I., p. 128

† A Treatise on the Ear, 1st Ed., p. 391.

‡ New York Med. Rec., July 5th, 1875.

§ Politzer, Diseases of the Ear, Am. Ed. p. 86.

Among boilermakers the continuous action of the ossicula auditüs renders them more liable to arthritis; and the exposure to draughts, lack of exercise of most of the other joints of the body, irregularity in taking food, which is often less assimilable than it should be, furnish other sufficient factors in the causation of this affection. These same influences obtain among ship-caulkers and locomotive engineers. That the affection may be manifest in no other joint is no sufficient reason against its attacking the ossicula, when they are most used. The affection may extend to the sutures of the cranial bones, and cause disturbance in their relations to each other, followed by a feeling of pressure, or "weight on the head," or, "as if there was an iron band around the head," or, "as if there was an iron axle between the ears," resulting from even slight distortion. It may thus so derange the cranial contents as to interfere with normal mental alacrity and the memory, of which some people with "progressive deafness" are so acutely conscious in the later stages. It may alter the shape and size of the cranial foramen to such an extent as to cause pressure upon the nerve thus finding exit, and so produce neuralgia in the region supplied by the nerve, this being one of the ways in which persistent neuralgia is supposed to be produced.

Arthritis deformans may occur at almost any age; at first, in the most exercised small joints, and if neglected it will progressively attack every joint in the body. It would rarely be recognized in the ear before the age of thirty, when the true function of the ear begins to be impaired in the late stage of atrophy, though it might have existed from the age of four or five years, at which time it would have been in its inflammatory stage. This stage would be marked by sudden onsets of pain, of spasmodic or neuralgic character, causing at short intervals sharp, quick cries, followed by a period of ease and quiet. Though during the day there is entire comfort, the attacks are disposed to recur at night, the child sometimes waking from a sound sleep with a cry of distress, and falling to sleep again in a short time. These attacks are supposed to be harmless, because they do not result in suppuration, immediate deafness, or any other material change in function or structure, for they pass off after several hours of intermitting pain, leaving some tenderness to touch, to recur perhaps the next night to follow much the same course. The m. t. may be hyperæmic, but is not thickened; the e. t. is as patulous as usual, and there is no appreciable increase of secretion; it may recur every evening of several days with entire subsidence of pain for the greater part of the twenty-four hours. These attacks differ in several particulars from the catarrhal affection resembling it, which causes almost continuous pain, thickening of the lining membrane, diminution or closure of the e. t., and increase of secretion, with bulging of the m. t., on this account; sometimes suppuration occurs, if the case is not promptly and properly handled; nearly always there is more or less impairment of hearing from congestive thickening of the tissues, or the presence of fluid in the cavity. After one catarrhal attack, there may never be another.

The same cause seems immediately productive of each, because each is more liable to happen at the change of the seasons, the child being more exposed to the cold and the damp air at these times.

Thus, even in childhood a differential diagnosis may be made from the catarrhal affection, and we may reason that the affection at the foundation of the atrophic process may begin *at any age*, although the atrophy is a malum senilis. V. Trölsch thought the disease *without catarrhal symptoms* should be given a different classification, but it is yet generally classed as a catarrh by authorities, though Pomeroy,* in a cursory way, says, "I believe that the rheumatic diathesis in many instances has much to do with the obstinate character of this affection; the rheumatic inflammation, according to its well-known predilection for fibrous tissues, finding a lodgment in the muco-periosteal lining of the drum."

Whether or not atrophy of the middle ear is of the same origin as arthritis deformans, it has a more extensive pathology than that generally accorded to it.

Treatment.—Arthritis is introduced by a chill (Bruce), followed by hemicrania, indicating depression of the nervous and circulatory systems. This action may be induced by cold, emotional disturbance, or physical shock; it concentrates at the cerebro-spinal nervous centres, and radiates therefrom to express itself in the organ of the least resistance in an individual, in the form of pain and trophic changes.

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* Dis. of Ear, p. 148.

The views of the writer in regard to local treatment in "progressive deafness" may be found in the *Am. Jour. Med. Sci.*, April, 1887, p. 413-423. Iodine vapor is our sheet-anchor for topical medication, but our efforts may be materially aided by constitutional and hygienic influences, under which head come climate, clothing, food, and other items of general treatment.

Climate should have special consideration in the choice of a winter residence. This should be moderate in temperature, and as dry as possible. The sudden changes of temperature in the higher latitudes are more deleterious, because they take place through a lower thermometric range, and passing from the inside to the outside of the house may produce violent circulatory disturbances during cold weather unless the cold is moderate. A climate distinguished by a decided difference in temperature between day and night is undesirable, unless this variation is guarded against by fire and clothing, which means thought and care on the part of the individual not likely to be taken.

Clothing is very important, as it should be of such character as to afford protection against the depression of climatic variation, that worn *next* the body requiring most thought, though at present it has least attention.

Three areas of the body are especially sensitive to changes of temperature and seem, to a great degree, to influence the comfort of the whole body. Such spaces are the *cervical region of the spine*, the *posterior aspect of the arm just above the elbow*, and *the nates*.

The ordinary dress of men protects them very well, the buttocks being most exposed, and from this region the body may be chilled or warmed. The exposure of this part of a chilled body to the grateful influence of radiated heat diffuses more general composure than warming the extremities, and every *man* will receive this suggestion feelingly. This effect is probably due to the superficial location of the sciatic nerve, and its short cutaneous branches.

Among women the cervical region of the spine and the arms are least covered, especially when in "evening dress." When entering a cold bath the body may be more quickly adjusted to the lower temperature by dipping the elbows and the nates than by wetting the head and neck, according to the usual custom, and in this we may find some proof of the above statement. Women, and particularly neurasthenics, often complain, at the menstrual period, of cold on the posterior face of the arm just above the elbow. When an individual has his arms bared he may be seen to hold his elbows with his hands unconsciously, unless he is at work. This habit may be observed among workmen and washerwomen, and sometimes among fashionable dames in bare arms. This part is supplied with cutaneous branches from the brachial plexus, and thus has more than local influence ; our instinct is to protect it from the cold. Women ordinarily have one thickness of dress upon the arm and neck ; sometimes two on the arms, and, in the coldest season, often *none* on arms or neck. The dress may be worn high and covered with wraps during the warmest part of the twenty-four hours, to be exchanged, frequently, for a decolleté habit when the temperature is lowest (slippers are not forgotten). May we not find in these facts some of the predisposing causes of the greater frequency of this aural affection among women ? Fashion is without discretion, and is a Moloch to which health is unpityingly sacrificed (self-sacrifice). Intelligent advice may be given in regard to dress, but fashion scorns it, and medication must be to little purpose without rational precautions on the part of the patient.

General Medication.—Avoidance of shock, mental distress, pregnancy, damp, cold, and whatever else greatly disturbs the balance of circulation, is to be advised. Nutritious food is to be taken regularly, and in such quantity as can be digested and assimilated. The moderate use of red wine is beneficial. Arsenic (Liq. potass. arsen.), in drop doses, taken for some months, promotes digestion and assimilation, in addition to its specific action upon the mucous membrane. In the same way it probably is useful in anæmia and certain forms of neuralgia. Syr. ferri iodidi is serviceable in cases dependent upon impoverished blood, and may be associated with arsenic in the same prescription. Any gain from the administration of cod-liver oil has not been apparent to me. Salicylic acid and the salicylates, in small doses, have had manifest influence in some cases. Due attention should be given to the proper performance of its functions by every organ of the body, and particularly to the action of the bowels. A habit of constipation must be corrected to aid proper nutrition.

The above outline, taken with the local manipulations heretofore described, are of most certain value in the treatment of cases of aural atrophy.

It is needless to say that a certain number of cases exist in which the structural injury is of such character and so established, that nothing short of re-creation will restore to the organ the conditions necessary to its intended duties; as, for instance, some cases of osseous anchylosis, or disarticulation of the ossicula.

DENTAL IRRITATION AS A FACTOR IN THE CAUSATION OF EPILEPSY.

Read before the Philadelphia Neurological Society, December 19, 1887.

By ALBERT P. BRUBAKER, M.D.,

PHILADELPHIA, PA.

IN all the wide divergence of view as regards the nature of epilepsy there is a general consensus of opinion that its essential feature is of the character of an explosive discharge from the higher nerve-centres, the nerve-force thus liberated bearing down upon the centrifugal distributions of the motor nerve-tracks with such an excess of energy that inco-ordination of movement reaches the stage of convulsion and spasm. Owing to the periodicity of the convulsive seizures, it has been assumed that in individuals predisposed to epileptic attacks the higher nerve-centres are in a state of high tension, of unstable equilibrium, and that it only requires a stimulus of a definite quantity or intensity to excite the explosive discharge.

Writers have generally laid it down as an established fact that the majority of the cases of epilepsy are idiopathic, without definite causation, and due solely to heredity ; but it can scarcely be doubted that these cases are properly so classed only as regards the pre-disposition, and that in them all a morbid action, even though slight in amount, is necessary to call forth the nervous discharge. The morbid process may be centrally located and beyond the reach of investigation, or it may be peripherically located and exciting the convulsion in a purely reflex manner. It is fully conceded by all that injuries to nerves, diseases of the ear, intestinal worms, phimosis, uterine troubles, etc., are all not uncommon peripheral causes resulting in epileptic attacks.

The question has been raised, however, as to whether a convulsive attack due to a peripheral irritation can be regarded as a true epilepsy, and whether it is not to be regarded rather as of an hysterical character. Without attempting to pass judgment upon this subject, it will suffice to quote the recent views of a very competent authority upon nervous diseases, Prof. H. C. Wood. In commenting upon the convulsion due to a peripheral irritation, he says, "It is almost invariably epileptiform in its general symptoms, and may conform exactly to the typical epileptic attack;" and, while admitting that many of these reflex convulsions partake largely of the hysterical character, he further says, "There are, on the other hand, convulsions which conform to the epileptic type, and which are the result of an organic peripheral irritation."*

A remarkable feature of the epileptic convulsion is its periodicity. Now, it is proved beyond question that the higher nerve-centres of the brain act not only as inciting but also as inhibitory centres to those of a lower level. They are at once reservoirs of nerve-force and regulators of its dispensation. If, therefore, a morbid process at the periphery continuously attack, through nervous intermediation, these higher nerve-centres, it follows that these in time must have their resisting power overcome at intervals and at successively higher levels, until a final one is reached, when control is no longer possible. The unremitting irritation having at last overcome the resisting power of the highest nerve-centres, their energy is suddenly liberated and the organism is flooded with waves of uncontrollable centrifugal energy, until exhaustion brings about a temporary equilibrium.

The object of this paper is to direct the attention of physicians to a cause of epilepsy which has not hitherto been estimated at its full value, inasmuch as in none of the standard works upon neurology is the subject even alluded to,—viz., pathological states of the dental structures. That dental inflammations and disorders are more often provocative of epileptic seizures than is commonly supposed appears quite certain from the following cases, and also from the character of the cause and its effect. Many reasons might be given why dental disorders are peculiarly adapted to call forth this periodical discharge, and why these disorders are habitually overlooked by the physician, but they need not be detailed here. As exemplifying these phenomena, some interesting and instructive cases are adduced.

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* "Nervous Diseases and their Diagnosis."

The following case occurred in the clinical service of Dr. Wharton Sinkler at the Orthopædic Hospital and Infirmary for Nervous Diseases, to whose kindness I am indebted for the privilege of recording it :

CASE I.—Mary L., æt. 9, was brought to the hospital in October, 1886, with a history of epilepsy dating from May of the same year. The convulsive attacks first made their appearance on the afternoon of the same day that the child had had three teeth extracted on account of repeated attacks of toothache. One decayed tooth, however, was left remaining in the lower jaw. Previous to coming to the hospital the epileptic attacks occurred two and three times a week. From all that could be learned from the mother, the symptoms were those of a typical epilepsy. There was no neurotic history in the family. She was placed upon from 3 to 5 drops of the fluid extract of cannabis indica for two weeks, during which period she had twelve attacks. The bromide of sodium was then given for two weeks, during which period she had twelve attacks. The bromide of sodium was then given for two weeks, during which she had sixteen attacks. From November 1 to March 1, 1887, she continued taking the bromides alone, in combination, and, finally, in conjunction with the infusion of digitalis. During the four months of steady treatment she had forty seizures. About the 1st of March the mother made the remark that the child was always extremely restless at night that she would lie awake for hours complaining of toothache : and even when asleep she would continually grind her lower teeth against the upper teeth. Examination of the mouth revealed a carious and inflamed condition of a molar tooth in the lower jaw on the left side.

From the history of the case, and the possibility tha the irritation arising from the diseased tooth might be the exciting cause of the attack, it was concluded to have the tooth removed. This was done under the influence of nitrous oxide gas. The night following the child rested much better, and from that time forward her sleep became natural, her appetite improved, and her general health became decidedly better. From the last week in February until the present time (Decemer 19, 1887,) she has not had a single symptom of an epileptic attack.

That a dental irritation should be capable of exciting an epileptic condition does not appear at all strange when it is fully comprehended how numerous are the recorded cases of ocular, aural, visceral, muscular, and nervous disorders which have been caused by the irritation arising from the pathological conditions of the teeth and associated structures.

The interest aroused by the result of the preceding case led to an examination of medical literature for reports of similar cases. I find that no less than sixteen cases, entirely and immediately cured by the removal of an irritating tooth, have been recorded by different observers, and which are here arranged in chronological order. It is not supposed that this collection embraces all the recorded cases, but it is hoped that it will elicit references to many others, and, what is more important, the reporting of many new cases.

The injurious effects of diseased teeth, and the irritation arising from them, in the production of many general diseases did not escape the acute mind of Dr. Rush. In a paper published in his collected works,* he records the following:

Case II.—"Some time in the year 1801 I was consulted by the father of a young gentleman in Baltimore who had been afflicted with epilepsy. I inquired into the state of his teeth, and was informed that several of them in his upper jaw were much decayed. I directed them to be extracted, and advised him afterwards to lose a few ounces of blood at any time when he felt the premonitory symptoms of a recurrence of his fits. He followed my advice, in consequence of which I had lately the pleasure of hearing from his brother that he was perfectly cured."

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* Enquiries and Observations, vol. i. p, 199.

Dr. Ashburner published,* in 1834, a number of remarkable cases of hysteria, spasms, convulsions, etc., due to diseased conditions of the teeth. Among others was the following case of epilepsy:

CASE III.—A young lady of highly nervous temperament was attacked with epilepsy in the eighth month of her first pregnancy. She had two attacks before her labor, which was a very favorable one. Seven months afterwards the fits reappeared, and occurred two and three times a week. Various methods of treatment were resorted to without success. For a while the intervals between the attacks were somewhat longer, and for a while they appeared twice daily. An examination of the mouth revealed seven carious teeth, which were at once removed. Three wisdom teeth were prevented from erupting on account of a cartilaginous condition of the gums. These obstacles were removed. The epileptic fits at once ceased, and after several years they had not returned.

CASE IV.—Albrecht relates† the case of a boy, æt 12 years, who for a period of six months suffered daily with general convulsive attacks. Just preceding the attack there was severe pain in the temporal region. No cause could be assigned for the seizures. Treatment was without avail. Examination of the mouth revealed an overcrowded condition of the teeth, which were in addition unusually large. After removal of some of the teeth the convulsions subsided, and in a short time entirely disappeared.

CASE V.—Dr. Tomes publishes‡ the following: "A lad, a farm-laborer from Windsor, was admitted into the hospital for epilepsy. The usual remedies were tried for six weeks without effect. His mouth was then examined, and the molar teeth of the lower jaw were found to be much decayed, and of some of these only the fangs remained. He did not complain of pain in the diseased teeth or in the jaw. The decayed teeth were, however, removed, and the fangs of each were found to be enlarged and bulbous from exostosis. During the eighteen months that succeeded the removal of the diseased teeth he had not suffered from a single fit, though for many weeks previous to the operation he had two or three per day."

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* On Dentition and some Coincident Disorders, p. 98.

†Casper's Wochenschrift, 1837, p. 125.

‡System of Dental Surgery.

CASE VI.—Dr. Baly records‡ the history of a case of epilepsy from dental irritation, occurring in a man, æt. 45. The patient was an employé in the Millbank Penitentiary; was of good physique; in good health, and had never suffered from vertigo, headache, or any form of nervous trouble. In the latter part of October, 1850, he began to suffer from toothache. On November 4th the tooth was examined by the medical officer, but on account of its carious condition and deficient light it was not extracted. Nitric acid, however, was applied, which gave the patient relief. On the 6th the muscles of the right side of the face began to twitch. The muscular spasms lasted four or five minutes, and occurred three or four times a day. "At these times, when the twitchings had reached a certain degree of intensity, the jaw became locked, and he lost the power of speech; but he had no pain in the head, giddiness, or sense of stupor. The paroxysm of spasm in the muscles of the right side of the face and jaws occurred the next day, and on the following day, the fourth after the examination by Mr. Chatfield (the medical officer), the twitchings became more violent, and his jaw locked. He had the sensation of all his teeth falling out, and then lost consciousness. A strong convulsive fit ensued, which lasted half an hour; the same night he had a second fit." These attacks were described as presenting all the features of an epilepsy. A third attack occurred before morning. The next day the tooth was extracted, together with a small piece of bone attached to the root.

For one month the patient was perfectly well, but on the 7th of December, in the middle of the day, he again experienced the spasmodic twitchings, and at the same time became conscious of the existence of something protruding from his jaw; with his fingers he removed a piece of dead bone. In the evening of the same day the spasmodic contractions of the face occurred several times. On the night of December 8th he awoke with a spasm in the cheek, and upon getting out of bed fell upon the floor unconscious; a general convulsive fit followed, during which there was foaming from the nose and mouth. At 6 A. M. a second fit followed more violent than the first, and lasted five minutes. In the intervals of these attacks there was considerable uneasiness and confusion of mind. The next night he suffered a return of the fit. Examination of the mouth revealed a swollen and tumid condition of the gum, but there was no discernible source of irritation. The patient was placed on calomel to prevent further mischief to the deeper-lying structures around the diseased tooth-socket. He remained well until February 22d, when he had, for the space of ten minutes, the same premonitory twitchings in the muscles of the face, but no real fit. A small piece of dead bone was extracted from the gum, after which the old wound healed, and the patient entirely recovered.

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‡ London Med. Gazette, xlviii., pp. 534-540.

In 1857, Dr. Sieveking read* before the Royal Medical and Chirurgical Society a paper entitled "An Analysis of Fifty-two Cases of Epilepsy." In the discussion that ensued Sir Charles Locock said he had noticed the omission of the paper of a very common cause of epilepsy, viz., dentition. He could not agree with Dr. Ashburner that all cases of the disease could be cured by the removal of the teeth; but he had certainly seen the affection cured in more than one instance by removing overcrowded teeth.

CASE VII.—Dr. Ramskill publishes† the following: "A boy, 13 years old, has had frequent attacks of epilepsy for the last eighteen months. Latterly, his mother noticed that some days he rubs his left cheek, complaining of face-ache, after which the fit follows. On examining the mouth, there is to be seen a molar tooth considerably decayed, with a swollen gum around it and partly growing over into the cavity: it is not very tender to the touch, and the examination does not give rise to toothache. On questioning,

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* Lancet, June, 1857.

† Med. Times and Gazette, 1862, vol. ii., p. 216.

I find the sensation which the boy experiences before the fit does not seem to be one of pain, but rather of indefinite uneasiness. He always has a fit the night this comes on. Has never felt it during the day; it is always about seven or eight o'clock. I desired the mother to have the tooth extracted, and ordered a simple saline, with one-quarter grain of belladonna, to be taken twice daily. This was in June. The tooth was extracted next day. I saw this boy once a fortnight from that time for four months, but he had no recurrence of the fits. In this case I believe an unfelt aura commenced about the gum surrounding the tooth, and was not recognized till some degree of inflammation arose, and thus a modification of pain became associated with the aura and directed attention to it."

CASE VIII.—Trousseau relates* the case of a patient, a young notary's clerk, under the care of Dr. Foville, who had been subject to monthly attacks of epilepsy for several years. Many remedies had been tried in vain. Dr. Foville suggested the extraction of some carious teeth which ached constantly. The suggestion was acted upon, and from that day the fits disappeared.

CASE IX.—Dr. Garrett related the following case before the Suffolk District Medical Society, and was reported by Dr. Page:† "A man, aged 40 to 50 years, had suffered with his teeth for years; these had been extracted and artificial ones substituted. He became paralyzed in the muscles of his face and tongue. There was a peculiar drawing of the mouth, from which the aura epileptica came just previous to the fit; the tongue was inclined to fall back within the mouth; he was fearful of swallowing it. In investigating the case, Dr. Garrett removed the false teeth, and found the soldering discolored; he went back to his dentist, had a rubber plate made, and had no further attacks of epilepsy; the paralysis gradually subsided."

CASE X.—W. H. Waite reports‡ the case of a young woman, æt. 18, who consulted him for treatment for a carious condition of the incisor and canine teeth of the upper and lower jaws. The teeth had been diseased for four years, and were very sensitive. For three years the patient had been subject to epileptic attacks, which were at first quite slight, but had gradually increased in severity. After removal of the diseased teeth and filling of others, the epileptic fits entirely ceased. After some months the fits returned, attended with sharp, shooting pains in the alveolus. Examination showed that several other teeth had become decayed. These were removed, and from that time on there was no recurrence of the epilepsy, and the patient increased in health and weight.

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* Clinical Medicine, New Sydenham Soc., vol. i.

† Boston Med. and Surg. Journal, November 8, 1860.

‡ British Journal of Dental Science, 1863.

CASE XI.—Dr. Nathan Field reports* the case of a boy, about five years old, who was suddenly seized with an epileptic fit. In two weeks he had a second attack, which passed away after a few minutes. In the course of the next ten days it was estimated that the boy had a thousand convulsions, occurring every few minutes. No cause could be assigned. It was finally observed that before the appearance of the convulsion there was a twitching of the muscles of the left side of the face. Finally, after a severe convulsion, while the child was unconscious, he drew up his upper lip, when it was observed that the canine tooth had, instead of causing absorption of the deciduous tooth, pushed it outward through the alveolus, the gum, and into the lip. The tooth was removed, and in less than an hour the convulsions subsided and never appeared again.

CASE XII.—Mr. Canton related† the history of the following case: "A strong, healthy boy, æt. 19, who had become the subject of epileptic fits, applied to Mr. Canton for treatment. As the cause of the fits could not be ascertained, it occurred to him that they might be due to the eruption of a wisdom tooth. The gum was freely incised, and the crown of the tooth laid bare. From that time the fits never returned.

CASE XIII.—Mr. Henry Moon related‡ the following case: "The patient, a girl, æt. 21, was brought as an outpatient to Dr. Fagge at Guy's Hospital, and he, finding that her teeth were in a very bad state, sent her to Mr. Moon. She had suffered from fits since she was fourteen, and lately they had become so frequent as to reduce her almost to the condition of imbecility. On examining her mouth, a third molar was found in process of eruption ; this he lanced freely. Some carious teeth were extracted and others were filled. Treatment by the bromides of potassium was ordered at the same time. The result was that the fits entirely ceased from the day of her first visit to the hospital. The girl recovered her intellect, and although she was kept under observation for several months, she had no return of the fits."

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* Western Journal of Medicine, 1869.

† Proceedings Odontological Soc. of Great Britain, 1880.

‡ Proceedings Odontological Soc. of Great Britain, 1882.

CASE XIV.—Dr. Schwartzkopf reported† the following case in the *Deutsche Monatschrift für Zahnheilkunde*, 1866 : "A man, æt. 27, suffered severe pain in the right upper central incisor, which was carious, and consulted a dentist, who filled it. Soon after this a swelling appeared in the hard palate, where an opening formed. The patient was now easy, but the tooth continued loose and tender when touched. The fistula also remained patent and discharging. Ten days after the tooth was filled the patient had an epileptic attack, and these recurred at gradually shorter intervals until, at the end of eighteen months, they occurred several times a week. During this time the patient was treated with bromides, atropine, etc., but without results. The tooth was then extracted, the fistula healed, and the fits ceased, and, at the time of reporting, the patient had remained free from them for four years."

The two following cases are reported* by Dr. Liebert:

Case XV.—Emil S., æt. 25, in good health and no neurotic tendency, began to suffer with attacks of vertigo in February, 1883. These attacks lasted several minutes, after which the patient appeared perfectly well. On one occasion, however, the vertigo was so severe that he was compelled to sit down to keep from falling. On one occasion he lost consciousness. By April 25th the attacks had greatly increased in severity. On this day he had had such a severe epileptic attack that Dr. Liebert was called in. The patient had been lying upon the floor for fifteen minutes wholly unconscious and most of the muscles of the body in a state of tonic contraction ; the pupils were of medium width and insensible to light ; there was also a fresh wound of the tongue. After careful inquiry, it was learned that just previous to the attacks the patient experienced a peculiar tickling or crawling sensation in the tongue, an inability to speak words distinctly, and some involuntary movements of the tongue. Immediately after there followed the giddiness, the fall, unconsciousness, etc. Despite large doses of the bromides, the attacks increased in frequency and severity. Finally, in June, he began to suffer with toothache. Examination of the mouth revealed several carious teeth, one of which was very sensitive to percussion. This was extracted, and from that moment all peculiar sensations and motions of the tongue ceased, and there has not been in the past two years a single epileptic seizure. This patient had in four months several hundred attacks of vertigo and eighteen or twenty typical epileptic convulsions.

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† Journal British Dental Assoc., 1886.

* Deutsche Medizin. Wochenschrift, September, 1885.

CASE XVI.—Young man, æt. 35, cabinet-maker. Began having epileptic attacks on February 3, 1862, which came on almost daily with increasing severity. On March 5th he had twenty-three seizures. With the exception of a toothache he had never been sick. Repeated inquiries elicited the information that from December, 1861, the use of his tongue was for some seconds, or even minutes, frequently rendered difficult, and this fact was coupled with a certain feeling of illness or vertigo. In the attack of February 3, 1862, these symptoms were exceptionally severe, the tongue being drawn to the right side and executing spasmodic movements. Immediately thereafter he became unconscious and fell to the floor in convulsions. The tongue symptoms were usually premonitory of the frequent subsequent attacks. Owing to the fact that the aura appeared to be connected with the mouth, it was determined to seek for the cause in that locality. As he had had toothache occasionally, several carious teeth were removed. The patient at once declared that he felt an unwonted freedom from a former oppressive feeling, and that he believed he would have no more of the seizures. His conjecture was correct, for he remained free from them from that time forth. This patient had epileptoid vertigo for three or four months and severe epileptic attacks for thirty-eight days.

A CLINICAL LECTURE UPON CERTAIN TYPES OF HYSTERIA

By LANDON CARTER GRAY, M.D.,

PROFESSOR OF NERVOUS AND MENTAL DISEASES IN THE NEW YORK POLYCLINIC.

I PROPOSE, gentlemen, to read you the histories of a mother and daughter, which will, I think, be of considerable interest :

The mother is 51 years of age, born in Scotland. She came to this country a number of years ago, and shortly afterwards had some trouble with her husband, who refused to support her, whereupon she became greatly excited and threw herself from a ferry-boat, and would have been drowned, but for the assistance of the boat hands. A considerable period of time following this event is an entire blank to her, but it appears that she was insane and was sent to the Ward's Island Asylum. At the present time, when speaking of this terrible time of her life, the woman utterly breaks down, and sobs and cries as if her heart would break. She seems to have come honestly by her defective nervous system, for a maternal aunt has been speechless and bed-ridden for several years, and several other members of her family in bygone generations have been afflicted with nervous disorders, of which I can obtain no precise details. After these neurotic phenomena of the mother's life occurred the birth of the daughter, Gracie, whom you see here, and whose history I propose to read to you in a moment. You will therefore perceive that, honestly as the mother came by her neurotic tendencies, the daughter came still more honestly by hers. Last June the mother and the daughter took a great dislike to one another, for no visible reason except that which may be found in a condition of the nervous centres of both of them. Neither one can tell me why they took this dislike, and I have excellent reason to believe, from having observed them both carefully for some period of time, that neither is withholding anything from me. Shortly after taking the dislike to each other both became afflicted with a peculiar difficulty of speech, which in the daughter became so pronounced that the mother took her to a hospital. After leaving the child there, the mother cried almost continuously for a week or more, and went absolutely to bed for three days, and for about two weeks was totally unable to speak. The present condition of the mother is this, as you see:

She is as timid as a hare, trembles at a sound, and fairly shakes with her mental reflexes when a disagreeable thought occurs to her. As you perceive, she speaks in an almost inaudible whisper. When, however, I speak to her sharply and harshly (much more so in fact than I care to do), I can force her to raise her voice into what may be described as an undulating whine. Further persistence in speaking to her sharply and harshly will force her to speak with considerable distinctness and without the whine. She tells me that she has had slight contracture occasionally, during several years, of the left big toe. She has absolutely no paralysis of motion of any of the muscles, whether of the head and face and buccal cavities or of the trunk and extremities; nor has she any objective sensory disturbances, in the way of impairment of tact, muscular sense, pain, temperature sense, sense of locality; nor any affection of her special senses, such as sight, hearing, taste, smell. Examined with an ophthalmoscope, her retinæ are found to be perfectly normal, the veins only being somewhat over-dilated, a condition observed in so many normal individuals as to be of no significance whatever. Further than an occasional slight headache, such as nervous women are especially subject to, there is no history whatsoever of cephalalgia. Nor does the most careful examination in other points, into which it is not necessary to enter, demonstrate the presence of any organic disease of the central and peripheral nervous system. This mother is extremely emotional, cries in talking,

—indeed, seems to have scarcely any self-control. She tells me that she has always been so, and has had hysterical attacks all her life at the menstrual periods.

So much for the history of the mother. Now listen to that of the daughter. Bear in mind, however, that, as I have told you, this daughter was born after the mother broke down into a suicidal attempt and insanity.

The daughter Gracie, aged 14, tells me that about thirteen months ago she began to notice, while at school, that first one and then the other heel would stamp involuntarily, this persisting till last June, *i. e.*, up to the time when the daughter and her mother took a dislike to one another, and the former was sent to the hospital. She claims that she noticed, in October, that when she had touched the hands to anything dirty or sticky, and then endeavored to wipe them, the wiping motion would involuntarily persist for some little time, until, as the child explains, "I could get something to keep my mind off." After lasting some eight months, this phenomena suddenly ceased. In April last, patient fainted away in church, but thinks she had no convulsion. During the few days immediately ensuing upon this she had some slight tremor and convulsive movements of the hands, all which suddenly ceased upon her being made to go out of the house and take exercise. When she and her mother took the great dislike to each other, her speech became affected at the same time as did her mother's, and she became well-nigh incapable of speaking; but hospital treatment for a few weeks relieved this aphonia entirely. Patient was then perfectly well for three months. At the end of this time there was some fracas in the house where she lived, and immediately the tremor recommenced. In about six weeks after this the difficulty in speech began again. At the present time, as you see, the patient talks huskily, indistinctly, pronounces individual sounds well enough separately. You will notice, also, that the left corner of the mouth is slightly drawn up, as if there were a few fibrillary contractures there in the *levator labii superioris alæqæ nasi* muscles. When she shows her gums, you will notice that this left side is distinctly less contracted than the other, demonstrating a slight paresis of these same muscular fibres. When she protrudes the tongue it points very slightly to the left, but distinctly. Uvula points slightly to the right and the left arch of the soft palate is evidently paretic. I have had her under observation for several weeks in the hospital, and the nurse tells me that she staggers occasionally in walking, which, however, I have never been able to perceive. The pupils are large, somewhat over-sized, but reacting well to light and movements of accommodation.

Now, gentlemen, when I first heard the histories of these two unfortunate human beings, I said to myself at once that they were cases of hysteria. But let me warn you never to make a diagnosis of hysteria with a flippant mind. Just think of the many hundreds, perhaps thousands of years, during which physicians set down as hysterical the lightning and the stabbing pains of locomotor ataxia. Just think of the periods of time during which physicians must have set down as hysterical the early symptoms of general paralysis of the insane. Just exercise your imagination a few moments and range over the whole wide range of nervous and mental diseases, and pick out those which for hundreds of years were set down as hysterical. And those of you who have been five or ten years in practice yourselves, just recall to memory the cases that you must have seen of grave organic affections that had been set down by somebody as hysterical. Scarcely a month of my life goes by that I do not meet with some case of acute suffering that has been pronounced to be hysterical by some member of the profession ; and I have assisted at more than one autopsy where an aneurism, or a tumor, or a caries of bone has been found to be the cause of so-called hysterical symptoms. For these and similar reasons I never make a diagnosis of hysteria hastily. But this case has many elements about it that tempted me in that direction, and these elements were :

1. The frank history of hysteria throughout life, given me by the mother ;

2. The extremely emotional condition of both mother and

daughter, evidenced by the mother's weeping and crying in conversation, by the violent and unreasonable dislike that mother and daughter had conceived for one another, by the causation of attacks in mother and daughter by emotion, by the fact that both mother and daughter could be made to speak distinctly when sharply and harshly spoken to ;

3. By the characteristic one-sided curl of the lip, due to a fibrillary contracture, which is quite characteristic of certain cases of hysteria.

But I was not satisfied even with these facts ; for it is possible, as you will admit upon the mere mention of the fact, that a hysterical patient might have organic disease. There certainly was in the mother a downright insanity, of a somewhat violent type and lasting for some little time ; and the daughter bore a slight lingual and facial paresis. Both the maternal insanity and the filial paresis were quite consistent with the diagnosis of hysteria, it is true, but it is also true that they were quite consistent with a diagnosis of organic disease. So I separated the mother and the daughter at once, took the latter into the hospital, made the former live alone at home, and did not permit them to see one another for several weeks. Then I set a nurse to work to make careful observation of the daughter. So I come to you at the end of that time, able to say that the daughter presents no other symptoms than those I have shown you, and that these can be made to disappear by the discipline of a well-ordered hospital ward.

These two cases are rather extreme types of hysteria, and are not met with very frequently in our clinics, although they have been well enough described by the books. The treatment of them is very often a very puzzling one, and becomes well-nigh impossible when the means of the patient are moderate. These graver types of hysteria are closely allied to those of many of the bed-ridden women throughout the country, who furnish forth so many paragraphs for the Sunday newspapers when they have been made to get up from bed and walk by clairvoyants, mesmerists, faith cure, or that craziest craze of them all which goes by the name of "Christian Science" or some equally biblical and meaningless term. To treat all this class of patients with any possibility of success, it is absolutely necessary to have them taken away from their friends. It is impossible to make a sympathizing layman or laywoman understand what hysteria is; indeed, it is almost impossible for any one to understand it until they have seen its eccentric and often really insane manifestations. It is therefore utterly useless to expect the co-operation of friends or relatives in any treatment that calls for self-control on the part of the patient. But if they are taken away from their home and put under the charge of a nurse who is trained to obey orders like a soldier, then we can enter upon the therapeutic struggle with some chances of success. Do not flatter yourselves, however, that you will gain an easy victory. By no manner of means! On the contrary, you must expect to have your temper, your ingenuity, your nerves tested to a degree that cannot be surpassed even by the great surgical operations. I maintain that the man who has the nerve and the tact to conquer one of these grave cases of hysteria has the nerve and the tact that will make him equal to the great emergencies of life. Your patient must be taught, day by day, to do what she has never done before, *.i.e*, to make her cerebrum act upon her muscles in the way that it is perfectly capable of acting, if she will only make it act. In other words, she must be taught to exert her will, not by preaching or sermonizing, but by steady, resolute, iron-willed determination and tact—that combination which the French writers somewhat melodramatically call "the iron hand beneath the velvet glove." It is utterly impossible for me to give you more than general directions of this nature, for each individual case will require a different application of the same general principles. You must, however, disabuse your mind of the prejudice that most physicians have against making use of mental impressions as therapeutic agents. In treating this class of patients, you may be perfectly positive of failure unless you have moral courage enough to make impressions upon the auditory and the optic nerves as well as upon the pneumogastric. In other words, if you *will* cling to the old idea that the body must not be treated except through the involuntary system of nerves ; that it is dishonest to put medicines into the body except you lodge them within the gastro-intestinal tract or beneath the skin ; that the great palpitating nervous mass of the brain, with its immense optic and auditory antennæ, is to lie useless,—then you may throw up the case at once. Your whole treatment must be based upon mental impressions. Drugs will be of no use whatever, unless your patient is wasted in health and strength. Should there be any necessity for tonics and nourishment, then you should make some application of the treatment which has been made so well known to the world by Dr. Weir Mitchell, under the name of "Fat and Blood Making," and which consists, as you doubtless know, of putting the patient absolutely to bed for six or twelve weeks, gradually increasing their food from two or three ounces of milk every two or three hours to three full meals in the day, adding malt extract and iron, and using electricity and massage to overcome the ill effects muscularly of this enforced rest.

Reviews.

DE L'EPILEPSIE JACKSONIENNE. Memoire couronne par la Société de Médecine et de Chirurgie de Bordeau. Par le Dr. E. Rolland, Medecin des Asiles "John Bost" de Laforce (Dudogne).

JACKSONIAN EPILEPSY. By Dr. E. Rolland, Physician to the "John Bost" Asylum. Published by the Progrès Médecal, 1888, pp. 181, with preface and 24 illustrations.

Dr. Rolland's work is the first monograph to appear on the subject of Jacksonian epilepsy. Since Hughlings Jackson, in 1863, published his minute and interesting observations on the disease which now bears his name, several other close observers, especially Fournier, Franck and Pitres, and Luciani, have studied the subject from anatomical and pathological standpoints; but until the present work appeared, no one has attempted to collect the results obtained, to classify them, and to formulate the conclusions which must necessarily be deduced by the careful study of the large number of cases Dr. Rolland has collected.

As physician to the "John Bost" Asylum, where many epileptics are confined, Dr. Rolland has had exceptional facilities for studying epilepsy in all of its forms. The first chapter of the work is devoted to a brief summary of the anatomy and physiology of the cerebral convolutions, especially the so-called motor convolutions, and is accompanied by the usual illustrations seen in all text-books which treat of this subject. It is but a slight sketch, and is simply introduced to show that the well-known experiments on cerebral localization on the lower animals are fully in accord with the pathological developments which produce the symptoms classified under the title of Jacksonian epilepsy.

The symptomatology of the disease is clearly and concisely set forth in a number of minutely described cases, several of which came under the author's personal observation. The description of the attacks are models of what such observations should be.

Under the heading of Pathological Anatomy and Physiology, the author has tabulated one hundred and twelve cases, arranged into five groups depending upon the localization of the spasm. In all of these cases the pathological lesion was discovered. A study of these cases goes to show that, while in a large majority of them the lesion was located in the cortex on one or both sides of the fissure of Rolando, in a small number the lesion was found either in the central gray nuclei (two cases) or else in the white substance of the occipital, temporal, and tempero-sphenoidal lobes (five cases). The differential features between the usual and the unusual types are made apparent in the tables.

In regard to the treatment, the author has nothing new to suggest. The operative measures instituted by Horsley are commended as affording the best means of relief, except in syphilitic cases, where, of course, specific treatment is advocated.

Though the volume, as a whole, presents few new features, yet Rolland's work is to be commended for the thorough and exhaustive manner in which the subject is treated.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Meeting held February 8, 1888.

The President, C. L. Dana, in the Chair.

Dr. J. B. Emmerson presented a pile of

RECTANGULAR PRISMS IN A SINGLE FRAME.

The pile consisted of Nos. 2, 4, 6, 8, 10, 12, 14, 16, which were fixed, and two additional prisms, Nos. 1 and 16 which were movable. By subtracting, Nos. 3, 5, 7, etc. were obtained, and by adding, Nos. 18, 20, 22, etc. The apparatus was used in the same way as ordinary prisms, the advantage claimed being increased facility in rapidly changing one prism for another. The muscles could be tested in one-fourth the usual time.

Dr. Birdsall asked what was the price of the instrument.

Dr. Emmerson replied that Meyrowitz furnished it for $16. Dr. Norris' rectangular prisms cost $18; the ordinary, set $12.

Dr. Dana asked whether the instrument would test fractions of a degree, referring to the fact that Dr. Stevens reports errors in fractions.

Dr. Emmerson replied that he had never tried to test errors in fractions but that the subtraction of a ½ or ¼ prism would enable this to be done.

Dr. Leszynsky considered the arrangement very ingenious. The principle of subtraction and addition was that of the opthalmoscope. He however thought tests by prisms unreliable. By means of the prism it was possible to create the appearance of a condition which did not exist.

Dr. Ira Van Gieson presented specimens stained by Golgi's method. Blocks 1 to 2c. c. in dimensions were subjected to the action of a 2 per cent. solution of bichromate of potash, or Muller's fluid. The time for hardening varied with the temperature. A constant temperature of 20°c. to 25°c. was preferred. In the summer hardening was completed in from 15 or 20 to 40 or 50 days. In winter 5 to 15 weeks were required. In changing the bichromate solution its concentration was increased up to 5 per cent.

After hardening, the specimen was placed in a solution of nitrate of silver at an average of ¾ of one per cent in strength. Where the hardening was incomplete a 1 per cent. solution might be required. Where it was more complete a ½ per cent. might be sufficient. A precipitate of chromate of silver required that this solution should be frequently changed. The specimen was kept in this solution for from 24 to 30 hours. It was then washed in water, embedded, washed in alcohol and cleared with creosote and turpentine. The same result could be obtained by immersion in a corrosive sublimate solution of 1 per cent. strength, but months were required by this method. The speaker also thought it less certain than the one described. Both methods had been modified by Pal of Vienna. The modification required the immersion of the silver or sublimate sections in a 1 per cent. solution of sodium sulphide, which produced a more complete precipitation in the finer processes.

The object of the method was the staining of ganglion cells and processes, the matrix being left clear. There was sometimes a tendency for the silver to precipitate in the pericellular spaces and in the channels around the processes so as to make them appear larger than they ought to be. It was however the only method which had stained the ganglion cell. The speaker had been successful in specimens taken from the cortex. In those from the cerebellum and spinal cord he had been less successful. It was essential that the specimen should not overharden. It was necessary every day or two to sacrifice a piece and try it in the silver solution to determine whether the hardening was sufficient.

Literature in connection with the subject was cited as follows:

C. Golgi, "Recherches sur l'histologie des centres nerveux," Arch. Italiennes de Biologie, Vol. III.–IV, 1883.

C. Golgi, "Sulla fina anatomia degli organi centrali des systema nervoso," Milano, 1886.

Prof. Forel, "Einige hirnanatomische Betrachtungen und Ergabnisse" Arch. f. Psychiatrie, Bd. XVIII, No. 1.

Bleuler, Correspondenzblatt f. Schweitzer Aertze, March. 15, 1886.

J. Pal, "Ein Betrag zur Nervenfarbentechnik," Med. Jahrbuchder K. K. Gesellschaft, '86, No. 8.

C. Mondino, "Recherche macro e microscopische dei cerebri nervosi," Torino, 1886.

Marchi, Sulla strutura dei corpi striati e thalmi ottici," (Memoir dal instituto Lombardo di scienzi e lettere, 1887.

Tara Tufi, "Sulla A Sull'Anatomia della retina." Internat. Monatschrift f. Anat. u. Phys. Bd. IV., H. 10.

L. Petrone, "Sur la structure des nerfs cerebro rachidiens." Internat. Monatschrift, f. Anat. u. Phys., Bd. V, N. 1.

Dr. Dana also presented specimens stained by Golgi's method. He had not so far been so successful as Dr. Van Gieson, but in a few specimens he had got staining which showed one interesting peculiarity of the method, viz : that it at times stained cells of one class ; at others, those of another. In his sections the connective tissue cells only were stained, but these were shown in a striking manner. Obersteiner had stated that by the sublimate method only about $\frac{1}{10}$ ° of the cells were stained. The speaker thought that Golgi's method was a truly epoch-making one, comparable to that of Wiegert for the nerve fibres ; but naturally great care would be required in drawing conclusions from what was seen. He presented Marchi's monograph on the structure of the optic thalamus and corpus striatum in which it was shown that the cells of the former ganglion were of the motor type and those of the latter of the sensory.

Dr. Sachs asked whether any facts had been demonstrated by Golgi's method which had not been shown by others.

Dr. Van Gieson replied that we have no other method which will stain the ganglion cell. Golgi's method showed the axis cylinder as a branch of the cell. It showed also that the protoplasmic processes do not inosculate.

Dr. Birdsall objected to this inference. We had no means of knowing that even by this method the whole of the process was stained by Golgi's method, we could trace the process further than by other methods, but we could not say that finer subdivisions which the precipitate failed to indicate did not exist.

Dr. A. D. Rockwell read a paper upon

NEURASTHENIA AND LITHÆMIA.

A diagnosis of neurasthenia was often made where the stomach and liver was chiefly at fault. Neurasthenia on the other hand was often mistaken for lithæmia. Mental depression was common to both ; but irritability was peculiar to lithæmia ; the tongue was coated and the pulse slow rather than fast. Neurasthenia presented many symptoms, those of lithæmia were few and cases were much alike.

In treatment lithæmia responded to mineral waters and restricted diet ; while in neurasthenia relief from work, and anxiety and increased feeding were required.

Dr. BIRDSALL remarked that the term neurasthenia was used to cover a variety of nervous disorders not due to organic disease. The term lithæmia also was commonly used in a vague and incorrect manner. He thought it impossible to make a division between the clinical features of the two conditions. There were many intermediate cases which would disprove any classification. All were probably but parts of a vast group of nerve disturbances based upon faulty conditions of nutrition. Absence and rest had a direct effect upon digestion. The results of treatment thus presented no ground for the conclusions named. Heredity was a marked factor.

Dr. SACHS stated that for a long time he had been skeptical of the existence of a true set of lithæmic symptoms, but that he had found them in private practice and had followed up a number of cases. He agreed with the previous speaker in regarding neurasthenia but a nervous disturbance of which lithæmia might be the ætiological factor. Lithæmia had in his experience been almost limited to women of the wealthy class, suffering from an inactive life. Nervous symptoms were proportioned to the amount of uric acid in the urine. With increased exercise or prolonged massage the uric acid disappeared and the patient improved. Referring to the question of heredity he considered cases with hereditary lithæmic tendencies gouty, and when neurasthenic he classed them as cases of neurasthenia upon a gouty basis.

In treatment he agreed with the reader of the paper. For lithæmia he used cholagogue cathartics, physical exercise and massage.

Dr. BIRDSALL explained that he had referred to the inheritance of neurasthenia, not of the lithæmic symptoms.

Dr. LESZYNSKY had met with cases which merited the name lithæmic neurasthenia. He recalled a case in which nervous symptoms had alternated with gouty manifestations. He had watched the case for two months, during which no diagnosis but that of neurasthenia could be made. At the end of that time a true gouty attack had developed. While the gout lasted the nervous symptoms were absent, but these returned when the gout disappeared. But little satisfaction was obtained from examining the urine in these cases. It was impossible to separate two classes by its indications. Concentration was common in neurasthenia and other states.

The speaker added that the late Dr. McBride had been accustomed to say that all neurasthenia was due either to lithæmia or muscular anomalies.

Dr. Emmerson had seen neurasthenia cured by the correction of errors of refraction and muscular anomalies.

Dr. Dana favored the classification of neurasthenic symptoms into further clinical groups, the subdivisions to be based upon the ætiology, symtomatology and the chief seat of the disorder.

Aetiologically: there were the hereditary and the acquired forms. Among the acquired were to be placed the lithæmic.

Symptomatologically: there were the irritative and depressed forms.

Anatomically: there were the cerebral, gastric, sexual and others.

In classification here, as in insanity, the various factors above indicated would have to be used.

Dr. Rockwell explained that he had not represesented neurasthenia and lithæmia as separate types of disease. He had not said that there was a distinct dividing line between them. He agreed with the lithæmic origin of neurasthenia in some cases. There was also, however, a distinct difference in the behavior of certain cases toward food. In the purely nervous case an abundance of albuminous food was indicated; while in lithæmia, on the contrary, the quantity of such food was to be curtailed. Notwithstanding the existenie of intermediate cases there were others which were distinct requiring distinctive treatment.

Dr. Dana asked whether Dr. Rockwell limited albuminous food in lithæmia.

Dr. Rockwell replied that he did. He had indeed cured himself of excessive irritability with lithæmia by dispensing with meat and living mainly on a farinaceous diet.

Dr. Dana referred to the fact that Dr. Draper had in the Practitioners' Society, about two years ago, recommended a purely nitrogeneous diet in those cases. He had used the same in his own practice but latterly with some doubt.

Dr. Birdsall stated that he too had followed Dr. Draper's rule. In defective digestion, sugars and starches were many times at fault.

Fats too, might be the offending agent. It was not even known that uric acid was the poison in these cases.

Dr. Sachs in his treatment preferred nitrogeneous to albuminous food.

Dr. Dana himself was skeptical of the value of albuminous food in lithæmia. It had to be remembered, however, that there were cases in which apparently only nitrogeneous food could be digested.

Reading Notices.

The remarkable restorative powers of the Buffalo Lithia Water make it a remedy of great value in the treatment of many nervous disorders. In mental overwork, in nervous dyspepsia, and in impotence and sterility the benefits derived from the judicious use of this water have been most marked. Its freedom from any unpleasant taste is also a great point in its favor. The large number of testimonials from the most eminent physicians in the country shows the high estimation in which this water is universally held.

Fairchild Bros. & Foster have gained an enviable reputation in the manufacture of their Pure Digestive Ferments. Their Pepsin in Scales, Extractum Pancreatis, and Essence of Pepsin cannot be approached in purity and efficiency by any other preparation we know of. A long experience in their use satisfies us that they accomplish all that is claimed for them. The Essence of Pepsin is to be particularly commended on account of its agreeable taste and rapidity of action.

Mr. Lorenz Reich, who is noted all over the country as the largest importer of the highest grades of Hungarian wines, occupies beautifully furnished offices in "The Cambridge," No. 334 Fifth Avenue. "The Cambridge" was built by Mr. Reich, and is a monument to his enterprise and ability. It is a pleasure to review the treasury of good words written by prominent men and women from all over the world indorsing the excellence and purity of his wines. Mr. Reich possesses a book containing over five thousand autograph letters from the most eminent physicians and laymen, extolling the virtues of his Tokayer Ausbruch.

The Rubinat Co. have every reason to congratulate themselves on the great success of this comparatively new water. Introduced but a short time ago, it has rapidly taken its place in the front ranks of cathartic waters. The great objections to waters of this class are their unpleasant taste and their debilitating effects on the system. The Rubinat water is free from these objections, and is quick and efficient in its action.

The following letter speaks for itself. It was entirely unsolicited and is therefore the more valuable:

HEADQUARTERS, DEPARTMENT OF TEXAS,
MEDICAL DIRECTOR'S OFFICE,
SAN ANTONIO, TEXAS, February 13th, 1888.

THE J. P. BUSH MFG. CO:

Gentlemen—I feel that I ought to inform you, for your own satisfaction as well as for the benefit of a large class of invalids we have in the United States, who suffer from anæmia, nervous debility (neurasthenia), and green sickness (chlorosis), that I have used your *Bovinine* in these diseases with unmistakable advantage. These cases all have the more or less gastric irritation, dyspepsia, nausea, and distressing sensations of fullness and weight in the stomach after eating, and all these symptoms are quite promptly relieved or lessened by the *Bovinine*, taken soon after eating. But the most striking benefit I have noticed from its use is in cases where agonizing pain (gastralgia) follows soon after eating; in these cases the relief from pain is sometimes more prompt and complete than when chloroform, morphine, and other anodynes are employed, but without the disagreeable after effects of such medicines. I was greatly surprised at the anodyne effects of *Bovinine* on the stomach when I first noticed it; but the explanation of this action would seem to be that the *Bovinine* contains the elements that the stomach, in its moments of distress, needs.

Yours very respectfully,
ED. P. VOLLUM,
Lt.-Colonel and Surgeon U. S. Army,
Medical Director.

The number of remedies whose unpleasant taste can be effectually concealed when combined with Maltine, seems limitless. The Maltine Co., of 182 Fulton Street, New York, advertise a number of excellent combinations with Maltine. Of these, perhaps the most important of all, is the combination of Maltine with cod-liver oil. Although this preparation contains about fifty per cent. of pure oil, the Maltine thoroughly disguises it, so that it cannot be detected either by taste or by smell. This renders it of inestimable value for children and for adults whose delicate sensibilities prevents them from taking the ordinary preparation.

Notice of Awards.—The American Institute has awarded the Medal of Superiority to the Jerome Kidder Manufacturing Company, No. 820 Broadway, New York, for their 1887 exhibit of "Electro-Medical Apparatus and Appliances and Instruments." For fifteen years the Jerome Kidder Machines have received the highest awards from the American Institute over all competitors and wherever exhibited in competition.

Their large, elaborately finished Cabinet Battery, unique in design, attracted much attention.

The materials and workmanship of their batteries are not to be excelled, and the experience of physicians is decidedly in their favor. The "Tip Battery," a specialty of the firm, is so constructed that it can be instantly thrown into or out of action, and there is no trouble from the handling of the elements, or the dipping and inconvenience incident to the ordinary Faradic apparatus.

Among the new remedies that have met with marked success in the last two years, *Salol* stands pre-eminently in the lead. Its action has been found to be speedy and free from the objectionable effects of the salicylic salts. W. H. Schieffelin & Co., the agents for this drug in America, have recently issued a treatise on Salol which cannot fail to interest the physicians who desire to keep pace with the progress of the day. The treatise embraces new and important experiences of eminent practitioners on the use of Salol. This treatise will be sent to any physician who sends his name and address to W. H. Schieffelin & Co.

IMPERIAL COLLEGE OF AGRICULTURE,
Sapporo, Japan, March 9th, 1887.

Messrs. Reed & Carnrick.

Gentlemen :—Will you kindly send me, as soon as possible, by express via San Francisco, One 5 lb. Tin of "Carnrick's Soluble Food." Forward to the care of Lohmann & Co., Yokohama, Japan. Perhaps a word of explanation for this order from far away Japan may not be without interest to you. My baby boy is now two months old and extremely strong and healthy, and is gaining in weight steadily at the rate of half a pound per week. Three weeks ago, however, he weighed half a pound less than at birth.

Forty-eight hours after birth, having received no nourishment, he was allowed a few drops of cow's milk and all the tepid water he desired. But the milk did not agree with him, producing the only symptoms of colic he has ever shown. On the third day, there still being no milk from the natural source, he was given two meals of "Carnrick's Soluble Food," from a trial package in my possession.

This nourishment agreed with him perfectly, but was discontinued on arrival of the mother's milk.

When he was about four weeks old, he showed signs of serious indigestion, passing material from the bowels closely resembling hard curds, and which analysis proved to be almost wholly unchanged casein. The most natural course was to attempt to remedy the difficulty by changing the diet of the mother, but, after two weeks of unsuccessful experiment, recourse was again had to the "Carnrick's Food," followed by immediate disappearance of all digestive trouble. However, with a supply of only four ounces of the remedy within 8,000 miles, and with the mother burdened with milk, some other means, as a permanent course, had to be adopted. The analysis of the mother's milk furnished the clue to the proper course.

The nutritive ratio (relation of albuminoid to carbo-hydrate constituents) was found to be too low ; the amount of fat and milk sugar present was not sufficient to enable the infant to digest the excess of nitrogenous food furnished. By supplying this deficiency by feeding soluble carbo-hydrates, the proper nutritive ratio was restored ; and the mother's milk, *thus supplemented*, is to-day accomplishing all that could be desired, and all that was gained by the use of the "Carnrick's Food" *alone*.

With this experience to judge from, I am convinced that the "Carnrick's Food" is as perfect and efficacious in practice as its composition is correct in theory. It appears to me to be compounded on thoroughly scientific principles, and in this respect differs from most of the other articles placed on the market for similar uses.

Assured of the superiority of your product, and feeling deeply grateful for the results of its use by my own child, I deem it only just to communicate these facts to you, with my sincere thanks for the benefits derived from "Carnrick's Soluble Food."

Believe me, very truly yours,

H. E. STOCKBRIDGE, Ph. D.,

Prof. of Chemistry and Consulting Chemist to the Imperial Japanese Government.

PERISCOPE.

BY DRS. G. W. JACOBY, N. E. BRILL, AND LOUISE FISKE-BRYSON.

ANATOMY OF THE NERVOUS SYSTEM.

CONTRIBUTION TO THE STUDY OF CEREBRAL LOCALISATION. By Prof. E. Leyden. *Deutsche Medicinische Wochenschrift*, Nov. 24, 1887.

The author after giving an historical sketch of the discoveries of Hitzig and Fritsch, of the observations of Goll and Spurzheim, of the fundamental observations of Broca on the subject of speech localisation (aphasia), calls attention to the work of Munk, Goetz, Exner, and others.

He formulates all the clinical experiences on the subject of cortical localisation into three groups.

1. The first group he calls the aphasic disturbances.

2. The second group represents the localisation of sensory functions, principally of sight.

The most frequent visual disturbance accompanying central cortical disease is homonymous bilateral hemianopsia. Total blindness results if the lesion involve both occipital lobes. In addition to hemianopsia, there is also described a peculiar visual disturbance which has been called "soul-blindness" (seelenblindheit). The patient may be able to receive a sensory impression on the affected retinal field, but is unable to form a perception therefrom; "he sees, but he does not perceive." The lesion is considered by Nothnagel to overlie that which produces hemianopsia.

3. The third group comprises the motor cortical centres about the central gyri.

Under this head the author calls attention to the subject of cortical epilepsy, and reviews Hitzig's experiments and Jackson's observations.

There is nothing new in the entire article, which seems to have been written for the purpose of presenting some of the author's cases which fell under the second and third groups. N. E. B.

AN INDIAN'S BRAIN.—The College of Physicians of Vienna were greatly interested, at a recent session, by the examination of the brain of an Indian. They found the cerebrum inferior to the cerebellum in development, and in the former a tendency to the formation of a convolution in the frontal lobe, which is characteristic of the horse and the ox. —(*Medical News.*) L. F. B.

HETEROPY OF THE GRAY SUBSTANCE OF THE SPINAL CORD.—Kronthal, *Centralblatt f. Nervenheilkunde*, Jan. 15th, 1888.

A plumber, 22 years of age, who had often been treated for lead-poisoning, and who died with cerebral symptoms (hallucinations), had a diseased nervous apparatus which showed the following:

Muscles normal, the only deviation being an increase in the nuclei. Radial nerve showed degeneration and dissolution of the axis cylinder and the medullary sheaths; ulnar and median nerves normal. The spinal cord microscopically presented an uncommon appearance. Two parts had a fluid consistency; above and below these appeared a swelling of the cord, which gave the idea that two tumors were present. The microscopical examination showed a very peculiar heteropy of the gray substance.

Evidently there must have been two distinct pathological processes. The signs of an old process were the intense vascular lesions, increase of connective tissue; of a recent process, foci of softening. The ganglion cells were only a little more opaque than usual. At different places the cord presented abnormal clefts and cracks. N. E. B.

THE ORIGIN OF NERVOUS SYMPTOMS IN ANATOMICAL ALTERATIONS OF THE SEXUAL ORGANS.

Dr. Engelhardt has availed himself of the abundant material that the Freiburg University provides in its gynæcological clinic, to prove that pathological alterations in the female sexual organs as primary cause of manifold nervous disturbances, has frequently been greatly exaggerated. He arranges his cases into four groups. 1. Those cases in which no nervous symptoms appear, not even dysmenorrhœa, notwithstanding important pathological conditions in or of, the sexual organs. To this class especially belong a considerable number of large tumors. 2. Those cases, by no means rare, in which the sexual organs are perfectly healthy and normal, but in which marked signs of affections of the lumbar portion of the cord with or without other concomitant nervous symptoms are manifest. Such patients, without exception, have never conceived. In forty per cent. the nervous symptoms are ascribed to hereditary predisposition of the gravest kind. In eighty per cent. the symptoms began in the earliest infancy, sometimes originating in nerves arising in the lumbar portion of the cord or in other nerves. In many of these cases dysmenorrhœa not due to local disease can be traced to various extraneous nervous influences, as early and severe physical labor, combined with inadequate nourishment, mental strain, etc.

3. This class comprises cases in which besides symptoms of affections of the lumbar cord, pathological alterations of minor degree were present, such as cervical catarrh, relaxation of entire ligaments, slight displacements, anteflexion with posterior perimetritis, retroversion, etc. In all these cases the local alterations were not the cause, and could only have aggravated the nervous disorders, for the patients had always been delicate women, the majority scrofulous from infancy.

4. To this class belong those patients in whom symptoms of lumbar cord affections were coexistent with serious local disease of the internal sexual organs. But in these

also, though not quite so definitely as in the former group, irritability and debility of the nervous system could be traced to early infancy.

These numerous investigations and direct observations established a certainty of congenital predisposition to nervous troubles. At the first commencement of menstruation, dysmenorrhœa occurred, and chlorosis was only exceptionally absent. The most frequent complications were relaxation of the uterine ligaments, softness and flabbiness of the uterus, catarrh of the several apparatus, in consequence of defective nutrition. In some cases psychical influences, in others masturbation, imperfect coitus, etc., have been assigned as a cause. Occasionally local affections of the sexual system are the direct cause of nervous troubles, as well as other injurious influences, such as depraved nutrition, loss of blood, etc. Diseases of the sexual organs that last for years may and do lead to grave general nervous disturbances.—*London Medical Record*, Nov. 15, 1887.

L. F. B.

PHYSIOLOGY OF THE NERVOUS SYSTEM.

ON THE CONCEPTION OF HYSTERIA. (Weber den Begriff der Hysterie.) By P. J. Möbius (*Centralblatt für Nervenheilkunde, etc.*, February 1, 1888).

Dr. Möbius, in calling attention to the various conceptions of this affection by medical men, takes exception to the name as misleading, and asserts that physicians are beginning to form a more unanimous idea of the character of this affection.

The first step towards this is in the recognition of the fact that this disease is a psychosis, or rather that the essential, the primary change is a morbid condition of the *psyche*. But inasmuch as some cases occur (especially in males) in which no demonstrable disturbance of the psychical functions is present, the essential characteristic may be found in the somatic symptoms. He says: "All those changes of the body are hysterical which are produced by the imagination." He adds that every child knows that the imagination may cause bodily changes, and gives as examples, crying, laughing, blushing, the secretion of saliva, vomiting from disgust, sweating from dread, collapse from fear, paralysis of movement from fright, etc., etc. The hysterical state consists equally in the fact that just as these changes may be produced to a greater or less degree by this cause, so may the fancy call forth somatic disturbances, which are not noticed in the healthy, e. g., hemianæsthesia. In many cases the form of the somatic disturbance has no direct relation to the causative fancy. But the character of the latter may determine the former to that extent that the attention may be directed to definite regions of the body. Thus a slight injury to the shoulder may be the occasion of awakening a severe injury to the arm—an hysterical paralysis of the arm may be the result. Most probably such a relationship is of more frequent occurrence than would appear on first thought."

Experience teaches that hysterical phenomena are often produced and as often destroyed by conceptions, and especially by those emotional phases connected with such conceptions. The experience gained from hypnotism in general, and especially the results of the phenomena of suggestion by means of which nearly all hysterical symptoms may be called forth at pleasure are on that account of much scientific value, because they throw light upon the existence of hysteria.

He continues to state that one might think that the definition of hysteria did not sufficiently limit it in reference to the phenomena of a healthy individual. But such a limit is not necessary. Hysteria is indeed the diseased increase of a condition which is present in everybody. "Everybody is, so to speak, a little hysterical."

Hence, in its practical aspect, the only therapeutics which can be applied for its alleviation must be psychical.

N. E. B.

UPON A PECULIAR DEFORMITY OF THE BODY, CAUSED BY SCIATICA.—J. Babinsky, *Archives de Neurologie*, p. 1, 1888.

In this article five cases are published, with illustrations, which prove that in certain cases of sciatica a peculiar deformity of the body occurs, which until now seems to have escaped observation. It is not simply a coincidence, but an actual relationship of cause and effect. The position of these patients is striking, and differs decidedly from the deformities caused by other affections (coxalgia), and therefore may even serve as a diagnostic point.

Case I.—Male, æt. 37; since two years left sciatica. A year ago commencement of the deformity. No affection of the hip-joint. When standing, the following position is taken. The body is inclined to the right, the side opposite to the affected limb, so that the weight of the body is supported almost entirely by the sound leg; in addition to the lateral inclination, a slight flexion of the body forward and a certain degree of rotation of the body upon its vertical axis are observed, so that the right shoulder is thrown forward and the left backward. The spinal column presents two curvatures, the one in the lumbar region, with its concavity to the right; the other in the dorsal and its concavity to the left. The right leg is slightly flexed upon the thigh. This deformity persists in the horizontal position, and all attempts to rectify it cause great pain. The author considers the possibility of a coxitis, but after mature reasoning discards it. The other cases present variations dependent upon the location of the sciatica, whether right or left. The mode of production is analysed. To the question whether the deformity may disappear entirely, the author answers affirmatively. G. W. J.

PARAMYOLONUS MULTIPLEX AND SPASMOPHILIA. (Neber Myoclonic Convulsibilität), by Prof. Seeligmüller, *Deutsche Medicinische Wochenschrift*, Dec. 29, 1887.

The author adds two more cases to the relatively rapid increasing histories of this affection, making in all three which have come under his observation. The history of the first of the latter case is briefly as follows :

Carl Köhler, æt. 41, laborer, dates his present trouble from a fall on the left side in June. 1887. In addition to symptoms of pain, anæsthetic zones, paræsthesiæ, etc. which must be ascribed to the effects of the fall, other symptoms made their appearance. Some time after the fall movements in the neighborhood of the thumb, involving in succession the muscles of the forearm, and the shoulder of the right upper extremity and to a lesser degree those of the left arm. These spasms increased in intensity until they reached the present stage. A few days later (October) during the night severe convulsions of the entire body and convulsive pains in the left thigh and calf supervened, and did not recur. During these spasms the arm movements are said to have ceased.

The spasms continue without special pauses during the day and night, only when the patient after much fatigue and exhaustion gets to bed and falls asleep do his limbs involunrily extend themselves. He is often awakened by severe starts, and has very little rest. It is stated that it is during quiet repose that the movements are most severe, although the author was unable to obtain the increase on having the patient lie down.

His previous history discloses that he had similar attacks but of short duration in 1870 and 1877, occurring after an over-indulgence in beer and sleeping in the open air.

The history is minutely given and expresses every detail concerning the character of the movements.

The author takes exception to the view of the French writers who regard this affection as a general convulsive tic, even though in his first case the facial region was involved. In his other cases no part of the face was affected. In convulsive tic, which differs according to Marie-Guinon's observations from ordinary tic, psychic phenomena are present and assume a prominent clinical feature, whereas in Seeligmüller's cases such were absent. The author considers the disorder due to an enormously increased irritability of the large ganglion cells of the grey anterior cornua of the spinal cord.

N E B

THERAPEUTICS OF THE NERVOUS SYSTEM.

GALVANISM IN THE TREATMENT OF INSANITY.—In the Journal of Mental Science, Dr. Joseph Wigglesworth has an article on the use of galvanism in certain forms of insanity. These are his conclusions :

1. That while the use of galvanism to the head is a proceeding which is certainly *not* going to revolutionize the treatment of insanity, this agent is, nevertheless, one that is capable of doing much good in certain selected cases, and that by its judicious employment we may every now and then cure cases which would drift into hopeless chronicity.

2. The class of cases which offer the best field for the employment of this agent is that which includes examples of mental stupor and torpor—cases which are grouped under the specific designations of melancholia attonita and so-called acute dementia. L. F. B.

TASTELESS PREPARATIONS OF CASCARA SAGRADA (RHAMNUS PURSHIANA.)—Recent investigation of the constituents of Cascara sagrada has led to the discovery of new principles and facts of great importance pharmaceutically and therapeutically.

The chief objection to Cascara sagrada heretofore has been its inherent bitterness. In the light of recent researches, tasteless preparations of this drug highly efficacious medicinally are now to be had.

These discoveries mark a distinct advance in pharmaceutical attainment and in the therapeutics of chronic constipation, since this remedy can now be much more generally and persistently administered, and its well-known tonic laxative action obtained without the drawbacks which seemed formerly inseparable from its employment.

The facts disclosed concerning this remedy deserve more than a passing notice, especially since they indicate the existence of principles and modes of action extending far beyond the subject indicated, and are well worth the close attention of the thoughtful and scientific physician. A valuable contribution to the knowledge of the chemical constitution of this drug appeared in the *American Journal of Pharmacy*, for February, 1888, which makes it possible not only to obtain a true interpretation of the various clinical observations, but clears up apparent anomalies and also indicates the reasons for observed effects, which have lately been disputed, but now admit of no further question or misunderstanding.

Among the discoveries referred to in this valuable paper, of especial interest to the physician, is the influence of a class of vegetable ferments and their recognition as the causes of various abnormal conditions, such as colic, vomiting, nausea, diarrhœa and dysentery, which occasionally attend the administration of certain drugs.

It appears that Frangula bark when fresh, contains such a ferment in excessive quantities and is, therefore, unfit for use until the ferment has exhausted itself—the process usually occupying several years. It also appears that Cascara contains some of this principle and this fact will account for the occasional untoward effects of the drug, which have been observed as consequent on the employment of a number of its preparations heretofore in the market. These facts are, therefore, not due, as has been supposed, to any idiosyncrasy on the part of the patient, or to the laxative or tonic constituents of the bark itself, but to a distinct objectionable principle, which once recognized can be rendered inoperative and harmless.—*Am. Journal Pharmacy, Feb., 1888.*

PSEUDO-ANGINA PECTORIS.—Prof. Roberts Bartholow lectured upon this subject recently at the Jefferson Medical College Hospital, Philadelphia. The patient, not yet thirty, of well-marked neurotic constitution, without history of rheumatism, syphilis, or diphtheria, presented but one abnormal condition—paroxysms of sudden pain in or about the præcordial region, shooting in all directions over the chest, especially into the left neck and shoulder, sometimes downward into the left arm. Respiration is gasping and shallow, cyanosis, an agonized expression, some protrusion of the eyes, and a cold, clammy sweat quickly appear.

First there is inhibition of the heart's movements, then the action becomes rapid and feeble, and irregular in rhythm; a deadly faintness supervenes, and loss of consciousness for an instant. Of late there have been muscular twitchings, complete unconsciousness, and the seizures are preceded by uneasy sensations. These warnings come from the organs connected with the solar plexus. May they be called an aura? Trousseau describes such cases as "marked epiiepsy." In treatment it is important to recognize the true nature of these attacks. The best results are had from combining remedies addressed to the true seat of the malady and to the organs suffering the most severe functional disturbances. Thirty grains of bromide of sodium three times a day, and a one per cent. solution of nitro-glycerine, dose increasing from one minim until the characteristic action of the drug is manifested, are the remedies Dr. Bartholow ordered, and strict attention to diet as well, a small amount of fresh meat once a day, one egg at breakfast, one vegetable at dinner, some fresh fruit at supper, and the only drink a moderate cup of warm skimmed milk.—*Medical News*, Dec. 10th, 1887. L. F. B.

ELECTRICITY AND NEUROTHERAPY IN THE TREATMENT OF CANCER.

In a letter to the editor of the *Medical Register*, Dr. C. H. Hughes, of St. Louis, says:

"Were the Crown Prince my patient, I would give him bromide of ammonium, muriate of ammonium, the hypophosphite and arsenic, and local and general static electrization; and I would use the static current, the direct static current, to the part, powerful enough to arrest the morbid cell activity, and remove the growth by electro-cautery, if it did not disappear. *Cancer is a neurotrophic disease, and the cancerous diathesis is neuropathic.*"

So far as we know, Dr. Hughes is the first and only physician who has ever proposed to treat cancer with electricity and a scientific neurotherapy. Views similar to those just enunciated as regards the origin and cause of the disease, were uttered in the St. Louis Medical Society ten years ago; but to Dr. Hughes belongs the credit of having first suggested the electric treatment.

Elsewhere he says:

"Inquiry and observation show the frequent interchangeability of cancer with grave diseases of the nervous system in families in which the neuropathic diathesis prevails. It is no uncommon history for one branch or one member of a family to show insanity, epilepsy, or organic paralysis, and another to reveal the neuropathic taint in cancer. It belongs to a period of life at which the tonicity of the nervous system is on the wane in many organisms; when the trophic, nutritive and assimilative functions are more liable to depression than in more vigorous periods. It is often quite rapidly developed after sudden and profound shocks to the nervous system. Its well-known metastatic peculiarities, especially after excision, are like those in certain well-known nerve diathesis with developed local disease. Lastly, the only real benefit derivable from treatment aside from palliating escharotic, and cleansing local measures, comes from plans of treatment addressed to the restoration of general nerve tonicity and consequent resisting power.

"Even where excision has been resorted to, the after-care and treatment reveals the fact that successful results have been due to this rather than to the knife, whose triumph has been chiefly due to the removal of the local strain and waste, without the prolonged irritation of the painful caustic processes and the neuropsychical rebound following the local relief, the hope revived, and the successful neurotherapy instituted.

"Cancer is probably as amenable to treatment as any other diathetic condition, if we recognize it as such . . . but the hope of conquering it, lies in recognizing its neropathic relations, and in an early, persistent, vigorous, and confident effort to improve them. The law of resistance to cancerous invasion is in the conservation of energy."

L. F. B.

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Nervous and Mental Disease.

Original Articles.

PARANOIA—SYSTEMATIZED DELUSIONS AND MENTAL DEGENERATIONS:

AN HISTORICAL AND CRITICAL REVIEW.

By J. SÉGLAS,
ASSISTANT PHYSICIAN OF THE HOSPITAL OF BICÊTRE, PARIS.

Translated by WILLIAM NOYES, M.D.,
ASSISTANT PHYSICIAN OF THE BLOOMINGDALE ASYLUM, NEW YORK.

FOR several years frequent descriptions have been given in the foreign journals, especially German and Italian, of the forms of insanity designated by the names *Paranoia*, *Verrücktheit*, and *Wahnsinn*. Although all these forms have been studied in France, the question has not been viewed from the same point; then, too, there have been difficulties in the way of fixing the value of certain particular terms and the place the facts they serve to describe should hold in psychiatry. On the other hand, the study of mental degenerations being everywhere the order of the day, and these having a direct connection with the question of paranoia, it seems as if a review of the principal works published up to this time upon this subject would not be out of season, and possibly would be of some service.

I.—To give at once a definition of paranoia would be difficult, not to say impossible. In fact, it is perhaps the one word in psychiatry that has the most extensive, but most ill-defined acceptance. Moreover, the term by itself does not have a very precise definition, as may be seen from its etymology (*παρα*, near to, at the side, near, across; and *νοεω*, to think, to be wise). Different authors, too, interpreting it some in one way, some in another, give it the meaning of deviation of intelligence or of incomplete intelligence. This ambiguity will be to others an evidence in favor of the excellence of the term, indicating, on the whole, a qualitative and quantitative alteration of intelligence. Let us simply say, for the moment, that we find the term *paranoia* employed by authors synonymously with the German words *Verrücktheit* and *Wahnsinn;* the first, however, approaching more to the meaning of paranoia in the sense that we have given it (systematized delusion grafted on an incompletely developed intelligence), while the word *wahnsinn* simply designates the qualitative alteration of the mind. This will be made clear in the exposition that follows; in the meanwhile, let us simply say that the terms may be rendered by the expression *systematized insanity* or *systematized delusion*, which includes all the forms of paranoia described by the authors. A feeble mental constitution may accompany the delusion or not, and there may or may not have been a previous psycho-neurotic state (mania or melancholia).

II.—The term *paranoia* has been employed in psychiatry for many years. In fact, if we may credit Bucknill and Tuke, Vogel used the word in 1764, and under the name *paranoia* included nine classes of neuropathies, among them mania. This is as far removed as possible from the modern conception of paranoia.

In 1818 we find the term *paranoia* again employed by Heinroth,* but he had only a vague and indefinite idea of it. Nevertheless, under the name of *extasis paranoica*, he has described certain secondary states of mental exaltation with fixed delusional conceptions and an exaggerated sentiment of personality.

We again find these ideas in Esquirol,† who describes the intellectual monomanias, and reports several observations in which he insists especially upon the ideas of grandeur.

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* Heinroth, Lehrbuch der Störungen des Seelenlebens, 1818.

† Esquirol, Des maladies mentales, t. ii., 1838.

In Germany, Griesinger * (1845) describes systematized insanity (Die Verrücktheit, p. 382), and considers it as a disease always secondary to melancholia or to mania. He describes, also, the small number of fixed delusions which the patient is constantly repeating.

These ideas, which always relate to the personality of the individual and to his relations with the world, may be active and exalted (ideas of grandeur), or passive (ideas of persecution). Two forms of systematized insanity are thus distinguished.

Finally, Griesinger classes this form of insanity among the secondary states of intellectual enfeeblement, thus explaining the formation and permanence of the delusion. Together with this secondary form we find another form, *wahnsinn* (p. 357), a state of mental exaltation with fixed delusions of an ambitious character. We see, also, that this exalted monomania is completely confounded with the prodromal period of general paralysis and certain states of mental exaltation.

At this same time Ellinger † admits, by the side of this secondary form, a form of primary systematized insanity, but without describing it.

Up to this time, then, we have nothing sufficiently clear on systematized insanity ; and although the idea has been advanced, it has not been described accurately and particularly. Lasègue now fills this gap, and we may agree with Witkowski ‡ in saying that it is he who deserves the first place in the description of systematized insanity by his study of the delusion of persecution § (1852). We now find systematized insanity in the works of Morel. In his " Études cliniques " ‖ (1852) he gives some observations on the transformation, among the hereditarily insane, of hypochondriacal ideas into ideas of persecution and afterward of grandeur. In his "Traité des maladies mentales" * (1860) he adopts the words "systematized insanity" as a substitute for the monomania of Esquirol and separates these states from dementia, the reverse of Griesinger. His first two classes of hereditary insanity comprise almost the entire outline of that which has since been described under the name of primary systematized delusions. He describes the fixed ideas, the eccentricities, and the oddities of these hereditary subjects, insists on the very great frequency of systematization in these forms, upon the rapidity of the appearance and disappearance of the delusional ideas in certain cases, and upon the slow but continuous development in others—all this developing on a constitution of original weakness, or rather on one of unstable mental equilibrium. The same author also describes the fusion of the two forms, the expansive and the depressive, in certain states of systematized delusion, comparable to the hereditary states, the passage of hypochondria into the delusion of persecution (transformed hypochondria), and from that to the delusion of grandeur; and the incurability of this form through an evolution that is sometimes remitting but continuous, and which terminates in dementia. In short, we see that through these two masters French psychiatry first described primary systematized insanity. We may cite, again, in further confirmation of this, the works of Lelut and of Voisin upon sensorial insanity, which certain authors have since regarded as an acute form of paranoia (Westphal). Since that time but small advance has been made in France on this subject, at least during recent years; and if we say that the theory of paranoia had its birth in France, we must acknowledge that it has been developed abroad, and especially in Germany.

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* Griesinger, Traité des maladies mentales. Traduction française de Doumic, 1865. (See, in English, Robertson and Rutherford's translation, published by the New Sydenham Society, 1867, p. 303.)

† Ellinger, Allg. Zeit. f. Psych., anno II., 1845.

‡ Witkowski, Berliner klinische Wochensch., 1876.

§ Lasègue, Arch. de méd., 1852.

‖ Morel, Études cliniques, 1852, t. i., pp. 163 to 166 and 363 to 367.

In 1863 Kahlbaum † remained faithful to the doctrine of Griesinger on the secondary origin of systematized insanity, although admitting the possibility of a primary form.

Snell, ‡ in 1865, is the first to describe clearly a fundamental

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* Morel, Traité des maladies mentales, 1860.

† Kahlbaum, Gruppirung der psychischen Krankheiten, Dantzig, 1863.

‡ Snell, Ueber Monomanie als primäre Forme der Seelenstörung (Allg. Zeit. f. ch., 1865, B. xxii., p. 368).

form, distinct from mania and from melancholia, and characterized by the primary appearance of a series of particular delusional ideas of a mixed nature (persecution and grandeur) and accompanied by hallucinations (primary or true wahnsinn). The delusions are not, as in the other forms, an echo of the whole mental life, a tendency to generalization. The most striking symptom is a delusion of persecution, with an exaggeration of the sentiment of personality, and a tendency to activity rather than to passivity, differing in this from melancholia. There are also delusions of grandeur uniformly primary, contemporary, or consecutive to the delusion of persecution, and bringing about a change in the personality. The development of these forms of insanity is slow, but sometimes they develop quickly on account of the mental excitement. The prognosis is bad, yet in these cases a true consecutive dementia is never observed.

In 1867 Griesinger,* retracting the opinion that he had formerly professed, admitted, with Snell, the primary origin of the mixed states (delusions of persecution and of grandeur), and he described them under the name of *primäre verrücktheit.* He described, in addition, the hypochondriacal and erotic forms.

Sander,† pursuing the subject still further, studied in 1868 a special form of *primäre verrücktheit* which he called *originäre.* He showed the degenerative characters common also to the other primary forms, and the distinctive feature consisting in the congenital origin. The patients are born with hereditary predispositions which they manifest from infancy (anomalies of intelligence, of character, of sentiments, and of physical conformation).

Arrived at the period of puberty they follow two routes: Some, too poorly equipped, fall in the struggle for existence; they are seized with hallucinations and delusions, and rapidly fall into dementia. The others resist for a long time, and live in society, where they are conspicuous by their oddities and eccentricities. They are emotional, mistrustful, misanthropic, and often onanists. In these cases illusions and hallucinations develop unexpectedly. The morbid subjectivity to which these subjects are the prey, connecting everything with themselves, increases; it is the same with the other parts of their character, which appear to hypertrophy, and then they develop systematized ideas of persecution, of poisoning, etc., varying in color according to the education of the patient and the surroundings of his life. Along with this slow and gradual development Sander also notes the frequency of remissions and the slight tendency to dementia. As regards the etiology of these idiopathic (*originäre*) delusions, we must look for it most frequently in heredity and in the nervous and cerebral diseases of childhood, arresting the normal development of the brain. Pederasty and sexual perversions would be characteristic of the *originäre* form of systematized insanity.

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* Griesinger, Vortrag zum Eröffnung der psychiatrischen Klinik zu Berlin, Mai 2, 1867 (Arch. f. Psych., B. i., S. 148, 1867).

† Sander, Ueber eine specielle Form der primären Verrücktheit (Arch. f. Psych., 1868–1869, B. i., S. 387).

In 1873 Snell described, under the name of systematized insanity (*Wahnsinn*) consecutive to melancholia, to mania, or to epilepsy, a secondary or improper systematized insanity, since adopted by Hertz,* Ripping, and Nasse.

Samt,† in 1874, described the hallucinatory variety of the *originäre* form of Sander, dividing it into two subvarieties. The first, depressive hallucinatory, often breaks out after a very long period of incubation at the menopause in women. Here the hallucinations of hearing are of principal importance. The ideas of persecution that accompany them are only a syndrome and are not characteristic, being found in other forms of insanity. Samt considers that the fixed ideas have the same pathological basis as the hallucinations, and are not an attempt at explanation on the part of the patient. Hallucinations of other senses are found, but only rarely of vision; the delusion, in which there are no signs of intellectual enfeeblement, has a very slow course, with exacerbations and remissions; it is only exceptionally that there is delusion of grandeur.

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* Hertz, Allg. Zeitschr. f. Psych., B. xxxiv.

† Samt, Die naturwissenschaftliche Methode in der Psychiatrie, Berlin, 1874, S. 38, 42.

The second subvariety, exalted hallucinatory, is distinguished by the predominance of visual hallucinations, following generally a state of excitement. Auditory hallucinations are also observed, but are very vague. The delusion has an irregular course and is not accompanied by signs of intellectual enfeeblement. As an example, we have the religious delirium and the true delusion of grandeur.

Westphal,* in 1878, first described the acute form, and offered a classification of systematized insanity (*Verrücktheit*), which he divided into four groups: 1. The *hypochondriacal form*, already described by Morel, having a chronic course with typical remissions. The troubles of general sensibility form the substratum of the delusion of persecution, accompanied by illusions and hallucinations. 2. The *chronic form*, having a slow beginning and a remitting course. The hallucinations and the delusion of persecution appear first—sometimes one, sometimes the other—and are not preceded by an hypochondriacal stage. At the end of a certain time the ideas of grandeur come into prominence. 3. The *acute form* is characterized by the sudden explosion of hallucinations, especially of hearing, accompanied by ideas of grandeur. At the height of the disease the incoherence is such that it suggests a febrile delirium. In certain instances there are impulses; in others, on the contrary, there is complete dejection. Westphal also places in this group many cases of melancholia with stupor and the katatonia of Kahlbaum.† These deliriums progress rapidly or slowly to a recovery. 4. The last form is simply the *originäre* form of Sander, and is the only one in which Westphal admits a basis of degeneration.

While certain authors, as we have seen, hold to the terminology of Snell, others adopt that of Westphal; these are Leidesdorf, Koch, Jung, Schuele, and Merklin. Murh,‡ in 1876, reported the autopsy of a case of systematized insanity, not originäre, in which he found atrophy of the right hemisphere of the brain. Leidesdorf[*] (1878) returned to the theory of secondary systematized insanities; but he went to the point of exaggeration in admitting as primary states not only psycho-neurotic states, but even some infantile diseases and traumatism, which are only causes. Fristh[†] studies the connections of delusions with the emotional state. In mania and melancholia the idea is secondary, and precedes the emotional state; this would be the inverse in systematized insanity.

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* Westphal, Ueber die Verrücktheit (Allg. Zeit. f. Psych., B. xxxiv., S. 252, 1878).

† Kahlbaum, Die Catatonie, Berlin, 1874.

‡ Murh, Anatomische Befünde bei einem Falle von Verrücktheit (Arch. f. Psych., 1876, B. vi., S. 733).

Kahlbaum[‡] (1878), struck by the differences of intensity that distinguish the secondary systematized delusions from the primary, proposed, in order that they might not be confounded, to keep the name of *paranoia* for the primary cases, and to give to the others the old name of *verrücktheit.*

Schuele,[§] in 1878, described verrücktheit among the degenerative forms, and placed wahnsinn among the psycho-neuroses between mania and melancholia on one side and dementia on the other. We shall have occasion to review more in detail the ideas that this author has advanced on this subject in the last edition of his book.

Emminghaus[‖] (1878) shares in these views.

In this same year a work appeared by Feaux[¶] upon hallucinatory systematized insanity, corresponding to the acute form of Westphal.

Merklin[**] (1879) and Schaefer[††] adopted the classification of Westphal, and described in addition an hysterical form analogous in its course to hypochondria, but without its typical remissions; in these cases the delusion generally has an erotic coloring.

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[*] Leidesdorf, Causistiche Beiträge zur Frage der primären Verrücktheit, In psych. Studien, Wien, 1877.

[†] Fristh, Psych. Centralbl., 1878. See, also, Fristh, Zur Frage der primären Verrücktheit (Jahr. f. Psych., 1879).

[‡] Kahlbaum, Sammlung klinischer Vorträge, No. 126, Leipsic, 1878.

[§] Schuele, Handbuch der Geisterstörung, 1878.

[‖] Emminghaus, Allgem. Psychopath., 1878.

[¶] Feaux, Ueber die hallucinat. Wahnsinn, Inaugural Dissertation, Marburg, 1878.

[**] Merklin, Studien ueber die primäre Verrücktheit, Inaugural Dissertation, Dorpat, 1879.

[††] Schaefer, Ueber die Formen der Wahnsinn, etc. (Allg. Zeits. f. Psych., B. xxxvii., S. 55).

Krafft-Ebing,* in the first edition of his "Text-book" (1879), and again in the second edition, gives a description of paranoia. He places the primary form among the mental degenerations; to his mind it is a morbid form that can only develop in an affected brain, heredity being the most frequent cause; the foundation on which it rests is formed by the delusions, of which the primary origin is well shown by the absence of all emotional basis or of a process of reflection that might give place to delusions. The disease has a uniform character and is thoroughly constitutional. It does not tend to dementia, but most frequently leaves the logical apparatus and thought intact. Krafft-Ebing studies the psychical constitution of these patients, and shows that in fact the delusion that breaks out later is only the exaggeration of their character, so that often the gradual development prevents assigning a precise date for the beginning. The dominating symptom in this disease is the morbid subjectivity already pointed out by Sander, and the exaggeration of the sentiment of personality.

Krafft-Ebing distinguishes two kinds of paranoia: First, that with delirium of persecution, which he fully describes, with its three periods of hypochondria, of persecution, and of grandeur, and its subvariety the "quarrelling insanity" (*folie de la chicane*), where not only the life of the patient, but his interests are in jeopardy, and in which he behaves in a fixed manner, becoming the persecutor instead of the persecuted. It is here that the degenerative taints are most evident. The second form, the delusion of grandeur, is also studied in its two varieties, the religious and the erotic (erotomania). The occasional causes are oftenest puberty, the menopause, uterine and intestinal affections, febrile diseases, and onanism. In studying the fusion of paranoia with hypochondria, the author describes, as a subvariety of paranoia, hypochondria with delusion of persecution (secondary form), the paranoia of masturbators, always developing on a neurasthenic base, and in which ideas of persecution by electro-magnetism and hallucinations of smell are often observed.* As for the systematized delusions that are often found among hysterics, epileptics, and, especially, alcoholics, these are not special characteristic forms, but ought to be attributed to the primary neurosis, or to the intoxication. In particular, Krafft-Ebing places among the alcoholic insanities the delusion of persecution of insane drinkers described by Calmeil and Thomeuf,† and by Nasse.‡ The fixed ideas are separated from the primary paranoia, at the same time being classed beside them in the mental degenerations.

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* Krafft-Ebing, Lehrbuch der Psych., Stuttgart, 1879, B. ii. See, also, Lehrbuch der gerichtliche Psycho-Pathologie, Stuttgart, 1881.

As for secondary paranoia, the author simply regards it as one of the possible terminations of the psycho-neuroses § and as a secondary state of psychical enfeeblement ; the delusion is quiet, monotonous, and unvarying, differing thus from that of the primary forms ; the secondary insanities would be especially the result of melancholic states rather than of maniacal states.

Krafft-Ebing entirely denies the existence of the acute form, and under the name of *hallucinatory wahnsinn* unites the acute primary paranoia of Westphal,‖ the acute hallucinatory form of sensorial delirium of Meynert,¶ the hallucinatory mania of Mendel,** the delusional stupor of Newington, and the acute and subacute general dementia of Tilling.†† In these psycho-neurotic forms there is always, according to him, a clear systemization of the delirium, which cannot be as lasting, and is not accompanied by, the permanent alterations of personality that are the rule in paranoia.*

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* See, also, Krafft-Ebing, Ueber primäre Verrücktheit auf masturbatorischer Grundlage bei Männern. Irrenfreund xx.

† Calmeil and Thomeuf, Gazette des Hôpitaux, 1856.

‡ Nasse, Allg. Zeitsch. f. Psych., B. xxxiv., S. 167, 1878.

§ We have seen above that among these forms is classed the hypochondriacal paranoia that Krafft-Ebing regards as one of the possible terminations of severe forms of hypochondria, the other being dementia.

‖ Westphal, loc. cit.

¶ Meynert, Acute Formen des Wahnsinns (Jahrb. f. Psych., B. ii., 1881).

** Mendel, Die Manie, 1882, S. 55.

†† Tilling, Psych. Centralb., 1878, Nos. 4 and 5.

Koch † follows the opinion of Krafft-Ebing on this subject.

Scholz,‡ in the same year (1880), renews the distinction already made by Samt, and distinguishes two principal forms of the disease: 1, The *originäre* form of Sander, and, 2, the hallucinatory form. His theory is briefly as follows:§ The systematized delusions cannot be explained without taking into full account the unconscious psychical life. If, under physiological conditions, the sphere of the unconscious is the foundation on which the elementary psychical processes organize themselves, the final results of which enter afterward into the domain of consciousness, then in these diseases the mental representations ought to be the definite result of the unconscious activity of the brain, but with this difference,

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[*] A brief summary of Krafft-Ebing's classification may make the preceding considerations more clear. The principal lines of his classification are here given:

A.

MENTAL AFFECTIONS OF THE WELL-DEVELOPED BRAIN.

- I. Psycho-neuroses.
  - 1. Primary curable states.
    - a. Melancholia.
    - b. Mania.
  - 2. Secondary incurable states.
    - a. *Secondary paranoia.*
    - b. Dementia.
- II. Psychical degenerative states.
  - a. Constitutional affective insanity (folie raisonnante).
  - b. Moral insanity.
  - c. *Primary paranoia.*
  - d. Fixed ideas.
  - e. Neurotic insanities.
    - Epilepsy.
    - Hysteria.
    - Hypochondria.
- III. Brain diseases with predominating mental symptoms.
  - a. Dementia paralytica.
  - b. Cerebral syphilis.
  - c. Chronic alcoholism.
  - d. Senile dementia.
  - e. Acute delirium.

B.

ARRESTS OF DEVELOPMENT OF INTELLIGENCE.

Idiocy and cretinism.

[†] Koch, Irrenfreund, 1880, No. 8, and Beiträge zur Lehre von der primäre Verrücktheit (Allg. Zeitsch. f. Psych., xxxvi., S. 543).

[‡] Scholz, Ueber primäre Verrücktheit (Berliner klinisch. Wochenschrift, 1880).

[§] See Buccola, Riv. Sper. di Fren., 1882, S. 80.

that these activities come from molecular anomalies of the nerve-cells. The psychical apparatus is then guided by false premises, and if the logical apparatus functions regularly it is without doubt because it has not undergone profound anatomical modifications. In the acute form, on the contrary, unconsciousness has no direct relation; the genesis of the disease is due to morbid perceptions which, according to Scholz, do not develop in the cortical centres, but in the peripheral centres or conducting tracts. But the brain must always be one that is pathologically disposed to transform the first excitation into false perceptions.

Then, too, the hallucinatory delusion develops more frequently in convalescence from febrile affections. The delusion then appears as a sequel to the hallucination.

Meynert * (1881) also describes the acute (hallucinatory) form of systematized delusion (wahnsinn).

Max Buch† (1881) reports a case of primary systematized insanity occurring in a young man who was an epileptic, and presented hereditary antecedents.

The work of Kandinski ‡ and the book of Weiss § (1881) give us nothing new on this subject.

Gnauck ‖ describes a form of epileptic paranoia which he separates from epilepsy by overlooking its pathogenic character.

Moeli ¶ (1881) describes some cases of systematized insanity developed after febrile diseases, the puerperal state, and alcoholic abuse.

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* Meynert, Die acuten (hallucinatorischen) Formen des Wahnsinns und ihr Verlauf (Jahr. f. Psych., B. ii., 1881).

† Max Buch, Ein Fall von acuter primärer Verrücktheit (Archiv f. Psych., 1881, B. xi., S. 465). In addition, this case was characterized by ideas of persecution, doubling of personality, hallucinations of sight and hearing, especially on the left side, due to an otitis media with perforation of the tympanum dating from infancy. The author makes this otitis responsible for everything, having observed an amelioration after local treatment.

‡ Kandinski, Arch. f. Psych., B. xi., 1881.

§ Weiss, Compend. der Psych., Vienne, 1881, Cap. iv.; Die Verrücktheit.

‖ Gnauck, Arch. f. Psych., B. xii., 1882, S. 337.

¶ Moeli, Falle von Verrücktheit, in Charitie's Annalen, vii., 1882.

Jung* (1882) gives the differential diagnosis between systematized delusions and primary affective forms (mania and melancholia), reasserting the views of Fristh (see p. 6). He claims that there has been an increase of paranoia of late years, and this transformation of the forms of insanity is due, in his mind, to the somatic and psychical degeneration of the human race that is going on from day to day.

Rauch † (1883) does not advance any new ideas on the subject.

Tuczek,‡ studying hypochondria, says it is not a true disease, but a symptom of melancholia or of systematized insanity. He differs from Krafft-Ebing in holding that the so-called hypochondriacal melancholia does not change into systematized insanity and that hypochondriacal systematized insanity does not tend to dementia.

Sakaki § (1883) describes the brain of a case of chronic systematized insanity with hallucinations.

Kroepelin ‖ (1883) distinguishes *primäre verrücktheit*, without a condition of mental debility and comprising the delusion of persecution, the delusion of grandeur, and the erotic and religious delusions ; then, 2, *secundäre verrücktheit*, grafted on a basis of degeneration, and developing rather after states of depression than after maniacal states. Like Koch, Pelman, and Krafft-Ebing, he emphasizes the monotonous and unvarying character of the secondary form, at the same time admitting some cases that have an acute course, recovering in some weeks or months. He describes in full the delusion of the litigationists, and regards this as a manifestation of moral insanity due to a lack of the hallucinations that are customary in systematized delusions, and due, also, to the absence of ideas of objective right and of the identification of personal interests with the general good.

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* Jung, Allg. Zeitsch. f. Psych., B. xxxviii., S. 361, 1882.

† Rauch, Die primord. Verrück., Leipsic, 1883.

‡ Tuczek, Allg. Zeitsch. f. Psych., B. xxxix., 1883, Annual Congress of the Society of German Alienists, Session at Eisenach, 1882.

§ Sakaki, Gehirn in chronischen Verrücktheit (Allg. Zeitsch. f. Psych., B. xl., 1883). The author found in this case an alteration of the pericellular spaces of the cortex, especially on the superficies of the convolutions and the presence of a flocculent yellowish substance; analogous to that already described by Mendel in general paralytics; the predominating seat of these lesions being the point of the tempero-sphenoidal lobule, the island and the ascending convolutions.

‖ Kroepelin, Compend. der Psych., Leipsic, 1883.

Arndt * places paranoia among the atypical psychoses or states of mental enfeeblement, and recognizes two forms: 1st, one, secondary, classed among the secondary atypical psychoses; 2d, the other, primary, classed among the primary atypical psychoses, together with idiocy, imbecility, and cretinism. This last form (idiopathic (*originelle*) primary paranoia) comprises, *α*, moral insanity; *β*, partial paranoia (rudimentary, or fixed ideas, which he first describes, and the delusion of persecution); *γ*, complete paranoia, which is the generalization of the preceding.

Mendel † (1883) gives a complete classification of paranoia, a term that he adopts definitely. He insists, especially, on primary paranoia, which he divides into simple and hallucinatory, each of which may be either acute or chronic. The acute form of simple primary paranoia generally comes on without prodromes, and is characterized by a delusion of vague persecutions without persecutors. The chronic form may be divided into three periods. The beginning of the first is difficult to indicate precisely, and often reaches back to youth; it is characterized especially by hypochondriacal tendencies. Then, in the second period, a delusion of persecution appears, developing slowly, with a sequel of delusion of grandeur; this delusion especially characterizes the third stage, which may terminate in dementia. As varieties of this form Mendel describes idiopathic (*originäre*) paranoia, always hereditary and degenerative, and the delusion of the quarrelling insanity, a weakened form of the delusion of persecution, and where the degenerative basis is very questionable.

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* Arndt, Lehrbuch der Psychiatrie, etc., Vienna, 1883. The principal features of his classification are as follows:

Psychoses.
- I. Typical.
  - *a.* Legitimate.
  - *b.* Circular.
  - *c.* Periodic.
  - *d.* Progressive paralysis.
- II. Atypical, or states of mental enfeeblement.
  - *a.* Secondary (to the typical forms).
    - 1\. Complete or dementia, *ανοια*.
    - 2\. Incomplete, *παρανοια*.
  - *b.* Primary.
    - 1\. Idiocy, cretinism, and imbecility.
    - 2\. Original primary paranoia.
      - α. Moral insanity.
      - β. Partial *paranoia* (rudimentary and delirium of persecution).
      - γ. Complete *paranoia*.

† Mendel, Eulenberg's Encyclopædia, November, 1883.

(To be continued.)

GLIOMA OF THE MEDULLA OBLONGATA.

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ROBERT B——, aged 32, laborer, colored, was admitted to the Philadelphia Hospital on the 4th of March, 1887. He was a well-nourished, muscular man, and gave a very good account of his condition and history. He knew little or nothing of his family; had lost his mother and all his brothers and sisters. He had a chancre two years ago, with secondary symptoms. With the exception of a very severe attack of headache with dizziness in 1885 he has been well until six or eight weeks ago, when he began to have fits, for which he was admitted to the Pennsylvania Hospital, where he remained a week. At first he had only one or two attacks a week; now they recur more frequently, and he has had three in the past six days; he begins also to feel a little uncertain on his feet.

Condition on the 6th, when first seen, was as follows: Is intelligent and answers questions promptly. Complains of headache, unsteadiness in walking, odd sensations over his body, and fits. There is no wasting, no paralysis. The grasp of the hands is fairly strong; muscular power of legs unimpaired. He complains of great stiffness and pain in the muscles of the back of the neck, and on getting up he carries the head and back stiffly, but turns the head easily from side to side. He walks without assistance, but says he feels "drunk," and he tends to sway. He paced the ward alone, and with the aid of an assistant's arm went to the ophthalmoscope room, fully 100 feet off. The co-ordination in hands is impaired, he does not grasp objects quickly, nor can he rapidly touch the tip of his nose. He gets out of, and returns to bed with great deliberation, like a man with lumbago. Sensation is everywhere retained; feels a pin-prick rapidly. Complains of numbness, tingling, and creeping feelings in the hands and feet. Says his legs "feel as if something had laid upon them and put them to sleep." Has also sensations of cold in hands and feet, and, to use his own words, "they are warm, but they feel so cold." This was a very frequent complaint. Sensation in region of fifth nerve normal. Special senses unimpaired; he hears the watch well at either ear; no affection of taste or smell. Vision good. The eye-grounds were examined twice; no neuritis; veins looked full, but there were no special changes. The headache was not constant, was chiefly occipital, and he did not seem clearly to be able to separate it from the painful feelings of stiffness in the nape of the neck.

Reflexes are present; patellar somewhat exaggerated. In the fits the movements are bilateral; he froths at the mouth; says he does not lose consciousness. This is probably a mistake. He fell out of bed last night in one and knocked his head. They last from five to fifteen minutes, and he comes out of them, as a rule, with the mind clear.

The appetite is good; he vomits sometimes; bowels regular. There is a loud apex systolic murmur, transmitted to axilla, and the pulmonary second sound is accentuated. Pulse fair in volume, 90 per minute. Urine clear; no albumen.

Taking into consideration the fact that he had had a chancre two years ago, the lesion was thought to be syphilitic, and he was given large doses of potassium iodide.

On the 7th and 8th he was better, but the pain in the back of the neck was severe. On the 9th the tingling and numbness of hands and feet were not so distressing, and he had less headache. Had a severe convulsion last night. There is increasing difficulty in getting in and out of bed. Pupils are dilated to-day. He talks clearly and says he is improving.

On the 10th, at 12 o'clock, he was given a dose of the iodide and immediately had a sort of fit, but he did not move the hands. At 12.45 I came into the ward and found him in the following condition: Is unconscious. Respirations very slow, three, four, and five in the minute. Inspiration is prolonged and quiet; expiration short and noisy. Pulse, 100–108, fair in volume. At 12.55 the respirations had fallen to two in the minute, and pulse stopped somewhat suddenly. No heart-beat or heart-sound could be detected after 12.55. Last inspiration at 1 o'clock.

AUTOPSY.—Twenty-four hours *post mortem*. Old scars on forehead and arms. Cálvaria normal, perhaps a little thick in the frontal region. Much blood escaped on removal of brain. Dura is adherent, sinus very full—on either side there is a line of fresh-looking pachymeningitis. Arachnoid is clear at base. Veins of pia dilated and full. Parts at base present following condition: Olfactory and optic nerves small, but have normal color. No effusion in interpeduncular space. Anterior margin of pons is very close to optic commissure. Vessels of circle of Willis contain blood; they are not atheromatous. The third, fourth, and fifth nerves look normal, and those emerging from the lateral part of medulla have a natural appearance. The crura were cut, and cerebrum removed separately. Vessels on the cortex very full; gray matter of pink-red color. White matter, in section, looks moist and glistening; no foci of disease. The ventricles contain a slight excess of fluid; lining membrane normal. Crura show no change. Pons normal. The fourth ventricle is dilated, particularly in the lateral recesses. The Fallopian aqueduct not enlarged. The floor of the ventricle looks normal above the level of the acoustic striæ, the right of which are not so distinct as the left. A large vein curls over the left margin of the medulla at the level of the left striæ.

The lower part of medulla and beginning of the cord are occupied by a large growth extending from below the calamus, projecting more on the left than on the right side. It is everywhere covered by pia. On the left side it has a reddish-brown vascular appearance; on the right side the white substance of the medulla is apparent. No trace to be seen of restiform bodies or of posterior pyramids. The olivary bodies are visible, but wider apart than normal, and the lower parts absorbed. The growth reaches to within 7 or 8 millimetres of the fissure separating the medulla and pons.

The cerebellum is a little compressed just above the tumor.

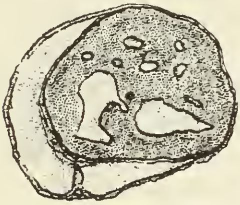

FIG. 1.—Section through the Tumor below level of Calamus. Natural size.

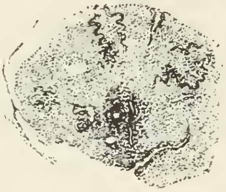

FIG. 2.—Section through the Olivary Bodies and uppermost portion of the Tumor.

No other changes. The upper part of cervical cord is soft and the postero-lateral columns have a very translucent aspect. The central canal is somewhat dilated. A cross-section just below the calamus has the appearance represented in Fig. 1 (actual size). The tumor is an inch in breadth by three-fourths of an inch in antero-posterior diameter. In fully one-half of the circumference it is in contact with the pia mater

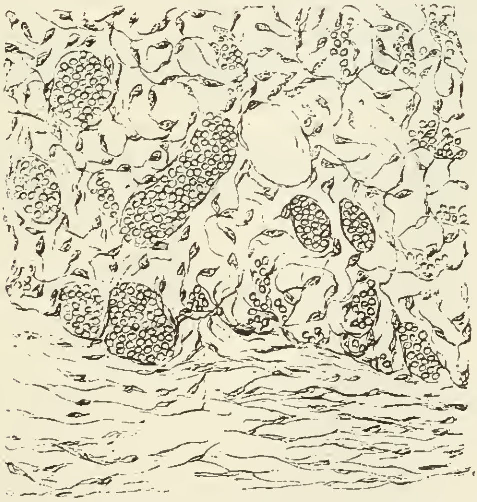

FIG. 3.—Section at the edge of the Growth, showing the gliomatous tissue and distended Blood-vessels Nos. 7 and 3.

of the left side ; in the rest of the extent, with the compressed and flattened columns of the cord. In the medulla it does not reach above the middle of the olivary bodies ; Fig. 2 represents the section at this level. The tumor was firm, of a red-brown color, with recent hemorrhages into its substance. The large lacunæ represented in Fig. 1 were filled with clots. Histologically, as shown in Fig. 3, the tumor is composed of a stroma of nucleated fibre-cells supporting blood-vessels which in places are so closely set that the appearance is that of an angioma. In other regions the gliomatous tissue is more dense and the blood-spaces less numerous.

The situation of the tumor, pushing aside and compressing chiefly the posterior columns, explains the disturbances of sensation and the inco-ordination which were the prominent features of the case. It is probable that the central hemorrhages, which looked recent, caused death by increasing the pressure and disturbing the respiratory and cardiac centres which lay just above the growth.

Gliomata of the medulla are rare. Sokoloff has recently described a case,* and has collected seven instances from the literature.

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* Deutsches Archiv. f. klin. Medicin, B. xli., H. 5, 1887.

HYGIENE OF REFLEX ACTION.*

By HENRY LING TAYLOR, M.D.,

NEW YORK.

MAN is the only creature that can live anywhere in the world. He can make shift to get along on an island, a mountain, or a tree-top; in a cell, a desert, or a city. He will live an average lifetime on rice, blubber, or clay, and will submit to the most diverse conditions imposed by nature or himself. His pre-eminence in this respect is due to the differentiation of the functions of his nerve-centres, and the capacity they have acquired for storing up energy, which can be drawn upon to meet the vicissitudes of his changing environment. The more varied and thorough the training and experience of his nerve-centres, the better will he be equipped. In every community we see a natural elimination going on of individuals who imperfectly adapt themselves to the conditions of their lives, and it is with those subjects who imperfectly react, whether temporarily or habitually, to the stimuli which reach their nerve-centres, that the physician has to do.

Without stopping to describe the special mechanisms by which these adaptations are accomplished, and confining this discussion mainly to the functions of those cerebro-spinal centres immediately concerned in the associated reflex movements of the trunk and limbs, let us consider in a general way the effect upon the individual of their various states and reactions, especially as observed in orthopædic practice.

By countless filaments connected with receptive surfaces and spaces, showers of impressions are constantly pouring in upon the centres, some leading to no visible effect, yet not without effect, others going to set up changes which are directly reflected to the muscles and glands to spur them to action, or passed from cell to cell, and all ultimately modifying in some degree the function of the remotest cell in the body. By far the larger portion of these in-streaming stimuli are never consciously perceived, but help to form that substratum of unconscious life upon which the fabric of our being rests.

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* Read at the meeting of the Neurological Section of the New York Academy of Medicine, held March 9, 1888.

There is, therefore, a continuous, unperceived alimentation and training of the nerve-cells of the cerebro-spinal axis, from the absorption of incoming stimuli and, in many cases, their reflection along various paths ; but, important as are the impressions received through the special sense-organs, particularly for consciousness, it is probable that the innumerable afferent impulses from skin, membrane, muscle, gland, and other tissues, responsive to changes in temperature and contact of the atmosphere and the blood, and changes in tension and position of the muscles and members, are equally fundamental and important.

A centre may have its activity re-enforced or interfered with by impulses from connected centres, and its function at any moment is the resultant of its own activity and all the re-enforcing and checking influences which reach it. Certain cells become specialized by inheritance or training, so that they store up the effect of impressions, giving out their impulses when properly stimulated. There is thus a selection of reflex arcs and paths in the evolution of the organism, so that, starting with but few and imperfect ones, many new ones are added according to its experience and necessities.

Reflex actions, as we observe them, are the products of the activity of many end-organs, fibres, and cells ; the latter connected with neighboring cells of similar grade and distant cells of higher function, and through their influence reacting to an ever-changing environment. The co-ordination of complicated movements, to a small degree innate, is for the most part painfully wrought out by innumerable repetitions, failures, and modifications, until our reflexes become what they are. A lady who has preserved the first little shoes of all her children tells me they exhibit evidence of the most varied use, one pair being worn at the toes, another at the heels, and others at the sides.

This acquirement of definite reflexes, *i.e.*, habits, is in the interest of an economy of force, and, once the needful associations are formed, volition is left free to initiate or control movements instead of laboriously executing them. "The digital struggle and facial contortion" of the youthful penman indicate the large number of nerve-cells necessarily involved in such an operation at first. Constant repetition and practice enable a much smaller group to do the work infinitely better, and at a fraction of the cost in protoplasmic wear. Talking, singing, playing on instruments, the use of tools, etc., and most of our oft-repeated actions, undergo similar improvement, to the economy of the organism and especially of the master-cells in the brain.

The reactions of the cord-centres will thus be seen to depend upon the nature and intensity of the stimuli received, so that we have a means of reaching and treating the nerve-centres by stimuli properly applied. When these are sufficiently varied, without being too numerous or too violent, the correlated peripheral and central areas readily act and react, each arc and group of arcs working out its experience and adjustment, until, in the same manner as a man's face reflects his character and experience, his hand or his back will acquire a form and expression largely dependent upon its neural life-history. Similarly, his carriage and attitude and the grace and dexterity of associate movements will depend upon previous training of the reflexes. We recognize a sailor or a case of Pott's disease at once from the gait, *i.e.*, from reflexes conditioned by special experiences.

The neural, and, to a great extent, the general vigor will depend upon the tonic action of large numbers of adequate adjustments and reactions well distributed over the different regions of the body. Unfortunately the environment supplied by our modern city life narrows the experiences of important regions of the body while it unduly quickens many purely cerebral processes, so that our wits are taxed more than our muscles. Shoulders, chest, back, and loins, and with them the thoracic and abdominal viscera, suffer particularly from our restricted and feeble muscular experiences, which lowers the tone of the centres, as in Langley's experiment on the frog, where "the ordinary reflex action produced by the stimulation of one sciatic is diminished by section of the other sciatic" (Foster, p. 605). Many persons, in being shielded from the necessity of any sufficient variety or vigor of purposeful and useful reflexes, either never emerge from, or lapse into, a state of spinal torpor, where the logical tendency would be toward a purely vegetative existence and the shedding of the appendages, as has already happened in some of the crustacea. Such conditions of torpor from "starvation" of the spinal nerve-centres may exist with considerable cerebral activity of one kind or another—overstimulation of the brain is apt to depress the tone of the cord—which leads to the remark that in practice an uneven or vicious distribution of stimuli is more common than absolute excess or deficiency. How we do a thing is not less important than what we do and how much. One-sidedness is the disease that is killing us.

In general, the kind of education which the spine and brain get from manual proficiency is very solid and very wholesome, but this has been much curtailed in the artisan, professional, and leisure classes by the excessive monotony of impressions from the sudden development of the extreme division of labor and use of machinery which characterize our modern civilization. We are just beginning to realize that the experiences of skin, membrane, and muscle-regions are as important and more fundamental than auditory or visual impressions. One-sidedness tends to overuse of the active region, often culminating in strain and exhaustion of the centres, which produces a profound detrimental effect, not only upon their function but on tissue-metabolism. It would seem that for every organism there is a certain range of stimulation within which it reacts readily and makes easy adjustments and assimilations. When the stimuli become too intense, too constant, or too disorderly, symptoms of wear and overwork, a sort of "neural dyspepsia," show themselves in the centres, and the nutrition of the body soon suffers, which in turn increases the protoplasmic distress in the centres and establishes a vicious circle, exceedingly difficult to break up. Disturbances of this kind beginning at one pole of the neural axis react upon the chain of connected ganglia, sweeping forward as a disturbing wave and exploding with peculiar violence at the terminal links. Thus among the numberless symptoms of brain-tire, debility and irritability of the genito-urinary and locomotor apparatus often occur, revealing the perturbed condition of the corresponding spinal centres. On the other hand, besides disturbed locomotor reflexes, states of profound mental depression, with a tendency to morbid religious notions, fear of suicide, and fear of insanity, at times amounting to melancholia, often result from imperfect sexual hygiene.

Health and mental vigor will be best secured by the exercise and co-ordination of large numbers of reflex arcs in widely separated parts of the body, and a rational distribution of neural reactions.

This view will present competitive athleticism, one form of one-sided use of the neuro-muscular apparatus, in a less favorable light as a health-promoter than has been claimed for it by some of its muscular advocates. Dissipation is the habitual excessive stimulation of nerve-centres, and will be detrimental according to the intensity, frequency, and duration of the molecular catastrophe and the grade of the cells involved. Thus medicinal, alcoholic, narcotic, alimentary, athletic, sexual, social, emotional, intellectual, and all other forms of intemperance are included from this point of view in one class as physiologically related.

The recent interesting experiments of Lombard show how delicately the knee-jerk reacts to the general condition of the body and the state of the centres. While they strengthen the view that the knee-jerk is not a true reflex act, its force seems to depend largely upon the state of the spinal centres, and one cannot help regarding its incessant variations as in some degree an index of the flushes of protoplasmic activity which there alternately glow and fade. These states will depend not only upon the stimuli directly received from the periphery, but also upon those retarded impressions received from the brain.

Stimuli projected against a background in the centres appropriate to the production of painful sensations referred to a particular spot, as well as imperfect mechanical conditions, will interfere with the usual and normal reflexes of a part. Thus unstable footing from any cause, or a pain referred to a limb, will modify locomotor reflexes.

Practically, nothing is more certain than that we can educate the spinal reflexes. Persons who have been prevented from walking for many months on account of joint-disease or other cause have their cerebro-spinal habits very much modified, and, on the disappearance of the general or local trouble, it is found that locomotion is lost or imperfectly accomplished, and largely by direct exercise of the will. Analysis of such conditions, with recognition of the cerebral and spinal elements, is often necessary, preparatory to establishing a more ready and perfect reflex response to locomotor stimuli.

When the functions of joints or muscles have been interfered with by disease or design for considerable periods the habitual reflex arcs become rearranged; we say a patient has a trick or habit of walking. Instinct is not always a safe guide in such cases. There are many cripples and invalids who have not acquired the associated movements which would enable them to make the most of the limited motion in a damaged joint or the limited power in weakened muscles. These considerations will apply to many cases among the hemiplegics, paraplegics, and paretics, whether from cerebral or spinal lesions, in which the vitally important question for the patient will be how to get the best service in associated reflexes out of his imperfect cerebro-spinal apparatus. The fact that the muscles are not sufficiently used, or even the circumstance that the accustomed neural paths are not kept worn smooth, is not the whole explanation of the difficulty; the lack of proper afferent impulses from sufficient peripheral stimulation is in all probability a most important factor. Such cord and brain centres, closely related to the member in question, as may still be intact, suffer from the lack of impinging stimuli and grow lethargic.

By a careful study and training of such capacity as exists, the physician will not only vastly improve the condition of these patients but give them the very best chance for increasing their powers by proper use. There are few cripples whose condition after the cessation of disease may not still be ameliorated with time and painstaking.

In the neuro-muscular degenerations following acute anterior polio-myelitis it is especially important to restore to the paretic extremities, so far as possible, the stimuli of locomotion and other normal associated movements without the inhibition of insecure footing and strained tissues. This should be the aim of mechanical treatment in such cases, and it is for the specific purpose of restoring to the damaged cord and muscles the cutaneous, muscular, and articular stimuli of locomotion that our apparatus are constructed. Even in mild cases a varus foot or wobbly ankle may produce such a sense of insecurity that the gait will be largely cerebral, consequently imperfect and exhausting, instead of mainly spinal and unconsciously performed; as in trying to walk on a slippery place or on rollers the postural reflexes of the knee, hip, and trunk—in fact, of the entire body—are rearranged by cerebral interference, and the joints held in special relations to afford a fixed point for the unsteady foot or to favor weakened muscles. Normal reflexes of locomotion are broken up and a wasteful and cumbersome set installed subject to constant cerebral interference in the efforts at balancing and progression, and additionally disturbed by the strain on weakened muscular and joint structures, which is rendered inevitable by the lack of balance between opposing groups. Thus the foot may be the key to the function and nutrition of the entire limb, and even to the health and carriage of the whole body.

Deformity having been overcome and the position of election given to the foot with exactness by mechanical support, and the direction and amount of motion precisely limited according to the indications, the sole is placed evenly upon the ground and the ankle held from lateral insecurity, so that the normal stimuli of pressure and motion are sent to the cord and reflected to the muscles, as well as the central lesion will permit. This mechanical protection with muscular training enables the patient to acquire a better set of reflexes and promotes the nutrition of the part. The special vascular paresis of this condition is most successfully corrected by the stimuli of very hot air locally applied, by means of a box heated by alcohol or a gas-jet.

This is the theory of treatment of infantile paralysis. If many reflex arcs are severed this stimulating influence can be less perfectly produced, and its effect on the muscles whose motor-centres are absolutely and permanently destroyed must be largely lost. But the ultimate fate of such muscles is not doubtful, and the stimulating influence of correct relations and reactions of the locomotor apparatus will be felt in the posterior columns, and radiated by them to neighboring motor areas and to the brain; the latter effect is very often marked.

I.—A little girl, 6 years of age, brought to me in the fall of 1886, had been unable to stand or to walk since an attack of infantile paralysis three years previous. She had always been perfectly content to sit quietly on her mother's lap without inclination to talk or to join in the activity around her, but on being placed upon her feet and enabled to walk, at the end of two weeks, she apparently experienced an entire change of disposition; she became talkative, lively, and ambitious to join in the plays of the children, and her mother had difficulty in restraining her.

These conditions of treatment cannot be realized without the greatest care in the design and construction of apparatus and perfect exactness and precision in their application, with such progressive changes as may be necessitated by the altering indications. If a paralyzed knee or ankle is allowed to yield a little from faulty construction or application of the apparatus, inhibitory influences are at once excited which interfere with that symmetrical development of associated reflexes which has been mentioned. While it is not claimed that muscles which have absolutely disappeared can be restored, the plan of treatment above outlined is a rational attempt to make the best use of what are left, and it is astonishing how helpless many patients are, whose condition is really far from desperate, for lack of a little well-directed assistance and training. The little girl just mentioned was brought to us two years before, and would then have come under treatment if the mother had not lost confidence by the favorable prognosis that the child would probably walk within two months.

II.—This little girl was severely paralyzed, but a boy of 9 years who came in March, 1886, and had never walked since his paralysis in November, 1885, was a case of only moderate severity. He had fair power in the right leg and could stand on it for a short time; the left leg was practically helpless; the knee was flexed 35 degrees, and abducted 20 degrees, and there was no power in the extensor muscles. There was a talipes equinus of 20 degrees at both ankles, as well as a tendency to valgus. These contractions were stretched by means of an apparatus, locked at the knee at the angle of choice and extending the entire length of the left leg, and an ankle-brace applied to the outside of the right foot with a screw-stop for progressively increasing ankle-flexion. These apparatus were so contrived as to be capable of progressive modification as the boy improved and to serve as supporting braces after the deformities were overcome.

The deformities were entirely rectified by the end of three weeks, with the exception of the knock-knee, which was so much diminished as to be no longer disabling. This patient has been walking freely ever since for considerable distances without other support than his braces, and his progress has been remarkable in other respects. The left (worst) leg gained, in the first year, 1½ inch at the top of the thigh, ½ inch each at the knee and calf in circumference, while the right leg gained 1½ inch, ⅝, and ½ inch respectively, the legs remaining equal in length. He was then able to walk with a free knee-joint and the lock was discarded, although there was no return of power in the quadriceps muscle. He could also walk very fairly without his braces, and it is, in my opinion, only a question of a moderate length of time before he will be able to discard mechanical support altogether.

The spasmodic condition of the neighboring muscles, resulting from the stimuli of the irritated tissues in progressive joint disease, presents a condition opposite to the one discussed. We cut off locomotor stimuli, in the acutest cases, by putting the patient to bed for a month or six weeks, and eliminate, so far as possible, by counter-extension the specially irritating and damaging stimuli occasioned by the rubbing and pressing of the inflamed surfaces. When the recession of the inflammatory action is fully inaugurated, we allow locomotion, with the weight borne on the perineal strap of the counter-extension splint, which takes the pressure from the joint and does not permit the foot to touch the ground, using crutches also if necessary. In the recovering stages it is desirable to permit a certain amount of stimulation, in order to promote nutrition, before the joint can safely bear pressure; we therefore give the joint motion, the amount of which is regulated by proper stops on a jointed apparatus, which still suspends the leg and carries the weight on a perineal strap. In this way the amount and kind of reflex stimulation which the leg receives is carefully regulated according to the indications. The withdrawal of stimulation causes muscular weakness and wasting, which is favorable to the joint in the active stages of disease, but the muscles improve as the joint recovers and the stimuli are readmitted. It is thus seen that bracing a paralyzed leg and a diseased one have entirely different objects and results.

Variously disturbed and faulty reflexes are a prominent and sometimes the paramount factor in many cases of so-called "chronic sprains," neuroses of the joints, neurasthenic hysteria, sluggish and irritable viscera, imperfect general nutrition with nervous symptoms and backache, often distinguished by the bedridden or partly bedridden condition. This comprises a large and somewhat heterogeneous class of invalids, many of whom are exceedingly helpless and very great sufferers, whose condition is the logical, we may say the inevitable, result of faulty training of the reflexes, in themselves as well as in their ancestors. Their nervous system, and perhaps general nutrition, suffering according to circumstances and temperament from the strain or relaxation of imperfect adjustments, affords favorable conditions for the formation of local disturbances of reflex action from causes sometimes so slight as to escape observation. The neuro-muscular machinery is vulnerable, and, given the proper soil, the abundant and varied crop of neural disorders easily germinates. Take a case of "chronic sprain." The patient presents himself to the physician usually with pain and tenderness in the affected part, often with wasting, rarely with heat or swelling, though a subjective sensation of burning and the puffiness of relaxed tissues are not uncommon. Disability of the most varied character and imperfect co-ordination of the neighboring muscular reflexes are among the most common symptoms, and the most characteristic one is the visible though often unconscious accommodation of the reflexes of the entire body to the condition of the disabled member. This is equally true of allied neuroses, and it is usually more distinct and more widely distributed than the secondary reflex adjustments in joint-disease, and somewhat different in character, possibly due to greater prominence of the cerebral element. If the patient have a lame ankle he is, so to speak, "ankle all over;" if it be a young woman with a backache, she presents every evidence in her conscious and unconscious life of the paramount influence of that region of the body. If we may speak of "care" as referring to attitude and movements in joint diseases, we may possibly characterize as "apprehension" the phenomena referred to in these functional troubles. The "care" of a diseased joint is most distinctly noticed in distant reflexes when the joint is hurt or threatened with violence. Pain, especially in the earlier stages of joint disease, is rather paroxysmal in character and often absent; the patient frequently forgets his trouble and hurts his joint by too spontaneous movement. In a neurotic joint affection, pain, while more constant, is not invariably a prominent feature, but, no matter what the distractions of the patient, the remotest muscular reflexes of the body are in a would-be-protective state of apprehension in a typical case. This influence can often be distinctly perceived in the expression of the face and the tone of the voice as well as in the peculiar mental attitude of the patient; the perceptions, emotions, and intellect will frequently revolve around a knee or a back for a centre as plainly as the muscular reflexes. So-called "chronic sprains," as I have seen them, usually resolve themselves into disturbances of the associated reflexes about the joint in question, whose starting-point has often been a real sprain or strain, but which had long before recovered, leaving disordered neuro-muscular action in its wake; these in turn interfere with the nutrition of the part and keep up the pain, which originally may have represented a slight organic lesion.

III.—A gentleman, aged 38, came in May, 1883, to have a brace applied to his right ankle, which he had sprained three months previously. There was pain and disability from the time of the accident. He did not use the foot for four weeks, and after that walked lame and only for short distances. The examination showed pain and tenderness about the ankle, especially the outside, with limited motion and irregular, spasmodic action of the muscles. Considerable motion at the ankle was brought out by finessing, much to the patient's surprise, as he could scarcely move it at all when told to do so. The diagnosis of disturbed reflexes about the ankle-joint without present organic lesion was made, and education of the reflexes by passive and active movements advised and begun. On the third day motion was nearly normal, with scarcely any pain, and the patient stated he had not felt so well since the accident. Three days later the patient was discharged cured, with normal locomotion, the movements of the ankle being perfectly natural and under control. Eight months after he was reported as continuing perfectly well.

IV.—A lady, about 50 years old, came to me in June, 1884. She had slipped on a piece of orange peel and turned the left ankle two years previously. This accident was followed by pain, swelling, and disability. She walked for the first time, six weeks after the accident, and then for a long time, from sickness in the family, she was obliged to be constantly on her feet and suffered from overexertion, anxiety, and broken rest. She asserted that pain, heat, swelling, and lameness had continued up to the time of examination, and had been worse during the previous six or eight weeks. The symptoms had been so severe that for a week before coming she had used crutches. The patient was a delicate woman, who had been worn out by mental strain and overexertion. She had had milk-leg on both sides several times, and varicose veins were present. The examination showed no swelling. She was able to relax the ankle and permit it to move naturally in all directions, and also to execute these movements voluntarily. She said she had never tried to move the ankle before, and did not know that she could. It was explained to the patient that the ankle was suffering from disuse and imperfect hygiene. The crutches were at once discarded, and physical, educational, and developmental treatment begun. In a week the patient stated that she did not know she had an ankle, and was able to lie on the left side, which she had not done before. She remained for several weeks for general tonic treatment, and has since been seen socially from time to time, and reported that her ankle had remained well and that only occasionally, after overexertion, was she reminded of the accident.

V.—While passing through Fall River in July, 1884, I was called to see a large, athletic young man, 17 years old, whose left knee had given out while tramping through Switzerland six months previously. He afterward limped when he tried to use it, and thought that it swelled. He had been better and worse by turns, but the knee had never ceased to trouble him, and for ten weeks he had walked on a crutch and a cane, bearing very little weight on the affected limb. Naturally energetic, he felt his condition very much, and chafed under the awkward work he made in hobbling around, and was exceedingly anxious to be relieved. The left thigh measured 1 inch less at the top, 1¼ inch less above the knee, ¾ of an inch less at the calf, and ½ inch less over the knee, although he believed it to be swelled. Mobility was good, and it was perfectly evident that the limb was suffering from nothing except disuse. After going through a few passive and active movements of the leg in various directions, I got him to stand up with his feet flat on the floor, and made him bear his weight on both limbs. Inside of five minutes I had him walking around the table without assistance, and, to follow up the impression, I took him a short turn in the street and up the front steps of the house. At the end of fifteen minutes he walked without a limp, and I took my leave of the bewildered family. This young man never had any trouble afterward, and played on the Harvard team in the intercollegiate football match last Thanksgiving.

VI.—A married lady, 44 years of age, came in May, 1884. She had suffered a great deal with her left knee for 30 years. While at boarding-school at the age of 14 her knee began to hurt her at times, especially on stepping up, but she did not remember to have injured it. She afterward met with a number of rather trivial accidents which had laid her up for months at a time and obliged her to use crutches; the knee also troubled her sometimes without known cause, so that it was often treated locally. Two years before coming she fell and struck her knee, and since then had used crutches constantly and kept the knee bandaged. Aching had been frequent and, since the last accident, located on the inner aspect of the knee below the patella. The patient held her knee in continuous complete extension, as had been her habit when it troubled her (I have noticed this peculiarity, impossible to the sufferer from synovitis, in several cases). Her right knee measured 1¼ inch, and the right calf 1¾ inch less than the left. She was of nervous temperament, but of fair physique and general health, and not morbid. Examination showed no organic change in the joint, except that incident to prolonged disuse. The knee-motion was of considerable extent and good quality, but voluntary control of the muscles moving the knee was deficient. She could hold the knee out when sitting, but not extend it from the flexed position. The diagnosis was atrophy of the limb and probable dryness of the knee-joint from disuse and disturbed reflexes. Graduated passive movements at the knee by special apparatus actuated by steam-power were given for half an hour daily, and gradually increased in extent and duration. She was also drilled in placing the foot squarely on the floor, in bending the knee, and in gradually increasing the amount of weight borne upon the limb as she walked with the crutches. This was followed in a few days by some increase of pain and considerable puffiness about the knee, though the exercises themselves were not painful. Nine days after beginning the treatment she was able to extend the leg from the flexed position; three days later she laid the crutches aside, walking readily, though with a slight limp. In a month after coming she was able to bend her knee and to walk considerable distances without crutches, and the pain had greatly diminished. She gained markedly in flesh and in spirits, and lost the drawn and anxious expression she had had. In five weeks from coming the affected knee had gained one inch in circumference and the calf 1¾ inch, and the patient returned to her home. She has since been frequently reported by members of her family as enjoying perfect health and locomotion.

VII.—A bright, active, intense woman, 27 years of age, came in May, 1885. She had fallen, striking the lower part of her left knee, seven months before. It pained her only moderately, and she went on teaching as usual, not walking much, until two weeks afterward the knee became red and swollen, and there was a pricking pain. She was put to bed and kept there four months, blisters and iodine being applied to the knee. During this time the knee was kept stiffly extended, and when she got up it was put in plaster for three weeks, and she walked on crutches, with a high sole on the right foot. She came on crutches, and had not borne any weight on the left leg since she went to bed. She had had burning, itching, and aching sensations in the knee, aggravated by motion, but no sharp pains. The patient's health had always been good, and she had had no previous sickness. She had felt the effect of her knee-trouble severely, but did not think she had lost much flesh; a few days after coming she weighed 97¾ pounds. Examination showed that the limb was held in complete extension by contraction of the quadriceps. There was about 10 degrees of voluntary and restrained passive motion, which was not very painful, though the patient was apprehensive; when her attention was distracted the motion was somewhat greater. The thigh and leg rotated outward when the patient was lying on her back; the muscles and even the subcutaneous tissues were very much atrophied, and the skin very thin, having the appearance of being drawn over the bones like parchment. The measurements were as follows: Above the knee, right, 13⅞ inches; left, 12; knee, right, 13¼; left, 12½; calf, right, 12; left, 10¼; there was no evidence of any inflammatory trouble in or about the joint, nor of any organic lesion anywhere. Diagnosis of restraint and abnormal reflexes following slight injury was made and the condition explained to the patient, who was then able, with a little preliminary training, to walk alone without crutches in a few moments, with only moderate discomfort and very little limping. Systematic passive movements and training of the reflexes were begun at once. There was some puffiness and muscular soreness during the first few days, but at the end of two weeks there was very great improvement in the condition and nutrition of the leg, which had already gained an inch in the calf and lower thigh measurements. Five days later she discarded her crutches entirely, and there was a gain of 1½ and 1¼ inch at the lower part of the thigh and the calf respectively, over the first measurements. The patient left in about six weeks, with perfect motion and good control over the knee, though it had not yet attained the strength of the right leg. Her general health was perfectly restored, and she had gained, in five weeks, 7¾ pounds.

With some care on the part of the patient the knee continued to improve during the summer, and in April, 1886, she called to demonstrate its entire restoration, the legs being then equal in size and function.

VIII.—A little girl, 9 years old, was brought in the fall of 1882, walking on crutches, which she had used for a year, during which time she had suffered from pain in the right hip with extreme flexion at the hip and knee. All efforts to straighten the leg caused such excruciating pain that they had to be abandoned. The muscles relaxed under ether, but on recovering from the anæsthetic they became as rigid as before. Her family physician had recognized the neurotic nature of the case, but all of his efforts at procuring relief had been completely baffled. She was an excessively intense, self-conscious child; her general health was fair, and she was very happy in running around on her crutches and joining in the plays of the children. Five months of training, which was not directed to the hip, as she was already hyper-conscious of that part, were followed by a complete cure, and she went home without pain and walking perfectly. This little girl had pain at the hip at long intervals for a considerable time, but she never had any recurrence of the functional trouble in that location.

She was brought to me three years afterward as a bedridden invalid suffering from backache and extreme mental depression. After a long and varied experience, she was taken home in an essentially bedridden condition and has never walked since. There is no question of any organic disease, she is simply floored by her chaotic reflexes.

IX.—A lady about 40 years of age, the wife of a physician, consulted me in September, 1885, about her left shoulder; she had wrenched it three months before while trying to save herself from falling on the stairs. She did fall, and bruised herself in several places, but not on the shoulder. Her arm was afterward stiff and painful, and she found it powerless at the shoulder and elbow; she carried her arm in a sling, and it had been treated electrically. At the time of the examination there was pain in the elbow when she raised the arm; she could not raise the hand to the face nor abduct the arm more than 45 degrees from the side. When passive movements were made the muscles about the shoulder resisted, and motion was not free. Diagnosis—of restraint and disturbed reflexes. Training of the reflexes by systematic movements was followed by marked improvement in mobility and usefulness of the arm, but after being treated for a week the patient was obliged to leave and went home with the arm still very much disabled. It remained in about the same condition until the death of her husband, which occurred unexpectedly a few weeks later. The shock was so great that she became entirely unconscious of her arm, and from that time it has been perfectly normal in every respect, as she was able to prove to me at her next visit.

Cases similar to the above are of very frequent occurrence in our practice. I recently saw a gentleman who had walked with the toes and inner border of the foot elevated for nine years, without organic lesion. Some time ago a lady reported who had walked many years with her toes voluntarily digging into the ground at every step. Only a few weeks ago I saw a lady in whom a rather severe injury to one finger had been followed by disturbed reflexes of the arm. I have under my observation at the present time a young lady in whom weakness of the knee from relaxed ligaments caused such severe pains across the back and down the thighs, and so much disability, that there seemed to be hesitation on the part of the patient and of her father, who was a physician, in accepting the diagnosis of disturbed neurility from knee-strain alone. Avoidance of locomotion for a few weeks caused an entire disappearance of pain in the back. Such instances might be indefinitely multiplied. I see more cases of functional joint troubles than of joint diseases ; they are exceedingly common in this country, and the importance of carefully differentiating between the two conditions can hardly be exaggerated, as many of these functional troubles will be indefinitely prolonged, with great distress and harm to the patient, unless recognized and properly treated.

I have already referred to the fact that there is a wide range of disturbance even in those cases where local trouble predominates. Not a few, beginning with a local disorder, degenerate into a condition of general invalidism, with scarcely any normally adjusted reflexes, as in the case of the child bedridden at thirteen. This is more apt to happen when the main disturbance is in the trunk, back, or viscera—of course, the primary and essential trouble lies in imperfect adjustments of the higher cerebral centres in a very large number of cases, but that element only enters incidentally into this discussion.

X.—A little girl, 12 years old, an only child, was brought in May, 1884; she had always been delicate and the object of great solicitude on the part of her parents. She had suffered at various times from chills and sick stomach ; the last time in November, 1883, when these symptoms, with pain in the back and jaundice, followed a fall. She had been allowed to walk but very little afterward, and, as she continued to complain of her back, caries of the spine was suspected and an apparatus applied, which she still wore. She was pale and thin, with an expression indicating solicitude, and was carried from the door to the office in a chair. It was perfectly evident that the entire family, including the sufferer, were intently engaged in watching for the development of expected symptoms. Examination of the spine showed it to be quite normal, with the exception of a slight bending occasioned by the shortness of one leg from asymmetrical growth of the extremities. Diagnosis of reflex debility, the effect of "too much mother," was confirmed by the rapid improvement which followed separation from the parents. Gentle exercises calculated to give tone to the cord and develop the associated reflexes of the trunk and limbs were given, and, the burden of constant repression and restriction being removed, it was a pleasure to see the pale, sad child taking her first taste of natural childhood. On the ninth day she walked two miles; on the tenth she walked upstairs for the first time, an effort which the spinal neurasthenic instinctively avoids. In three weeks from coming she was riding on horseback, and at the end of two months she returned home in perfect health. I am confident she would have suffered a relapse had we not undertaken the education of the parents, who had become completely demoralized by the abnormal relation to an only child, and who were trained with difficulty not to watch nor repress her. She returned for inspection in five months, natural and well in every respect, and having gained considerably in weight and height.

XI.—In May, 1886, a gentleman, 28 years old, who had always been rather delicate and had broken down at college five years before, came with a variety of complaints, of which backache and general debility were very prominent. He had not been able to do any work since leaving college, and had been growing steadily worse, until he was unable to sit up for his meals, and even talking made his back ache unbearably. Pain and apprehension, with introspection and mental and physical demoralization, made him a helpless wreck. The attempt was made to tone up the centres and restore the equipoise of the various functions by properly directed exercises. He proved a difficult case, but the attempt was so successful that he was able to leave for home comparatively restored at the end of two months. I continued to advise him by letter from time to time after the discontinuance of the treatment, as is my invariable custom in such cases. The following fall he took a position as civil engineer with a field-party, which he has held creditably ever since, and when I saw him a few months ago he was a perfectly healthy young man.

XII.—A case similar as to the general condition, approaching the bedridden state, with excruciating pain in the back, great prostration, disinclination to exertion, and abnormal reflexes of the back muscles, was that of a young lady who came to me in February, 1886. The pain was so great—"as if her back would break in two," she expressed it—that it had led to the diagnosis of Pott's disease, and the application of a plaster jacket, which she was still wearing. In spite of the protest of the patient, for her reflexes had accommodated themselves to the rigid casing, the jacket was at once removed and the usual means for the development of neuromuscular tone were employed. This case returned home at the end of two months in fair health and much relieved, but I have recently heard that she has relapsed.

XIII.—The next case was that of a lady, aged 34, who had been confined to the bed and a wheel-chair for seventeen years. She stated soon after coming, December, 1884, that exactly seventeen years before she had gone to church for the last time and attended five services. Spinal pain and weakness had been prominent symptoms throughout the case, and had resulted in a readjustment of the reflexes to the abnormally restricted condition. That this patient was walking within a few days and improved steadily in all respects was largely due to her own intelligent and hearty co-operation, once the condition was explained to her. For some weeks her main difficulty was in accustoming the soles to bear the pressure of use, for the feet had lost their form and character, and required to be reshaped. She went home at the end of four months, walking freely and in fair health. A month after she reported in fine condition, having gained twenty-five pounds in four months and a half. While not robust she leads a tolerably active life, visiting, shopping, and attending to her domestic and social duties at her pleasure.

It is not necessary to prolong this enumeration of type-cases, where, from causes depending upon local conditions and upon states of the centres, the associated reflexes of the trunk and limbs, or of special areas, have become too keen, too sluggish, or too disorderly. The object of these outlines is to indicate that such detrimental conditions do exist in various localities, either alone or associated with organic or functional troubles, and that they are susceptible of analysis and rational treatment. Sometimes the consideration of faulty reflexes will not be important, in view of more urgent indications, but there are cases of serious local or general disturbance where the best results have been obtained by progressive, systematic training and development of associated muscular movements. By thus feeding in appropriate stimuli, we can fill up gaps and reclaim barrens in the centres, balancing and distributing nerve-force as may be desired. The steps must often be so gradual as to commit the centres to a certain line of action, stimulate consciousness of power, and promote nutrition, without exciting the inhibition of undue fatigue, pain, apprehension, or resistance, but in certain cases very much may be accomplished by sudden and profound impressions. The training will often be directed to regions remote from the part affected; for instance, a functional spasm in the lower extremity may be favorably affected by the exercise of the trunk and arms as a physiological diversion. The effect of local treatment in exaggerating the attention already fixed upon the affected part should be carefully considered.

We are frequently obliged to draw up a physiological balance-sheet, and, if necessary, place the system in the hands of a receiver, going over the assets and liabilities, finding where the former can be increased and the latter diminished, where idle capital can be made to yield interest, and wasteful extravagance checked. These patients have the right to expect something more than the prescription of drugs, diet, braces, or exercise. Quantitative analysis of the various activities of the organism will be needed, and a complete system of physical economies adopted, which shall recognize and regulate, so far as may be desirable, all the functions and all the conditions of life. Change of moral atmosphere, separation from the family, the acquisition of definite aims and purposes, the control of emotional excess, the introduction of order and system into daily habits, are examples of what is meant.

Much of this will best be done indirectly by the modifying influence of neuro-muscular training on the organism and its higher centres. We wish to practise economy in the organism, not that we may spend less, but more in the long run. Analysis should reveal weak points in order to strengthen them and make the organism more efficient.

In concluding, I will mention some of the means which we have found useful in promoting reflex hygiene: Daily rest, lying down at a stated hour, with complete relaxation of mind and body.

Systematic heating of the legs from above the knees by the hot-air box, kept at a temperature of about 130–140° F.

Drill in the "standing frame," with knees or hips, or both, supported, thus training the centres without the disturbing influence of balancing the body, and enabling the physician to throw more or fewer muscle-groups into active use while giving all the stimulus of standing.

Drill in locomotion, free or between parallel bars.

Localized active and passive movements by hand and by means of special apparatus, susceptible of accurate adjustment of the resistance and amount of motion, among the most useful of which for the purpose considered are:

Passive alternate, right and left flexion of the trunk by means of steam-power apparatus, patient lying; 46 complete movements a minute; 2,760 an hour.

Active flexion and extension of trunk through lumbar region, patient lying; the upper or lower half of the body fixed as desired.

Passive (steam-power) flexion and extension at the knees, patient seated. This apparatus gives 25 movements of flexion and extension a minute; 1,500 an hour.

Passive (steam-power) flexion and extension at hips and knees, patient partly reclining; 23 complete movements a minute, or about 1,400 an hour.

Active extension at the hips and knees, patient partly reclining; and flexion and extension at ankle, both against graduated resistance.

Weight and pulley for arm-movements.

Artificial respiration by means of an apparatus known as the "respirator" (steam-power), which produces full inspiration and expiration, by drawing the arms of the patient strongly upward, the chest being at the same time arched back; the patient is reclining and passive, except as to grasping the handles of the apparatus. This machine has rendered us yeoman service in regulating reflexes and distributing nerve-energy, besides which, it develops the chest, oxygenates the blood, equalizes the circulation—warming the extremities—and acts as a general tonic to the system. We use two apparatus, one giving 13 and the other 16 respirations a minute.

The exercise-room is provided with couches, and all patients are required to rest before and after each movement.

What I wish to emphasize as the central idea of this paper is the development and use of associated reflexes as a practical means of modifying nerve-centre function. The spinal and cerebral factors are to be recognized and differentiated, in order to send re-enforcing or inhibiting impressions into appropriate areas, by applying or removing particular stimuli, and thus to effect an advantageous redistribution of their energy.

REVIEWS.

LEÇONS SUR LES FONCTIONS MOTRICES DU CERVEAU, ET SUR L'ÉPILEPSIE CÉRÉBRALE. Par le Dr. François Frank. Cours du Collège de France, 1884–85, pp. ix., 570. Paris: O. Doin, 1887. LECTURES ON THE MOTOR FUNCTIONS OF THE BRAIN AND ON CEREBRAL EPILEPSY. By Prof. François Frank.

Prof. François Frank has made a number of important contributions to physiology, some of which are more original than the present work; but none have been more thorough and careful than those recorded in this volume. In it he gives the result of extensive experimental researches upon the motor functions of the brain, going over the same ground which has been covered by the classical works of Fritsch and Hitzig, Munk, Ferrier, and Luciani, and coming to conclusions which agree very fully with theirs. His experiments have been upon the cortex of the brain in dogs and monkeys, and he has irritated the various areas by the faradic and galvanic currents, producing irritative manifestations. He reaffirms what has been so often proven, that the motor area is about the transverse fissure; that its upper third is related to movements of the leg; its middle third to those of the arm, and the lower third to those of the face, of the opposite side; but he does not localize the movements any more exactly than this, as has been done by Ferrier. He finds that the motor tract is not as excitable as the cortex, it being necessary to use a stronger current upon the fibres in the centrum ovale or internal capsule to produce the same movements. Irritation of the basal ganglia does not cause movements, an assertion which differs from that of other observers. A number of electric shocks to the cortex, any one of which is insufficient to cause a motion, will, if repeated at very short intervals, produce one ; a fact to which he gives the name of the cumulative action of the cortex. And a long-continued excitement reduces, and finally suspends, the excitability. These results of experiment are carefully recorded by the graphic method, which has not before been used in such experiments ; so that the degrees of muscular contraction, and the time elapsing between irritation and result, are here accurately determined. And the results reached are then compared with the phenomena presented by patients suffering from cortical epilepsy. It is this feature of the book which adds greatly to its value—the comparison of clinical and experimental facts being fully worked out. One interesting fact from a number may be selected as an example. He found that moderate irritation of the motor area will produce an epileptic fit in an animal, and that, after this fit has occurred, the animal is thereby rendered susceptible to the recurrence of such fit on irritation in any area of the cortex ; whereas, irritation of the occipital lobe in an animal which has never had such an artificially induced fit will not produce a convulsion. This is compared to the well-known clinical fact that the occurrence of a single epileptic convulsion predisposes the individual to a subsequent attack under sufficient physical or mental excitement. That the irritation of the cortex in order to produce a fit, must either originate in, or extend to, the motor area of the brain, is also conclusively proven by the author. Cortical epilepsy begins with a tonic spasm, followed by clonic spasms, which are at first slight, then severe. Reflex epilepsy, on the other hand, begins with large clonic spasms, and if a tonic spasm occurs it is in the midst of the fit, as an evidence of cumulative excitement. No one who is familiar with the clinical features of Jacksonian epilepsy can fail to be exceedingly interested with the experiments and conclusions bearing on this disease.

The effects of electrical irritation of the cortex upon the respiration, vascular tone, heart action, pupillary contraction, secretion of saliva and sweat, and excretion of urine, are very carefully investigated, and in the lectures upon these experiments much that is new is to be found. Frank does not consider it justifiable to conclude, as Ferrier does, that because irritation of a part of the brain causes secretion of saliva, that, therefore, that part contains the "gustatory centre." He finds that the influence of cortical irritation on secretion is wholly indirect.

The results of the destruction of motor areas are considered much more briefly than those of irritation. The author holds that physiology must here yield the palm to pathology, since conclusions regarding loss of function are less reliable when derived from the observation of animals, than when based on the statements of patients. He prefers, therefore, to draw practical conclusions from such collections of cases as have been made by Charcot and Pitres, rather than to record the results of experiment. In this he is supported by Charcot, who commends, in his preface to the work, the willingness of the physiologist to give a place to clinical observation.

The book should be studied carefully by those who are interested either in nervous physiology or in the subject of Jacksonian epilepsy.

M. A. S.

Society Reports.

THE PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, January 23, 1888.

The Vice-President, Charles K. Mills, M.D., in the Chair.

Dr. F. X. Dercum reported a case of

CHOLESTEATOMA, WITH REMARKS ON THE ORIGIN OF THE TUMOR.

P. J., aged 43, unmarried, an Englishman by birth, was admitted to the nervous wards at the Philadelphia Hospital on August 3, 1887. He was dull and stupid. He answered questions slowly and imperfectly, and gave a very poor and disconnected account of himself. He said that about nine years ago he had had a wen removed from his scalp at the German Hospital. Otherwise he had been in good health.

Careful examination of the scalp failed to reveal any evidence of a former operation, and an exhaustive search among the records of the German Hospital failed to reveal any history of it.

His present trouble had commenced about three months ago, with trembling of the hands, and more or less mental confusion. He had what he called a " rigmarole of things " in his head. On admission he complained of constant and persistent headache, which he invariably referred to the base and the back of the skull. He was, in addition, quite weak, but in no sense paretic. Sensation seemed to be normal. The knee-jerks were markedly increased. An examination of the eyes revealed double optic neuritis. The pupils at this time showed no decided inequality.

Brain-tumor was deemed probable, but, owing to the absence of special symptoms, it was not possible to locate the lesion. There had at no time been any localized palsy, any convulsion, or, in fact, any symptom referable to this or that part of the cortex.

The patient was under constant observation up to the 31st of August, when he died. During this time his mental condition gradually grew worse. He became more and more stupid, and at times wandered. On the day of his death he commenced vomiting, the vomit consisting merely of mucus and bile. He rapidly became less and less conscious, and finally lay perfectly quiet, with the exception of occasional movements of the head and arms. These movements were, however, not in any way convulsive. When the hands or arms were pricked with the æsthesiometer he would draw them away, but further than this he could not be aroused. His right pupil was now seen to be widely dilated, while the left responded but sluggishly to light. His pulse was 64, soft and compressible, and his respiration was 28. His general appearance was that of a man in a sound sleep, but whose face was flushed. Toward evening his depression deepened, and at 7.20 P.M. he died.

At the autopsy it was found necessary to remove the brain entire with the calvarium, all of the tissues, the brain, dura, and bone being very much adherent to one another. An examination revealed this adhesion to exist in an area some two or three inches in diameter in the right frontal region. In forcibly separating the dura from the bone a rough prominence was discovered on the latter, occupying the middle of this area. The prominence measured about two-thirds of an inch across, and about one-sixteenth to one-eighth of an inch in height. The outer surface of the calvarium revealed no change other than a slight deepening of color—a faint purple tinge—at the site corresponding to the prominence on the inner side. The dura mater, on the other hand, exhibited a depression, a shallow pit, the counterpart of the bony prominence. Beneath this pit, and for the radius of an inch or more around, the tissue felt much firmer than normal, and a rounded mass was readily outlined by the finger. On attempting to raise the dura it was found to be intimately united with the underlying tissue, not only over the tumor, but also over the entire vertex. The pia, in turn, was everywhere intimately adherent to the cortex. Its meshes were quite œdematous, as was also the brain substance. Some of the convolutions, especially at the base, were much swollen. The walls of the ventricles were exceedingly pale. The right lateral ventricle was much compressed. The choroid plexuses were cystic. The vessels at the base were apparently healthy. The veins of the pia were quite full.

The tumor was found to be situated between the first and second frontal convolutions, on the one hand, and the anterior central, on the other. It had developed downward, and gradually separated these convolutions. The pia was intimately adherent to the edge of the growth, and did not separate it from the brain substance. A number of vessels could be seen passing from the former directly into the latter. When incised the tumor had a whitish or pinkish-white color. It was quite friable, and a pinkish-white juice could be expressed from it.

The microscopical examination reveals its general structure to be that of a sarcoma, while scattered through it in varied profusion we find typical pearly bodies.

Dr. Dercum had not been able to find in the literature of endotheliomata or cholesteatomata an exactly similar case. The formation of the pearly bodies in the midst of a sarcomatous tissue is exceedingly interesting, especially in the present instance, as the locality of the tumor gives abundant opportunity for the origin of the endothelioid formations. As seen in the drawing, they closely resemble the pearly bodies of the epitheliomata, so much so that some writers still believe them to be of epiblastic origin. Their occurrence, as in the present instance, in the depth of a sarcomatous tissue, is certainly another argument against this view.

On examining them closely their concentric, onion-like structure is readily seen. Their centre appears to be made up of a granular and nuclear material which is probably cell detritus. Toward the periphery individual cells, more or less preserved, are still to be distinguished. They are sharply differentiated from the surrounding tissue; though the vacant space seen about them is probably the result of the hardening process.

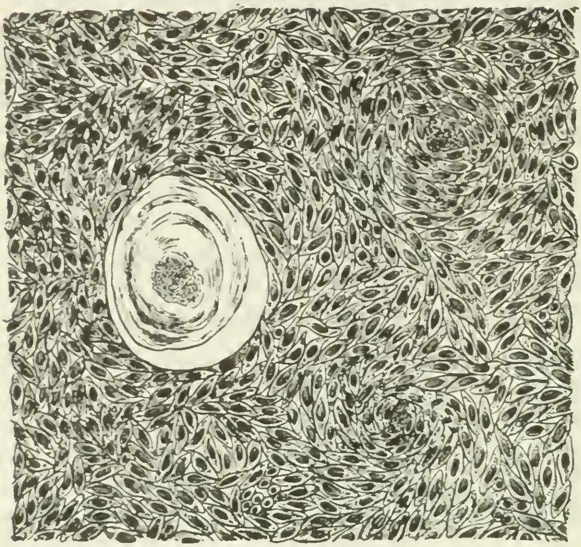

Fig. 1.

Clinically this tumor is interesting from the absence of symptoms enabling us to localize it. Dr. Dercum's impression was that it was either far forward in the frontal region or back somewhere in the occipito-temporal region, not interfering with the cuneus. He favored the latter supposition on account of the basal and occipital pain. He regretted that the scalp was not shaved, in order that evidences of traumatism could have been better sought for.

The man had an exostosis on the inner table of the calvarium involving the upper portion of the frontal bone somewhat in advance of its junction with the upper portion of the right parietal. There had been inflammation of the dura about the exostosis, with adhesion to the pia. From this focus of irritation the tumor appears to have developed.

Dr. William Osler said that a short time ago he reported a case in which the pearly bodies were even more distinct than in the specimen shown, and in which the origin from the endothelium of the floor of the third ventricle and infundibulum was evident. In this tumor, as in Dr. Dercum's, there were many spindle-cell elements. Cholesteatomata are more common at the base than on the cortex.

Dr. Charles K. Mills said that Dr. Dercum's case was of interest as bearing upon the localization of growths in the second frontal convolution. In the case on which Dr. Keen operated, the tumor was in much the same position, perhaps a little lower. In a case which he reported to the Pathological Society the tumor was situated in much the same locality. In all these cases there were eye symptoms. In his own case he noted a certain fixity of the eyes with dilatation of one pupil. These cases to a certain extent carried out the idea of Ferrier and others, that the oculo-motor centres are located in the second frontal convolution.

Dr. Dercum said that his patient had no specific symptoms of the locality of the tumor. He did not place the same interpretation upon the eye symptoms as did Dr. Mills. This tumor did not press directly upon the cortex, nor did it irritate the cortex. The man was killed by œdema of the brain, and not directly by the tumor itself. The mass grew downward between the first and second frontal convolutions and the ascending frontal, and it separated these so gradually that no irritation was produced. The eye symptoms were of late occurrence. The tumor became so large that the venous return was interfered with and the brain became œdematous, and was so found at the post-mortem. As a result we should expect oculo-motor symptoms from transmitted pressure. The size of the tumor was probably also increased by œdema. The right half of the brain was not as œdematous as the left, as it had not so much room to swell. The left base was much swollen.

Dr. Osler presented

A CASE OF LOCAL SYNCOPE AND ASPHYXIA OF THE FINGERS.

The patient came to the Infirmary for Nervous Diseases a few days ago, with her hands in the following condition: The second, third, and little fingers of the left hand were like marble, the line of demarcation being at the second joint. The thumb and index fingers, with those of the right hand, were livid, particularly the terminal phalanges. The condition is aggravated by exposure to cold. The local syncope is so marked that he thought the members would be interested in it. The condition is variable, the syncope in a short time is followed by asphyxia. This is the mildest grade of Reynaud's disease, which in extreme cases may result in gangrene of the affected finger-tips.

He thought that the local syncope is caused by vaso-motor spasm. Following this there is relaxation of the spasm with extreme engorgement. Such a condition produced by the local action of cold is closely related to chilblains. Many of these cases have had chilblains. Last winter he had at the University Hospital a child, aged thirteen or fourteen, who had presented for many years this condition of local asphyxia of the terminal phalanges of both hands. It did not vary in any degree while under observation.

DR. EDWARD N. BRUSH reported

A CASE OF PORENCEPHALUS, WITH SPECIMEN.

J. K., aged 57, a native of Pennsylvania, married, by occupation a laborer, was admitted to the Pennsylvania Hospital for the Insane, June 16, 1885.

The following is an abstract of the history of the case: At birth the patient was observed to be unsymmetrically developed. The left side was smaller than the right, the limbs were shorter and their circumference less. No history of infantile paralysis could be obtained, and the patient is said to have walked at about the usual age, though with a limp which continued through life, owing to the shortness of the left leg.

At the age of nine, while playing in a barn, he fell to the floor from the hay-loft, a distance of some sixteen feet, striking the back of his head. For some weeks following this fall he complained of headache, but his general health did not seem to be affected.

As a young man he was intemperate in the use of liquor, but he had been, for some years prior to admission, of temperate habits.

A maternal uncle had been insane, and several relatives are said to have died of "dropsy of the heart."

About nine years before admission to the hospital he had sunstroke, and was from that time frequently troubled with severe headache, especially during the hot weather.

He had always been a man of violent temper and rough in conversation, and, following the sunstroke, is said to have become worse in these respects. By some he was regarded as not possessing a thoroughly balanced mind. His friends had not noticed any active mental disturbance earlier than a week prior to his admission to the hospital, when he suddenly became noisy and violent, and gave expression to extravagant delusions. His condition, however, on admission pointed to a much longer period of mental disturbance, as he was in advanced general paresis. Upon inquiry, it was found that as early as December he had been talking in a boastful manner of his power and possessions, and particularly of the speed of his horse. It was also found that he had not, on account of his imaginary wealth, made any charge for his services as a carter since spring.

On admission he walked with a slight halt, the left leg was shorter than the right—as was also the left arm ; there was some asymmetry of the skull, particularly in the occipito-parietal portions—the left arm was stiff from an old fracture. In addition to the limp, his gait was somewhat ataxic. He swayed to a marked degree when standing with his eyes shut. The tendon (patella) reflexes were absent. The pupils were unequal, the left being the smaller. They responded to light very slowly, and not at all to accommodative changes. No other examination of the eyes was made at the time, and the patient was never in a condition subsequently to permit any. There was commencing cataract in each eye.

The patient gave expression to very extravagant delusions of wealth, and was lavish in his gifts of checks to all about him.

There was marked tremor of the lips and tongue, and of the hands and fingers as well. He pronounced his words in a hesitating, drawling fashion, and frequently stumbled over words of many syllables.

The case progressed without incident for some weeks. He was frequently noisy, shouting and singing. His gait grew more disturbed—he manifested a tendency to fall forward, and, when once started, would almost run at times to keep from falling. At the end of a month his pupils had become almost pin-points. His speech was more hesitating and his delusions more extravagant. He said that he had a horse which could trot a mile in a minute and a quarter less than no time—and another that could go around the earth in two minutes and a quarter. He had hallucinations of sight and hearing.

For a time the patient gained in flesh, but soon lost, and in the fall was quite emaciated, though eating heartily. The tendency to fall forward continued, and it became necessary to support him in walking. When he could be induced to stand still, he was able to support himself fairly well.

To omit unnecessary detail, it will suffice to say that the patient became noisy, untidy, and very destructive of clothing—at the same time failing mentally and physically. In November, five months after admission, he was quite demented, and his speech was hardly intelligible.

On December 2, 1885, the ward notes state that his morning pulse was 48; temperature, 97°; P.M.: pulse, 56; temperature, $97\frac{8}{10}$°; in bed, quite feeble. On the day previous he had been up and had taken some exercise in the ward with the assistance of an attendant.

December 3d, A.M.: pulse, 60; temperature, $97\frac{8}{10}$°; P.M.: pulse, 64; temperature, 98°; heart sounds muffled; respiration, 14.

December 4th, A.M.: respiration blowing, 10 to 12; pulse, 48; face pale; body generally blanched. Is comatose. At 2.30 P.M., the respirations were but eight per minute and wholly diaphragmatic; pulse, 54. Died at 8.15 P.M.

Autopsy sixteen hours after death. Examination made of brain only. On removing the calvaria the dura in the right occipito-parietal region protruded, bulged out as if from internal pressure. Dissecting the dura carefully from the brain, to which it was strongly adherent over the vertex, this protrusion was seen to be due to a large accumulation of serum beneath the arachnoid. In removing the brain, the arachnoid was accidentally ruptured and the fluid escaped. About six fluid-ounces were collected, and found to differ in no respect from the ordinary cerebro-spinal fluid.

Enlarging the opening in the arachnoid, the cavity which is shown in the specimen was found. It will be observed that it communicates with the lateral ventricle, the posterior cornu of which is enlarged, and that it is lined, except where it communicates with the ventricle, with the pia mater. This cavity, it will be observed, occupies the position of the parietal lobe and encroaches also upon the occipital and temporal.

The ascending parietal convolution is, in its upper portion, almost wholly absent, a small amount of cortical substance held in the meshes of the membranes alone remaining.

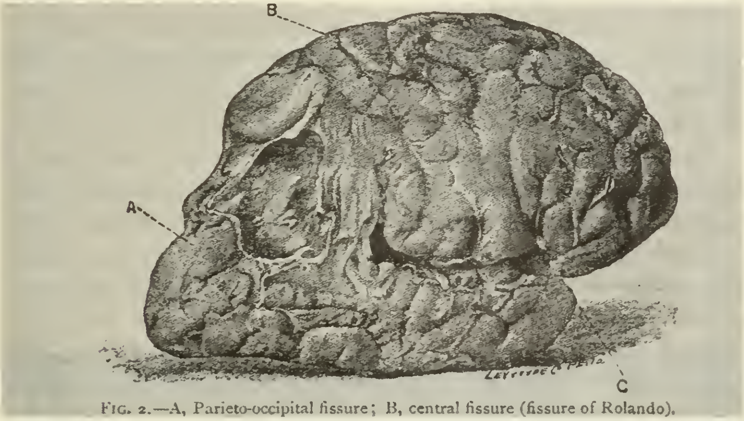

FIG. 2.—A, Parieto-occipital fissure; B, central fissure (fissure of Rolando).

The superior parietal lobule, or a portion of it, rather, forms the roof of the cavity. Nothing of this lobule remains but a mere shell of cortical substance and membrane which overhangs the cavity.

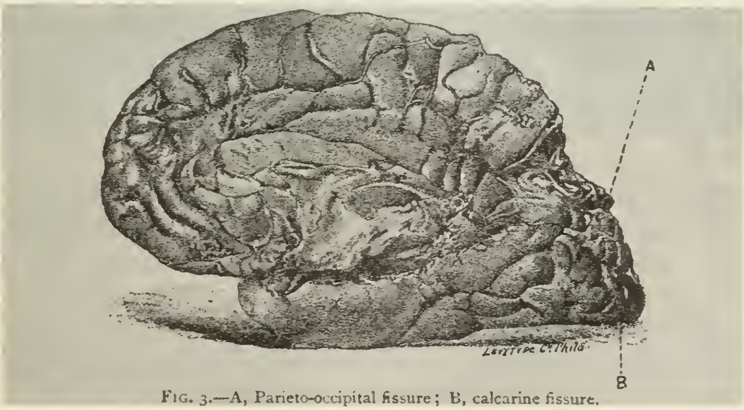

FIG. 3.—A, Parieto-occipital fissure; B, calcarine fissure.

The posterior portion of the superior temporal convolution is absent, and nothing but a thin membrane exists between the cavity and the fissure of Sylvius, which is turned abruptly upward. The inferior occipital convolution appears intact, but of the superior and middle convolutions but a small portion remains.

On the inner surface of the hemisphere the cuneus is simply represented by a shell of brain substance, and of the quadrate lobule nothing remains but a thin stratum of brain substance at its anterior portion.

If the right hemisphere is compared with the left, the portion not encroached upon by the cavity is markedly smaller, and the convolutions are not as well developed. The difference in weight between the two hemispheres was, when removed and thoroughly freed from fluid, nine and one-eighth ounces.

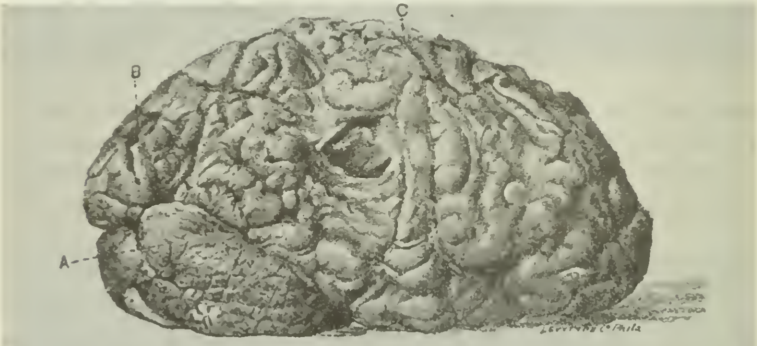

Fig. 4.—A. Right half of cerebellum, not symmetrical with the left; B, occipital lobe, left hemisphere; C, central fissure (fissure of Rolando).

The cerebellum has developed unsymmetrically, the right half extending upward and forward considerably in advance of its fellow, owing to the absence of resistance from the right cerebral lobes. It is to be regretted that the maniacal state present at the admission of the patient, and the subsequent dementia into which he passed, prevented a more careful clinical study of the case, and especially that no opportunity was afforded for recording the eye symptoms.

Dr. E. T. Bruen also presented a specimen from a

CASE OF PORENCEPHALUS.

The specimen of the brain which was presented was removed from the body of Mary C., aged 32, who died in the

Philadelphia Hospital September 21, 1887. On removing the dura mater four cysts were found, situated upon the cortical portion of the brain near the occipital lobes of both sides.

There were two cysts on each side of the median fissure, those on the right side were irregular in shape, and somewhat larger in size than a walnut. They were situated mostly over the occipital lobe, but the outer one particularly extended over a portion of the parietal lobe. In the left cortex the cysts were much larger, the one close to the longitudinal fissure extended forward three inches, and was an inch in diameter. The smaller cyst was two inches long by three-quarters of an inch in diameter. The inner and larger cyst extended over the occipital and parietal lobes, parallel with the longitudinal fissure. The outer and smaller cyst occupied a position over the occipital lobe, but extended somewhat over the parietal lobe. The walls of the cysts were composed of thin lamina of membrane, and contained a clear fluid. Owing to the pressure occasioned by the cysts, extensive atrophy of the convolutions of both occipital lobes had occurred, but upon the left side the convolutions of the superior portion of the parietal lobe had also been decidedly involved in the atrophy consequent upon the pressure. This process was so marked that the surface of the left cerebrum was depressed half an inch below that of the right, and the entire left hemisphere occupied by the cysts appeared distinctly smaller. The close proximity of the cysts upon the left side to the fissure of Rolando, shows that the motor tract on this side of the brain must have been subjected to irritation by pressure during the development of the cysts.

Dr. Osler had seen several specimens of cysts in the brains of adults which were evidently, from the history, due to embolism or thrombosis, with secondary atrophy. Such defects may be large, and form examples of true porencephalus; but the condition which Heschel first accurately described, and to which Kundrat has devoted a large monograph, more often originates in fœtal life, either in an error of development or as the result of disease. Encephalitis, obliteration of arteries, traumatism at birth—any condition, in fact, which in the fœtus or child leads to destruction of brain tissue—may produce porencephalus. Kundrat states that in the congenital cases the arrangement of the convolutions about the defect is radial. Bilateral lesion, as in Dr. Bruen's specimen, is rare, but the convex surface in the neighborhood of the motor zone is the most common seat. Intellectual defects and spastic paralysis are almost invariable associations when the condition is congenital or due to disease during infancy.

PERISCOPE.

By Drs. M. A. STARR, G. W. JACOBY, N. E. BRILL, and LOUISE FISKE-BRYSON.

ANATOMY OF THE NERVOUS SYSTEM.

Note on the Ascending Antero-lateral Tract. H. H. Tooth, M.D. (Saint Bartholomew's Hospital Reports, vol. xxiii., 1887).

In this paper the author describes a small tract in the antero-lateral region or " mixed zone " (Flechsig) which was first recognized by Gowers, but which other observers have regarded as a part of the cerebellar tract. Bechterew and, lately, Sherrington have shown that this tract is composed of fibres which acquire their medullary sheath at a definite time in the developing fœtus. The lesion in the case consisted of a rapidly growing sarcoma, involving the membranes and compressing the cord from about the mid-dorsal to the mid-lumbar regions. That part of the cord involved in the tumor was completely disorganized and softened, as were also the lumbar and sacral regions. The sections were cut from the cervical region, which was in excellent preservation. The cord, after being cut into segments, was hardened in potassium bichromate, and was then stained in the mass, in Weigert's hæmatoxylin solution, for four days. They were then washed and kept in potassium ferrocyanide solution for four days. After careful washing they were dehydrated and saturated with solid paraffin. Sections made in this way showed the degeneration very plainly. All of the sections showed degeneration of the posterior median columns and degeneration in the antero-lateral tracts. The latter degeneration could be traced up to a level with the lowest part of the olive. The author goes on to state that the ultimate fate of the antero-lateral tract is a point of considerable interest, more especially as there is every reason to believe that its fibres are concerned in the conduction of pain sensations. "One thing seems certain, viz., that these fibres do not pass to the cerebellum *via* the restiform body. What, then, becomes of them? There are two possible ways in which they may terminate. They may have passed into the central part of the medulla, and thus become lost in the mass of fibres forming the bulk of the formatio reticularis, or they may have become connected, fibre by fibre, with the numerous ganglion-cells lying about in that part of the medulla, more especially with that group of them known as the nucleus lateralis. The nucleus lateralis is the remnant or upper termination of the lateral horn of the gray matter of the cord. If we accept as possible the conclusion that this tract is ultimately connected with the cells of the nucleus lateralis in the medulla, there is no reason why its fibres should not be received from time to time in its upward course into the cells of the lateral horn of the cord lower down. Thus we have some sort of anatomical evidence that the gray matter of the cord is concerned in the conduction of pain sensations."

PHYSIOLOGY OF THE NERVOUS SYSTEM.

Experiments on Special Sense Localizations in the Cortex Cerebri of the Monkey. By E. A. Schäfer, F.R.S. (*Brain*, x., 362, January, 1888).

It is well known that experiments upon animals regarding the visual area of the brain have led to different results in different hands. Ferrier was the first to localize this area, and he found it in the angular gyrus. Munk, Luciani and Tamburini, and Schäfer and Horsley all agreed in localizing the visual area in the occipital lobe, contending that the angular gyrus had nothing to do with vision. All cases of cortical blindness in man support the last view, and indicate that in monkeys bilateral hemiopia rather than unilateral blindness should be the effect of occipital lobe lesion. In the last edition of his work on "Functions of the Brain" Ferrier admits that the occipital lobe has something to do with vision, and admits that its lesion causes hemiopia, but he still claims for the angular gyrus an important part. It was in order, if possible, to finally settle this controversy that the present series of experiments was undertaken by Professor Schäfer and Dr. Sanger Brown. They seem to prove conclusively that the visual area lies wholly in the occipital lobe, each hemisphere being related to both eyes, so that a unilateral destruction produces bilateral hemiopia. They further show that the cortex of the angular gyrus has nothing to do with vision, but that beneath it passes the visual tract leading to the occipital lobe, injury of which tract was probably responsible for the effects observed by Ferrier (a probable explanation which was first offered by the reviewer, in 1884, in the *American Journal of the Medical Sciences*, and which has met with approval and adoption elsewhere since). These experiments ought to put an end to the controversy, since they bring clinical facts and experimental results into complete harmony.

The results regarding the auditory area are entirely negative. Ferrier located this area in the superior temporal gyrus, but Schäfer and Brown have destroyed this gyrus in six monkeys without in any way affecting hearing. They gently condemn any conclusions as to sensory areas from irritation of the brain, and depend on the results of destruction in their conclusions. It may be stated that very little, if any, reliable clinical evidence can be cited to show that the auditory area in man lies in the first temporal convolution; a case of L. C. Gray's, recently reported, having demonstrated that complete softening of both temporal lobes does not necessarily produce deafness. On the other hand, word-deafness is undoubtedly caused by such lesion in the large majority of cases; and recent anatomical investigations seem to indicate that the intra-axial course of the auditory tract ends in the temporal lobe.

The animals in which the entire temporal lobe was removed showed no evidence of loss of taste or smell.

In regard to these results, it must be stated that they do not harmonize with those of all the other observers, who unite in assigning these functions to the temporal lobe. In regard to tactile sensibility, Schäfer found that removal of the gyrus fornicatus caused hemianæsthesia of the opposite side, except of the forearm and hand in one monkey. This persisted for seven months after the operation. Any conclusion from a single experiment is not, however, warranted.

M. A. S.

THERAPEUTICS OF THE NERVOUS SYSTEM.

On the Treatment of Hydrophobia by Hyposulphites. Dr. A. H. Newth (*Maryland Medical Journal*, March, 1888).

Nearly thirty years ago Professor Polli, of Milan, suggested the use of sulphurous acid in cases of icorrhæmia. He proved by experiment that dogs who had putrid blood injected into their veins quickly died. But if hyposulphite of sodium was previously mixed with the blood they were not affected. Further, if the hyposulphite was administered to the dogs either before or immediately after the injection of putrid blood, they did not suffer.

I have used this remedy repeatedly in cases of blood-poisoning with most marked success. For instance, a patient has received a punctured wound, which has inflamed, the lymphatics have become swollen and reddened, the parts are extremely painful, and there are rigors. Within a short time after the exhibition of the hyposulphites the pain has decreased, the parts are less inflamed, and all the symptoms of poisoning have abated.

I find even children take hyposulphites readily, and I have never met with the slightest unpleasant symptoms from their use. Probably this may in some measure be due to the fact that I am in the habit of prescribing the hyposulphites in combination with bicarbonate of soda and sulphate of magnesia in peppermint-water. For children I simply give it with sirup and caraway-water. I would suggest a fair trial of this remedy, not only when hydrophobia has developed itself, but as a prophylactic. After a bite by a mad dog I would give five or ten grains of the hyposulphite of sodium or magnesium (the latter is richer in sulphurous acid) for the first three or four days every four hours; then three times a day for a week; then twice a day for another week; then every morning early for one month; recommending a Turkish bath twice a week. When the disease has developed I would prescribe the hyposulphite every hour or every two hours, with vapor or dry hot-air baths or prolonged warm-water baths containing some hyposulphite in solution. The hypodermic injection might also be tried, especially if the patient is unable to swallow.

THE TREATMENT OF NEURALGIA IN GENERAL PRACTICE. Dr. Gustavus Elliot, A.M., M.D. (*in a paper read before the Ninth International Medical Congress*, September 8, 1887).

Of all the drugs which have been recommended and tried in the treatment of neuralgia, three are invaluable. They are morphine, quinine, and iron. In acute attacks of marked severity, morphine is almost indispensable. It is preferably administered, if the patient is seen while severe pain continues, by hypodermatic injection. Given by this method, in doses of one-quarter or one-half grain, at the onset, or during the persistence of a paroxysm, the relief which follows is prompt, grateful, and often complete. Thus used, it is always palliative, and sometimes curative, a second attack never occurring.

It is, however, rather unusual to see the course of neuralgia immediately arrested by this treatment, if it is commenced after more than one paroxysm has occurred. In these cases it is desirable to prescribe, if possible, some remedy which is more directly curative. The drug which acts more positively in this direction than any other, which more justly than any other might be called a specific, is quinine. It should be given in large doses, and is of value in all cases, but especially in those which are markedly paroxysmal, and most of all in those which are dependent upon malarial poisoning.

In a considerable proportion of cases anæmia is not a prominent feature. The patients are well-nourished and full-blooded subjects. In them, neuralgia is an expression of an exhaustion of nervous force, perhaps due to cold or to over-exertion ; or of a toxæmia, perhaps malarial ; or of some reflex irritation. In the latter cases, the removal of the source of irritation is, of course, indicated. In the other cases, in connection with proper hygiene, including, as of highest importance, rest and good food, a combination of quinine and morphine is of the greatest value. In such cases, seeing the patient in the interval between the paroxysms—when, of course, the hypodermatic use of morphine is not indicated—it is often convenient to give the two drugs together. One drachm of sulphate of quinine and one grain of sulphate of morphine, having been thoroughly mixed, may be divided into twelve powders. Of these, one may be given two or three hours after each meal, and two (or more, if necessary) at once, one or two hours before the paroxysm is expected.

In another large class of cases anæmia is the predominant characteristic. These cases are often exceedingly obstinate, and likely to recur. For them, no drug is more valuable than iron When given continuously, as the quality of the blood improves, the neuralgic tendency grows weak.

As remedies of secondary value may be classed gelsemium and aconite. They certainly deserve to be ranked as valuable adjuncts to the remedies previously named. The danger of producing unpleasant toxic effects with either drug is, however, so great as to render it inexpedient to rely upon either as a single remedy in ordinary cases. The uncertainty in regard to the strength of the various preparations, as manufactured by different pharmacists, is also so great as to constitute a serious hinderance to the general use of either drug. Furthermore, the varying susceptibility of different patients introduces another element of uncertainty. I desire, however, to bear personal testimony to the utility of five-drop doses of the fluid extract of gelsemium, repeated every four hours, and given in connection with quinine and morphine. These three together will often give most favorable results.

As remedies of the third class may be mentioned arsenic, nux vomica, belladonna, phosphorus, and iodide of potassium. These have long been highly commended. I name them rather in deference to popular opinion than because I consider them of great value. I have used them all, but have failed to obtain with them results which warrant me in commending them. Arsenic, in particular, has been recommended by many authors. I have never seen very marked improvement follow its use. On the contrary, I have seen symptoms of considerable severity develop in spite of its administration in full doses, advised with the hope of preventing the attack. As an adjuvant of iron in cases of anæmia, it is unquestionably of value in curing the anæmia. Thus, indirectly, it may contribute to the cure of neuralgia. Similarly, nux vomica and its alkaloid, strychnine, are of use as stomachic tonics, when the general nutrition is impaired by indigestion.

External applications are of considerable utility as adjuvants to internal medication. Counter-irritants have been largely used. In obstinate cases, particularly of sciatica, blisters and cauterization sometimes do great good. In cases of moderate severity—as in intercostal neuralgia—sinapisms, applied either over the seat of pain or over the origin of the affected nerve, often afford some relief. Various sedative applications have some value as palliatives. I have frequently used a mixture containing one part of oil of gaultheria and two parts of olive-oil, or a combination of equal parts of oil of origanum, tincture of opium, spirit of ammonia, and olive-oil —known as Fahnestock's liniment—warmed and thoroughly rubbed along the course of the affected nerve and its branches, hot flannel being superposed, and have found that either will produce a palliative effect in many instances.

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Original Articles.

LOCOMOTOR ATAXIA CONFINED TO THE ARMS: REVERSAL OF ORDINARY PROGRESS.*

By S. WEIR MITCHELL, M.D.,

E. C. B., M.D., of California, aged 55. (Private Note Book; Case No. 1167.)

This gentleman was in active practice until two years ago. He has no constitutional disease of any description.

The patient, by his own description, was a well man up to July, 1887, possessing nearly perfect health. Near to that time he observed that, in writing with a steel pen, his hand began to change—becoming unsteady. There was no tremor, certainly none manifested in his hand writing. He only discovered that there was slightly increasing numbness in the cushions of the finger ends of both hands, extending through the hand, up into the arm, through the chest and down over the belly and back. About September 20th, this was slightly present in the toes, but more distinctly afterwards. There seems to have been no preceding eye or bladder symptoms, nor, indeed, any warning such as usually attends an outbreak of locomotor ataxia. The character of the case has not greatly changed since the first attack; it has only shown more decisively its nature. Perhaps the best description I could give of it would be that which I take from my note book of January 15th, 1888. To make clear what follows, I will state that the case is in my experience exceptional. I have seen none which I could place along side of it. It consists of an attack of locomotor ataxia affecting practically the upper extremities. In other words, the legs now show the same condition which one is apt to see in ordinary cases of this disease in the arms after the legs are pretty well on their course of degenerative change.

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* Read before the Philadelphia Neurological Society, March 26, 1888.

Nutrition.—This gentleman weighs 138 pounds; has lost 20 pounds; his height is 5 feet 9 inches; his limbs are thin; throughout the body the muscles are more or less flabby, notably in the upper extremities, but nothing to be called atrophy; the legs are liable to swell slightly during the day; the arms are more liable to be swollen after sleep.

The patient, an accomplished physician, declares the swelling not to be at any time œdematous. It offers slight resistance to pressure, and but a part of the swelling is due to serum.

There is an appearance of yellow pallor about him which caused me to request Dr. John K. Mitchell to make an examination of the blood. The result surprised me. The corpuscles amount to 3,980,000 per C. C. M.; the coloring matter is 75 per cent. of the normal, by Fleischl's test. Certainly the appearance of the skin would seem to indicate a want of blood not shown to exist by accurate test, repeated again and again. This condition of things I have several times seen in post. sclerosis, a not excessive want of color or corpuscles, and a whiteness of skin and mucous surfaces, which once we would have accepted as sure evidence of anæmia.

Sensation.—There is no pain of the character of neuralgia. He says that since his boyhood he has been liable to aches in the great trochanter and between the shoulders.

Touch.—At a half inch the compass points begin to be felt as two in the finger of either hand. There is no remarkable difference between the two hands. The sensation of numbness with which the disease began are, perhaps, more pronounced in the legs now than at first; but about the clavicles the sensation in all kinds appears to be normal; also it is fair for heat and cold, but decisions as to these are less rapid in the arms than in the legs. The pain sense is everywhere preserved. I ought to add that when the left hand is chilled it is apt to cause pain to run up the arm on that side. The special senses appear to be normal.

Dr. G. E. de Schweinitz gives the following statement of the eye conditions: "Vision in each eye with correcting glasses, normal; no disturbances of accommodation and his reading glasses allow comfortable range; 3 degrees of insufficiency of the interni; no other muscular anomalies, except slight drooping of the right upper eyelid. Pupils present normal reactions for light and accommodation; ophthalmoscope shows fine floating vitreous opacities; distinctly gray optic discs with hazy edges and general epithelial choroiditis. Form fields are normal; there is concentric contraction of the color fields. From these facts I conclude that there is beginning gray degeneration of the optic nerves, as well as the disturbances of the chroid and vitreous humor."

Motion.—Motion is generally weak; when he walks any distance he moves slowly, and in action the arms tire more easily than the legs. He stands well with his eyes open; but with his eyes shut he sways one inch to the right and an inch and a half forward, so that his station may be stated to be good for a man in his condition. It certainly shows how little ataxic trouble there is in the lower limbs. He moves the legs well, and the common actions of the feet exhibit little inco-ordination in these parts. On the other hand, the upper limbs are awkward in their movements. There is a great lack of co-ordination in every effort to make delicate adjustments, as when he attempts to touch his nose or ear with the eyes shut, or to bring the forefinger of his right hand in contact with the tip of the little finger of the left hand. He fails also in the test of weights; he is unable to tell the difference between an ounce placed in his palm and a half ounce, or an ounce and a half. I took tracings of his efforts to hold a pencil steady. They showed no tremor—less than is common, and this negative symptom is a remarkable one in ataxics. The effort to draw *a* from right to left resulted in a singular illustration of dejective co-ordination.

Electrical Reactions.—There are no unusual peculiarities.

Muscular Reaction.—All the muscles of the arms react to a direct blow, but are incapable of being reinforced by motion. The leg muscles have, as I judge, about the normal amount of response to the hammer blow, but are readily reinforced by hand motions.

It is interesting to contrast these conditions of the leg and arm muscles. Elbow jerk is lost on both sides, and non-reinforcible by motion or sensation. Knee jerk is *excessive* on both sides and is easily reinforced. Ankle jerk is also extreme, and when powerfully reinforced a slight clonus exists. Thus, if the tip of the foot be pushed upward so as to stretch the tendo Achillis, the blow on it results in two or three forward movements of the foot at the same time, the patient makes a powerful motor reinforcement. I remark in this case, as I have done in others, that a blow on the left patellar tendon gives rise to motion in the left leg, also to what seems to be a reflex jerk of the left arm. I suspect that this phenomena is due to the strong reinforcements produced by emotion; for, while it is frequently seen in the first interview with the patient, it may be impossible to evolve it at another sitting.

Secretions.—Generally normal; occasional diarrhœa; no painful gastric crises. There is no trouble with the bladder or rectum. Sexual power is lost.

Skin irritations of the soles of the feet appear to cause no motion. Testicle reflexes absent, as also abdominal reflexes.

Remarks.—This case stands alone in my experience. It is a locomotor ataxia beginning in the arms. These lose their tendon reactions, but show excess of direct mechanical excitability. In the legs we find excessive knee jerk and normal mechanical responses. The conditions seen in common cases of ataxia are here reversed. I have little doubt that in the earliest stages of this disease the tendon reactions are excessive.

PARANOIA: SYSTEMATIZED DELUSIONS AND MENTAL DEGENERATIONS.

AN HISTORICAL AND CRITICAL REVIEW,

By J. SÉGLAS,

ASSISTANT PHYSICIAN TO THE HOSPITAL OF BICETRE, PARIS.

Translated by WILLIAM NOYES, M.D.,

ASSISTANT PHYSICIAN TO THE BLOOMINGDALE ASYLUM, NEW YORK.

[*Continued from last Number.*]

THE acute form of *primary hallucinatory paranoia* begins with a prodromal period ot insomnia, irritability, or depression; but sensorial troubles (of hearing or smell) develop unexpectedly, together with sudden delusions of grandeur and of persecution, either combined together or alternating, and accompanied by excitement; a period of quiet may follow, but the hallucinations persist with ideas of poisoning, persecution, etc. This acute form may terminate in recovery or may pass into the chronic state. It may follow acute diseases, such as the puerperal state, hysterical or epileptic attacks, or the abuse of alcohol or morphine.

The *chronic form* is especially characterized by the persistence of the sensorial troubles, illusions, and hallucinations, with a fixed delusion of persecution. The most important variety is hypochondriacal *paranoia*. In fact, Mendel returns to the acute form, upon which he insists; and, on the other hand, he greatly limits the degenerative element in the systematized delusions, since he recognizes as a distinct form only the idiopathic (*originare*) variety of primary *paranoia*. Finally, he also admits *secondary paranoia* in his classification, but at the same time entirely transfers it to the second scheme.

In another work* he had already insisted on its rarity (five cases in one hundred and fifty). Yet he reports in his memoir three observations of secondary *paranoia* developed secondary to primary melancholic syndromes, and to all appearance reaching the full limit of their evolution. He describes the resemblance that exists between the delusional conceptions of melancholiacs and those of the systematized insanities. The difference is that the one class find in themselves the material for their complaints and accusations, while the others draw it from the external world.†

Meyser‡ (1885), returning to the study of the *hallucinatory delusion (Wahnsinn)* of Krafft-Ebing, whose ideas he fully shares, finds however that the expression of this author is not happy since alienists do not agree whether they ought to call *paranoia Wahsinn or Verrücktheit*, and, on the other hand, the actual language identifies *Wahnsinn* with *Verrücktheit*, while *hallucinatory Wahnsinn differs completely from paranoia.* It is therefore necessary to find a special title for this well characterized malady.

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* Mendel, Ueber secondare Paranoia (Berliner Gesellschaft f. Psych. und Nerven. Sitzung, 9 Avril, 1883.—Neurologisches Centralblatt, No. 5, 1883).

† At the conclusion of the reading of this paper before the Society of Psychiatre and Nervous Diseases of Berlin (April, 1883), a discussion followed at the session of June, 1883, which we think it will be interesting to resume, Jastrowitz said that he had never seen true melancholia change into systematized insanity; but he had seen some patients with systematized insanity at advanced period of their disease (dementia) have hypochondriacal melancholy symptoms.

Westphal, recognizing fully that melancholiacs generally accuse themselves, had also observed it in systematized insanity. It is not the character of the ideas but their genesis that is of greatest importance. As in Mendel's cases, a certain interval elapses between the existence of the melancholia and the time of the appearance of the ultimate systematized insanity, and it might be thought that the same individual had been successively attacked by different independent psychoses. For these very same facts, where the establishment of a direct connection appears to be clearly justifiable, are open to the following objections: where the systematized insanity has the appearance of taking its origin in a melancholia, hypochondriacal ideas are always prominent at the same time; now, these last have invariably constituted the point of departure of ultimate conceptions of the systematized insanity. Moeli held that the character of the ideas alone sufficed for the diagnosis between melancholia and systematized insanity. Mendel replied that it was the genesis of the conceptions that he insisted on. The interval that elapsed between the melancholia and the systematized insanity had never been in his patients a period of perfect health, and the new psychical complexus had been shown from the first week after the melancholic state. It is extremely difficult to demonstrate the psychopathic connection for each particular case, but one of them is clear (a female melancholiac began all at once to accuse her parents and to have later ideas of grandeur and of persecution); without doubt two of the observations gave evidence of hypochondriacal conceptions which dominated the scene, but who can determine the line of demarkation between pure melancholia and hypochondriacal melancholia? (See Arch. de Neur., 1884, No. 23.)

‡ Meyser, *Wahnsinn hallucinatorischer* (All. Zeitsch. f. Psych., Bd. xlii, 1, 1885): This would be a *general delusion* of the asthenic order, similar to the postfebrile psychoses of Kroepelin, sometimes acute, sometimes chronic, resembling

In this scheme, Meyser makes the hallucinatory mania of Mendel, the disorder in the hallucinatory ideas of Fristh, the disorder in the pseudo-aphasic ideas (Meynert, Schlangenhausen), and the disorder in the curable hallucinatory or acute primary ideas (Meynert and Fristh), identical in themselves with the acute *Verrücktheit* of Westphal, the acute systematized delusion *(Acuter Wahnsinn)* of Schaefer, the first group of acute partial insanity of Kretz,* the delusions of exhaustion of Voigt, and the case of primary systematized insanity of M. Buch.

Witkowski† (1885) devotes an entire treatise to the nosography of *Verrücktheit* in its connections with melancholic depression. The fundamental process of *Verrücktheit*, he says, is always the production of delusional ideas with tendencies to systematization. But, by the side of this process, certain phenomena from time to time may come in a permanent manner to occupy the first place, and of such a nature that it is necessary to reserve for them a separate place in the terminology, instead of inventing such terms as *hypochondriacal hallucinatory*, *stuforous*, and *melancholie Verrucktheit*, which are all forms that do not exclude one another, but may exist concurrently or succeed each other.

There exists also an *illusionary Verrucktheit*, in which

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the maniacal form of agitation with disorder in the ideas, and ideas of persecution, based upon multiple hallucinations and slight intellectual weakness, or that of periodic insanity.

* Kretz, *XVe Congress des Alienistes de l'Allemagne du sud-oest.* Session at Carlsrhue, October, 1882.

This group is characterized by the primary appearance of hallucinations or illusions, while in the second group it is the delusion that controls the scene, the hallucinations coming afterwards reinforce this and sustain it as in the chronic forms of primary systematized insanity.

†Witkowski, *Congrès annuel des Medicins alienistes Allemands.* Session at Baden, 1885. (Allg. Zeitsch. f. Psych., Bd. xlii., 6, 1886.

the hallucinations of hearing and the hypochondriacal complaints lose their importance in comparison with the forgetfulness of persons and things; while, at the same time, transiently or permanently, the depression may play a fundamental rôle. There exists, in addition, a form intermediate to true melancholia and *Verrucktheit*. These are the people permanently depressed (those who are constantly making negatives, the sceptics, the damned, and the ones who are rotting).

But in the majority of cases it is the *Verrucktheit* which constitutes in them the basis, which gives rise to the ideas, systematizes them, and brings about the abnormal conceptions and the modifications of temperament absolutely independent of the melancholia. The *Verrucktheit* brings about a condition of mental debility.

Such are these cases of systematized insanity, which are partial and stationary (among the persecuted whose intelligence preserves for a long time a high degree of vigor), and by the side of these cases are found some forms of progressive systematized insanity, generally tending to dementia. Very often also, among the congenitally weak, delusional ideas are found more or less distinct, sometimes reaching a high degree of systematization, at other times degenerating into absurd and fanciful creations. The author concludes that, in fact, *Verrucktheit* is an insanity with concrete permanent delusional ideas, with a tendency to systematization that is more or less clearly marked and more or less perfect. We may note here the work of Vejas* (1886) upon epilepsy and systematized insanity, and that of Schmidt† upon systematized morphine insanity, analogous to alcoholic. We have already seen these forms described by Gnauck, Moeli, and those distinguished by Krafft-Ebing.

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* Vejas, *Epilepsie und Verrucktheit* (Arch. f. Psych., Bd. xviii, 1, 1886).

† Schmidt (Arch. f. Psych., Bd. xvii, 1886).

[*To be Continued.*]

OCULAR SYMPTOMS IN DISEASES OF THE SPINAL CORD.*

By WILLIAM OLIVER MOORE, M.D.

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OF Nature's minute wonders the human eye is the paragon. But it is not the apparatus which the delicate knife of the anatomist reveals, the retina and lenses, or even their combined arrangement, that most strikingly indicates the subtile workmanship involved in the little fleshy globule we call the eye; it is the effects they produce, the purposes they subserve, the results they accomplish. Far greater are these than the careless crowd dream of, or the imaginative fully realize. The phenomena of sight is indeed sufficiently extraordinary; not less so are the minor missions which the visual organ fulfils. The eye speaks with an eloquence and truthfulness surpassing speech. It is the window out of which the winged thoughts often fly unwittingly. It is the tiny magic mirror on whose crystal surface the moods of feeling fitfully play, like the sunlight and shadow on a stream. How aptly has the eye been called the "window of the soul"; instinctively it is raised in devotion, and bent downward in shame. When enthusiasm lends fire to the soul the eye flashes; when pleasure stirs the heart the eye sparkles: when deep sorrow darkens the bosom the eye dispels hot tears; when confidence stays the mind, the eye looks forth proudly; when insanity desolates the brain, the eye roves wildly; and

> "O'er the eye Death most exerts his might,
> And hurls the spirit from its throne of light."

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* Read before the Section on Neurology, N. Y. Academy of Medicine, March 9, 1888.

Eye language is thus a part of our study in medicine, and should be more generally understood than it is, not in diseases of the brain and cord, but in general affections. The unfortunate know a friend and are reassured; the timid recognize a master spirit and are strengthened; the guilty know their accuser, and quail. Beware of the man whose eye you can never meet.

That the eye should show changes in the diseases affecting the brain, with which it is so closely connected, is clear, but why, in those situated in the spinal cord, it is not quite so easy to comprehend. Ever since the diseases of the "golden bowl" have been the subject of close study, ocular symptoms have been noticed, and of late years these same symptoms, associated with affections of the "silver cord," have claimed much attention and enquiry.

I regret that I am unable to make any new contribution to our knowledge of the pathological anatomy of these diseases, but hope that in the discussion it may be evoked.

In entering this vast and interesting field one must be very careful to make minute and accurate observations, especially in reference to pupillary signs, as Iwano has found that out of 134 young persons examined, 131 had assymetry of the face, and that the pupils were irregular in 122: and that the largest pupil occurred most frequently on the left side, which was usually the smaller side of the face.

We shall not attempt to cover the above field, which the title of the paper indicates, but only take up certain points that have always interested us.

Your attention will be more particularly called to atrophy of the optic nerve, and ophthalmoplegia interna, with but only a passing notice to ophthalmoplegia externa, and nystagmus.

Atrophy of the optic nerve comes chiefly under the notice of the ophthalmologist, while the affections of the iris more commonly under the observation of the physician.

The ocular symptoms in spinal cord disease are to be looked upon as associations and not as effects; and that they are also always the result of degenerative processes in the cord.

The usually slow progress of the spinal symptoms is evidence of this fact.

Among the many degenerative diseases of the cord it is strange that most of the eye symptoms should be with one of them alone, with tabes dorsalis. The fact that they are associations and not effects is clearly stated by Gowers in the following manner :

1. "That disease of any nature may exist in any part of the spinal cord without the occurrence of ocular symptoms, if we except the very rare paralysis of the dilators of the pupil in disease of the sympathetic tract in the cervical region.

2. "The ocular symptoms which may be absent when the cord disease is advanced may exist in extreme degree when such disease is in a very early stage.

3. "With the single exception of the sympathetic symptoms just mentioned, we know of no anatomical connection or functional mechanism by which the spinal cord disease can produce the ocular symptoms."

INJURIES TO THE SPINE.

Experimentally in laboratories, injuries to the spinal cord of animals have shown ocular symptoms, and these naturally lead us to expect similar results in man, where injuries of the spinal column have inflicted pressure on the cord.

Mr. Erichsen,* of London, has done much to bring this to notice, and all but considered it sufficiently frequent to establish a causal relation between the two events.

It is stated on authority, that Alexander the Great was in danger of losing his eyesight from the blow of a heavy stone on the back of the neck. Thirty-six per cent. of Erichsen's cases showed undoubted eye symptoms.

These are more commonly : difficulty of seeing in poor light to read, blurring of the type, floating bodies before the eyes, occasionally diplopia, with photophobia. The ophthalmoscopic appearances are usually negative.

Wharton Jones† considers the eye symptoms in these cases due to the disturbance in the cilio-spinal centre, and the sympathetic filaments springing from the dorsal and cervical cord.

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* Erichsen, "Concussion of the Spine," London, 1875.

† "Failure of Sight after Railway and other Injuries of the Spine and Head," London, 1869.

Other authors have reported severe eye symptoms from comparatively slight injuries of the spine.

I have seen but two cases of concussion of the spine, and in each of these eye symptoms were present in the shape of asthenopia. The fundus in each case was normal, and distant vision good, yet difficulty was experienced on reading; both were females, and each had loss of accommodation, that is to say, they could not use the ciliary muscle long without fatigue, and a convex glass had to be given to rectify this difficulty. In neither case was muscular disturbance of the extrinsic muscles of the eye found.

In Caries of the Vertebræ, when of traumatic origin we frequently find intra-ocular symptoms, and the ophthalmoscope usually shows a condition of engorgement of the vessels of the optic disc and surrounding parts.

Dr. Chas. S. Bull * reports the histories of eleven cases of traumatic caries of the spine, mostly occurring in the cervical, but some also in the dorsal and lumbar regions.

Seven had engorgement of retinal veins, with no changes in the arteries: in two choked disc was noticed, and in two anæmia seemed to be present, although the veins were enlarged.

I have examined fifteen cases of ocular disturbance in vertebral caries of traumatic origin, and found the following conditions: In two, optic neuritis, with swelling: three, hyperæmia of the disc, with blurred outline, but no œdema; eight had enlarged veins, and arteries normal in size or only slightly enlarged, the changes were symmetrical in each eye; two had floating bodies in the vitreous, with enlarged retinal veins; these changes are probably due to the sympathetic filaments springing from the dorsal and cervical cord.

LOCOMOTOR ATAXIA.

The frequency with which optic nerve atrophy occurs in tabes is differently stated; Leber found it present in 26 per cent., Gowers, in 20 per cent., and Nettleship, in 50 per cent.

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* Trans. American Ophthal. Society, vol. ii.

During the past ten years we have made notes of 80 cases of atrophy of the optic nerve, where no other history could be obtained, and of this number 32 had the signs of tabes as shown by the absence of the knee-jerk; 18 had ataxic symptoms when first seen. In examining the reports of many of the ophthalmic hospitals of this country, we are unable to tell the proportion of optic nerve diseases due to locomotor ataxia, owing to the method of recording diagnoses. Erb found only 12½ per cent. of his cases to have atrophy of the optic nerve; so that, although it is admitted by all writers as a frequent association, the percentage is at great variance. It has been said that the percentage given by the ophthalmologist is too high: I do not think so, but rather that it is due to the fact that the patient seeks advice for failing vision, when no signs of ataxia are yet present, and that the observers being on the alert for a cause of the optic atrophy, enter into an examination of the general condition of the patient and discover tabetic symptoms. I know this has been my personal experience in this direction. Atrophy of the optic nerve belongs to the more frequent of the complications of tabes; it commonly arises in the initial stage, and may be the first manifestation; the amblyopia produced by it may last for as many as ten years, before other symptoms of tabes appear, as in the case reported by Charcot. Gowers has also reported similar cases; in one the optic atrophy preceded the locomotor symptoms twenty and in another fifteen years. That optic atrophy ever occurs before the loss of the knee jerk, I am not satisfied; in all the cases under my observation it was absent—and I believe it is well recognized that the loss of the knee jerk may precede for a long time other locomotor symptoms.

When the disease was supposed to be situated and limited in the posterior columns of the cord, the association with it of a peripheral degeneration of the optic nerve was an anomaly. But since the recent pathological researches of Pierret, who has shown that the degeneration of the optic nerve is not the only peripheral lesion, and that that in the cord is not the only central change. He has shown that there is often an independent degeneration in the cutaneous nerves, commencing in the extremities, and that the optic nerve change is strictly analogous ; that there may also exist a degeneration at the central termination of the optic as well as of other cranial nerves, similar to that existing in the posterior columns of the cord. From his standpoint tabes is considered a "wide sensory neurosis," in the course of which the optic nerve atrophy occurs. Déjerine and Westphal have also enlarged our former narrow conceptions of the tabetic process. During the period when there is no affection of the patient's gait, and only the loss of the patellar reflex, unsteadiness on standing with bare feet close together, with closed eyes, and with lightning pains, there is no doubt that optic atrophy often commences and advances to a considerable degree, and according to Gowers, it occurred during this period twice as frequently in this than the later stages of the affection. In my cases, 14 out of 32 had only loss of knee jerk and rheumatic pains in the legs. It is rare for it to occur when the ataxia is so great that the patient cannot walk.

The atrophy usually begins in one eye before the other, and may reach a considerable degree before the fellow-eye suffers. The immediate cause of the amblyopia in tabes is the gray degeneration of the optic nerves—a degeneration similar in all respects with the changes in the spinal cord. One disease begins at the periphery of the trunk of the optic nerve, and gradually attacks the central fibres ; it always begins on the trunk of the nerve or the portion nearest the eye, and from thence toward the optic tract. The narrowing of the visual field would lead us to this conclusion, had not post-mortem changes already shown this condition.

The disease of the optic nerve manifests itself by diminution in the acuteness of vision ; the field which is at first normal becomes gradually narrower, the field being irregularly contracted. The color field is usually limited before the visual, the perception of green being lost first, then red, and then yellow and blue. Ophthalmoscopic examination shows in the early stages a dirty-looking nerve, which is indicative of a low grade of neuritis, and this continuing produces the final full marked appearances of white atrophy of the optic nerve, with narrow arteries and other parts of the fundus appearing normal.

The optic nerve atrophy occurring in tabes is usually progressive, and leads to complete blindness, though I have seen two cases where vision was not completely destroyed. And one may observe no ophthalmoscopic change in the fundus, and yet amblyopia of a high grade present ; in such cases doubtless degeneration is retro-bulbar.

The next most frequent ocular symptom in tabes is the pupillary change. Of the four muscular actions, contraction of the pupil on stimulation of the optic nerve, contraction of the sphincter, in association with that of the ciliary and internal recti muscles, and contraction of the dilator fibres of the iris on stimulation of the skin, and contraction of the ciliary muscle on accommodation, some or all may be lost in association with spinal disease. These changes depend upon or leave three centres capable of separate action, all of which probably lie in the tract beneath the aqueduct of Sylvius. Experiments made by Hensen make it likely that the anterior portion of the tract governs accommodation, and the centre next behind it the reflex contraction of the iris. On the outer side of the latter is a centre on which depends the reflex sensory dilatation of the pupil. The efferent path of the two former are through the third nerve. As yet we know little as to the centre for the contraction of the iris which is associated with accommodation, not knowing whether the nucleus for the ciliary muscle is connected with the mechanism for contraction of the pupil at the centre, or in the lenticular ganglion, or in the ganglionic mechanism within the eye. It is more probable that the connection is in the lenticular ganglion.

The path by which stimulation of the skin causes reflex dilitation of the iris is circuitous. The afferent impulse reaches the centre by the cervical part of the spinal cord when the skin of the neck is stimulated, and the efferent impulse descends the cervical cord thence to the superior thoracic ganglion of the sympathetic, and then ascends the sympathetic to the eye.

These pupillary symptoms are as common in tabes as they are rare in other spinal diseases.

The most common being the loss of reflex action to light, while the pupil still contracts to accommodation, the "Argyll-Robertson" pupil or reflex iridoplegia.

Associated with this symptom is also a loss of the dilitation of the pupil on stimulation of the skin. Next in frequency, but very much less common, is paralysis of all the intrinsic muscles of the eye, the ophthalmoplegia interna of Hutchinson.

The rarest is loss of accommodation, cycloplegia, without loss of reflex action.

If we embrace all of the pupillary symptoms, both slight and transitory, and the more profound, we will find them present, according to Erb, in more than one-half of all tabetic cases. Gowers, in 72 cases of primary degenerative ataxia, found the internal muscles of the eye affected in 92 per cent.

The percentage of his cases with pupillary signs was in the first stage 84, in the second 93, and in the third 100.

In forty-one of our own cases already spoken of, the "Argyll-Robertson" pupil was present.

When the light reflex is lost the pupils are often small, but not necessarily so.

When there is loss of accommodation they are rarely very small, less than 2½ mm., and are often 4 or 5 mm. in diameter.

The reflex dilatation of the pupil when the skin is stimulated is a phenomenon closely allied to the contraction of arteries, which may be produced in animals by the stimulus of pain. The dilatation of the pupil may be obtained by irritating the skin of the face or the neck ; it is double, that is, the stimulation of one side causes dilatation of both pupils.

The skin reflex is usually absent when the light reflex is lost ; the skin reflex may be retained when cycloplegia is present.

Dr. Hughlings Jackson has reported a case where vision was absolutely lost by optic atrophy, in whom the "Argyll-Robertson" pupil existed, and when the patient "made believe" look at the clouds the pupil enlarged, and contracted when he made the effort to look at a near object.

In examining the reflex dilatation which accompanies stimulation of the skin, precaution as follows should be observed: the eye should be shaded from a glare of light, voluntary movements on the part of the patient should be arrested during the test, as any movements being made the pupils being shaded will cause dilatation of the pupils.

Urthoff* found reflex immobility of the pupil, combined with preservation of reaction on convergence, in 67 per cent. of all tabetic cases, and also the pupils were found to be unequal in one-fourth of the cases; one-sided cycloplegia was found in only five out of 166 cases. Reflex immobility of the pupil, without reaction on convergence, was found in thirty cases.

We must not omit the paralysis of the externe muscles of the eye, that are so commonly seen in tabes, usually either the abducens or the motor oculi, and rarely the fourth nerve, which give rise to various symptoms of dizziness, diplopia and strabismus. Graefe has pointed out that tabetic patients show little disposition to fuse the images in binocular vision, and this is taken as sign of the central origin of the affection. These muscular paralyses are frequent in the early stages of the disease; they are transient often in character, and this fact has not been very readily explained. Ptosis is also present when no other branch of the third nerve is involved.

In 203 cases of tabes collected from various sources, paralysis of ocular muscles occurred in 52, or over 25 per cent.

GENERAL PARESIS OF THE INSANE.

In this disease ocular symptoms are quite prevalent and in great variety. Mr. W. B. Lewis, of Wakefield, England, has collected the histories of sixty patients, with the following result: Loss of reaction of the pupil to light, 78 per cent.;

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* Berlin, "Klinisch Wochenschr." 1886, No. 3.

movements on accommodation, associated iridoplegia, 43 per cent.; reflex dilatation to cutaneous stimulation was lost in 63 per cent.; complete fixity of the pupil, without impairment of accommodation, was found in 15 per cent.; in 7 per cent. of ophthalmoplegia interna occurred, although it was rarely complete as regards the ciliary muscle.

He summarizes as follows:

1. A loss of reflex dilatation to sensory stimulation occurs in a very large proportion of cases.

2. Reflex iridoplegia (loss of action to light) next to the preceding is the most frequent accompaniment of the disease.

3. Complete loss of movements of accommodation occurred in 25 per cent.

4. Cycloplegia associated with the latter in four of the 60, (more or less complete) and was found only in the advanced stages of the disease.

And he concludes that the sequence of morbid phenomena occurring in the iris, is as follows:

Paralysis of reflex dilitation to cutaneous stimulation, reflex iridoplegia probably shown at first by an initial contraction, followed by dilitation under full focal light, and passing into a later stage of immobility, and occasionally complete, ophthalmoplegia interna. Optic nerve atrophy occurs, and in 22 cases examined by Dr. Lawford, London, Eng., three were found thus affected.

Nystagmus is associated with tabes, but is a rare symptom in this affection, and is usually found in multiple-sclerosis; it has been ably set forth by Friedreich, who states that it is always bi-lateral, and that the movements of the eyes are rotatory and irregular, as seen in the case presented to the Society this evening:

Richard Purcell, æt. 42, watchman. Sent to me by Dr. A. C. Coombs, of Newtown, N. Y., Feb. 13, with the following history: That for the past fourteen months he has noticed a difficulty with his eyes; that they "jump" and annoy him very much by causing objects to move rapidly in front of him. Has no pain. In 1872 he was struck on the left shoulder by a falling telegraph pole; this knocked him over and caused a severe contusion of the arm and shoulder, so that he could not use it for many weeks, and he never has had freedom from pain in it since, although he has moderate use of the member. Has not used tobacco since 1886, but has used alcohol in moderation.

Present condition, Feb. 13, 1888, well nourished and all the functions good. R.$=\frac{15}{15}$, L.$=\frac{15}{30}$—2.5 D,$\bigcirc$—2.75 D, cy. ax. 30°. Abduction, 8°; adduction, 15°; no vertical deviation by the prism test, nor hyperphoria at 20 feet. With the correcting glasses in position the nystagmus is the same as without them. There is no contraction of the visual or color field as tested by the perimeter.

Both eyes show a marked nystagmus both around a transverse as well as an antero-posterior axis, which gives the eye a peculiar, swimming appearance. When the patient looks downward the ocular movements cease, as they also do when he fixes the eyes on a given object, but as soon as they are removed the oscillations begin with marked vigor.

Pressure on the nape of the neck by the fingers, or throwing the head back so that the neck impinges upon the collar, causes the motions of the eye to cease. When lying in bed the patient states the eyes are quiet.

The fundus in each eye shows a dirty-looking optic nerve, but one which would not be considered abnormal by many; yet I am inclined to consider the nerve in a state of neuritis of a low grade which will produce atrophy; the ophthalmoscopic examination is very difficult, owing to the rapid movements. The pupil responds to light and accommodation.

The knee-jerk is present so far as my testing has proved; the patient complains of swaying while walking, and that his feet do not have the proper sensation on touching the floor. I present him as an example of nystagmus probably due to multiple sclerosis, from the injury of the shoulder years ago.

It does not occur during rest, but always on an attempt to fix the eye.

Frederich considers it due to a form of ataxy of the movements of the eye, and speaks of it as ataxic nystagmus, and the cause to be due to a disturbance of the co-ordinatory tracts, which lead from the centres of co-ordination to the nuclei of the nerves of the ocular muscles lying on the floor of the fourth ventricle, and that it does not occur in spinal disease until the medulla is involved.

Pierret explains these movements on the ground of the primary disease of the sensitive root tracts of the trigeminus in the medulla.

Nystagmus is a very common symptom of multiple sclerosis, and it is increased by any effort of the will or any violent emotion ; according to Charcot it is met with in one-half of the cases.

In multiple sclerosis of the cord, we have temporarily a permanent diplopia due to paralysis of the various ocular muscles ; amblyopia is also observed, but it rarely, as in tabes, leads to positive blindness, with optic nerve atrophy, although in rare cases it does ; the difficulty is probably due to nodules of sclerosis in the optic tracts and optic nerve.

The paralyses of the external muscles of the eye due to spinal cord disease, we will reserve mention of till a future occasion, as the subject of ophthalmoplegia externa is of much scope and interest.

ATAXIC LATERAL SCLEROSIS.*

By J. G. PRESTON, M. D.,

PROFESSOR OF GENERAL MEDICINE IN THE BALTIMORE POLYCLINIC.

UNDER the names *Ataxic Paraplegia*, *Combined Sclerosis*, *Mixed Sclerosis* &c., have been described a quite definite group of symptoms for which I would suggest the term *Ataxic Lateral Sclerosis.* Since the time (1875) that Erb, and soon after Charcot, described *primary lateral sclerosis*, cases have been reported, possessing most of the characteristic symptoms of this disease, with certain additions. Some of these cases fell under spastic paraplegia, others again were described as abnormal cases of *posterior sclerosis.*

No account of this disease will be found in works on general medicine, and few writers on nervous diseases (if we except to R. Gowers, who has admirably described it) have done more than alude to it.

The disease appears to be more common in men than in women, and begins usually about the thirtieth year. So far as has been observed it does not follow any neurotic tendency, and appears to be entirely unconnected with syphilis.

Most writers suggest over exertion, sudden cooling of the body, excesses, &c., as probable causes. In the case which I will relate, over exertion was the only assignable cause. It is not improbable, as has been suggested, that some cases of sclerosis are really traumatic, produced by the rupture of spinal arteries, with secondary degeneration.

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*Read before the Clinical Society of Maryland, Mar. 16th, 1888.

The symptoms of the disease are usually easy to bring out. There is a history of a tired, weak feeling, beginning in the legs, which lasts for months. The patient notices that he is more easily fatigued, and that his legs feel stiff on rising. Often there is a good deal of ataxia present at this stage, and the history will show that the patient complains of staggering when he attempts to walk at night, and has to keep his eyes on the ground.

As the disease progresses spastic gait becomes more and more marked. Owing to the over action of the gastrocnemius the heel is drawn up and the toe thrown forward. The patient is thus obliged to adopt a "waddling" gait, to prevent the toe dragging. Going up and down stairs is very difficult, and rough ground makes progress very tedious. Sometimes, however, this spastic gait is somewhat modified by the ataxic symptoms. There is no *girdle* pain, or *lightening* pains, so common in posterior sclerosis, though there may be sensations of numbness, and slight pains in the legs. Sexual power and control over the sphincters is usually much impaired. The reflexes, superficial and deep, are greatly exaggerated. The knee jerk is much increased, and ankle-clonus is readily obtained. Usually the pupils react normally. As the disease advances the spastic conditions become more marked, until the slightest attempt at motion causes a spasm of the extensor muscles. Finally paralysis more or less complete comes on. The arms are rarely attacked, but may show to a less degree the symptoms so characteristic in the lower extremities. As illustrative of this disease I will relate the following case, now under my care.

R. M., æt. 33, engaged in putting up stoves, ranges, &c. No neurotic history; never had syphilis or rheumatism; has always been healthy; worked hard since boyhood, and in the course of his business had much heavy lifting.

About two years ago he noticed that his legs were getting weak—had slight pains and numbness. Continued to get gradually worse; noticed that he staggered when attempting to walk at night. Sexual power has now almost disappeared, and he has lost perfect control over bladder and rectum ; often has cramps in his legs ; no pain or tenderness over spine ; no girdle sensation. Sways slightly when standing with eyes closed, and walks with difficulty. Muscles are well nourished, and react rather more than normal to Faradaic current. Gait spastic ; drags his toes ; says he always wears out his shoes at the toe—right leg rather more affected than left—no impairment of sensibility; knee jerk much exaggerated, and ankle-clonus well marked.

In this case, as often happens, the ataxic symptoms, prominent in the early stages, are obscured to some degree by the spastic and paralytic phenomena.

In this particular case the ataxic symptoms are in the background, while the spastic phenomena are prominent. The sensory and ataxic disturbances, and the loss of control over the sphincters would tend to discredit the notion of pure lateral sclerosis, while the absence of girdle pain, or in fact any localized pain, together with the fact that the symptoms appeared simultaneously on both sides would make against the diagnosis of chronic myelitis.

While this disease is hybrid, so to speak, and should not be considered as a perfectly distinct affection, still it is convenient for diagnostic purposes to separate it from the two forms of sclerosis, *posterior* and *lateral*, which it so much resembles.

The diagnosis is to be made between the affection we have been considering and which we would call *Ataxic Lateral Sclerosis*, and several diseases of the cord. Chronic *myelitis* and slow compression often present symptoms similar to the ones here described, but in these conditions we have nearly always localized pain, girdle pain, marked interference with sensation, and after a period of a few months tendency to recovery or marked destructive lesions. Posterior sclerosis presents, at least during the greater part of its course, no paresis or spasmodic phenomena, marked pupillary reactions, lightning pains with trophic changes and other marked special symptoms. Simple, primary lateral sclerosis shows no ataxia, no changes in sensory conduction, and according to Erb, Charcot, Ross and others, no interference with sexual powers and no loss of control over the sphincters. There is another form of sclerosis, which from the nature of the lesion, is often impossible to distinguish from ataxic lateral sclerosis, namely, disseminated, or multiple sclerosis. The sclerotic patches may of course occupy any position, and consequently give rise to a great variety of symptoms. One of Charcot's cases, diagnosed ataxic lateral sclerosis, proved on autopsy to be a case of very general multiple sclerosis.

The autopsies on patients dying from this form of sclerosis have not been numerous, but have gone far to bear out the clinical picture. Westphal, in a very typical case, found (Archiv. f. Psyc. Bd. XV.) post-mortem, sclerotic degeneration in the lateral columns and also in the columns of Goll. Hamilton (N. Y. Record, XV, 1879), Omerod (Brain XII, 1885), Dana (Med. News, 1887-1), and a number of others have reported autopsies of cases presenting the symptoms enumerated above, in which were found degenertion of both lateral and posterior columns.

The degeneration is not always found to be strictly systemic, but may spread over adjacent regions to a certain extent. Generally the most marked sclerosis is in the dorsal region in the pyramidal tract, and in the columns of Goll. The posterior root zones are rarely much affected. The direct cerebellar tracts may or may not be included in the degeneration. Of course there may exist great variey in the extent and intensity of the process, just as we see in most other forms of sclerosis, and secondary degeneration may follow the primal lesion.

The prognosis of ataxic lateral sclerosis is very favorable as regards life. It is the most chronic of all the scleroses, and rarely ever of itself produces death. It may apparently be arrested in its course, and cases have been reported in which improvement and even recovery have taken place. Generally speaking, however, we can rarely hope for cure in this more than in other forms of sclerosis.

As to treatment, rest, warm baths, arsenic, massage, calabar bean, bromides and general tonics are to be recommended.

The bladder must be carefully watched, and the general health of the patient attended to. Often a sea voyage proves beneficial. Electricity is probably hurtful and if used at all should be employed cautiously.

The patient whose case I have reported has certainly improved on arsenic, warm baths and massage.

I have refrained from describing minutely the symptoms or pathology of this disease, since they are both well-known or at least have been often described in the two forms of which this affection is the hybrid. I wish simply to call attention to this interesting variety of sclerosis.

Society Reports.

PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, February 27, 1888.

THE VICE-PRESIDENT, CHARLES K. MILLS, M. D., IN THE CHAIR.

DR. WILLIAM OSLER reported the following case of

ENLARGEMENT AND CONGESTION OF THE RIGHT ARM FOLLOWING EXERCISE OF ITS MUSCLES.

J. B., aged forty-eight, white, a robust, vigorous-looking man, applied at the University Hospital on Novemebr 4, 1887, complaining of swelling of the right arm when he worked.

His family history was good, and he had always been healthy with the exception of several attacks of sciatica, which he had suffered with lately. He was married and had five children, all healthy. When he was young he had had the smallpox. He entered the navy in 1861 and left it in 1863, and has been a storekeeper ever since. He had gonorrhœa in 1858, but there was no history of syphilis. About two days before he applied at the hospital he began to work as a carpenter, but had to give it up in two or three days on account of the swelling in the right arm. The arm also became tender and blue, but on raising it over his head the swelling and dark color would disappear. There was no history of sprain or injury, and otherwise he appeared to be in excellent health.

Before exercise the measurements were as follows: Right arm, 11.4 inches; forearm, 11 inches; left arm, 10.25 inches; forearm, 11 inches.

The patient was a well-developed, well-nourished man, with chest well formed, sternum a little prominent over manubrim, subcutaneous veins somewhat prominent, those over the anterior aspect of the left shoulder more than the right. His pupils were equal. The heart apex beat was just visible below and inside the nipple. There was dulness at the lower border of the fourth inside nipple line, and the right border of the sternum. The sounds at the apex were clear; at the base somewhat feeble first; second distinct, not specially ringing or metallic; and not more distinct toward the right than the left clavicle; no pulsation on the vessels of the neck and no murmur. Radials were equal; no atheroma; no increased tension, and no stiffness in the vessel wall were apparent. The right arm and hand looked larger than normal; and when at rest the skin was the same color in both; the muscles were well developed; the panniculus was the same on both sides. There was no distention of the veins, and no œdema. The brachial artery could be easily felt, and the axillary artery was not very deep on either side. No difference in the pulsations of the two axillary arteries could be detected, and no murmur could be heard in either.

After exercise the measurements were as follows: In a few minutes the right arm measured 11 inches; forearm, 12½ inches. It became livid; there was capillary injection, and the distended veins felt like cords. The radial pulse was only just perceptible. Auscultation of the axillary gave a distinct murmur not audible on the other side. When the arm was held up the congestion would disappear in a few minutes. There was a feeling of fulness, but no pain. Deep percussion in the upper axillary region was negative. There was no enlargement of the axillary glands; the expansion at the apices was equal. No pain was experienced on deep pressure in the neck. The side of the face did not flush with the arm, and the opposite side was not affected. Percussion was the same on both sides behind.

Dr. Osler wished to place this case on record, as it presented very unusual features. He thought at first that the condition was due to obstruction, perhaps from pressure, on the subclavain or axillary veins, or possibly occlusion with reëstablishment of a collateral circulation sufficient for the ordinary blood flood which occurs during active use of the muscles. There was, however, no evidence whatever of any such obstruction, nor were the veins of the shoulder-girdle distended. The glands were not enlarged ; there was no subclavicular dulness, or evidence of disease at the right apex. The condition had remained unchanged during the four months in which the patient had been under observation. He was now more inclined to believe the condition one of faulty innervation of the vessels of the arm during exercise, a defect in the local regulating mechanism controlling the supply and outflow of the blood, the circulation of which is, as we know, enormously increased by contraction of the muscles.

DR. JAMES HENDRIE LLOYD presented the following

REPORT OF A CASE OF RAPIDLY FATAL EXOPHTHALMIC GOITRE.

On August 12th he was summoned to a case which was described as cholera morbus. The patient was a single woman, aged thirty-nine, who had been suffering for some hours with diarrhœa and vomiting. Pulv. opii and plumbi acetat. were prescribed. On the following day the diarrhœa was checked, but obstinate, persistent vomiting continued. This vomiting was quite remarkable, allowing not a morsel of food or even a small quanity of water to remain on the stomach. A careful physical examination failed to detect any direct cause for this. There was a slight tenderness in the epigastrium, but no hardness or evidence of any growth ; no enlargement of the liver or spleen ; no evidence of hernia ; no abdominal pain, or any tympanites. A very marked derangement of the vascular system was, however, detected. The whole abdominal aorta to its bifurcation was throbbing intensly, with a pulsation somewhat expansile. The heart's action was rapid, but there was no intracardial murmurs. It was now observed that the patient's eyes were very prominent and staring, which symptom had escaped observation at the first visit on the preceding evening (possibly due to the poor light in the room). Further examination revealed a much enlarged and rather soft thyroid gland.

Careful inquiries were now made of the parents and near relatives about the patient's previous health. They had regarded her as a perfectly healthy woman up to this attack. They had, to be sure, noticed the altered appearance of her eyes, but it had not made any impression upon either them or her, and had not been of long duration. She had had a spell of illness about six months previous, the exact nature of which he could not determine, but it had been accompanied with some irritability of the stomach and prostration (as she recollected). Since then she had been apparently healthy, bright and active ; had not complained of dyspnœa ; and had been out on the day of the onset of the so-called "cholera morbus," for which he had been called to her. The vomiting continuing as the most distressing symptom, a carefully regulated diet was ordered, and tinct. ipecac. gtt. ij was ordered every hour. The pulse was beat-as high as 100 per minute.

The patient passed a restless second night. In the morning the vomiting was somewhat relieved, but not gone. The prostration was growing worse, and the case had assumed a threatening aspect. The pulse had risen to 170. Cyanosis of the hands and feet were observed. Consultation was now held with Dr. W. G. Porter, who concurred in the diagnosis. Calomel was ordered in one-half grain doses every hour ; ex. opii, gr. ½, by suppository every two hours until quieted ; digitalis, which had not been given because of the extreme gastric irritability, was also determined upon ; brandy, as much as the stomach would bear. At the evening visit the pulse was found, as nearly as it could be counted, 70 to the third of the minute, 210 per minute. The vomiting had ceased. Ordered one-half ounce of milk, with which tr. digitalis, ♏. x, was to be put, every half hour, the digitalis to be given only every hour after the fourth dose. Collapse was imminent ; the patient has had several fainting spells. On the morning of the third day the pulse had fallen to 160, apparently under the digitalis. Dr. Porter again saw the patient. Ex. bellad., gr. ⅓, was added to the suppositories. Later that day the patient's pulse again failed. At the last visit the heart was beating in a very tumultuous way, and no pulse was perceptible at the wrist. The mind was perfectly clear. She died at the end of the third day of her ilness.

The autopsy in this case was, unfortunately, not conplete, owing to the jealousy of the family, who refused to allow the skull-cap to be removed. He therefore reported it more for its clinical interest than for any light it throws upon the pathology of this interesting but obscure disease. The examination was made by Dr. Dock, in the presence of Dr. Porter, Dr. J. H. Musser, and himself. The heart was a little dilated in the left ventricle, and slightly hypertrophied. The lungs, kidneys, and suprarenal capsules were normal ; the liver was not abnormally fat. The stomach was slightly congested, due probably to the excessive vomiting. There were no other abnormal appearances in the chest or abdomen, which were alone opened.

It may be noted that this patient had had urticaria, a fact recorded by Dr. Bulkley* in two cases of the same disease. The special interest which attaches to this case, however, is its very acute character and rapidly fatal termination ; for while there was some evidence of the disease having been making insidious approaches for several months, it had not impressed its presence upon the notice of either the patient or the family until it culminated in a violent crisis, which quickly deprived the woman of her life.

DR. WM. OSLER said that these acute cases of exophthalmic goitre are rare. Were any mental symptoms present? Recently, in Dr. Henry's ward in the Philadelphia Hospital, was a case of this disease, in which acute symptoms suddenly developed, and death took place with marked mental trouble.

DR. J. H. LLOYD said that in his case the mind was perfectly clear, nor was there the slightest hysterical character about the symptoms. The patient was naturally much agitated over her condition.

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*Hammond : Diseases of the Nervous System, p. 812.

DR. W. B. JAMESON made the following remarks on a case of

CARDIAC ANEURISM FROM AN INSANE MAN.

The heart which he exhibited was from an insane man who died in Dr. Lloyd's wards in the Insane Department of the Philadelphia Hospital ; and it was through his courtesy that he was able to present it. The man was fifty-six years of age ; a native of Switzerland. He was probably a priest, but for several years before admission to the hospital was engaged as a teacher. He had no friends in this country, and it was impossible to obtain a definite history. He was arrested on the streets and sent to the hospital. When admitted he was in an excited state ; had evident delusions of persecution, believed that his food was being poisoned, and also had hallucinations of sight and hearing, and would often preach, and sometimes chant. His physical condition was good. No physical lesions were discovered on admission. The heart sounds were clear, distinct, and without murmur. It was thought that there was possibly a pleural friction sound, but this was not definitely determined.

When Dr. Jameson went on duty in the Insane Hospital six months ago, the man was apparently in perfect health. He was quiet, speaking pleasantly when spoken to, but nothing more. He was well advanced in dementia, although perfectly cleanly in his habits. One day while Dr. A. A. Stevens and Dr. Jameson were in an adjoining ward, they were summoned to see this man who was said to be dead or dying. They found him lying on the floor absolutely pulseless, face white, extremities cyanotic and cold, and respiration gasping. Brandy was given and ether hypodermatically. He was then put in a bath with the temperature of the water at about 117°. He shortly improved, became quite rational, and talked clearly. He complained of no pain whatever. He was kept in the bath three-quarters of an hour, when he began to sink, and was taken out and soon died.

Three hours after death the autopsy was made. The brain showed nothing special. There was nothing of interest found in the abdominal cavity. When the first costal cartilage was cut,.blood gushed out, raising a distance of at least two inches. The whole mediastinal space was filled with fluid and partially coagulated blood. Extensive pleural adhesions, more particularly on the right side, were found. The space that was left was filled with blood. The total quantity was estimated at about two pints. The aorta was examined carefully with the parts in place, but nothing abnormal was found. The pericardial sac was found to be one-third filled with fluid blood. An aneurism was found at the apex of the right ventricle, and just at the septum there was a small opening where a rupture took place. The opening in the pericardium was not positively located.

In looking over the literature, Dr. Jameson found but one or two references to the subject. One case of rupture of the left ventricle was reported in the *American Journal of Insanity* for January, 1885. In this article reference is made to three other cases which had been reported in the *Edinburg Medical Journal* for February, 1884. In all these cases the patients were about seventy years of age.

In this case there was no atheroma of the aorta. There was an ulcerated condition along the lateral border, also on a spot about the epicardial surface, three-fourths of an inch broad by one inch long, which presented the same appearance as the aneurism. Whether this spot was connected with the ulcer inside he could not say.

DR. WM. OSLER said that it was very unusual to find aneurism of the right ventricle of the heart. This condition is most common on the left side. The majority of aneurisms are the result either of local weakening, due to pericarditis, which is the less frequent case, or to a fibroid condition of the left apex. There is one other condition which may lead to the rapid production of aneurism of the heart, viz., myomalacia or anæmic softening, due to arterial changes. He would suggest that sections made from this locality might throw some light on the nature of the condition. The fact that we have in the immediate vicinity of the aneurism another spot of disease, would seem to point in this direction.

DR. J. MADISON TAYLOR exhibited a

NEW FORM OF PERCUSSION HAMMER

devised to serve as nearly as possible all ends for which a hammer is likely to be-called into use by clinicians. This feature at least would, he thought, commend it. In shape it is a cone flattened on the opposite side, with apex and base carefully beveled or rounded, of about the thickness throughout of the human index finger. The material is moderately soft rubber. It is held by an encircling band of metal midway between the apex and base transversely, and from it, on the edge, depends the straight handle. The handle is rigid though light, it being Dr. Taylor's opinion that this had better be under the full control of the wielder. If elastic, as recommended by some, an element of uncertainty enters in the degree of force used in the blow. The special feature of this hammer is that the shape of the striking surface is like the outer aspect of the extended hand, palm downward, which is most often used in obtaining tendon jerk. The rounded apex end is adopted to reach the biceps tendon at the bend of the arm. The last has become important in diagnosis. In fact, in the light of recent investigations, the study of tendon jerks of knee, biceps, and Achillis tendons, of the jaw jerk as discovered by Dr. Morris Lewis, has become a valuable addition to our means of unravelling the causes of nerve maladies. The matter is even now engaging the attention of the foremost writers—Jendrássik, Weir, Mitchell, Lewis, Lombard, and others. Especially is this true now that the field is immensely widened by the corroborative symptoms of reinforcement.

This little tool will also well serve to elicit chest sounds, to percuss the abdomen, and in fact, is useful whenever an elastic hammer is needed. The material being of soft rubber, the blow does not hurt the intercepting fingers as does the hammer usually employed to strike the tendons and muscles. It is made by Snowden, of Philadelphia.

DR. CHARLES A. OLIVER made

A FURTHER DEMONSTRATION OF WERNICKE'S HEMIOPIC PUPILLARY REACTION.

It was his intention to present a patient with well-marked left homonymous hemianopsia, in whom the hemiopic pupillary inaction of Seguin could be beautifully seen, but this was not possible. He gave a rough demonstration upon the blackboard of a few observations worthy of notice. The case was seen a few weeks ago in consultation with Drs. Mills and Turnbull. The fields, which were carefully taken for form, yellow, blue, red, and green, showed the distinctive and typical limitation of hemianopsia, in which the appearance of a double bow-like curve—the smaller one sweeping around and avoiding the point of macular fixation, and the second continuing irregularly both above and below to the outside in the left field and to the inside in the right field, was plainly visible ; the sequence of the fields following the order of the colors just noted. Both of the remaining fields were regularly contracted, without indentations or scotomata ; the series on the left side being but one-half the size of those on the right side, although the intensity and brightness of the colors seen in and around the macular fixation of the left side were more vivid and pronounced. Vision for form was reduced in each eye, but slightly more so in the right ; this was partially accounted for by marked astigmatism with a low degree of hypermetropia, which, upon being excluded by proper lenses, showed that its vision almost equalled that of its fellow. With or without the correcting lenses, the pupil of the right eye did not respond to light stimulus so freely as the left when equal amounts of light were projected into the interior of the organs from the different portions of the existent field areas. The pupillary reactions, which were pursued in the ordinary method of concentration of narrow, yet strong beams of artificial or natural light from various directions upon the pupil, whilst the organ was feebly illuminated to a sufficient degree to observe the actions of the irides, were not only typical of the hemiopic type, but were peculiar in the fact that in nearly all the experiments there seemed to be an absolute ratio established between the amount of pupillary reaction and the degree of color saturation seen ; this being true both in monocular action and consensual ; this was plainly shown by the amount of reaction in the left iris being at least one and a half millimetres greater upon exposure of its own arc, than when its fellow arc—that of the right eye—was similarly stimulated.

Another instance which beautifully illustrates the same point, was seen in a case of brain-tumor which he examined in consultation with Drs. Keen and Mitchell, in June of 1887. There were unequal pupils corresponding in direct ratio with unequal amounts of hemianopsia, in which the greater percentage of lost color manifested itself in the smaller field ; there was at least one and a half to two millimetres greater response in one or both irides when the arc holding the better conception was stimulated ; this condition has been carefully studied, and will be combined in a forthcoming report of the case.

This observation, therefore, is of value in adding another partially objective factor to the various means employed in gaining better information in reference to the now all important question of cerebral localization, as it distinctly shows which arc has the greater amount of destructive or irritative lesion, and thus becomes an additional sign of special and distinctive usefulness.

DR. J. LLOYD presented

SPECIMENS FROM A CASE OF PROBABLE ALCOHOLIC MULTIPLE NEURITIS, WITH BRAIN ENVOLVEMENT.

M. L. W., white, thirty-seven years of age, a dressmaker by occupation, was brought into the hospital February 21, 1888, in a delirious state. When first seen she was unable to move her left leg, but moved the right one with some difficulty. The day following both legs were rigidly extended, and she was apparently unable to move either. She complained of severe pains in the calves upon passive motion. The muscles of the face kept twitching all the time. The arms were constantly thrown about, the hands flexed, and the thumbs turned inward. On the night of the 22d, she became very violent, raving constantly. She would catch hold of the attendants and beg them piteously to keep her from falling. She was given, during the night, ninety grains of potassium bromide and twenty grains of choral. The next morning she was very much depressed. She had retention of urine ; about one pint was obtained by catheterization in twenty-four hours. The urine was dark colored and filled with urates, but no albumen or sugar. On account of the small quantity of urine passed, one-fourth of a grain of pilocarpine was administered hypodermatically. The abdomen was much distended with flatus. After the administration of pilocarpine she became rather weak, and digitalis and whiskey were then given. The pulse rate became better, falling from 135 to 90 per minute. Hyoscine was then administered with the intention of securing sleep : $\frac{1}{120}$th of a grain was given at 9 P. M., $\frac{1}{240}$th at midnight. At 8 P. M. of the 24th $\frac{1}{120}$th of a grain was again given. On the morning of the 24th she seemed much more rational and quiet, the hyoscine having had the desired effect, but toward noon the pulse rate again became very rapid—from 135 to 160 per minute. The lungs quickly became œdematous, and she died at 1:45 P. M. of the same day. The left pupil was perceptibly larger than the right. There was little rise in the temperature, the highest being about 101.6° F. During her illness she retained very little food or medicine, vomiting it as soon as it had been administered. Her bowels were constantly moving involuntarily. The heart sounds were normal but very feeble. Her family could give no account of her at all, as they had not seen her for years ; they surmised that she had been leading an immoral life for a long time. She died in about twenty-four hours after she was first seen by Dr. Lloyd.

At the post-mortem examination very few gross changes of any kind were found. There was some congestion of the pia mater, particularly along the longitudinal sinus. In addition there was marked adhesions of the dura mater.

The peripheral symptoms simulated those of multiple neuritis, and she had, in addition, a maniacal and delirious condition. She had cutaneous hyperæsthenia and muscular tenderness. She had paralysis of the extensors, which is characteristic of multiple neuritis. Dr. Lloyd's opinion was that the case was one of alcoholism and not of epidemic cerebro-spinal meningitis, some cases of which latter disease have recently occurred in this city.

DR. CHARLES K. MILLS made the following remarks on

THE PROBABLE OCCURRENCE OF MULTIPLE NEURITIS IN EPIDEMIC CEREBRO-SPINAL MENINGITIS.

Recently a number of cases of cerebro-spinal meningitis have appeared in Philadelphia, of which he had seen several in consultation, three quite recently, two with Dr. Cahall, of the Falls of Schuykill, and one with Dr. Dick. They had some of the typical symptoms described by authors. The particular point to which he wished to call attention was that in two of these cases markedly, and in the third to some extent, there were symptoms which seemed to point to multiple neuritis, or, at least, to neuritis involving certain portions of the body. In one case the most decided complaint of the patient was pain and soreness in his legs. He found soreness over the nerve trunks ; and also, in addition, muscular and cutaneous hyperæsthesia. He also had the appearance of the legs often seen in multiple neuritis, the equino-varus position of the foot. The knee-jerk was diminished. In another case, outside of the mental condition, the pain and tenderness in the leg were the most distinct symptom. The conditions were exactly those which are regarded as diagnostic of multiple neuritis—that is, tenderness over the nerve-trunks, and pain in the muscles with tenderness on pressure. In going over the literature of multiple neuritis lately, Dr. Mills found that this affection had been associated with or caused by neary all the infectious diseases, but no cases were reported in connection with so-called cerebro-spinal fever. In this disease we have a true meningitis, and he thought we might have a perineuritis, or a neuritis. In some cases the multiple neuritis might alone be present. In this way certain irregular cases might be accounted for. Stillé and others refer to a neuralgic form of cerebro-spinal meningitis. The point which he wished to make was that true diffused neuritis, either alone or in connection with other conditions, probably occurred as a result of the infection.

DR. W. OSLER had recently had an interesting case of cerebro-spinal meningitis, which proved fatal. The patient, a young girl, when admitted to the hospital was thought by the resident to be hysterical. She was nervous and twitched on attempting the slightest movement. He thought at first that it was a case of subacute rheumatism. She subsequently developed well-marked symptoms of cerebro-spinal meningitis. The case proved fatal, and the autopsy showed the characteristic leisons of the disease. There was extensive recent exudation down the whole posterior surface of the cord. In the brain it was limited to small patches on either side of the pons, but there was the most intense congestion of the cortex. It is remarkable in Dr. Mills's cases, if they be instances of multiple neuritis, that, unlike other examples of this complication of specific fevers, the symptoms have appeared early in the course of the disease. In other affections, as typhoid fever, pythisis, and diphtheria, the neuritis appears as a late complication.

He would like to make one remark with reference to central nerve irritation producing symptoms of cerebro-spinal meningitis. There are cases of typhoid fever with marked meningeal symptoms, and he has known such cases to be diagnosed by careless observers as cerebro-spinal fever, and the autopsy has shown the specific lesion of the intestines, and only congestion of the spinal and cerebral meninges. The diagnosis of meningitis, either in the brain or the cord, may be extremely difficult. Unless the nerves at the base are involved, it may be impossible to say whether or not there is exudation. This is well illustrated in the meningitis of pneumonia, which may produce an ever-deepening coma, not to be distinguished from that which accompanies cases with intense cerebral congestion, unless pressure on one of the nerve-roots give the clew ; and this led to the diagnosis in one of the eight cases of the most intense meningitis in this disease, which were observed in the Montreal General Hospital between 1876 and 1884.

NEW YORK NEUROLOGICAL SOCIETY.

Meeting of March 5th, 1888.

The President, DR. C. L. DANA, in the Chair.

THOMSEN'S DISEASE.

The President reported a case, in some respects atypical, and showed a specimen of muscle. The patient was a man, thirty-five years of age, single. He had been delicate as a child, and at the age of fifteen or sixteen years had masturbated, but not to excess. At the age of fourteen years he had been affected with talipes varus in the left leg, but it was cured by tenotomy. The symptoms of the disease in question had not appeared until the twentieth year, when the patient began to notice stiffness of the hands and difficulty in opening them when closed ; also stiffness of the leg and of the muscles of mastication. He tired easily, especially in the arms. Until recently he had a high-pitched child's voice and a child's larynx. Erections were incomplete, and there was mental difficulty in concentrating his thoughts. The muscular development was good. Measurement around the biceps gave ten inches in each arm. The dynamometer showed 40° in the right hand and 38° in the left, the normal measurement by the president's dynamometer being 43° to 50°. There were tonic contractions of the calf muscles and of the pillars of the fauces. Striking the muscles of the arm or forearm caused tonic contractions in those muscles. Striking the biceps with the percussion hammer would cause a welt and a myoid tumor as well. There was no increase of irritability to mechanical stimulation in the nerves. By galvanism, Erb's reaction was obtained. There was increased muscular irritability, also a closure tonic contraction which persisted as long as the current continued to be passed. There was closure tetanus both to the cathode and to the anode. There was no opening contraction to either pole. A peculiarity of the reaction to faradaism was that in the arm the contractions persisted after the current ceased to be passed. There was no ankle clonus, no increase of the reflexes, and no spastic condition. In the eye the fundus was normal, but there was a fibrillary contraction of the muscles of the lid. There was vasomotor weakness ; the hands and feet were red, and easily became cold.

A piece of the supinator longus had been removed ; also, for comparison, a piece of the same muscle from the president's own arm. These specimens had been placed in weak alcohol, and stained with picro-carmine and Bismark brown. The specimen taken from the patient showed an increase of the nuclei of the sarcolemma, an increased number of fibres, and, in addition, a dichotomous division of the fibres such as were found in muscle of the heart. This was characteristic of Thomsen's disease, and was supposed to indicate reversion to an earlier type. The tonic contractions of this disease, too, were characteristic of unstriped muscle fibre.

DR. GEORGE W. JACOBY had examined the specimens, and compared them with those from his own case. He did not consider this the typical case which Erb's monograph had described. Erb had excluded all the published cases but eight. Erb's typical case showed no disease of the central nervous system. In the president's case there was at least a suspicion of such disease. The reactions of the muscles, too, were incomplete, or failed to fulfill the requirements given by Erb. The speaker did not, however, believe in Erb's lines. He thought they were too close. While this case did not come under the heading as limited by Erb, it did not come under the name as understood by others. Erb's theory was that of a disorder of the muscles themselve, a congenital malformation of the muscular system. If the fissuring of the muscles and the increase of nuclei in the case under discussion were dependent upon a central affection, this alone was an interestiog fact. It would demonstrate that, microscopically, alone, a diagnosis of myotonia congenita could not be mode. Our knowledge of primary muscle affections was not yet on a solid basis. It was possible that there was first trouble in the central nervous system, from which the other proceeded.

DR. C. HEITZMAN had examined the specimens with a low power, and had been impressed with the belief that this was not a genuine case of Thomsen's disease. In a typical case the nuclei of the muscles were augmented. The president had made Erb's mistake when he spoke of the nuclei of the sarcolemma. The sarcolemma was a structureless membrane. There was also augmentation of the sarcoplasts, or muscle-corpuscles. There was too much muscle substance from the earliest period ; hence the name myotonia congenita. The president's case, on the contrary, could not be called congenital, as the disease had not developed until the twentieth year. Moreover, the muscle-fibres were not distinctly augmented in size ; compared with those in Dr. Jacoby's case the difference was marked. Besides, the fissuring was not prominent. For these reasons he was loathe to accept the diagnosis. He was, on the other hand, unable to tell what else the condition could be.

DR. J. B. EMERSON had examined the patient's eyes. There was no insufficiency of the muscles, and the pupils had reacted normally.

DR. M. A. STARR realized that we could not yet lay down any positive deductions in regard to electrical reactions. In degeneration we observed the pure reaction of degeneration, an intermediate reaction of degeneration, and the normal muscle reaction. Erb had given the reactions for three cases only. Other cases might not substantiate those results. He considered the president's case valuable and one to be put on record. For reliable data a large number of cases were required. Even Dr. Heitzman's objection of muscular anomalies was not fatal to the theory of its being a case of Thomsen's disease.

The PRESIDENT explained that he had not called the case one of myotonia congenita, but an atypical case of Thomsen's disease. It remained to be proved whether myotonia was always congenital. He had recently seen a case which commenced at the eighth or tenth year. Since the appearance of Erb's book four additional cases had been reported besides his. He agreed with Dr. Heitzman that the enlargement of the fibres was not positive. He had measured the fibres with a stage micrometer in the specimen from Dr. Jacoby's case, in that from the case under discussion, and in that from his own arm. He had found the fibres in the first $\frac{1}{400}$ to $\frac{1}{300}$ of an inch broad ; in the second, $\frac{1}{250}$ to ^{1} ; and in his own, $\frac{1}{450}$ of an inch. Thus, in the case under discussion, some of the fibres were larger and some smaller than normal. The increase of the nuclei, however, was shown in some of the specimens as typically as in Erb's plate. He acknowledged that the fissuring might have been produced artificially by tearing. Electrically, there was greatly increased irritability of the muscles both to galvanism and to faradism. The contractions were tonic with closure tetanus. There was normal excitability of the nerves. Clinically, the patient presented the phenomena of Thomsen's disease. It remained to be proved whether the phenomena of Thomsen's disease could be produced by disorders in which the central nervous system was involved.

TUMOR OF THE BRAIN.

DR. STARR presented a specimen. The patient was a woman, fifty-six years of age at the time of her death, who for two years had presented the general symptoms of tumor of the brain—vertigo, projectile vomiting, dulness of the mental faculties, and optic neuritis. During the last five months she had been examined carefully, but without revealing any evidence as to the locality of the tumor, except on one occasion, when during an attack of vertigo she fell forward and to the right. This was not a prominent symptom, and it was the only evidence pointing to cerebellar disease. The tumor was the size of a hen's egg. It was found on the lower surface of the tentorium, simply resting upon and compressing one lobe of the cerebellum. There was no adhesions and it was unfortunate that no symptoms had pointed to its location, as it might have been removed easily. In 1878, Nothnagel had remarked that tumors of the lateral lobes of the cerebellum did not give rise to the symptoms of inco-ordination common to other cerebellar tumors.

BASEDOW'S DISEASE.

DR. J. WEST ROOSEVELT presented the report of a case, with that of the autopsy. The patient had been admitted into the Roosevelt Hospital on May 25, 1887. She was a widow, forty-seven years of age, a housekeeper. Both the personal and family history were good. Two years before, she had begun to complain of palpitation, dyspnœa upon exertion, and swelling of the throat, which was largest upon the right side. She could not lie upon that side. In the course of a year the eyes began to protrude, and at the time she entered the hospital she had a profuse watery diarrhœa. The pupils were found to be equal, and the reaction to light and to accommodation were good. The lids did not follow the eyeballs. The neck measured thirteen inches around the lower thyroid region, and ten inches and a half around the upper thyroid region. There was dysphagia to solid food. The pulse was from 100 to 120, and the respiration 38. The apex-beat was found in the fifth space, in the nipple line. There was epigastric pulsation and the area of dullness was slightly increased. There was a short systolic murmur at the apex. There was a systolic thrill over the jugulars, also a continuous venous hum. The pulmonary resonance was exaggerated. The veins of the retina pulsated, but the arteries did not. There was no tremor. The patient lived until May 31st. She suffered from watery diarrhœa and restlessness, but was not otherwise sick. In walking to the bath-room one evening, she fell dead on the floor. The autopsy showed the thyroid reduced in size, but still moderately enlarged and of a pink color. The kidneys showed a trace of fibrous tissue, but otherwise the organs were normal. There was apparently nothing abnormal in the medulla nor in the sympathetic or vagus nerves. Microscopic examination of these parts also showed nothing.

Dr. W. O. MOORE had seen twelve cases, all in women of the average age of forty years, the youngest being thirty-one and the oldest forty-five. Ophthalmoscopic examination had simply shown enlargement and tortuosity of the blood-vessels. Great relaxation of the bowels had been present in one of the cases, as many as ten evacuations taking place in the day, which it was impossible to control. Electricity had been of no avail in these cases. All had presented the three characteristic symptoms, exophthalmia, thyroid enlargement, and rapid action of the heart. All had shown the symptom to which Von Graefe had first called attention, namely, a disturbance of the usual co-ordination of the movements of the eyeball and the upper lid, so that when the patient looked downward below the horizontal meridian the lid no longer followed the eyeball in its motion, but halted in its course. This fault in the action of the lid was supposed to be due to some defect in the orbicularis, and was not present in patients having prominent eyes from other causes. Occasionally the prominence was so great as to cause the eye to be exposed at all times, whether the patient was awake or asleep. In one case in his experience suppuration had occurred and the eye had been lost. The patient, aged forty-five years, stated that when a child she had lost the sight of the right eye by an accident, and that one year before coming under observation, she had noticed commencing enlargement of the neck, dyspnœa upon exertion, and prominence of the left eye. Six months later, vision for near objects began to fail, and four weeks before coming under notice the left eye had become painful and inflamed. An examination, December 13, 1886, showed in the right eye phthisis of the bulb, total corneal leucoma, and exophthalmia so marked that, although the eyeball was atrophied, the lids were as full as in the usual healthy state. The eyelids on this side covered the globe fully when shut. In the left eye the exophthalmia was so great that the lids were retracted to their full extent and the eyeball was dislocated through the commissure of the lids. The ocular conjunctiva was chemotic and the cornea was cloudy through its whole extent. At the upper border of the cornea there was a serpiginous ulcer; in other words, there was a keratitis from loss of nutrition and exposure to the air. The pulse was irregular at 110. The patient was admitted into the Post-Graduate Hospital, the outer canthus was cut, hot-water applications were made, and the parts were protected by lanolin. In spite of treatment, perforation took place, with escape of the vitreous and lens. The eye began to recede and phthisis bulbi developed. Had this case been seen earlier, the speaker would have united the upper and lower lids, thus covering the eyeball. At the end of a few weeks the lids would have been reopened, when, as a rule, the exophthalmia would be found improved and the corneal trouble removed. For constitutional treatment the patient should receive digitalis, ergot, and tonics. This case was remarkable from the facts that an eye was lost by suppuration and that this loss was associated with phthisis in the other eye. The loss of an eye from exposure was so rare that the speaker knew of but ten reported cases in this country, while Wells had reported only one case.

Dr. STARR said that the fact that no lesion had been found in the sympathetic in Dr. Roosevelt's case did not, of course, prove anything conclusive. Ross had reported eight cases in which such lesion had been found out of twelve cases, as far back as 1882. The pathology, however, was not clear. It was difficult to understand how any one lesion could produce all the phenomena of this disease. The hypothesis accepted by Gowers was that of lesion of the vagus nucleus in the medulla. The rapid pulse would be accounted for by the loss of the inhibitory power of the vagus. It was known, too, that vaso-motor disturbances were produced by irritation of the medulla in this region. The speaker had some time since collected twenty-one cases of lesion of the medulla, in eight of which the lesion was in its upper part, in the region of the nucleus of the tenth nerve. In all those cases there were subjective flushings and objective increase of perspiration, while in the thirteen in which the lesion was in the lower part of the medulla there were no vaso-motor symptoms whatever, thus substantiating the hypothesis of the physiologists that there was a vaso-motor centre in the medulla and that this centre was in the neighborhood of the nucleus of the tenth nerve.

The speaker had personally observed seven cases, five in the female and two in the male. In all but one palpitation of the heart had been the first symptom. This disproved the theory that the goître was primary, and that the other symptoms were due to the pressure of the tumor upon the pneumogastric nerve. The pulse had ranged between 90 and 155. In all but one the eyes had been prominent. In six there had been nerve symptoms, in four tremors, in four Von Graef's symptom, and in six flushes. Mental disturbance had been present in one case, in which there had been delirium every night for several months, and subacute mania for several weeks. The first symptom in this case had been insomnia, which resisted treatment. The speaker supposed it to have had its origin in a condition of the vessels of the brain similar to that in the back of the eye and the thyroid gland. Digitalis had done no good in his experience. Ergot and bromide had quieted the tremor, but no drug which he had tried had reduced the action of the heart. He had used electricity according to Benedict's recommendation, but without result. He had himself, when in Vienna, watched the treatment of three cases in which Benedict had given a good prognosis, but had failed to find any reduction of the pulse while the galvanism was being used. In his own cases he had tried every method described as galvanization of the sympathetic ; he had placed the poles upon either side in front of the sterno-cleido-mastoid, behind the sterno-cleido-mastoid, and at the back of the neck and at the epigastrium, carrying the current as high as nine milliampères, which was as strong as could be borne with a small electrode, and he had never been able to produce any retardation of the pulse. Dr. Janeway had expressed himself as having had the same experience.

Dr. A. D. ROCKWELL had met with about thirty cases of this disease, and in nine cases he had observed an approximate cure. He had employed diet, galvanism, and very full doses of digitalis, bromide of zinc, ergot, and iron. He had authentic records of his results. He believed that the cases not benefited by treatment were organic, and that those benefited were functional in origin. Those having all the cardinal symptoms were more often responsive to treatment than those in which the symptoms were more incomplete. He recalled a case in which there was a pulse of 110 reduced to 80. There was puffiness of the eyelids in that case. In the galvanic applications one pole had been placed over the eyelid, and the other behind the sterno-cleido-mastoid muscle at its upper third. In another case the pulse had ranged from 130 to 150 for several years. There was dilatation of both pupils, and there was a pulsating swelling over the solar plexus. This patient was placed upon the use of a milk diet and persistently treated for many months, when the pulse fell to below 100, and the swelling of the thyroid and the exophthalmia had become less.

Dr. STARR asked whether Dr. Rockwell had observed a reduction of the number of the heart-beats while the current was being used, also whether he had ever seen paling of the face and dilatation of the pupils—phenomena which followed galvanization of the sympathetic when needles are used.

Dr. ROCKWELL replied that he had not made his observations during the application, but that subsequently such slowing had been demonstrated. Lowering of the pulse was also a very common result of general faradization.

Dr. JACOBY considered exophthalmic goître a rare disease. For eight or nine years he had seen in his dispensary from six to seven hundred neurological cases a year. Not more than twelve of them had been cases of exophthalmic goître. The patients had been regular in attendance and the treatment had been persistent, but he had tried every means heralded without result. Subaural galvanization had been without effect upon the color or the pulse. He considered the disease a hopeless condition. Bodily and mental rest constituted about all that could be done. He had tried faradization according to the recommendation of Vigouroux, but also without result. He thought the exophthalmia the least constant symptom. Where goître was present, one side of the neck was usually larger than the other. Some said that the right was always the larger. This he could contradict, as in a case which he had recently seen with Dr. Birdsall the enlargement had been equal upon both sides. Von Graefe's symptom was not always present. Tremor was often the first symptom. In one case in his experience tremor had existed for a year before the development of the other symptoms. At the end of the second year these had become well developed, and bronzing of the skin also was present. Vigouroux had stated that the electric resistance was diminished in all cases, and even in the commencement of the disease. This, if true, would be an important diagnostic point. In twenty cases Wolfenden had also found this lowered electrial resistance. Histories of two of these cases had been published in full. In one of the latter the resistance was only 300 ohms. It was mentioned that there was profuse sweating in this case. In the second, the resistance was 200 ohms. Of this case it was said that there were clamminess of the surface and sweating. In the other eighteen cases the resistance was from 500 to 1,300 ohms. The speaker said that the standard of resistance, however, varied according to the method used. It had been given as 300,000 to 400,000 ohms by Jolly; Gärtner gave it as 30,000 to 40,000; and here we estimated it at a third or a quarter. He had recently tested the resistance in three cases, his method having been to place the body in the circuit, the electrodes being equal in size and wet. When the galvanometer needle ceased to be deflected, the body was taken out of the circuit and a resistance coil inserted until the deflection of the needle was again brought to the same point. The amount required equaled the resistance of the body. Comparative observations were at the same time taken upon himself. The first case was that of a woman, twenty-eight years of age, with exophthalmia, palpitation, and sweats. The resistances obtained were as follows:

Through the palms, - - -	Patient,	6,000	ohms;
	Self,	8,000	"
Through the goître, - - -	Patient,	1,200	"
	Self,	2,400	"
Through the posterior part of the neck, - - - - -	Patient,	1,400	"
	Self,	2,400	"

The second case, Dr. Birdsall's, presented goître with palpitations and exophthalmia, and the comparative observations were taken upon Dr. Birdsall and himself. The resistances obtained were as follows:

Patient, through the hands, 5,000 ohms; through the goître, 800 ohms; through the posterior part of the neck, 1,000 ohms. The measurements in himself were respectively, 5,500, 2,400, and 2,000 ohms, and in Dr. Birdsall, 8,000, 1,000, and 1,000 ohms.

In the third case the resistances were:

	Patient.	To control subjects.
Through the hands	5,000 ohms	11,000 and 10,000 ohms.
Through the thyroid	1,0co ohms.	4,000 and 3,000 ohms.
Through the neck, antero-posteriorly	1,500 ohms.	3,000 and 3,0co ohms.

The reduction was thus scarcely a quarter, certainly not a half, and not more than would be accounted for by the maceration of the skin due to the abundant perspiration in these cases, or, as in the goître, to the fluxion of blood. The speaker failed to see how any importance could be attached to the test as a symptom.

The PRESIDENT was surprised to hear that there was any dispute in regard to the possibility of lowering the pulse by galvanism. In a case of Basedow's disease in Bellevue Hospital a pulse of 140 was found lowered fifteen or twenty beats after the current had been applied. The sedative effect of galvanism was generally admitted, though we could not say whether it was produced through the pneumogastric or through the sympathetic nerve. Dr. Starr's theory he thought incorrect. Lesion of the nucleus of the pneumogastric should give the same symptoms as division of its trunk, and this never gave rise to the phenomena of Basedow's disease. It might perhaps be said that this disease was due to lesion of the nucleus of the pneumogastric and neighboring parts. He thought that the resistance was diminished in these cases—at least slightly, perhaps 1,200 ohms. In testing he placed one electrode on the region of the seventh vertebra and the other on the sternum.

Dr. ROOSEVELT had had the same experience as Dr. Starr and Dr. Jacoby. He had used both strong and weak currents without any influence on the sympathetic nerve. He was surprised to hear digitalis recommended. He believed that digitalis was without value for heart failure except from organic disease. He had had five cases of exophthalmic goître under observation. Two of the patients had improved, but both were young anæmic girls. The measurement of electrical resistance he thought a difficult problem, because the factors varied.

PERISCOPE.

By DRS. G. W. JACOBY, N. E. BRILL, AND LOUISE FISKE-BRYSON.

PHYSIOLOGY OF THE NERVOUS SYSTEM.

A Certain Kind of Insomnia.—The two most reliable signs of perfect health are the absence of pain and the ability to sleep tranquilly and soundly. One kind of insomnia is frequently met with and is most difficult to treat—the insomnia caused by malaria. First, we have a group of patients who sleep neither day nor night. They count each stroke of the clock all night, lie awake tortured by their own thoughts, rise in the morning all tired out, and unfit for daily duties. In another form of this complaint, after a rest of two or three hours, the patients waken and get no more sleep. This short slumber refreshes them, they can attend to business, giving out, however, quite frequently during the day, their limbs seeming to give way under them. To a third class belong those who fall asleep and sleep well up to a certain hour or moment. Their short rest seems to have perfectly refreshed them, but they are frequently subject to slight chills and fever and sweating, also to neuralgia and lumbago and exhaustion.

It seems probable to me that the microbes of malaria may produce a similar condition to that of certain alkaloids —caffeine, theine, theobromine, etc., that cause wakefulness. It is evident that these malarial microbes are contained in great quantities in the veins, and also in the smaller vessels of the pia mater and the large gangla of the brain. Here they may act as a delicate "reagent," by means of which the existence of malaria may be proved, viz., by their effect in producing the insomnia of the patient. Massage, hydropathic treatment, and quinine combined with sodium bicarbonate or dilute phosphoric acid are of great service in such insomnia. Morphine, chloral, etc., are dangerous and non-curative, heightening rather than diminishing the disease, and producing an overwhelming sensation of excessive fatigue and mental depression, accompanied frequently by increased sexual desires and a morbid anxiety about business and the affairs of life.—F. Eklund, M.D., Stockholm, Sweden, in *Therapeutic Gazette*, Dec. 15, 1887. L. F. B.

PUERPERAL INSANITY.

The term puerperal insanity is employed in a comprehensive sense, embracing (1) the insanity of gestation ; (2) post-parturient insanity, and (3) lactational insanity, or cases caused by long nursing. It is the most favorable form of mental derangement. The forms that it may assume are melancholia, mania, delusional insanity, and dementia. Its causes are predisposing and exciting, as in other diseases. Heredity, and an acquired predisposition from former attacks of insanity, play the most important part. In 765 cases collected by Dr. Ripping, 16.4 belonged to the first type (insanity of gestation), 50.6 to puerperal insanity, and 33 per cent. to the lactational type. The first form usually makes its appearance about the end of the third or the beginning of the fourth month of pregnancy. Griesinger attaches considerable importance to the emotions of women pregnant for the first time. He also says (quoting Marce's "Folie des Femmes Enceintes) : "Slight mental disorders, hysterical humors, irresistible longings, foolish jealousy, and kleptomania are more frequent during pregnancy than fully developed insanity." Depression, or mild melancholia with anxious delusions, is the form that mental derangement generally assumes at this period. Puerperal insanity begins within thirty or sixty days after delivery, occurring more frequently during the week following parturition than at any other time, usually on the third or fourth day. "Mental incoherence during natural labor" (Montgomery) is well known to every accoucheur, and is only an evanescent state. Melancholia, delusional insanity, and even dementia may occur, although mania is the most common form of insanity following parturition, and is the most dangerous to life. Tense cases very often resemble an acute phrenitis. The suicidal tendency is not uncommon, especially in melancholia. The character undergoes a complete change. The woman may evince a profound aversion to her nearest and dearest, talk incessantly, give way to the most violent anger, utter the most awful oaths and imprecations, and act as though possessed by demons. Anxiety, severe and tedious labors, convulsions, or great loss of blood, are among the exciting causes of this unhappy state. Insanity of lactation occurs in delicate women who have borne children rapidly and suckled them a long time. The patient's thoughts assume a sad and sombre hue, and soon are only on one subject—herself. Delusions, self-reproach and self-accusation of the " unpardonable sin " are common. The majority of such cases are of the melancholic type, but exceptional cases of mania do occur. Whatever the form of insanity due in some way to reproduction, each patient should be studied as a unique specimen, and the case treated according to the symptoms it presents. In acute maniacal cases, the first and most important indication is to allay the excitement by subduing the irritation of the nervous system. This is best accomplished by free catharsis, followed by sedatives aud anodynes. Chloral, the bromides, and hyoscyamus are more to be depended upon than opiates. When the febrile excitement has been subdued, tonics—iron, cinchona, quinine, and simple bitters, with good, nourishing diet and wine—are indicated. Warm bathing, rest and quiet, together with moderate systematized exercise, are also in order. In cases of profound melancholia, with stupor and apathy, blisters to the temple and nucha are frequently beneficial. Lactational insanity requires the weaning of the child first and tonic treatment next. With insanity of gestation there is usually a strong hereditary predisposition, and recovery is doubtful before delivery. Active treatment is contra-indicated. Diet, exercise, and hygienic surroundings are the only safe measures to be adopted.—J. E. BOWERS, M.D., in *Northwestern Lancet*, Feb. 1, 1888.

L. F. B.

PARAPLEGIA FOLLOWING PNEUMONIA.

In an article published in the *Gazette Hebdomadaire*, Dr. Carré has collected the various cases of paraplegia following pnemonia thus far published, eight in all, and adds one observed by himself. From a comparison of these cases it seems that they are very dissimilar. Most frequently a general weakness is noticed; the paralysis may, however, be complete, particularly in the lower extremities. Sometimes the muscles of the chest, of the shoulders, of the larynx and of the pharynx are affected. Paræsthesia, and even anæsthesia, has been noticed. In some cases the paralysis began in the lower extremities and ascended; in others the course is descending, affecting the four extremities without any regular sequence. Occasionally the symptoms are purely spinal, at other times the intra-cranial nerves are also affected. In all the cases the paralysis occurred during convalescence from the pneumonia; in one half the cases the termination was fatal. Carré believes the cause to be infectious, and considers the existence of microbes in the nervous centres probable. (*Journal de Médecine*, March, 1888.) G. W. J.

PARALYSES FOLLOWING RAILROAD ACCIDENTS.

Onimus, in his "Traité d'électricité Medicale," devotes a chapter to the above subject. It seems that particularly those persons are subject to spinal lesions who travel seated with their back to the engine. Persons sleeping at the time of the accident escape to a great extent. The causes of both these facts are purely physical ones. This spinal concussion is of importance, because it dominates all other symptoms following the accident and often produces paralysis after days or even weeks. Also characteristic is the long duration of the affection. Onimus cites a case, noteworthy on account of the smallness of the lesion, which consisted only of slight atrophy of the trapezius and contracture of the scaleni and which during years, although the atrophy had disappeared, remained in a state of contraction, rendering all movements of the neck difficult and painful. The principal difficulty in diagnosis consists in the exclusion of simulators. The electric current serves as a guide. (*Journal de Médecine*, March, 1888.) G. W. J.

RETROGRADE AMNESIA FOLLOWING INTENSE EXCITEMENT.

Under the name of retrograde amnesia, Azam has described the following facts: A healthy patient receives a severe cranial injury, he loses consciousness, and when this is regained, it is found that the recollection of the accident, as also the memory for events preceding the occurrence are entirely lost. Two, three or four days prior to the accident, and the circumstances of the accident itself are entirely obliterated from the subject's memory; in every other respect his memory is perfect. Little by little the recollection of occurrences anteceding the accident is regained until finally there is only a hiatus as regards the occurrence itself. Arnozan has described an analogous condition occurring in a patient after intense emotion due to the receipt of bad news. From the moment of the mental shock, without any nervous symptoms or other intellectual disorder, every fact connected with the subject disappeared from his mind, and he was unable to comprehend any of the details explained to him or to appreciate the consolations offered him. When seen by Arnozan he presented loss of memory for all actions and facts which had taken place during three days. In every other point his memory was normal. The disorder was only transitory, for in twenty-four hours all traces of the affection had disappeared. (*Journal de Médecine*, March, 1888.) G. W. J.

CEREBRAL TUMOR WITH HÆMORRHAGE.

(E. J. Sidebotham, M.B., in Saint Bartholomew's Hospital Reports, Vol. xxiii., 1887.)

The patient, æt. 20, apparently in perfect health, was suddenly attacked by pain in the head. A physician who was sent for found him comatose. The pupils were contracted to a pin-point, and there was divergent squint. He soon regained consciousness, and the pupils became natural. He complained of pain on the right side of the head, the movements of the left arm and leg were feeble, and the knee-jerk was absent on the left side. There was hiccough, retching and vomiting. During the next four days the following symptoms were observed: There were wide hysteroid movements of the right limbs, purposeless as a rule; profuse sweating; pupils equal; divergent squint; paralysis of the left arm and leg, sometimes rigidity of both arm and leg; the left knee-jerk was absent, the right normal; the temperature varied from 100.8° to 104°; there were several attacks of opisthotonos with spasm of the left limbs, also side-arching of the body, the concavity of the arch being to the left side of the body, but in one attack it was to the right. He died on the fifth day. The evening before his death the hysteroid movements ceased. The whole body was periodically thrown into tonic spasm.

At the post-mortem examination, a large irregular hæmorrhage was discovered in the right hemisphere of the brain, whose origin was obviously a tumor, which was situated on the surface of the angular gyrus. The hæmorrhage extended from the surface of the brain to the posterior part of the internal capsule, and had neither burst into the lateral ventricle nor externally. The tumor was an inch in length by three-quarters of an inch in diameter. It consisted of a close plexus of blood vessels. These vessels appeared to be derived from the artery upon which they lay, reinforced by branches from neighboring arteries. The tumor must therefore be classed as an angioma or nævus.

REFLEX PSYCHOSIS FROM TRAUMATISM.

At a meeting of the Berliner Gesellschaft für Psychiatre und Nervenkrankheiten, held on January 9th, 1888, Dr. Thomsen presented the following case:

W., an invalid soldier, with heredity history, but himself always healthy, received a gun-shot wound in the right upper extremity, in 1870. In 1884, attacks of pain appeared in the arm; these gradually increased, and in 1885, during every attack of pain, an hallucinatory mental disturbance made its appearance; of short duration and with pure intervals at first, but later more intense and less clearly periodical, the intervals being filled with fearful dreams, emotional depression, etc. During the paroxysm the patient sees forms and animals, men covered with blood, dogs, buffaloes, etc., which threaten him, so he believes. During the severe attacks he becomes confused and very much excited; during the mild ones, merely seized by fear; during the intervals he is wholly mentally sound and has full knowledge of his condition.

During the attack there is right-sided hemianæsthesia, involving both the skin and the organs of special sense; smell and taste are gone on the same side, the field of vision is enormously contracted on both sides, colors are distinguished very poorly or not at all (excepting red); there is deafness in left ear.

During the interval all disturbances of sensation disappear.

On December 27th, 1887, the cicatrix was cut out, two days after which there was a return of hallucinatory attack lasting but twenty-four hours, and ever since then a return to mental health and normal sensation. The patient never had epilepsy. The speaker concluded, therefore, that the patient was a case of pure peripheral reflex psychosis. (Centralblatt für Nervenheilkunde, etc., February 15th, 1888.) N. E. B.

TABES DORSALIS.

Dr. Martins (Deutsche Medicinische Wochenschrift, March 1st, 1888). The following case presents some exceptional points of interest:

The patient was perfectly well up to his fifty-third year, never had syphilis; was taken ill in June, 1885, with typhoid fever, which confined him to his bed for six weeks, at the end of which time the first symptoms of tabes appeared. These consisted of paræsthesia, exclusively confined at first to the upper extremities, especially to the hands and finger tips (feeling of stiffness, fur sensation, and the like). It was not until two months thereafter that the lower extremities became involved. Soon thereafter a palpable feeling of weakness in the lower extremities appeared, and it was this that brought him to the hospital (Charité).

At his entrance the history was as was given, paræsthesiæ in hands and feet, weakness in knees. Objectively, the internal organs were found to be healthy; in the motor sphere, no signs of ataxia, nor was the walk ataxic; pupils reacted normally to light, no swaying on closing eyes; knee phenomena present on both sides and easily worked. The only demonstrable symptom in the sensory sphere was that, on closing the eyes, the patient could not distinguish with his hands little objects, the feeling being as if he had thick gloves on his fingers. There was a disturbance of pressure sense, less of temperature sense.

Tabes was thought of, but the diagnosis could not be substantiated, for the classified symptoms, viz., the Romberg, Robertson, and Westphal signs were all absent. It was only after two months that the girdle sensation appeared.

At the end of March, the patient died of an intercurrent pneumonia, on the third day of the disease. During the pneumonia, reflex iridoplegia appeared, but the patellar reflex remained until death.

The spinal column appeared microscopically normal; it was only after it had been hardened in Müller's fluid that definite fields of degeneration in the spinal cord could be distinguished. The fields showed a different area than is usual in tabes, and the degeneration was most extensive and intense in the *cervical* region, and very little, if any at all, in the lumbar cord. N. E. B.

NERVOUS SYMPTOMS OF SECONDARY SYPHILIS.—Prof. Fournier, *Journal de Médecine*, p. 15, 1888.

Individual conditions have a great influence upon the nature of the disorders produced by the syphilitic virus. Sex also plays an important role; women being more prone to general nervous disturbances than males, also more often develop general neuroses as a consequence of syphilis.

These occurrences Fournier has divided into headache, disturbances of sleep, neuralgias, affections of sensation, general neuroses, neuroses of the great sympathetic, and visceral neuroses. The headaches are not any more frequent in females than in males. The disturbance of sleep occur as a symptom of secondary syphilis very frequently in women, and are independent of any pain or headache: it is an essential insomnia without any recognizable cause. In some patients all symptoms of neurasthenia occur, and are then only relieved by antiluetic remedies. Neuralgias are more frequent in females, and differ from ordinary neuralgias in the pain being more neuralgiform than neuralgic. They appear in three types—fascial, very frequent; intercostal and sciatic, less frequent. In the fascial form the infraorbital branch is most frequently affected, in the sciatic also, only a single branch is involved. The diagnosis is of importance, but not always easy. The disorders of sensation which occur as a result of syphilis are not generally known, and are often confounded with symptoms of hysteria. The occurrence of analgesia, anæsthesia, and loss of temperature sense is referred to, and their duration said to be very long, from several to fifteen months. G. W. J.

THERAPEUTICS OF THE NERVOUS SYSTEM.

TREATMENT OF NERVOUS AND MENTAL DISEASES BY SYSTEMATIZED ACTIVE EXERCISE.

While massage and electricity have received a large share of attention from neurologists, these have neglected too much the use of medical gymnastics, particularly systematized active exercise. In a large number of nervous and mental cases, the improvement of general nutrition is the one thing needed to bring about relief or cure. To this end a most effective aid is found in systematized active exercise. The movements may be classed as passive, duplicated active (operator and patient both taking part,) and active exercises. Treatment should be carefully individualized. It is usually necessary to combine respiratory with muscular movements. "On the two powers, muscular and respiratory, depend the ability to perform all bodily exercises" (Maclaren). Inherent nervous power has also something to do with the capacity to perform bodily exercise. Want of respiratory power is certainly either at the root or is an essential constituent of many morbid nervous conditions. Often when of apparently equal muscular ability, there will exist in different pessons marked difference. of respiratory power. Inspiratory exercises insure further muscular development, greater æration of the blood, and increased control over nervous and muscular effort.

For gout and lithæmia, to promote excretion and nutrition ; for anæsnia and spanæmia, to assist assimilation and further oxidation ; for headache, sleeplessness, and nervous irritability, to soothe and calm the nervous system ; to aid elimination in cases of lead, arsenic, mercurial, and other metalic or toxic diseases ; for diabetes, to favor activity of the skin and increase combustion, systematized active exercises have a value which cannot be too highly extolled. Also in curable ataxias, as in those which follow diptheretic or exanthematous diseases and in the hysterical varieties, systematized active movements, the patient lying down at first, then sitting, then standing, have proved of great service. The advantage of any treatment that involves specific direction and the adroit calling out of the volition of a patient must be evident to any one who has had experience with hysteria in its manifold forms. Whatever view may be taken of the much mooted question of neurasthenia, without doubt both respiratory and muscular power are often deficient, and the nerve centres themselves can be strengthened by exercising these two powers. Those forms of nervous palpitation which are dependent upon a neurasthenic condition, associated or not with digestive disorder, are greatly benefited by systematized movements. Special forms of gymnastics have been employed with advantage for the treatment of chorea. Napoleon Laisné,* a French professor of gymnastics, and evidently an earnest and enthusiastic worker in his chosen field, under the direction of Dr. Blache and other physicians of Paris, has used gymnastics largely both for chorea and other convulsive disorders. In 1865 he published a book in which his methods are set forth. Both Schreiber and Dujardin-Beaumetz refer to his labors and successes. His method in mild cases, as described by Schreiber, is to place the child before him, steadying it between his knees, and then take its hands in his and perform rhythmic movements with each arm, keeping time by counting, or, better still, singing out loudly—"one," "two," "three," etc. The child, at the same time, is also urged to keep time with the movements, and not to make them irregularly.

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*Applications de la Gymnastique a la Guerison de quelques Maladies. Paris, 1865.

"Care must be taken in the beginning to prevent, as much as possible, the coincidence of involuntary movements with the rhythmic ones. When the arms have been exercised, similar movements are undertaken with the legs. From time to time, a pause for rest is made, during which the limb must be held firmly enough to prevent the occurrence of involuntary motions. The child is then laid on its back upon an inclined ladder, the feet being held by an assistant ; then grasping a rung above its head, it holds on in that position as long as it is able. This is to be repeated several times, and to be followed by a short rest. Afterward, the shoulders, back, and legs are rubbed and gently kneaded."

Lengthy details of treatment will be found in Schreiber's Manual of treatment by massage and methodical muscle exercise (translated by Walter Mendelsohn, of New York).

In patients suffering from multiple neuritis, or some curable forms of myelitis, advantage should be taken of the first signs of motor improvement to begin with active exercises, while the use of electricity and massage is continued. The particular point upon which I desire to insist, is that the attempt to join the will of the patient to the long unused muscles, shall not be deferred a moment longer than is necessary.

In the treatment of various forms of paralysis, that systematized active movements may be employed with advantage has long been known. Even in paralysis from organic brain disease, a clear method of using gymnastic treatment will be found to serve an excellent purpose. Such paralysis is usually the result of hemorrhage, embolism, thrombosis, tumor, abscess, or depressed fracture ; less frequently of meningitis or cerebritis, of atrophy or arrested development, and still more rarely of uræmia. Sometimes in cases of sudden lesion, as hemorrhage or embolism, the assault upon the nervous system is so violent, or the destruction is so great, that death results quickly, or the patient is reduced to a state of helplessness, for which, practically, nothing can be done. In many cases, however, soon after the attack, or even at a later period, the amount of palsy is disproportionate to the cerebral lesion by which it has been initiated. Many cases of monoplegia and hemiplegia illustrate this truth. Little by little some of these patients regain muscular power to such an extent as almost to induce the belief that they will get entirely well ; indeed, in some cases of hemorrhage, tumor, traumatism, syphilitic meningitis, and uræmia, complete or almost complete recovery does not occur. We should, therefore, not disregard entirely the treatment of such patients.

A method of gymnastic treatment which I have often employed with benefit in cases of monoplegia and hemiplegia, is to cause the patient, first, to make a movement upon the unaffected side, and then instantly to perform the same movement with the paralyzed member, following this quickly with an attempt to do the same thing with both limbs. It is surprising the curious results that will sometimes be obtained in this way, if the leg is but little affected, and the patient can stand while these movements are performed by the upper extremities. To exercise the legs, the patient, of course, should be placed in an easy position, and one that will allow the movements to be performed with the greatest convenience. Exercises of this kind probably have some effect in bringing the paralyzed side of the body under control of the uninjured side of the brain through comissural channels in the spinal cord.

For some of the arthritic neuroses, and for rheumatic neuritis, or muscular rheumatism, these exercises are of undoubted value. I have seen three cases of a form of rheumatic neuritis affecting the deltoid and adjoining muscles, in which the progress to complete recovery was much assisted by an early resort to dumb-bell exercises and pulley-weights. Cases of this kind are best treated by using large doses of oil of gaultheria, or sodium salicylate, with hypodermic injections of morphia in the most acute stages ; a little later resorting to massage, electricity, or both ; and then to exercise with light dumb-bells or pulley-weights. Here, again, the point I wish to impress is, that such active exercise should not be deferred too long.

For the group of diseases which fall to the lot of both the neurologist and the orthopædist, cases of curvature, deformity, atrophy, etc., systematized active exercises have long been used by the best authorsties.

In the treatment of ataxic affections, even sometimes when dependant upon organic diseases of the cerebro-spinal axis, the use of what may be called balancing or acrobatic gymnastics is of some value. Dr. Mortimer Granville, in the *Practitioner* for 1881, and subsequent to his monograph on "Nerve Vibration and Excitation," discusses a method for the regeneration of the nerve elements by exercise on the basis of the law of development through function, holding that the ataxic subject is reduced by dissolution to the postion of a child just learning to stand or walk. His plan is to direct the patient to stand with his eyes closed in his bath, after pouring a small can of water down his spine, or applying a mustard poultice over the full length of the spine for ten minutes or a quarter of an hour, and, as his state improves, for half an hour every morning. He is to be furnished with a chair or rail at hand, to which he can cling in case of need, but is instructed to avoid using it except when in danger of falling The exercise must be continued diligently for weeks before success can be obtained.

Dr. Chas. Fayette Taylor, who published as early as 1861, *The Theory and Practice of the Movement Cure*, thus speaks of the combination of rest and exercise.

"The true remedy," he says "is rest and exercise. Let the rest be complete relaxation of all muscular effort—not the entertaining of company, sitting bolt upright, so that the spinal muscles must be constantly acting, or reclining in a 'graceful' attitude on a lounge, with a book in hand, but a completely sustained position, when all the muscles must cease to act. Then the exersises to follow should be short, varied, and taken with some vigor."

The now generally accepted views with reference to cerebral localization throw some light upon the manner in which systematized active exercises, or other forms of gymnastic treatment, improve, or repair the nervous system, and especially the brain. This fact has not been overlooked by authorities in neurology and gymnastics, as by Emil Du Bois-Reymond, Schreiber, Crichton-Brown, and others. In the brain are represented both a differentiation and an integration or solidarity of function. Centres for speech, for vocalization, for particular movements, for the special senses, for the muscular sense, for organic sensations, for some of the higher faculties, as of attention and inhibition, are now, with reason, claimed to have been isolated. For the localization of some or these, as of speech, motor, and some of the sensory centres, the facts and arguments are practically incontrovertible. In the plainest of terms, if brain centres which determine certain movements exist, the performance of these movements must develop and train not only muscles concerned in these actions, but the cerebral centres with which they are connected.—CHARLES K. MILLS, M. D., in the *Maryland Medical Journal*, Feb. 11, and 18, 1888.

L. F. B.

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Original Articles.

PARANOIA: SYSTEMATIZED DELUSIONS AND MENTAL DEGENERATIONS.

AN HISTORICAL AND CRITICAL REVIEW,

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[*Continued from last Number.*]

SCHUELE* (1886) gives a very detailed description of systematized insanity. He distinguishes in the most formal manner *Wahnsinn* from *Verrücktheit*, as will be seen from the place he assigns this in his classification. It is, in fact, in the second class, that of psychoses based on an incomplete development or on hereditary degeneration, that he places Verrücktheit. It appears there between the hereditary neurosis and simple hereditary insanity (insanity with delusions of possession by evil spirits, *folie du doute*, and quarreling or quibbling insanity, *folie de la chicane*), and moral insanity and idiocy; this *originare Verrücktheit* is, according to this author, grafted on an abnormal hereditary constitution characterized by original psychical anomalies; that is to say, it manifests itself at an early age; it would thus be only the hypertrophy of the original character. On the other hand, it is in the first class (psychoses in the completely developed individual), but in the second group

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* Schuele, *Klinische Psyschiatrie—Specielle Pathologie und Theropie der Geisteskrankheiten* (Leipsic, 1886). As this passage may seem obscure, we have

(cerebro-psychoses or diseases of the *invalid* brain) that we find *Wahnsinn.* This word is only a generic term which denotes the existence of delusional conceptions more or less united, systematized and associated in groups more or less connected, and forming a more or less crystalized whole.

Schuele has divided the chapters relative to *Wahnsinn*, properly so called, as follows: this primary *Wahnsinn* may be, first, *chronic;* second, *acute;* third, *stuporous.* The author describes the typical chronic form by saying that all the chronic cases tend to systematization, properly so called; a special chapter is devoted to chronic depressive *Wahnsinn*, comprising two sub-chapters: (*a*, delusion of

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thought best to give a tabular view of his classification to facilitate comprehension.

I. PSYCHOSES GRAFTED ON THE COMPLETE ORGANS—PSYCHIC DEVELOPMENT.

A. Psycho-neuroses.

- 1. *Of the sound brain* (psycho-neuroses properly so called)—
  - *a.* Mania, } with their secondary states.
  - *b.* Melancholia, }
- 2. *Of the "invalid" brain* (cerebro-psychoses)—
  - *a.* Mania, grave (furor);
  - *b.* *The systematized delusion* (*Wahnsinn*):
    - 1. Acute,
    - 2. Chronic,
    - 3. Stuporous.
  - *c.* Acute primary dementia; hallucinatory stupor.
  - *d.* Hysterical, epileptic, and hypochondriacal insanity; the periodic insanities, circular and alternating.

B. Insanities consecutive to extra-cerebral somatic diseases (fever, puerperal state, and intoxication).

- 3. *States of pernicious exhaustion of the brain*—
  - *a.* Acute, dangerous (acute delirium).
  - *b.* Chronic, with destructive degeneration (progressive general paralysis).
  - *c.* The psychic cerebropathies, or psychoses consecutive to subacute or chronic organic cerebral diseases, diffused or localized.

II. PSYCHOSES GRAFTED ON AN INCOMPLETE OR CONSTITUTIONAL ORGANO-PSYCHIC DEVELOPMENT, WITH HEREDITARY OR CONSTITUTIONAL PREDISPOSITION.

- *a.* Hereditary neurosis with transitory insanities.
- *b.* Simple hereditary insanity (*folie du doute et du toucher; folie impulsive;* delusion of persecution (litigationists).
- *c.* Idiopathic systematized insanity (originäre Verrücktheit).
- *d.* Degenerative hereditary insanity (moral insanity).
- *e.* Idiocy.

persecution; *b*, expansive form). The variety of acute *Wahnsinn* comprises four sub-varieties. The first is acute sensorial *Wahnsinn* (hallucinatory).

Here is described: *a*, the hyper-acute and acute hallucinatory *Wahnsinn* with exaltation (menstrual diseases); *b*, the sub-acute maniacal delusion of grandeur; *c*, the acute and sub-acute hallucinatory delusion of persecution; *d*, the depressive and then acute expansive *Wahnsinn; e*, the acute and at the same time depresso-expansive *Wahnsinn; b*, the acute hypochondriacal *Wahnsinn; c*, acute or sub-acute cerebro-spinal *Wahnsinn*. The second sub-variety is the acute melancholic *Wahnsinn* (demonomania); the third sub-variety, the expansive maniacal *Wahnsinn;* the fourth, the acute stuporous or stuporo-hallucinatory *Wahnsinn*. The stuporous form of *Wahnsinn* (attonita and katatonia), comprises three sub-varieties: *a*, expansive religious insanity; *b*, depressive religious insanity; *c*, a form resting on a basis of constitutional hysteria, without prepossession from that which describes in the chapter on hysteria. Here, in fact, Schuele describes an hysterical systematized insanity (*Wahnsinn* or *Verrücktheit* indifferently), which may consist of several types: *a*, a type approaching the idiopathic hereditary systematized insanity (*originaria hereditaria Verrücktheit*); *b*, a type that is the hypochondriacal systematized insanity (*hypochondriascher Wahnsinn*); *c*, a type characterized by fantastic caprices; *d*, a type characterized by a vague delusion of persecution without great systematization, and the character of which varies; *e*, a type described as a condition of acute abortive systematized insanity (*Wahnsinn*), presenting the form of sudden irresistible thoughts; *f*, katatonic systematized insanity (*Wahnsinn*); *g*, chronic incurable hysteria with symptoms of degeneration.

III.

Such are, in a word, the ideas expressed in the principal German* works upon systematized insanity; other countries have followed the impulse. Thus in Russia we find the conception of systematized insanity in the works of Tiling (1878-1879), Kadinski (1881), and Max Buch (1881), already cited, and of Rosenbach[*] (1884), who is of the opinion that *paranoia* can only develop on a basis of mental debility, because the sensorial troubles can be interpreted in the sense of a delusion which arises spontaneously elsewhere, and the elements of which these sensorial troubles do not furnish. Still further, the ambitious ideas are not a logical consequence but are often contemporary to the ideas of persecution which already indicate an exaggertion of the personality.

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*We desire here to thank our colleague Dr. Keraval, physician of the Colony of Vancluse, for the hearty manner in which he has assisted us; his profound knowledge of the German language has been of the greatest assistance in our bibliographical researches.

Dr. Greidenberg[†] (1885), studying acute hallucinatory systematized insanity (*paranoia hallucinatoria acuta*) distinguishes two forms of this; the one hereditary, and the other, the more frequent, asthenic, producing in their train sometimes an intellectual enfeeblement, and sometimes a true dementia, or tending to recovery. In England, Bucknill and Tuke[‡] (1879), reject monomania and describe *delusional insanity*, the word *delusion* of the English alienists designating the primary delusional conceptions (*les conceptions délirantes primatives*), the original lesions of ideation.

Maudsley[§] (1883), when he describes the *insane temperament*, especially in its *suspicious* variety. only describes in full the character of patients suffering from *Paranoia* or certain weakened forms. In America, Spitzka[‖] (1880-1883) adopts the conception of *paranoia*, which he describes, although he adopts the word monomania.[¶] He admits that this form of primary delusion is the expression of a true intellectual enfeeblement; he also classifies it in the group of states of mental enfeeblement, which are almost always hereditary and constitute a sort of chain whose extremities are formed on the one side by idiocy and on the other by insanity with primary systematized delusions. Between these he classes imbecility, moral insanity and epileptic insanity.* In another work,† the same author has given us a classification of delusions which he divides into *systematized* and *non-systematized delusions*. The systematized delusions are of two forms : first, *the expansive systematized delusions* (megalomania), subdivided into *a*, systematized delusions of social ambition; *b*, systematized delusions of an expansive erotic character; *c*, systematized delusions of an expansive religious character. The second form is that of *systematized depressive delusions*, subdivided into, *a*, systematized delusions of depressed social ambition; *b*, systematized delusions of a depressive erotic character, usually of persecution; *c*, systematized delusions of a depressive religious character.

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[*] Rosenbach.—*Messager russe*, 1884.

[†] Greidenberg.—*Messager russe*, 1885.

[‡] Bucknill and Tuke. *A Manual of Psychological Medicine*, 1879.

[§] Maudsley.—*The Pathology of Mind*, 1883.

[‖] Spitzka.—*A case of Original Monomania* (Medical Times and Gazette, February, 1881), and *Manual of Insanity*, New York, 1883.

[¶] [In the edition of his *Manual of Insanity* in 1887, Spitzka definitely adopts the term *Paranoia*. W. N.]

Regarding the *non-systematized delusions* these are incoherent delusions resulting from the destruction of the power of association, and the emotional delusions dependent on the exaltation of the mental sphere by a violent emotional trouble.

We may also mention in America the works of Beard,‡ Fenn,§ and Hammond.‖

IV.

But, after the Germans, the Italians have occupied themselves most in the study of *paranoia*. In a first memoir, Buccola¶ (1882) undertakes the study of primary systematized delusions and seems to concur with the opinion of Krafft-Ebing.

These delusions are to his mind the expression of a feeble mental state as shown by their etiology, their continuous and

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*Spitzka.—*St. Louis Clinical Record*, 1880, VII.

†Spitzka.—*Insane Delusions; Their Mechanism and their Diagnostic Bearing* (*Journal of Nervous and Mental Disease*, 1881).

‡Beard.—*Monomania and Monohypochondria* (*New York Medical Record*, March, 1882).

§Fenn.—*Original Monomania*, (*American Medical Weekly*, August, 1882).

‖Hammond.—*A Treatise on Insanity*, London, 1883.

¶Buccola.—*I deliri sistematizzati primitivi* (*Riv. Sper. di Fren.*, 1882, p. 80).

remitting course, etc. Moreover, complete physiological exercise of the mental functions cannot be judged solely by the persistence of the logic, but by the nature, the quantity and the association of the psychical energies, and the harmonious relations that should exist between the ideas, the sentiments and the acts.

In this work Buccola studies the genesis of the delusion, and remains undecided on the subject, questioning whether the hallucinations are primary and the delusional ideas only the interpretation of them or arise from unconsciousness. He studies the course of the systematization, especially in the delusion of persecution, of which he reports two cases.

Morselli and Buccola* (1883) show the special development of these delusions, their chronic course without dementia properly so called, and their limited curability. Regarding the delusion, two forms may be distinguished: first, the delusion of persecution, of a variabie nature according to the age, temperament, and education. In this form would come the quarreling insanity, a true delusion of active persecution. The second form would be the delusion of grandeur, associated at first or existing alone, most frequently with an erotic or religious coloring. The fixed ideas should be regarded only as an abortive form of these delusions, they being differentiated by the fact that the patient has a consciousness of his condition.

Regarding the clinical nature of these delusions Morselli and Buccola place them among the *degenerative psychoses*, and divide them into two classes : first, primary systematized delusions with anomalies of the development of the psychic individuality (P. originäre of Sander); second, systematized delusions showing themselves in a psychic individuality already developed. Then intervene some occasional causes (acute diseases, menopause, and traumatisms). Even in these cases, however, hereditary influence exists in the majority of cases.

The forms that Morselli and Buccola place under the head of acute primary insanity are the following:

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*Morselli et Buccola.—*La pazzia sistematizzata. Giorn.* della R. Academ. di Torino, 1882, p. 210.

1st. The intellectual monomania of Esquirol.

2d. Sensorial insanity, when the hallucinations are not brought on by mania or melancholia but from delusions, from an original lesion of the perceptive centres.

3d. The so-called cases of lypomania with delusion of persecution, in which the melancholic state is secondary.

4th. The hypochondriacal insanities, in which the synesthetic hallucinations are the pivot of the delusions, and where a delusion of persecution is often concealed under hypochondriacal ideas.

5th. Certain cases of hysterical insanity that present an erotic delusion without remission (Merklin and Schaefer).

6th. Certain cases of claustrophilia or claustrophobia that have been wrongly interpreted, and concealing a delusion of persecution. A certain number of analogous forms, all characterized by the predominance of a given group of ideas and tendencies constituting the abortive forms of primary systematized insanity, while others form the group of fixed ideas where consciousness remains.

7th. The forms intermediate between sanity and insanity (the insane temperament), the graphomanias with concealed ideas of grandeur.

8th. Certain cases of *folie lucide*, or *folie raissonnante.*

9th. The eccentric and original individuals.

Amadei and Tonnini* (1883) give us a very complete description of *paranoia*, showing that the delusion is only a phase and the culminating point of the disease. The development, characteristics, course, transformations or associations, and the termination of the delusions are clearly studied. But the most original point of the memoir is the classification. The authors admit by the side of a degenerative form a psycho-neurotic form, and they bring forward the following arguments:

1st. Absence in these cases of a constitutional element from which the disease could be foreseen; no usual symptom of neuropathy.

2d. Frequent existence of occasional causes or of temporary predispositions that may explain the *paranoia*, without which there would be a necessity of seeking a predisposition in the antecedents.

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* Amadei and Tonnini.—*La Paranoia et le sue forme* (*Arch. ital. per le malattie nervose*, 1883–1884.

3d. Often these cases recover, sometimes there results a certain mental enfeeblement.

4th. In these psycho-neurotic forms there is neither more nor less heredity than in mania or melancholia.

5th. The duration of the acute forms of the disease and the beginning of the psycho-neurotic forms is in contradiction with the former mental life of the patient, while this is not so in the degenerative forms.

Here is the classification of *paranoia* that they propose:

- I. DEGENERATIVE PARANOIA:
  - *A. Idiopathic (originaire).*
    - *a. Simple:* delusions of persecution, ambitious, religious, and erotic.
    - *b. Hallucinatory:* delusions of persecution, ambitious, religious, erotic, and hypochondriacal.
  - *B.* Late.
    - *a. Simple:* delusions of persecution and of quibbling, ambitious, religious, and erotic.
    - *b. Hallucinatory:* delusions of persecution, ambitious, religious, erotic, and hypochondriacal.
- II. PSYCHONEUROTIC PARANOIA:
  - *A. Primary.*
    - *A. Acute and curable.*
      - *a. Simple:* delusions of persecution, ambitious, religious, and erotic.
      - *b. Hallucinatory:* delusions of persecution, ambitious, religious, and erotic.
    - *B. Incurable,*
      - *a. Simple:* delusions of persecution, ambitious, religious, and erotic.
      - *b. Hallucinatory:* delusions of persecution, ambitious, religious, erotic, and hypochondriacal.
  - *B. Secondary.*
    - *a.* Post-maniacal.
    - *b.* Post-melancholic.

As is seen by this table, degenerative *paranoia* alone would be always primary, the secondary being solely the termination of a psychoneurotic state. This secondary form is considered by these authors, together with Krafft-Ebing, as a state of mental enfeeblement consecutive to some of the primary forms of psychoneuroses.

Regarding the degenerative defect in *paranoia* of the first category, this is never the most profound, and does not reduce the patients to the last degree of the scale of the degenerates. Schuele also, with good reason, separates these forms of degeneration, in the strict sense of the word, to make, as we shall see, the neuroses with a degenerative basis. But this is not simply a question of degree, and there should be intermediate forms between the degenerative forms of *paranoia* and certain simple delusional outbreaks observed in imbeciles, and also between certain states of mental debility and the idiopathic (*originaire*) *paranoias* where the delusion is but little accentuated.

In conclusion, Amadei and Tonnini hold that the sensorial delusion* (*Wahnsinn* of Krafft-Ebing), the true type of non-systematized delusions may pass into *paranoia* through the stage of fixed ideas, which is a rudimentary or prodromal form of *paranoia*, differing from this, however, by the consciousness of the subject.

Finally, we may distinguish (as Krafft-Ebing has done) true *paranoia* in certain more or less systematized delusions of epilepsy, hysteria, alcoholism, etc.

Raggi † (1884) considers that the rôle of the degener-

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*[In explanation of this term (*délire sensoriel*), Folsom's description (Primary Delusional Insanity, in Pepper's *American System of Medicine*) may be of service: "Transformed delusions (*sensorielle Verrucktheit*) arise usually in some anomally of sensation, which probably directs the delusions already forming in a mind in the early stage of disease rather than causes the disease. The causes lie in a deep-seated exhaustion of the nervous system, especially in the neuropathic constitution and profound hysteria. Various anomalous sensations give rise to a belief in delusions as to their being caused by individuals for a purpose, or to their being an indication of all sorts of impossible and most extraordinary changes in the part: the chest is of stone, the leg of brass, the head on fire, the hand of ice, and so on indefinitely. Hallucinations and a cataleptiform state are common. The variety of delusions which may arise is almost endless, and they may have their origin in the unhealthy action of any organ in the body; one of the most troublesome forms, called ovarian insanity by Skae, causes single women of severely continent lives to imagine all sorts of impossible marital relations with men whose lives are equally beyond scandal and above suspicion."—W. N.]

† Raggi.—*Dell' elemento degeneratio nella genesi dei cosi detti deliré sistematizzati primative (Arch. ital. per le mal. nerv., 1884).*

ative element in the genesis of *paranoia* is far from being demonstrated; he denies all the distinctive characteristics drawn from the etiology, evolution, symptomatic complexus, and the course.

To his mind heredity plays no larger rôle here than elsewhere; the priority of the idea in date to the troubles of sensation is not in the least demonstrated, and, on the contrary, the slightest amount of emotional trouble in these patients suffices to change or excite their delusions. Moreover, all the authors do not agree upon the symptoms, some regarding the hallucinations as primary, others as secondary to the delusion, which is one of persecution with some and of grandeur with others. Regarding the course, do not some authors admit that the delusions have an acute course, a thing that is incompatible with an idea of degeneration?

All these arguments are very specious, and it seems to us that it is sufficient to cite them to show how few of them should be taken into consideration.

In this same year (1884), Tanzi* published an historical study on *paranoia*, a kind of introduction to a monograph on this form of insanity made in collaboration with Riva.†

To Tanzi and Riva *paranoia* is a functional psychopathy founded on a degenerative basis, characterized by a particular deviation of the highest intellectual functions, implying neither a grave decay nor a general disorder; it is almost always accompanied by hallucinations and by permanent delusions more or less systematized, but independent of all definite occasional cause or of all emotional morbid condition, which pursues a course neither uniform nor continuous, but nevertheless essentially chronic, and generally does not in itself tend to dementia.

In only fourteen cases out of a hundred, according to Tanzi and Riva, heredity was unknown but not excluded, and in eighty-six other cases the *paranoia* had a degenerative basis either from heredity (77), or from diseases of infancy implicating the development of the individual (9.5). From this it is not unfair to conclude that *paranoia* is a form of mental debility. It is, as already shown by Amadei, Tonnini and others, simply a degenerative psychosis due to an hereditary or constitutional defect, as shown by its chronic course and insidious beginning and its variable symptomalology.

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* Tanzi.—*La Paranoia (delirio sistematizzato) e la sua evolutionei storica (Rev. sperim. di freu., 1884).*

† Tanzi et Riva.—*La Paranoia contributo alla storia delle degenerazion psichiche (Riv. sperimen. di fren., 1884, 1885, 1886).*

The psychical constitution of paranoiacs can be put in evidence only through the systematized delusion which rises on the mental constitution and is the exaggeration of this; and meanwhile this constitution is all important, constituting sometimes in itself the whole disease (indifferent *paranoia*), and showing forth again in the prodromes and in the periods of remission.

It consists especially in anomalies of the intelligence (associations of odd ideas and absurd judgments) or of the affective sentiments (egoism, defiance, romanticism, irritability, emotionality, sexual perversions, etc.).

The psychical characteristics of this constitution develop with the years until they reach a *degenerative maturity* at the age when a sane man is at the height of his intellectual power (thirty-two years on an average). It is then that the delusion generally develops, but sometimes it is lacking (in eccentric and original individuals), or it is insufficient to disturb the psychic equilibrium; the patient has no delusion properly so called, but he reasons falsely aud is paradoxical (*folie raissonnante*,—indifferent type).

In fact *paranoia* is a morbid constitutional form, and the delusion is only a symptom. Moreover, it is not absolutely specific and it may be found in other psychopathic forms without distinct psychological characteristics, but in these cases it is independent of the psychic constitution and arises under the influence of an incidental somatic cause; circulatory (mania or melancholia), inflammatory (general parlysis), toxic (alcoholism), etc.

As regards the genesis of the delusion it develops unexpectedly without a previous emotional morbid state, and it is accompanied by hallucinations that are secondary, affecting most frequently the sense of hearing, afterwards the general sensibility,—visual hallucinations being extremely rare. The delusion may undergo transformations, becoming either multiple or indetermintae, or be entirely wanting.

From this point of view paranoia may be divided as follows:

1st. Paranoia with delusions of persecution..
2d. Ambitious paranoia.
3d. Religious "
4th. Erotic "
5th. Intermediate " (Quarrelling insanity; *paranoia* without delusions).
6th. Mixed "
7th. Rudimentary " (Fixed ideas).

From the point of view of the onset two kinds of *paranoia* may be distinguished (an artificial distinction it may be, the ground remaining always the same):

1st. Idiopathic[*] *paranoia* (*originare* type of Sander).

2d. Late *paranoia*: *a*, post puberal; *b*, of the menopause (these two varieties following the biological evolution of the individual); *c*, simple (independent of the biological evolution).

Regarding the course, which is essentially chronic, it may be divided, according to the delusional symptom, into uniform (same type of delusion) and variable, and according to the mode of succession of the symptoms into continuous, remittant, and with exacerbations. All these varieties may be combined, and we shall have these a course:

- Uniform
  - Continuous (delusions of persecution).
  - Remittant.
  - With exacerbations.
- And variable.
  - Continuous (transformations of the delusion of persecution into ambitious delusion).
  - Remittant.
  - With exacerbations.

The exacerbations may be brought about by psychoneurotic attacks (mania, melancholia, or stupor).

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[*] In this translation the word *idiopathic* has been used to designate the *originäre* and *originaire* of the Germans and French, thus leaving the *primary* for the corresponding *primäre* and *primative*. W. N.

As regards the terminations, mental enfeeblement is little frequent, and absolute dementia is very rare. When it exists it may show itself under three aspects: first, premature senility, the expression of the rapid failure of the degenerated organism; second, dementia due to intercurrent psychoneurotic attacks (mania or melancholia); third, apparent dementia may appear in two forms: in the first the patient, seeing the uselessness of his ideas, remains calm, loses confidence, and becomes indifferent; in the second he concentrates himself more and more in his delusion, becomes exalted and extravagant, and gives himself up to disordered and incoherent actions.

Regarding the place *paranoia* occupies among the degenerations, Tanzi and Riva place it in the purely psychical forms (that is to say, without disturbances of motion or sensibility), called by Morselli *paraphrenias*, and in this sub-group may be distinguished, first, the intellectual psychical degenerations with or without delusions, that is to say, *paranoia;* and second, the affective psychical degenertions (moral insanity, congenital delinquency, and sexual perversions).

Furthermore, and notwithstanding the opinion of Bonvecchiato,* who finds this classification too systematized, Tanzi and Riva willingly admit mixed forms, both intellectual and affective, all resting on the same degenerative basis.

During the course of the publication of this long memoir, other works on this same subject have appeared in Italy.

Salemi-Paci (1885) distinguishes two kinds of *paranoia:* one, *simple paranoia*, independent of all degenerative element; the other, *degenerative paranoia;* he describes also a form of *consecutive* or *secondary paranoia*, but he does not see the necessity of making a particular form of it, as do the other two authors.†

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* Bonvecchiato.—*La pazzia sistematizzata primitiva.* Venice, 1875.

† Salemi Pace.—*La classificazione dela frenopathie. Il Pisani, 1885.*

This distinction, which is apparently very simple at first sight, is much less

One may well have doubts of the existence of *simple paranoia* when he sees an author place it by the side of moral, impulsive and sensorial insanity, and emotional delusions.

Angelo-Zuccarelli* (1885) reports an observation on primary *paranoia* with delusions of persecutions of a chronic form, that he held to be of a non-degenerative nature.

Guiccardi† (1886) agrees with the ideas of Tanzi and Riva regarding the interpretation of the psychical phenomena that characterize the paranoiac personality.

B. Battaglia‡ (1886) cites a case of *paranoia* with ambitious delusions, that is at least open to criticism. We shall content ourselves with remarking that the author pretends not to have found hereditary antecedents, nor signs of

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so when the following *résumé* of his classification is considered:

- GROUP I. *Cerebro-neuroses.*
  - Nervous insanity.
  - Hypochondria.
- GROUP II. *Dynamic cerebro-insanities.*
  - General delusions.
    - Simple insanities.
      - Melancholia.
      - Mania.
      - Circular insanity.
    - Diathetic insanities.
      - Specific.
        - Pellagous insanity.
        - Puerperal "
        - Syphilitic "
        - Alcoholic "
        - Rheumatic "
      - Neurotic.
        - Epileptic "
        - Choreic "
        - Hysterical "
        - Erotic "
  - Partial delusions.
    - Impulsive instinctive insanities.
      - Kleptomania, pyromania, agoraphobia, dipsomania, suicidal and homicidal insanity, etc.
    - Sensorial insanity.
    - Moral insanity.
    - Intellectual insanity
      - Metaphysical insanity, insanity of doubt, delusions, of touch, *simple paranoia.*
- GROUP III. *States of cerebral defect.*
  - Imbecility, idiocy, cretinism.
- GROUP IV.
  - *Paranoia—degenerative, consecutive, or secondary.*
  - Primary dementia.
  - Consecutive or secondary dementia.
  - Senile dementia.
  - Paralytic dementia.

* Angelo-Zuccharelli.—*Contribution a l'etude medico-légale de la Paranoia (Il manicomio, 1885).*

† Guiccardi.—*Psychologia e psychatria (Riv. sper. di fren., 1886, p. 531).*

‡ Bruno-Battaglia.—*Contribuzione alla casistica della Paranoia.* (*La psychiatre*, 1886, fasc. 3 and 4, p. 354.

degeneration. Nevertheless, he tells us that his patient had a feeble mind, was ill-balanced, loved the marvellous, was unstable, and was disgusted with life without good reasons; he lacked, he says, the faculty of adaptation to his social circle, and the spirit of rational criticism. Now, are there not here sufficient signs of a state of mental degeneration?

Morselli* (1886) reports a case of rudimentary impulsive *paranoia*. We have already seen that Arndt was the first to describe this form of *paranoia*.

This rudimentary *paranoia* (or rather the fixed ideas) has been divided by Tamburini† into three classes: first, the *simple fixed ideas* (ex: pure *folie du doute*), without a tendency to transform themselves into acts; second, the *emotional ideas* with simultaneous actions (ex: *folie du doute avec delire du toucher*); that is to say, with a tendency to the exteriorization of their motor content; third, the *impulsive ideas*. Morselli admits only two classes, uniting into one the last two of Tamburini, because in these cases there always exists, according to him, the tendency of an ideational representation to transform itself into an act. Regarding the ground on which these ideas develop, there is likewise much contest.

Krafft-Ebing,‡ Cantarano§ and Andriani‖ regard these forms as being always manifestations of degeneration. Others, with Berger,¶ Kroepelin,** Tamburini, Amandei and Tonnini, and Tanzi and Riva, admit that they may develop on a neurasthenic constitution, but are not always hereditary. Morselli inclines to this latter opinion and classes them in the *Paraphrenias* of the second group. The ideas

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[*] Morselli.—*Paranoia rudimentale impulsiva* (*Riv. sper. di fren.*, 1886, f. 4, p. 495.

[†] Tamburini.—*Sulla pazzia del dobbio* (*Riv. sper. di fren.*, *1883*).

[‡] Krafft-Ebing. *Lehrbuch der Psych*, 1879.

[§] Cantarano. *Contributo allo studio delle psicosi degenerative.* (*La psychiatria*, 1884)

[‖] Andriani. *Contributo alla conoscenza delle psicoside generative* (*idee fisse*) *La pschiatria*, 1885.

[¶] Berger. *Grubelsacht ein psychopathische symptom.—Grubelsacht und Zwangvorsellungen* (*Arch. f. Psych.*, Bd. vi. and viii.).

[**] Kroepelin. *Comp. der Psych.* Leipsic, 1883.

of this author will be made clearer by an explanation of the place that the different forms of *paranoia* occupy in his classification of mental disease. Properly speaking, Morselli admits only two typical forms of *paranoia* ; first, *idiopathic (originaire) degenerative paranoia* with its three varities of persecution, grandeur, and the erotic form (erotomania); second, *rudimentary paranoia* with its two varieties, ideational and impulsive. Both are classed among the *paraphrenias* (anomalies of cerebral evolution with abnormal formation or perversion of the personality). But although idiopathic (*originaire*) degenerative *paranoia* is a part of the sub-group of Paraphrenias formed by the *psychical degenerations* (paraphrenias depending on a psychopathic constitution most frequently of hereditary origin), rudimentary *paranoia* is classed under a second sub-group, that of *constitutional psychopathies* (Paraphrenias depending on a psychopathic constitution most frequently congenital).

Regarding the forms of acute *paranoia* and secondary *paranoia*, admitted by some authors, these are completely separated from the preceding and classed among the *psychoneuroses*, a sub-group of phrenopathies (diseases of the completely developed brain with morbid changes and alteration of the personality). The one, *paranoia*, called acute or hallucinatory or curable, is described under the name of *acute sensorial insanity* and placed by the side of maniacal or melancholic states. The other, *secondary systematized* insanity* (so called secondary *paranoia*), with its two forms of persecution and grandeur, is not considered, with dementia, as a terminal condition,—a conditional of intellectual enfeeblement. (Synonym, incomplete dementia.)

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*It should be noted that Morselli designates these forms under the names of of acute sensorial insanity (*Frenosi sensoria acuta*) and secondary systematized insanity (*Pazzia sistematizzata secondaria*). reserving for the idiopathic (*originaire*) and rudimentary forms the term *paranoia*, which seems thus ssociated in the author's mind with the idea of a neuropathic constitution, which may be congenital or hereditary.

[*To be Continued.*]

OPHTHALMOPLEGIA EXTERNA PARTIALIS.

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Read before the Neurological Section of the Academy of Medicine, April 13, 1888.

THE condition of paralysis of all the muscles moving the eyeball, *ophthalmoplegia externa totalis*, is well known and easily recognized. Its lesion consists of a general destructive process of a subacute inflammatory or degenerative nature in the nuclei of origin of the sixth, fourth, and third nerves upon the floor of the fourth ventricle and aqueduct of Sylvius. It is always bilateral. The destruction of the sixth nerve nucleus on either side causes a paralysis of conjugate movement of both eyes toward the side of the lesion ; and when both sixth nerve nuclei are involved together, the eyes look directly inward and cannot be turned from side to side. The destruction of the fourth nerve nuclei produces a paralysis of the act of looking upward and inward; and that of the third nerve nuclei deprives the patient of all other movements of the eyeball and upper lid, so that in the terminal stage of this disease double ptosis and immobility of both eyeballs are present. The motions of the iris are, however, unaffected in this disease, and the nuclei governing the movements are not involved in the degenerative process, though they lie quite near to and just in front of those governing the other muscles supplied by the third nerve. Twenty-seven cases of this affection have been collected by Mauthner in his lectures, a number of them accompanied by autopsy; and other cases published since 1885, some of which were brought together by Mittendorf in 1887,* have established the existence of this disease and the invariable character of its lesion.

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* Journal Nervous and Mental Disease, 1887, p. 78.

It is not to *total* ophthalmoplegia externa that I desire to call attention in this paper, but to a condition of paralysis of but one or two of the muscles moving the eyeball, a condition which may be termed *ophthalmoplegia externa partialis* to distinguish it from the condition already described.

It is of course well known that a paralysis of the external rectus muscle of one or of both eyes is a common occurrence due to a lesion affecting one or both sixth nerves in their long course upon the base of the brain. Upon this form of partial ophthalmoplegia I do not care to dwell, as it is well understood. Nor need I refer to paralysis of the patheticus or fourth nerve which occasionally occurs, but is extremely rare. It is especially to paralysis of one or two or more of the muscles governed by the third nerve that I desire to call attention. For there are several points of interest which arise in its study, and which have not been fully discussed either in neurological or ophthalmological literature.

Paralysis affecting but one or two of these muscles moving the eyeball is so rare as to warrant a report of every case observed. And the fact that six such cases with autopsies are now on record warrants an attempt at the localization of the lesion during life.

The following case has been under observation for a year, and has been in a stationary condition for the past nine months. It offers a number of points of interest for discussion.

S. D., a Frenchman, aged 56, a painter by occupation, and a resident of Providence, R. I., was brought to my clinic April 18th, 1887. He had been a healthy man all his life, with the exception of occasional attacks of rheumatism and frequent attacks of migraine. He had never contracted syphilis, and denied all symptoms of pulmonary, cardiac, gastro-intestinal and renal disease, although a physical examination revealed the existence of slight aortic obstruction producing a systolic murmur heard at the base and associated with slight ventricular hypertrophy.

He stated that about the first of April, 1887, he had been seized very suddenly with double vision and vertigo, objects appearing to move up and down constantly, so that he was much bewildered and unable to stand or to walk alone. He managed with help to reach his home, but has no recollection of what occurred during the three following days, during which, according to the statement of his family, he lay in a somnolent condition, but not comatose or paralyzed. He was then able to get up, but felt stupid, dizzy, and walked with difficulty, it being impossible for him to fix any object with his eyes; all objects being seen double and in motion. These symptoms have improved slightly, but he still feels weak, has vertigo and double vision. He has never had headache, nor has he felt any sensation of numbness or cold or pain in his body, and he has had no paralysis, tremor, or spasm.

Examination shows a well-nourished, intelligent, active man, whose facial expression is rendered peculiar by the position of his eyes. When at rest, they diverge slightly, and the right eye is turned upward, and the right pupil is slightly larger than the left. When the eyes are moved it becomes apparent that the motion is defective. The eyes can be turned from side to side together perfectly, but such motion soon produces lateral nystagmus of the right eye. They cannot be converged to an object nearer than two feet, because of slight weakness of the right internal rectus muscle. They cannot be turned downward below the horizontal line either together or when tested separately. When asked to look up, the right eye follows the object above the horizontal line, but the left eye does not. Both eyes, however, turn up and in, though this motion produces rotary nystagmus. The reaction of the pupils to light and in accommodation is prompt, though the right pupil contracts in accommodation more slowly and less completely than the other, and remains slightly larger. Tests by secondary deviation and double images confirm the conclusion reached by this examination, viz., that in the right eye there is paralysis of the inferior rectus and paresis of the internal rectus; and that in the left eye there is paralysis of the inferior rectus and superior rectus. There is no ptosis. There is no paralysis of the oblique muscles or of the external recti. One week later the paresis of the right internal rectus, the difficulty in convergence, and the difference in the size of the pupils had disappeared, but all the other conditions remained, and they have persisted until the present time (April, 1888). He is still suffering from general weakness, vertigo, and double vision, although the latter symptom no longer troubles him excepting when he attempts to draw lines in painting, when he finds that he does not draw accurately, and hence has had to give up his work. He often staggers in walking, but this is due to vertigo and not to ataxia, and it is not constant. He walks as well with eyes closed as with them open. Attempts to turn the eyes up or down, or upward or inward, produce nystagmus of a rotary kind, more marked in the right eye, and this always makes him dizzy. He has developed no further symptoms, is not paralyzed, has equal and normal tendon reflexes, and has no loss of sensation, vision or hearing. The diagnosis made is embolism, from the aortic valve, in the small arteries entering the posterior perforated space between the crura cerebri, and resulting in one or more small foci of softening in the tegmentum cruris.

This diagnosis can be reached by exclusion ; for it is impossible for the symptoms to have been caused by a tumor or a meningitis upon the base of the brain in the course of the third nerves. Such a lesion would not have come suddenly or have remained stationary, and would have involved the nerve as a whole, impairing all its functions and not affecting merely a part. Nor have we here a condition of acute inflammation with hæmorrhage in the floor of the aqueduct of Sylvius,* nor such a condition as occurs in a true ophthalmoplegia externa ; for there is no tendency manifest toward an extension of the symptoms, or to complete immobility of the eyes.

It is true that a hæmorrhage in the same region is with difficulty distinguished from an embolism, but in hæmorrhage some evidence of pressure upon the adjacent sensory or motor tracts is usually shown by unilateral symptoms, which have been wanting here; and here there is a roughened aortic valve to give rise to an embolus. The nature of the lesion is therefore easily determined.

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* Polio encephalitis of Wernicke.

The chief interest in this case lies in the fact that it seems to aid us in the localization of the oculo-motor nerve nuclei when taken in connection with other cases recently published, and accompanied with autopsy.

It is well known that by experimental irritation of the floor of the aqueduct of Sylvius in dogs, Hensen and Völckers claimed* to have located in rabbits the nuclei governing the various functions of the third nerve in the following order from before backward:

Ciliary muscle, accommodation.
Sphincter iridis, light reflex.
Rectus internus.
Rectus superior.
Levator palpebræ.
Rectus inferior.
Obliquus inferior.

If, however, the cases upon record of paralysis of individual muscles in combination be considered, it is evident at once that this order of the nuclei cannot be the one which obtains in man. In the case just related, for example, the rectus internus and rectus inferior of one eye, and the rectus superior and rectus inferior of the other eye, were paralyzed together, and no other muscles were affected. A single lesion would not explain the symptoms in either eye were this scheme true. It may be admitted, however, that the centres governing the movements of the iris do lie anterior to all the others. For Westphal has recently published a case in which all the nuclei governing the motions of the eyeball were destroyed, but in which two nuclei lying in the floor of the aqueduct of Sylvius near to its opening into the third ventricle were found intact. And to these nuclei he assigns the function of governing the motions of the pupil which were preserved in his case.* And a complementary case has recently been recorded by Bernhardt, in which the action of the pupils in light and accommodation was permanently lost, with only temporary affection of the other ocular muscles on one side.† And Leube has recorded a case,‡ with autopsy, in which the action of the right pupil was suspended and the right levator palpebræ alone of all the ocular muscles was paralyzed; the lesion lying far forward beneath the corpora quadrigemina anterior. By the kindness of Dr. Seguin, I am allowed to record a case similar to these two, in which there was a paralysis of the iris for three weeks, no other muscles being at all affected.§ The conclusion from these cases with and without autopsies is that the pupillary action is controlled by centres lying independent of and farther forward than all the other third nerve nuclei.

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* V. Graefe's Arch. f. Ophthal., 1887, vol. xviii., p. 1. They admit that the evidence is not wholly satisfactory.

The independence of the iris centres from those governing the ocular muscles had been reached several years ago by the investigation of Hutchinson‖ upon ophthalmoplegia externa, in which it was shown that paralysis of the ocular muscles implied a progressive destruction of the various nuclei concerned in motions of the eyeball from the sixth nerve upward through the third nerve. Hutchinson supposed that a lesion in the ciliary ganglion alone produced pupillary paralysis. But this conclusion was soon shown to be erroneous, and subsequent investigations proved that when the entire gray matter of the floor of the aqueduct was involved the iris was also affected. And subsequent cases, with autopsies, have confirmed this statement. The independence of the centres of the iris from those of the eyeball does not therefore imply their wide separation.

The question remaining for settlement is the relative order in which the remaining nuclei of the third nerve are arranged. What are the facts? There is, first, a case of Leube, (1) in which the paralysis of the pupil was accompanied by paralysis of the levator palpebræ, on the right side only. There is, secondly, a case of Bernhardt, (2) in which the paralysis of the pupil was associated with paralysis of the levator palpebræ, recti superior, inferior and internus, the latter four muscles all recovering.

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* Westphal, Arch. f. Psych., 1888, vol. xix., p. 858. It is true that the light reflex was lost in this case, but this W. explains by the existence of posterior spinal sclerosis.

† Bernhardt, Berliner Gesellsch. f. Neurologie; Arch. f. Psych., 1888, xix., 505.. Case 1.

‡ Leube, Deut. Arch. f. Klin. Med., 1887, xl.

§ See this Journal, this number, page 317.

‖ Hutchinson, Medico-Chirurg. Trans., 1879, vol. lxii., p. 307.

There is, thirdly, a case of Thomsen,* (3) in which a temporary paralysis of both pupils and both levator palpebræ was accompanied by permanent paralysis of both superior recti.

And, lastly, there is a case of Steffen,† (4) in which double ptosis was associated with sluggish action of the pupils, both ascribed to a destruction of the corp. quadrigemina.

These cases seem to prove that the centre governing the levator palpebræ lies next in order to the centres for the iris. Let us see what muscle is most frequently affected in conjunction with the levator palpebræ.

Bernhardt‡ has recently reported another case, in which the levator palpebræ on the right side, (5) and the levator palpebræ and superior rectus on the left side, (6) were paralyzed.

Mauthner§ cites a case of v. Grafé, in which congenital abscess of the iris was accompanied by partial ptosis and imperfect elevation of the eyeballs, (7) and remarks that he has seen a similar condition without absence of the iris (8).

A year ago I published‖ the history of a patient who was suddenly seized with paralysis of the left levator palpebræ and superior rectus, (9) and with slight right hemianæsthesia, all of which symptoms remained as long as the patient was under observation (five months).

In 1881, Kahler and Pick published a case,¶ with autopsy, in which the levator palpebræ and rectus superior and obliquus inferior were totally paralyzed on the left side, (10) and the rectus inferior and rectus internus were weak. The lesion in this case was a hæmorrhage in the tegmentum, involving the left red nucleus and the fibres of the third nerve which pass through it, especially those in its lateral part, the median part being unaffected.

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*Archiv. f. Psych., xix., 185.

† Cited by Nothnagel "Topische Diagnostic," p. 214.

‡ L. c. Case 2.

§ Mauthner, Vortrage ii., 4, 370.

‖ JOURNAL OF MENTAL AND NERVOUS DISEASE, 1887, p. 115.

¶ Zeitschrift f. Heilkunde, 1881.

To this case they added another, (11) in which a temporary paresis of the levator palpebræ, rectus superior and inferior, was accompanied by a paralysis of the rectus internus, which persisted until death. The obliquus inferior was not affected. The autopsy showed a small hæmorrhage in the right half of the tegmentum, involving the red nucleus and the fibres of third nerve passing through its median portion.

In the case of Thomsen already cited, the temporary paralysis of the pupils and levator palpebræ was accompanied by a permanent paralysis of the superior rectus on both sides.

From these cases it seems evident that the levator palpebræ and superior rectus are so often associated together in paralysis as to indicate a close proximity of their respective nuclei.

There have been a number of cases observed, in which the superior and inferior recti were associated together in paralysis, other eye muscles escaping. Thus a case of Wernicke* (12) is recorded, in which after a left hemiplegia there was a disturbance of the movement of both the eyes of such a character, that looking up or down was almost impossible, while lateral motions were well performed, there being at rest an evident paralysis of the left superior rectus (13). There was no ptosis. The autopsy showed an old contracted cicatrix in the right corp. quadrigemina and optic thalamus, which had resulted from a softening. It will be remembered that in the second case of Kahler and Pick (11) there was a paresis of the levator palpebræ rectus superior and inferior, as well as a paralysis of the rectus internus. In the case here presented (14) there was a paralysis of the left superior and inferior recti, no other muscle being affected. And in these cases the obliquus

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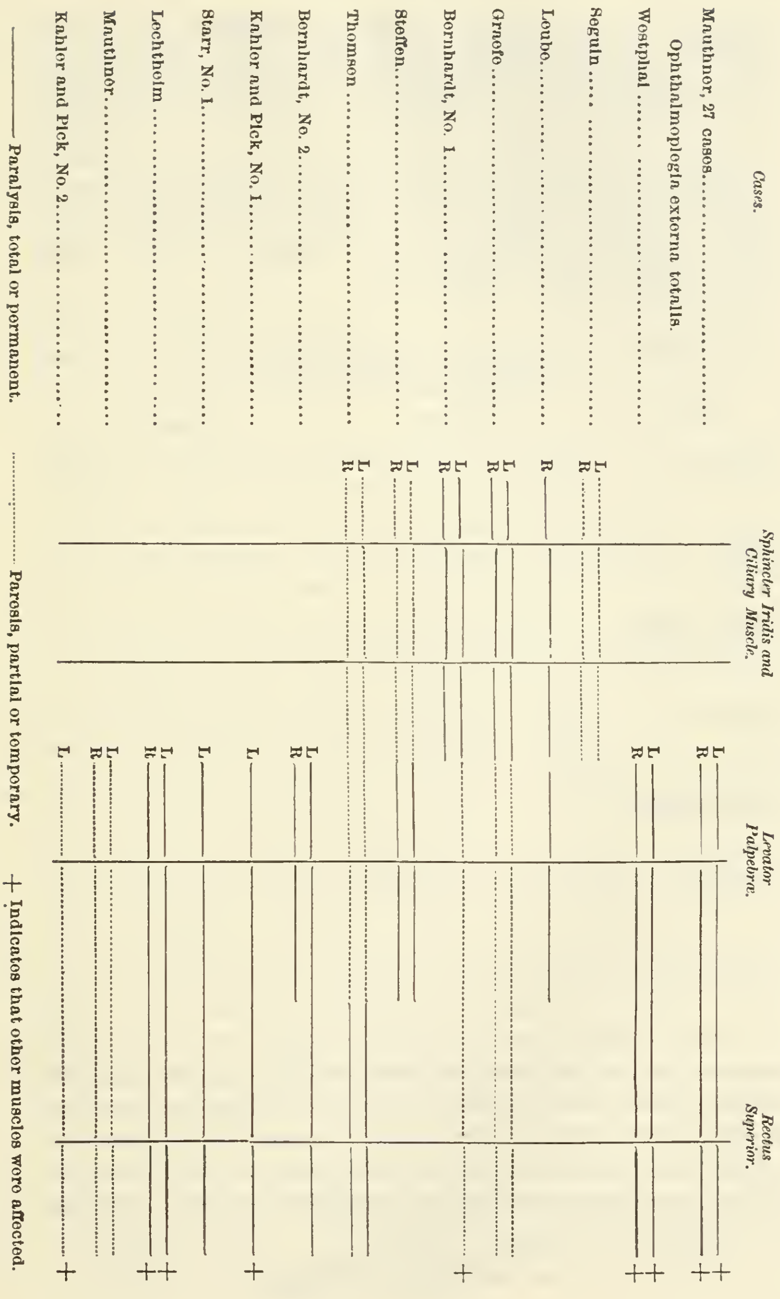
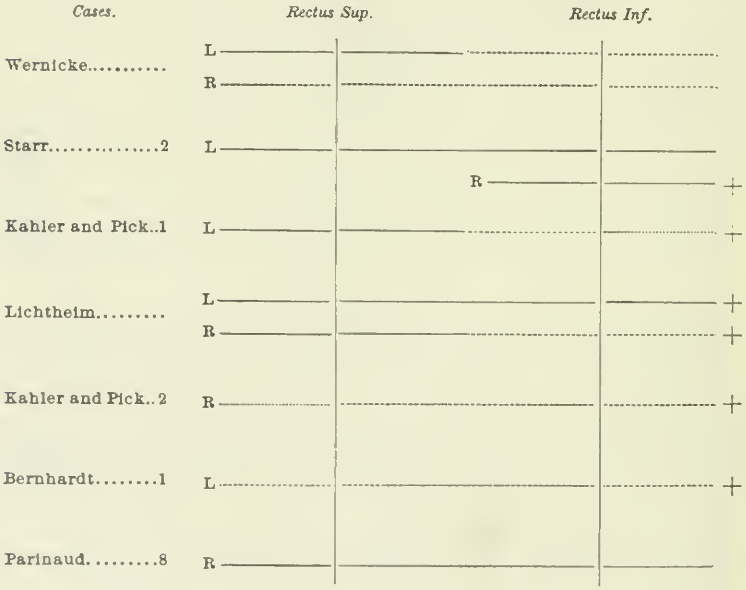
* Wernicke, Berl. Klin. Woch., 1876, No. 29, and Arch. f. Psych., viii.

DIAGRAM I.—To Show the Associated Paralysis of the Iris Muscles, Levator Palp., and Rectus Superior.


Cases.
Sphincter Iridis and Ciliary Muscle.
Levator Palpebræ.
Rectus Superior.
Mauthner, 27 cases
Ophthalmoplegia externa totalis.
Westphal
Seguin
Leube
Graefe
Bernhardt, No. 1
Steffen
Thomson
Bernhardt, No. 2
Kahler and Pick, No. 1
Starr, No. 1
Lechtheim
Mauthner
Kahler and Pick, No. 2
L
R
+
Paralysis, total or permanent.
Paresis, partial or temporary.
+ Indicates that other muscles were affected.

inferior was not involved when the superior rectus was affected. It is well known that the action of these two last-named muscles is homologous; but they are not, therefore, necessarily affected together, and cases recorded by Henoch,* (15) by Gowers,† (16) and the case of Thomsen already cited, in all of which the only permanent paralysis was that of the superior rectus in one or both eyes, prove that the obliquus inferior may, and often does, escape when the superior rectus is paralyzed.

DIAGRAM II.—To Show the Associated Paralysis of Rectus Superior and Inferior.



From these cases we may argue that the centre of the inferior rectus lies next in order to that for the superior rectus. But since in some cases the obliquus inferior has been paralyzed with the superior rectus and without an affection of the inferior rectus, it is necessary to locate its centre equally near to that of the superior rectus. This can only be done by assuming that the centres for the superior rectus and inferior rectus lie side by side, while that of the inferior oblique lies next to but behind that of the superior rectus.

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* Henock, Berl. Klin. Woch., 1864, No. 13.

† Gower's Dis. of Nerv. Syst., 1888, vol. ii., p. 174.

The last muscle to be considered is the rectus internus.

The fact that the recti interni are concerned in the act of accommodation has led to the supposition that its centre lies in close proximity to the centres for the pupil. And this view has prevailed in spite of the fact that its associated action with that of the abducens of the opposite side would lead to the hypothesis that the centres of the rectus internus and rectus externus would be near together. Paralysis of one rectus alone has not yet been observed, but it has been noted that a paralysis of the act of convergence may occur when the action of the muscles in other movements is preserved, and in six cases published by Parinaud,* the paralysis of convergence was accompanied by defective pupillary action. These cases would indicate that the nucleus of the internal rectus is near to that of the iris. A few cases have been recorded, in which paralysis of the internus has occurred with that of other muscles. Thus is the second case of Kahler and Pick the rectus internus was permanently paralyzed and the levator palpebræ, superior rectus and inferior rectus were paretic. In a case of Lichtheim,† (17) the right rectus internus, levator palpebræ and rectus superior were paralyzed, and the rectus inferior and obliquus inferior were paretic. In a case of Graefe ‡ the right rectus internus, levator palpebræ and rectus inferior were paralyzed, (18) and the left rectus internus was weak, while the left levator palpebræ was paralyzed. (19) In my case here published, the right rectus internus was temporarily paretic, and the right rectus inferior was permanently paralyzed. (20) In the cases of Kahler and Pick (No. 1), and Bernhardt (No. 1), the rectus internus was paretic as were also the rectus inferior and levator palpebræ, and rectus superior.

It is evident from these cases that the rectus internus centre is closely related to several other centres, viz., to

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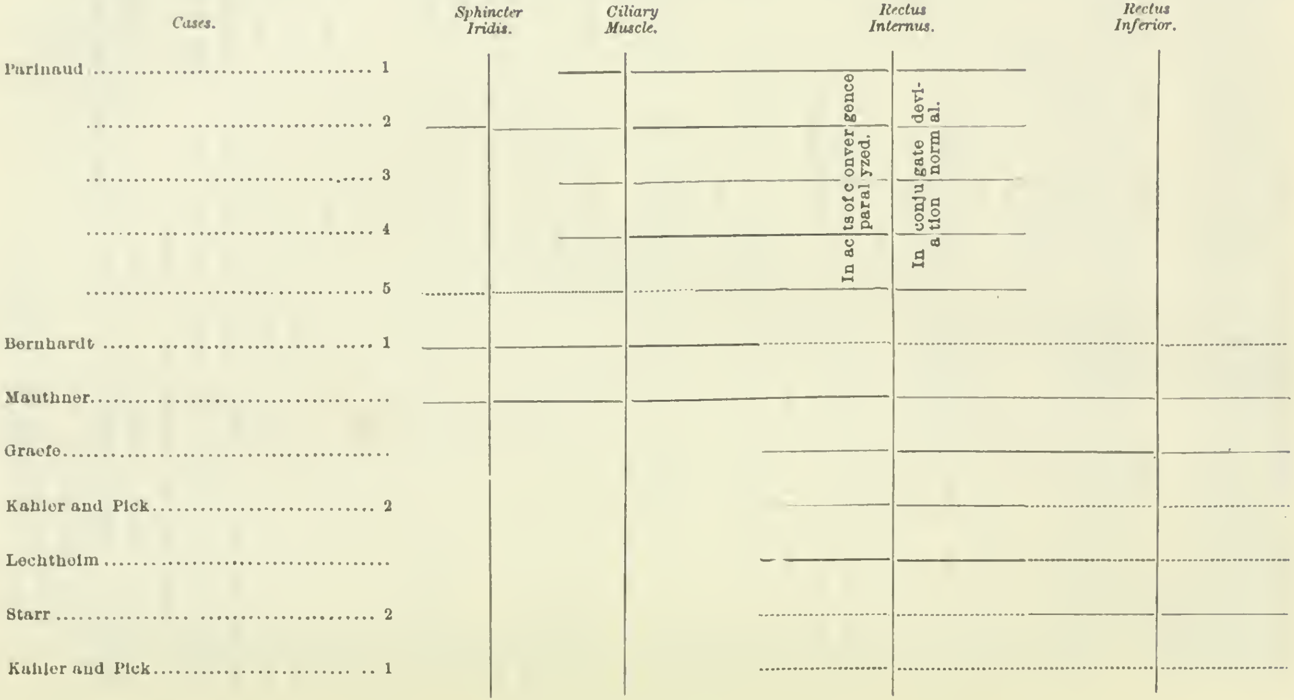
* Brain, Oct., 1886.

† Lichtheim, cited by Mauthner. Case 17, l. c.

‡ Graefe, cited by Mauthner. Case 28, l. c.

DIAGRAM III.—To Show Associated Paralysis of the Iris, Ciliary Muscle, Rectus Internus, and Rectus Inferior.

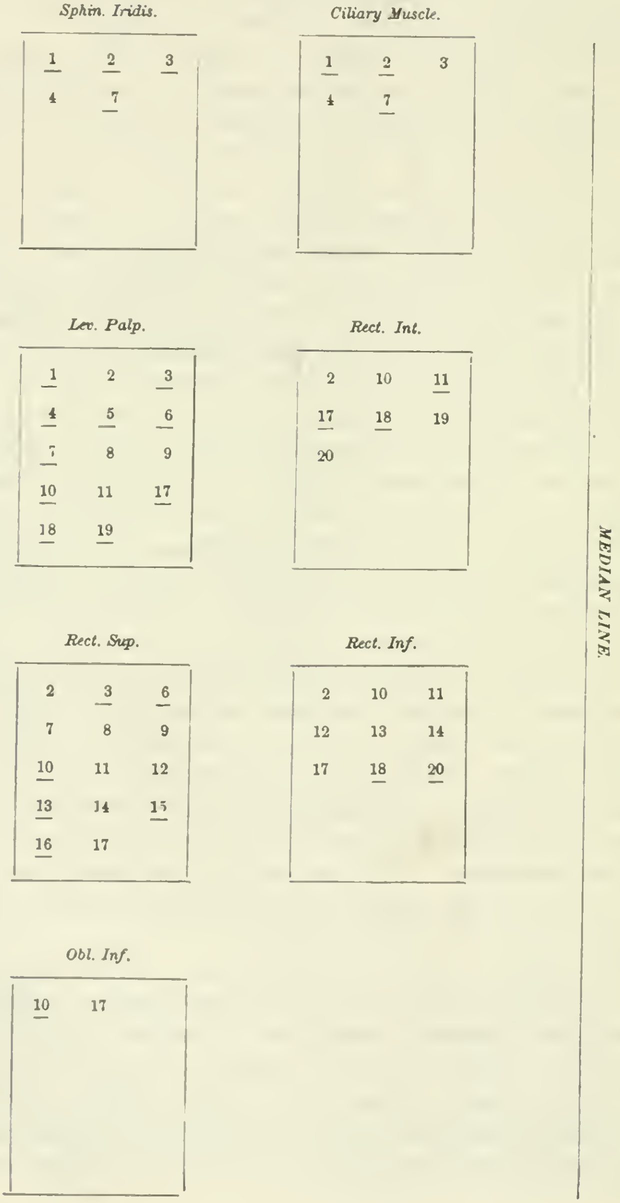


that of the iris, to that of the levator palpebræ, to that of the rectus inferior, and to its fellow on the opposite side. A central position with these muscles on its four sides is therefore the only one which will conform to the facts.

Can any diagram be made out which will explain all these cases. In 1881, an arrangement of the nuclei of the third nerve was proposed by Kahler and Pick, which was based entirely upon the two cases with autopsies here cited. And it must be confessed that when those autopsies are studied it seems as if the conclusion drawn was hardly warranted by the premises; for the lesions in each case involved the nerve roots of the third nerve rather than their nuclei; and this has been the case in most of the cases of partial ophthalmoplegia externa hitherto published. But the arrangement proposed by these authors has shown itself competent to explain the various cases as they have arisen since 1881, and the cases here brought together afford a brilliant confirmation of the essential part of the diagram of Kahler and Pick, and establish it, I believe, for the first time, upon a sufficient basis. It is true that Mauthner, in his lectures, has approved of Kahler and Pick's arrangement in preference to that of Hensen and Völchers', but he cites but five cases, and admits that the evidence is scanty. The cases here collected are twenty in number, each eye being counted as one case, unless both eyes were affected alike. The following diagram shows the relative position of the nuclei, and the extent and degree of the paralysis in each case cited, the numbers corresponding to the cases numbered in the text, and being underlined when the paralysis was complete or permanent. For convenience, the arrangement on the left side of the raphé only is represented, all the cases being tabulated as if on the left side.

The relative position of the centres for the sphincter iridis and ciliary muscle is still an open question. The observation of Westphal is sufficient to indicate that they lie side by side. Kahler and Pick placed the centre for accommodation in front of that for the light reflex. But the observation of Parinaud cited would indicate that the centre for accommodation being usually affected with that of the

DIAGRAM IV.—*The Position of the Third Nerve Nuclei on the Left Side.*


Sphin. Iridis.
1 2 3
4 7
Ciliary Muscle.
1 2 3
4 7
Lev. Palp.
1 2 3
4 5 6
7 8 9
10 11 17
18 19
Rect. Int.
2 10 11
17 18 19
20
Rect. Sup.
2 3 6
7 8 9
10 11 12
13 14 15
16 17
Rect. Inf.
2 10 11
12 13 14
17 18 20
Obl. Inf.
10 17
MEDIAN LINE.

rectus internus lies nearer to it than does the centre for the iris. The number of fibres passing from the corpora quadrigemina to the lateral of the two nuclei described by Westphal seems greater than the number passing to the median nucleus, and as these fibres probably complete the reflex arc for light reflex joining the optic and iris centres, I have placed the sphincter iridis nucleus outside of that for the ciliary muscle. It will be noticed that this arrangement brings the ciliary muscle centre near to that of the rectus internus as the cases of Parinaud require. Another fact which can be deduced from the diagram is the relative frequency of affection of the various muscles, it being evident that the superior rectus and levator palpebræ are most frequently paralyzed, and the superior oblique is rarely involved.

One pathological fact of importance remains to be noticed. In all the cases recently examined after death, in which one or more of the muscles of the third nerve was paralyzed, but in which total ophthalmoplegia externa was not present, the lesion found did not involve the nuclei of the third nerve in the aqueduct of Sylvius, but the roots of the nerve on their way from those nuclei through the tegmentum to their exit from the crus. These roots pass through the red nuclei of the tegmentum. These nuclei are closely connected with the superior peduncles of the cerebellum. It is not at all unlikely, therefore, that the vertigo which so frequently occurs in such cases is due not so much to the disturbance of vision, but to the lesion of these nuclei, and is comparable to the vertigo of cerebellar disease. In my own cases the patients complained of vertigo in the dark and when the eyes were closed, as well as when they were open. It seems warrantable, therefore, to conclude that in any case of partial ophthalmoplegia externa associated with vertigo the diagnosis of a lesion in the tegmentum cruris affecting the red nuclei may be made, provided the vertigo is not ocular in origin. While in cases of total ophthalmoplegia externa in which vertigo is not a marked symptom the diagnosis of a lesion in the gray matter of the aqueduct of Sylvius above the tegmentum is justifiable.

CONCLUSIONS.

Therefore, in any case in which the muscles moving the eyeball are involved, it may be possible to locate the lesion. If the iris alone is affected, the lesion is small, and lies either in the ciliary ganglion in the orbit, or just at the opening of the aqueduct of Sylvius into the third ventricle. If all the muscles of the eyeball are affected together, the external rectus and superior obliquus, as well as those supplied by the third nerve, *excluding the iris*, the case is one of ophthalmoplegia externa totalis, and the lesion lies in the gray matter of the floor of the fourth ventricle and of the aqueduct of Sylvius. Both eyes are then involved. If all the muscles of the eyeball supplied by the third nerve are affected, *including the iris*, the case is one of total peripheral paralysis of the third nerve, and the lesion lies on the base of the brain, and may in time implicate other cranial nerves. One eye is usually alone affected.

If one or two of the muscles of the eyeball supplied by the third nerve are affected, others escaping, the lesion lies in the tegmentum of the crus cerebri, between the nuclei of origin and the point of exit of the third nerve. One eye or both may be affected, but both eyes are rarely affected in the same manner.

There is but one exception to the last conclusion, and that is in the case of post diphtheritic ocular paralysis, in which the peripheral branches of the third nerve are affected after the entrance of the nerve trunk into the orbit. And here the existence of a diphtheria preceding the paralysis will establish the diagnosis.

NOTES OF FIVE CASES OF OPHTHALMOPLEGIA.

By E. C. SEGUIN, M.D.

THESE notes were read before the New York Neurological Society in the course of the discussion of Dr. Starr's valuable paper on the subject. The cases present points of interest which perhaps justifies their publication.

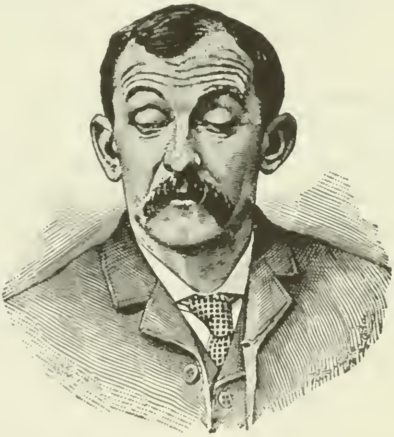
Two cases presented areas of anæsthesia in the distribution of the trigeminus; and one case developed during my observation paralysis and atrophy of the muscles of mastication. This coincidence of lesion of the motor and sensory roots of the trigeminus is, I am informed by Dr. Starr (who has had occasion to search the bibliography of the subject thoroughly), almost if not quite without precedent.

Case V. was one in which only the ciliary apparatus was affected, and is especially interesting from a therapeutical standpoint.

I. Ophthalmoplegia externa et interna bilateralis.

A man, æt. 31 years, first seen at the Manhattan Eye and Ear Hospital, January 4th, 1884. Nine years ago had a chancre, followed by secondary symptoms, treated by two well-known New York physicians. After working in a basement and committing various excesses, one morning, about two years ago, he suddenly discovered double and dim vision, and external strabismus of the left eye. Afterward, along with symptoms of general ill-health, he suffered from pains, dull and shooting, mostly in the legs, occasionally in the arms; without paresis or numbness. Hair has come out. About three months ago suddenly lost consciousness, and fell, cutting his head. About five months ago the left testicle became enlarged and hard. Has recently returned from Hot Springs, Ark., where he took mercury and much K I.

Present condition. Partial double ptosis, needing help of frontalis to expose the pupils. In the right eye all the muscles supplied by N. III. are more or less paralyzed; the superior and inferior recti acting feebly. The external rectus (N. VI.) is normal. In the left eye (which has improved during preceding treatment) all muscles supplied by N. III. act, though feebly; the external rectus is normal. The frontalis is constantly and automatically active to secure vision. The pupils are of medium size, the left larger (left eye first affected); they are completely motionless to light and to accommodative effort. Examination in the ophthalmic classes gives the following results: Right V. $\frac{20}{70} + \frac{1}{18} = \frac{20}{40}$. Left V. $\frac{20}{50} + \frac{1}{18} = \frac{20}{40}$. Accommodation right $\frac{1}{16}$, left $\frac{1}{14}$. No lesion of optic nerves; fields not tested.



Engraved from a photograph of Case I. Automatic action of frontalis, ptosis, and divergence of eyes shown.

Besides the ocular symptoms there were slight signs of crossed paralysis. The left cheek seemed a little inactive, the tongue deviated slightly to the right and the grasp was, R. 42° and 44°, L. 45°, which in a right-handed man meant paresis. The knee-jerk is exaggerated on both sides. Stands well with eyes closed (claims that while at Hot Springs he staggered when standing with closed eyes). The left testicle is hardened, and slight hydrocele is present. Alopecia is present, though less than a few months ago. Heart and the viscera normal, except bladder. It was subsequently noted that the power of muscles supplied by the left N. III. was somewhat variable.

As regards the bladder, the patient complains of both feeble and involuntary micturition. A No. 12 (Engl.) sound passes into the bladder without the least resistance. The act of rising from the recumbent posture causes escape of urine.

Treatment with increasing doses of K I, and galvanism to eyes and lumbar region.

In May, 1884, it is noted that sharp pains recur from time to time in the legs, and in the right hypothenar eminence. Eyes and bladder substantially as before (temporary improvement in eyes in February). Hoarseness appeared about this time, and an examination in the throat department revealed a scalloped ulcer on the right vocal cord, with inaction of this organ. Under local treatment and prolonged use of mercury and iodide of potassium this ulcer was cured by the end of September, but some hoarseness remained. Eyes and bladder as before. In November it is noted that he has had severe fulgurating pains, with hyperalgesia [illegible] spots where pains occurred, in thighs. In February, 1885, the bladder continues feeble. Pupils are as before. The ptosis is nearly total on the left, partial on the right side. On the other hand, the ocular muscles on the left side act almost normally, while those on the right are as before; internus completely paralyzed, superior and inferior recti weak. Occasional fulgurating pains in legs; knee-jerk good; no numbness. In April the fundus is noted as normal. The patient complains that "after chewing awhile, his chin becomes powerless." Examination shows distinct paresis and atrophy of the temporal and masseter muscles on both sides. The last note made of this case was in March, 1886, when it is stated that eyes are as before (ptosis perhaps greater); the bladder is still paretic, and the muscles of mastication show objective as well as subjective paresis, and atrophy; masseters very thin. No marked facial paresis; can whistle and hold water or air in mouth. The tongue is flabby, but not wrinkled; is still a little hoarse. A photograph of the patient taken about this time shows very well the compensatory automatic constant contraction of the frontalis to secure some vision. (*Vide supra.*)

This case shows signs of extension of the lesion to nuclei of the motor roots of the trigeminus. It also exhibits a trace of crossed paralysis; face on left side, limbs on right.

The fulgurating pains, with hyperalgesia, and the fall in degree of knee-jerk, during two years of observation, would seem to justify a suspicion of incipient posterior spinal sclerosis.

II. Ophthalmoplegia externa et interna bilateralis.

A woman, æt. 20 years, sent for examination to my class at the Manhattan Eye and Ear Hospital, Nov. 4th, 1887. The history is simply that of progressive paresis of both levatores palpebrarum; the left more affected. There has been occasional diplopia. The right pupil is active, the left sluggish. No other nervous symptoms. Patellar reflex fairly good. The post-cervical glands are slightly enlarged, but no history of syphilitic symptoms can be obtained.

Nov. 18th.—Has had KI. in doses of fifteen grains three times a day. Pupils much better; the left much better (this pupil is a little wider at rest and in medium light). Palpebræ as before. Patient was not again seen.

III. Ophthalmoplegia externa et interna bilateralis.

A man, æt. 40 years, was referred to me, July 27th, 1887, by my friend, Dr. J. B. Isham, for treatment during his temporary absence. In the last five or six years has been troubled by imperfect vision. Eighteen months ago external strabismus of the right eye, and later ptosis, appeared. About four months afterward the external rectus was cut, and a month later a piece of skin excised from the upper eyelid, but neither operation did good. Some four or five months later still, slight improvement occurred in the eyelid. Six weeks ago ptosis gradually developed on the left side, progressing to complete closure of the eye, but in the last few days a return of power has begun. With the onset of first symptoms had diplopia. No marked headache until recently, when a dull pain has been constant in the left parietal region; not distinctly nocturnal. Micturition has become slow; the legs tire easily; he is perhaps a little unsteady on legs, more especially in putting on his trousers. Denies sharp pains and numbness in limbs; but in the last two months there have been slight numbness and anæsthesia in the distribution of the left trigeminus. No injury to head. Twenty-two years ago had a double chancre, but never any secondary symptoms.

Examination: Almost complete ptosis on left side; on right the pupil is just visible, and there is some voluntary power in the levator. Eyeballs are slightly divergent. In the right eye all muscles suplied by N. III. are quite inert; the external rectus is normal. In the left eye all the muscles are paralyzed, the external rectus least, the superior obliques on both sides act. The pupils are equal and of medium size, but react neither to light nor to accommodation. Vision, R. $\frac{15}{40}$, L. $\frac{15}{50}$. Optic nerves appear normal, but vessels are small. The tongue is straight but tremulous; the left facial muscles act less well than the right; no weakness of extremities. Patellar reflex exaggerated. Sensibility is normal except in distribution of the left trigeminus (excepting the mental branch); the left half of tongue feels numb. Memory good. Has had inunctions of mercury to soreness of gums.

Ordered saturated solution of KI. at bed-time, sixty drops, increased by five daily; also a mixture, each dose of which contains $\frac{1}{25}$ grain of red iodide of mercury and fifteen grains of iodide of potassium, after each meal.

Sept. 9th.—Has reached dose of one hundred and twenty drops of KI. after breakfast and two hundred and fifty at bed-time (mixture omitted some time ago), and has had galvanism to eyes and cervical region. For a few days in August there was some return of power (?) in left eyelid.

No improvement on the whole; eyes same, anæsthesia as above, micturition slow. The iodide was subsequently increased to four hundred and fifty drops (equal to about four hundred and fifty grains) a day; then stopped and strychnine ordered.

There was in this case extension of disease to the sensory root of the left trigeminus, or possibly to the trunk of the nerve.

IV. Ophthalmoplegia externa bilateralis.

A man, æt. 42 years, was referred to my class at the Manhattan Eye and Ear Hospital by Dr. Carey, Assistant Surgeon in the Ophthalmic Department, on November 14th, 1884. Has been a sailor, but is now in the life-saving service. Has enjoyed good health; denies syphilis; has healthy children. No injury to head. Last March, after much exposure, and straining eyes in reading signals, he noticed some dimness of vision for distant objects. In May the right eyelid drooped, and the left also in June. Before this, not long after the first symptoms, he had noticed numbness of the skin in the right temple near the angle of the eye, and since, the numbness had extended to other parts of the right face. No deafness or loss of taste; no dizziness or headache. No peripheral pains or numbness.

Examination: Presents peculiar physiognomy of paralysis of levatores palpebrarum, with automatically acting frontalis, and half-shut eyes. No facial or lingual paresis. In the right eye all muscles supplied by N. III. (except inferior rectus?) are paretic, the external rectus is normal. In the left eye all the muscles are paretic, especially the external rectus (N. VI.) and the superior rectus. Has a feeble degree of conveyance. The pupils are equal, of medium size, and react well to ordinary daylight and to accommodative effort. According to Dr. Carey's report, vision is normal, except for slight presbyopia; reads Jaeger No. 2 at 12". The right side of face, temple and ear present slight anæsthesia and analgesia. Knee-jerk is exaggerated, and a distinct wrist-reflex is obtained.

On December 1st, after having taken strychnine in doses

$\frac{1}{30}$ and $\frac{1}{20}$ grain, three times a day, it is noted that there is improvement in the ocular muscles; the frontalis is less active. Patient returned to his post on the Jersey coast, and has not reported.

This case is remarkable as showing besides paresis of both third nerves, a slight lesion in the sensory nuclei (or in the trunk) of the right trigeminus, and a marked lesion of the nucleus of the left N. VI. The filaments of the third nerves which innervate the iris and ciliary muscles escaped.

V. Ophthalmoplegia interna bilateralis.

A married woman, æt. 35, was referred to me by my friend, Prof. C. R. Agnew, on January 16th, 1888, for an opinion as to the pathology of the symptoms she presented, viz., double mydriasis and paralysis of accommodation. R. V. $\frac{20}{200}$, L. V. $\frac{20}{200}$; no improvement by glasses. Reads Jaeger No. 14 without glasses, No. 5 with + 28 (Agnew). Dr. Jas. A. Booth examined the patient first, as I then happened to be confined to my room by illness. He obtained the following history: Cataract has occurred in her mother's family. When nine years old, after a coasting accident, her eyes were much swollen for a time, and they have "troubled her ever since." Eleven years ago "blisters appeared on the eyelids," which affection was cured by Dr. Williams, of Boston. (Dr. Agnew thinks that this may have been phlyctenular keratitis.) Fourteen months ago, suddenly found that she could not see clearly—everything was blurred; and she noticed that her pupils were dilated. This condition has continued. Denies all symptoms, direct and indirect, of syphilis. No injury to head since that, twenty-six years ago.

Examination; Pupils extremely dilated, only a small rim of iris being visible in both eyes. They do not respond to light or to accommodative effort. The optic nerves appear slightly grayish, or at least not normal. Denies having used any drops in eyes or belladonna outside. In Dr. Agnew's note it is stated "that the fundus scanned with the ophthalmoscope has a queer look—astigmatic, and yet we cannot improve her V. with any glass." The ocular muscles act well, except possibly the right external rectus.

Stands well with eyes closed; patellar reflex high. No other symptoms. Eserine solution to be dropped in the eyes twice a day.

January 23d. Temporary pupillary contraction and better vision for near objects resulted from use of eserine. I now see the patient for first time, and verify the correctness of Dr. Booth's examination. No eserine has been used this morning, and the pupils are both extremely dilated, much more than in cases of third nerve paralysis. They are about equal, measuring about seven mm. in diameter, and with about one mm. of the iris visible. I am strongly impressed with the patient's peculiar muddy quasi-cachetic appearance, so suggestive of constitutional syphilis, and question her closely about evidences of this disease. All that is admitted is, that three years ago she suffered from pains in the right upper arm, worse at night, for three or four days. No miscarriages. The post-cervical glands are, however, distinct. I direct the continuance of eserine drops in the eyes twice a day, and prescribe a solution, each dose of which contains $\frac{1}{8}$ grain of red iodide of mercury, and fifteen grains of iodide of potassium, to be taken after each meal. (Patient states that the right pupil was first dilated.) On January 27th, Dr. Booth ordered the dose of mixture to be doubled. February 1st. Has had no eserine since 7 P. M. yesterday (fifteen hours). The left pupil measures four mm., the right six mm. Both pupils now act in accommodative effort, but not to light. Ordered a new solution, each dose of which contains $\frac{1}{6}$ grain of red iodide of mercury and sixty grains of iodide of potassium after each meal. February 4th. No eserine since last note. Right pupil six mm., left four mm. in repose. Both act fairly well in accommodation. February 10th. Very great improvement: the left pupil is of normal size, the right is still larger; both act well in accommodation, and the left shows a distinct reaction to light. Patient left for home with instructions to take bichloride of mercury $\frac{1}{30}$ grain after each meal for two weeks, then fifty drops of saturated solution of iodide of potassium after each meal for two weeks, and so on alternately. On same day, eyes re-examined by Drs. Agnew and Webster. R. V. = $\frac{20}{70}$ in each eye; no improvement with glasses.

Reviews.

A MANUAL OF DISEASES OF THE NERVOUS SYSTEM, By W. R. Gowers, M. D., F. R. C. P. American Edition, with 341 Illustrations. P. Blakiston, Son & Co., 1888.

From a small beginning a great work has gradually been evolved. Less than ten years ago Gowers put forth a very modest little book on the Diagnosis of diseases of the Spinal Cord, which was soon followed by an equally modest treatise on Diseases of the Brain. Two years ago the first half of this manual appeared, comprising Diseases of the Spinal Cord and Nerves, and now this manual of diseases of the entire nervous system is placed before us.

The present volume is an exceedingly clumsy one of 1357 pages, so voluminous indeed, that it is almost impossible to hold it in the hand for even a few minutes with any degree of comfort. Why the American publishers should have insisted on putting this vast amount of matter into a single volme, we cannot understand. To make matters worse, the typographical appearance of the book has suffered, the type is often blurred and the illustrations unnecessarily indistinct. Publishers should remember that students of a book such as Gowers', will not refrain from buying a work because it happens to appear in two volumes. With this protest we can pass on to the work itself.

The most conscientious reviewer cannot be expected to read 1357 pages of a manual from beginning to end, but he can put the book to a different yet satisfactory test. The present writer has had Gowers' manual at his elbow for the last six weeks, and during that time has made it his chief book of reference on matters neurological. During this time a very wide range of subjects has been consulted; among these were neuritis, locomotor ataxia, myelitis, acute infantile paralysis, cerebral hemorrhage, ataxic paraplegia, pseudo-hypertrophic paralysis, brain tumors, tetanus and neuralgias, and many more. The work has not been found wanting in any respect.

All these chapters have been written with unusual care. The author's wide clinical experience enables him to lend the charm of personal observation to the discussion of the most abstruse forms of nervous disease, while every earnest student will stand aghast at the author's wide reading of neurological literature. He has liberally consulted English, German and American articles, a little to the neglect, we think, of French writings.

Besides being a thorough clinician, Gowers is an admirable pathologist, and in no work on nervous diseases that we are acquainted with has the pathology of nervous diseases been so thoroughly discussed as here. Not quite so much can be said of Gowers' therapeutic suggestions; they are meager rather than otherwise. Yet it is questionable whether the author has not after all shown sound judgment in limiting himself to therapeutic measures of known value instead of entering upon the efficacy of this or that drug in this or that special little disease.

Gowers has followed in the footsteps of Strümpell and others in beginning his book with diseases of the peripherial nervous system, preceded by a general introduction on symptomatology; next in order come diseases of the spinal cord; then diseases of the brain, and lastly general and functional diseases of the entire central nervous system.

By way of introduction to diseases of the spinal cord and brain, Gowers gives excellent chapters on the anatomy and physiology of the cord and brain. These chapters are of a very high order of excellence, and as far as the brain is concerned no better and more concise description of its anatomy has been given in any English text-book. For the purpose of the student or clinician, all that he needs to know on this head, he will find lucidly stated in this chapter.

We are a little surprised that even Gowers should have slighted the anatomy of the peripheral nervous system. Every teacher of neurology will agree with the experience of the present writer that the average medical man knows just a little more (to be sure it is not much at that) of the anatomy of the spinal cord than he does, say, of the "make-up" of the brachial plexus. A few more good illustrations and a short descriptive account of the peripheral nerves would have added to the value of the first section of this book.

The discussion of the special diseases of each of the three large divisions of the nervous system is preceded by a chapter on the symptomatology of that division. The plan is a good one and well executed, but it increases the number of cross-references. Thus, to get a complete idea of excessive knee-jerks, the student must seek this information in part in the chapter on symptomatology of diseases of the spinal cord, and in part in the chapter on primary spastic paraplegia. For the differential diagnosis of apoplexies, he must look up apoplexy and find a little but by no means as much as he had a right to expect under cerebral hemorrhage.

But we will not quibble with the author regarding the arrangement of his book. There is room for a difference of opinion on such points. The author has shown unusual discrimination and restraint in giving due consideration to each subject according to its merits; and not enlarging unduly upon subjects which the author had made special subjects of study, and yet his special studies tell in the admirable chapters on muscular paralysis, on optic nerve symptoms, and on spinal scleroses. Functional nervous diseases are slighted just a little, and we are astounded that but one page out of 1357 should be devoted to neurasthenia. The author denies the justice of considering neurasthenia a clinical entity. This challenges the conviction of those of us who are thoroughly familiar with this *morbus Americanus*, and who know that there are cases of neurasthenia which are not merely cases of "neuralgia, headache, cephalic sensations, hysteria or hypochondriasis." It is surely as distinct a clinical conception as hysteria is.

Gowers' Manual is herewith recommended to the general and to the special student. It is not too detailed for the former, while for the specialist it is explicit enough as a first book of reference. It is, on the whole, an admirable treatise.

B. S.

Society Reports.

PHILADELPHIA NEUROLGICAL SOCIETY.

Stated Meeting, March 26, 1888.

The President, S. WEIR MITCHELL, M.D., in the Chair.

Dr. S. WEIR MITCHELL read a paper on

LOCOMOTOR ATAXIA CONFINED TO THE ARMS: REVERSAL OF ORDINARY PROGRESS.

Drs. CHARLES K. MILLS and W. C. CAHALL reported

SIX CASES OF EPIDEMIC CEREBRO-SPINAL MENINGITIS.

Dr. WILLIAM OSLER said that it was interesting to note that in this city cerebro-spinal meningitis had been endemic for so many years. Dr. Stillé states that every year since 1863-64, deaths from this affection have been reported. Many of the cases were not cerebro-spinal meningitis. Mistakes in diagnosis were very common in this affection. Three or four instances of this had come under his observation. One case of smallpox was diagnosticated as cerebro-spinal meningitis. Cases of what the French term the cerebro-spinal type of typhoid fever, were frequently diagnosticated as cerebro-spinal meningitis, and at the post-mortem the brain was deeply congested, while the intestines exhibited the characteristic enteric lesions. The fifth case reported by Drs. Mills and Cahall resembled typhoid fever with severe cerebro-spinal manifestations. In certain of these cases he did not think that it was possible to make a positive diagnosis, unless the base of the brain was involved.

Dr. MILLS said that the fact that in the fifth case the patient became blind was a strong point in favor of cerebrospinal meningitis.

Dr. H. A. HARE spoke with reference to the use of salicylic acid and quinine in cerebro-spinal meningitis. He had always understood from his reading and teaching and had found it practically correct, that these drugs are contraindicated in inflammation of the meninges of the brain and of the brain itself on the ground that they produced congestion of these parts. Studies have shown that in animals killed with quinine intense inflammation of the meninges of the brain was the typical post-mortem lesion.

Dr. WILLIAM W. WELCH had been much pleased in listening to what had been said. It was, he said, shortly after the war that he first saw in this city cases of cerebrospinal meningitis, and they answered to the description given that evening. Since then he had only occasionally met with a case. He was inclined to agree with Dr. Osler that not unfrequently a mistake in diagnosis was made in regard to this disease. He had seen more than one case of smallpox mistaken for cerebro-spinal meningitis. About a year before, a young Swedish girl, suffering from this disease, had been brought to the Municipal Hospital from a ship arriving at this port. Of course he did not see her early in the disease nor was he able to obtain any history, but when admitted there was cephalalgia, hyperæsthesia, and marked stiffness of the neck and along the spinal column. She was totally deaf and in the course of the disease a purulent discharge came from the ears. For a time she seemed to improve, but finally lapsed into coma, and finally died after being in the hospital thirty-six days. No post-mortem was made. There had been under treatment in the hospital two cases which came from the locality mentioned by Drs. Mills and Cahall. These cases were not sent to the hospital until they had been sick about two weeks. He learned, however, that in both cases the sickness came on suddenly with a chill followed by fever, nausea, headache, intolerance of light, delirium, and stiffness amounting to rigidity of the cervical muscles. On admission most of these symptoms continued. It was about seven weeks since the attack occurred; in one case there was apparent improvement, but the condition of the other was still critical. The convalescence from this disease is always protracted. Indeed, he had sometimes questioned whether in severe cases perfect recovery ever takes place.

Stated Meeting, March 26, 1888.

The President, S. WEIR MITCHELL, M. D. in the chair.

In opening the discussion upon the subject of the

FORCIBLE FEEDING OF THE INSANE,

introduced by the translation of Dr. Rader's paper, and by the paper of Dr John B. Chapin.

DR. S. PRESTON JONES, of the Stockton Sanitarium, Merchantville, N. J., said he had never seen any serious results from forcible feeding. He found it necessary in about one out of every one or two hundred cases. Some patients refused food because there was actually no appetite and a loathing of food. These patients were in bad health, and in such cases forcible feeding he thought did harm and the patients would mostly die at any rate. If the patient was in good general health and refused food, he did so because he thought it was poisoned. It could be forcibly administered, and digestion as a rule was good. He had seen such patients steadily improve under such a course and sometimes get well. Patients sometimes had queer reasons for refusing to take food. One man under his care had told his wife as she was about leaving him, that he would never eat a mouthful in the hospital. We let him go for a week or ten days and then began to use the stomach-pump. He soon began to improve and would have gladly taken food had it not been for the fact that he had made a vow not to eat voluntarily. At the end of three months he left the institution restored to health. Another of our old patients was very fastidious about his food and unless he got just what he wanted he would not eat. This became troublesome and on one occasion we used the pump, not in the gentlest manner. And there was no further trouble.

Formerly the stomach-pump and tube were used, but he now employs the nasal-tube. This was easily done, did not injure the œsophagus or stomach, and the food passed into the stomach much more slowly than with the pump. He thought that possibly some damage might be done by pumping a large quantity of fluid rapidly into the stomach. The use of the stomach-tube was sometimes done in a rough manner, causing much discomfort. The mouth had to be forcibly opened, and sometimes this was a serious matter.

Dr. Jones had never before heard of a patient being strangled under the operation. He thought that formerly patients were often forced to take food too soon. At one time it was taught that the patient should not be permitted to go more than one or two days without taking food. He now had a patient who was rapidly recovering, who had been fed twice a day for six months He had not the slightest doubt but that he would have died if he had been left alone.

Dr. E. N. Brush, first assistant physician in the Male Department of the Pennsylvania Hospital for the Insane, said that it had been his habit for the past ten years in the two hospitals with which he had been connected to use forcible feeding. He had employed the nasal-tube, the stomach-tube and injections. He had never regretted feeding a patient, but had sometimes regretted that he had not done it. He thought that sometimes patients were not fed soon enough, and not often enough when we do feed them. In some hospitals the routine custom was to feed two or three times a day. He thought that in some cases it would be better to give smaller quantities six or eight times a day. It was an easy thing to use the nasal-tube, or if there was some deformity of the nose, or other reason contra-indicating its use, the mouth could be readily opened if we went about it in the right way. It had been said that if the patient was a lady, the best plan was to get her to talk. In other cases, the index finger could be passed between the cheek and the teeth and inserted behind the last molar, and the jaws could then be separated.

The importance of this matter should be impressed on the general practitioner. We frequently see cases reported in newspapers where death has resulted because artificial feeding had not been employed. Physicians had a fear of this simple and ordinary operation. He frequently used the tube as a siphon, the tube being provided with a bulb by which the flow might be started. A similar arrangement might be used for washing out the stomach in case of poisoning. The bulb was without valves, these being extemporized by the operator's fingers. In a certain proportion of cases washing out of the stomach as part of the feeding operation—the washing being done some time before the introduction of food—resulted in an improved condition of that organ and a voluntary resumption of eating.

Almost all of the ordinary articles of food might be given through the tube. Mashed potatoes could be given if mixed with a little milk and some preparation of malt. The same might be said of the farinaceous foods. Powdered beef and other preparations of meat are easily administered.

Dr. Brush on more than one occasion fed patients by the rectum. When the stomach rejects food, or when the injection of food caused pain, this method deserved a trial. He had employed various articles by this method. Some years ago he tried defibrinated blood. It acted satisfactorily, but gave rise to such an offensive òdor that it was discontinued.

Various methods have been suggested for the feeding of patients. One of the most striking was that proposed by an Italian physician. He suggested that the food be prepared in the form of a bolus, which was placed in the back of the pharynx, and then an electric current was passed through the neck, causing the mass to be swallowed. He claimed to have accomplished this.

The length of time that a patient can be kept in good condition by forcible feeding probably depended upon the other conditions present. He saw a patient of Dr. Yellowlees, in Scotland, who had been fed daily for six years, and was still in good condition. He had fed a patient for eighteen months. The patient was then transferred to another hospital, where at last accounts she was still being fed. Dr.

Westphal preferred the use of a funnel with a stomach-tube. So do some of the other German authorities. Some of the English alienists still use the stomach-pump. Dr. Yellowlees used a bottle with the tube attached to its side, at the bottom.

In the matter of tubes, Dr. Brushe's preference was for the soft rubber ones. For nasal feeding he used a soft rubber catheter, with the opening in the end. He had various sizes of stomach-tubes of the same material.

Dr. J. C. Hall, Physician-in-Chief of the Frankford Insane Asylum, Philadelphia, said the ground seemed to be pretty well covered by those who had taken part in the discussion. He must say that he did not agree with Dr. Rader in regard to the advisability of not feeding. He had never seen any bad results from the practice, and he thought that a mistake was often made in waiting too long before beginning forcible feeding. He should not like one of his patients to go more than twenty-four hours without taking food if he thought his condition required it.

At the present time, they had an epidemic of not eating in the institution with which he was connected. About ten per cent. of the cases refused to take food. He found that one with a good deal of strategy will influence others to follow his example. Some he thought had taken up the matter by imitation. He employed the nasal- tube, although he preferred the stomach-tube where it could be used without too much annoyance, on account of its greater rapidity. He used with this a funnel. The only objection that he had met with was that the patient would occasionally regurgitate the food. He thought that the main thing to be taken into consideration was not to allow the patient to go too long without food. He recalled one case of melancholia in which food was refused under the delusion that it was poisoned. This patient was fed three or four times a day for eight or ten months, and finally recovered, left the hospital and went into business. He has seen many other cases in which the advantages of forcible feeding were clearly illustrated. Dr. Chapin had well covered the ground, and he agreed with him on most of the points presented.

Dr. J. Willoughby Phillips, of Burn Brae, Clifton Heights, Pennsylvania, said that during the past ten years he had had about fifteen cases in which forcible feeding was called for. He had never seen any accident; the only untoward occurrence that he had known of was a convulsion during the passage of the stomach-tube. He had used both the stomach and nasal-tube, and either can be employed with ease if properly managed. He used the tubes simply with a funnel. He had then in charge a lady who had not taken food voluntarily since a year ago last December. The nasal-tube was used twice daily during the entire period. She had not gained in weight, neither had she lost. During the operation the patients should be so thoroughly under control that there can be no possible chance of their injuring themselves or interfering with the operation. This can be accomplished by having plenty of assistance.

Of the foods used in such cases, milk and eggs head the list; with these may be combined beef tea, mutton broth, and strong comsommé, vegetables in liquid form, and occasionally extracts of malt and spirits, according to the requirements of the case. When patients are debilitated and run down, prompt and liberal feeding is clearly indicated. His practice was to administer nourishment twice daily, the amount being at least a pint and a half at each meal.

Dr. William Osler said that in general practice he often had occasion to feed patients with the tube. In the course of some years' observation in the post-mortem room, he had seen three or four instances of deglutition pneumonia, which had been referred to in the paper. He saw such a case not long ago. A girl was admitted to the hospital in a comatose condition, and it was necessary to feed her with the nasal-tube. At the autopsy a double deglutition pneumonia was found. It was extremely important that the operation should be properly performed. The tube should be entirely emptied before it is withdrawn, and tsken out carefully. In the insane, accident is not so likely to result, for the patient generally coughs the foreign matter from the larynx, but the comatose patient dose not recognize it, and the fluid passes into the bronchial tubes.

Dr. E. N. Brush said that the danger to which Dr. Osler referred should always be borne in mind. His invariable custom was to pinch the tube if it was soft, or if it was stiff, to place his finger over the opening while removing it. Dr. Hall had referred to the fact that he had had an epidemic of refusal of food. It was found that if other patients knew that there was a patient being fed with a tube there would soon be other cases, especially among those of a hysterical tendency. A curious fact may be mentioned that many of the case which refused to eat, would eat if they had a chance to steal sufficient to live upon; and acting upon that, he had often avoided the necessity of feeding, by directing the nurse to leave food where these patients could surreptitiously gain access to it.

Dr. Charles K. Mills said that he regarded the subject of the forcible feeding of the insane as one of great practical importance to general practitioners of medicine, as well as to those who had charge of the insane institutions. When we read in the *Medical and Surgical Reporter* the favorable editorial comments on Dr. Rader's paper, advocating non-interference when insane patients refused food, he felt that the subject would be an excellent one to bring before an association like the Philadelphia Neurological Society, which counts among its members neurologists, alienists and general physicians. He did not, however, feel that he could add much to the discussion; but he would like to emphasize the importance of forcibly feeding the insane who are treated at their homes, or not in institutions especially intended for such patients. He saw many cases of insanity in consultation, and was frequently called upon to treat such patients at their homes, either alone or in connection with other physicians. He could recall a number of cases of acute mania, melancholia, delusional monomania, and stuporous dementia, in which he was confident that fatal results, or absolute failure to succeed in treatment at home, were due to carelessness or tardiness or indifference as regards forcible feeding. Occasionally cases of hysterical insanity will either intentionally, or in spite of themselves because of their morbid impulses, carry their refusal of food so far that their stomachs will not respond properly to the stimulus of food when given, and serious results will then ensue. He had had under his charge for several years an intelligent young man, but the unfortunate victim of a form of paranoia, chiefly exhibiting itself in abulia, inchoate delusions, and imperative conceptions, nearly all circling about a fundamental delusive idea with reference to the sinfulness of having blood entering in any way into his food. This patient was fed 400 to 500 times forcibly with the œsophageal tube in the course of about two years. Dr. Mills had but little doubt that his life was saved by the procedure; and not only so, but, as the patient himself had more than once declared, the forcible feeding had probably prevented him from passing into a state of acute mania, great excitement having frequently resulted from the terrible conflict precipitated by the struggle between the desire to take food owing to pressing physical necessity, and the resistance to the inclination by which he was delusively dominated.

As to the methods of feeding by force, his experience was in favor of the nasal-tube. As this discussion was intended in publication to cover the subject of forcible feeding, he would close his remarks by quoting from his little book on the "Nursing and Care of the Nervous and the Insane," a few remarks on this subject of nasal feeding: "The number of patients who cannot be fed by the nose is very small; occasionally, however, a patient is found whom it seems impossible to feed in this way, owing to the choking and strangling produced. This may be because of some peculiar anatomical conformation, or some special idiosyncrasy on the part of the patient. Such a patient will choke or strangle with nasal feeding when he will not when the stomach-tube is resorted to. If, when the attempt is made to pass the well-oiled tube through the nostril, resistance is encountered, and if, after a few trials, the tube cannot be made to pass, great force should not be employed by the operator, but the tube should be at once withdrawn and the effort should be made to pass it through the other nostril. In nearly all cases where special resistances is offered on one side, the tube will pass with ease upon the other, and this, in most instances, is because, if hypertrophies or projections exist upon one side, there will be upon the other corresponding or compensating depressions and enlargements. Sometimes, but rarely, the mucous membrane is exceedingly irritable. After the nasal-tube has passed through the nostrils, it seems to have a peculiar tendency in some cases to drop into the glottis, the patient struggling and attempting to scream meanwhile. Some patients will spit or force the tube out into the mouth ; and attendants can sometimes through the mouth, keep the tube, which has been passed through the nose, in position. Occasionally the nose is made sore by the use of the tube, but this is not likely to occur if the tube is always perfectly cleaned and well oiled. If it is of the proper kind ; that is, a soft tube, there will be no danger of injuring the parts by breaking or perforating the mucous membrane. In using the nasal-tube, great care should be always exercised to see that at least fifteen to sixteen inches of the tube has been passed before beginning the feeding. This will make it certain that the entrance to the windpipe has been passed. Of course care should be taken to observe that the tube has not doubled itself." He would add one remark, namely: Great care should be taken not to administer the food too hot. He knew of one accident occurring in this way.

DR. S. S. SHULTZ, Physician-in-Chief of the State Hospital for the Insane at Danville, Pennsylvania, sent the following letter to Dr. Mills as his contribution to the discussion :

DANVILLE, PA., March 23, 1888.

My dear Doctor:—I give you herewith, as requested by you in your favor of the 17th inst., briefly my views in regard to the forcible feeding of the insane.

1st. Is it ever absolutely necessary to administer food against their will to any class of the insane ? Is life prolonged or restoration to reason promoted by such a course of treatment ? It must be admitted that this question does not allow a mathematical demonstration either way. It is easy to claim when bodily health is restored or the mind improved under compulsory feeding that this would have happened without such treatment, or when death occurs, or insanity becomes chronic, under the expectant plan that such results were inevitable. In medicine, few problems could be solved by such a method of reasoning. The majority of patients who come into hospitals, from country districts at least, suffer from impaired nutrition. Until there is improvement in the pasty tongue, the want of appetite, anæmia and emaciation, it is in vain to look for improvement in the symptoms of insanity. Impoverishment of the blood seems to be the condition which gives many of the so-called causes of insanity their importance. These may be incurable, as, for instance, the remains of injuries to the skull, or disease of the heart, and yet if the nutrition can be improved and the blood enriched, the mental disorder often for a time disappears. When insanity is the result simply of defective nutrition, progress towards permanent restoration keeps pace with the improvement of the blood resulting from better nutrition. If this torpid condition of the nutritive functions is permitted to remain a long time, the irregular mental habits become chronic, and the risk of incurability rapidly increases.

This much to show my deep convictions that poor blood plays an important part in the causation of many cases of insanity, and that the prompt correction of this will give the best chances of recovery. A german writer defending the expectant plan, sees no danger in fasting when it is not prolonged over fourteen days without taking water, not over fifty days when water is taken, nor so long as 60 per cent. of the body weight remains. The practice of such a rule or anything approaching its extremes, it seems to me, must lead to the sacrifice, not only of the chances of recovery, but of life itself. It can certainly not be the part of wisdom to allow the boat with its living human freight to drift to the very brink of the cataract, without attempting to arrest it at the beginning of the rapids, where it can be done with so little risk.

Insane patients having organic disease of the digestive apparatus, as inflammation of the pharynx or cancer of the stomach, are likely to refuse food earlier and more persistently than the same in similar conditions, and the measures suitable for those whose fasting is the result of delusion need modification for these. The melancholic who fast from religious or suicidal motives, or the delusion that there is no room for food, or that the passages are closed, most often carry their purpose to a dangerous extent, and thwart persuasion, reasoning, coaxing, no matter how skilfully or persistently plied. No rule based on the element of time of fasting is applicable; but as there has been usually for weeks an insufficient amount of food taken, it is safe to begin the feeding as soon as the purpose of abstinence has shown itself to be settled, and refusal to yield to other resources, and both bodily and mental symptoms become worse. The more the character of the patient while in health was marked by resolute purpose and stubbornness of will, the less likely is delay to be of any use.

Of course food introduced into the stomach in this mechanical and compulsory manner is of less value than when taken at the promptings of natural hunger; but one must choose the lesser of two evils.

Nutritive enemata may answer for a time when fasting instead of being the result of a fixed purpose has its origin in the loss of the feeling of hunger.

Patients suffering from melancholy no doubt must often require artificial feeding, but other forms of insanity may demand the same treatment. When no physical condition can be detected that would justify abstinence, the forcible administration of food should not be delayed to the point of starvation in any form of disease. When emaciation has surely set in, the breath has become characteristically heavy and foul, and the strength is diminishing, active measures should be no longer postponed, when the will of the patient cannot be persuaded.

With reference to the manner of carrying out the indication, little need be said, as the nasal tube is now universally preferred to that by the œsophagus. It has the advantage of making resistance less possible; and injury to the teeth and soft parts cannot occur.

It is possible that the tube may enter the larynx through an awkward position or movement of the patient. If the tube is pervious and haste is avoided, such a misadventure will be defeated through the restlessness of the patient and the escape of the air through the outer end of the tube.

Very truly yours,

S. S. SCHULTZ.

NEW YORK NEUROLOGICAL SOCIETY.

Meeting held Tuesday Evening, April 3d, 1888.

THE PRESIDENT, C. L. DANA, M.D., IN THE CHAIR.

DR. A. ROCKWELL presented two cases of

BASEDOW'S DISEASE,

illustrating results which could not infrequently be obtained by treatment. The first case was that of a young lady who for a year and a half past had presented the three symptoms—protrusion of the eyes, swelling of the neck, and increased frequency of the pulse. The neck had measured fourteen inches, and the pulse had varied between 140 and 160, or above. Respiration had been 35, as a rule. After four months of treatment, consisting of dietetic regulation, internal medication, and electricity to the neck, the size of the goitre had been reduced one inch, the exophthalmus had diminished, and the pulse had diminished 60 beats. The patient's whole physical condition also had been changed, so that she could engage in the occupations and the enjoyments of life. At the end of treatment the pulse was 75 or 80. It had intermitted from the first, and it still retained this characteristic. Five years had elapsed since the discontinuance of treatment, and there had been no relapse.

The second case was that of a gentleman still under treatment. Out of thirty cases in the speaker's experience this was but the second in which he had encountered the disease in the male. The case was of an incomplete type. Great improvement had followed treatment.

Dr. W. M. LESZYNSKY presented a case of

ACUTE IDIOPATHIC NEURITIS OF THE BRACHIAL PLEXUS,

and read a paper embracing the history of the case, with carefully recorded electrical tests.

The patient was a man who had had an attack several years before, causing a painful and wasting paralysis of the right shoulder. This was followed recently by a second attack, involving the left shoulder. The areas of anæsthesia, local tenderness, and electrical reactions showed that nearly all the branches of the brachial plexus were involved. Syphilis, lead, and trauma were excluded.

DR. BIRDSALL, while acknowledging the difficulty of following the details of a written history, had the impression that the case presented was one of periarthritis, with neural involvement starting from the joint disease. Dr. Jacoby had reported a number of similar cases.

DR. JACOBY was inclined to the same view; yet he had understood that there had been no antecedent joint trouble either in the shoulder or the wrist.

DR. BIRDSALL added that in some cases the pain, the joint involvement, and the paralysis appeared together. Especially in the case of the shoulder there was often no antecedent history of joint involvement.

DR. DANA referred to Dr. Jacoby's article and asked whether the degeneration reaction had not been absent in his cases of arthritic paralysis.

DR. JACOBY replied that it had.

DR. BIRDSALL added that, while this had also been the experience of Erb, in some of his own cases the degeneration reaction had been obtained.

Dr. PUTNAM JACOBI asked how the electric resistance had been measured.

DR. LESZYNSKY replied that one electrode had been placed upon the hand, and the other upon the back of the neck. In reply to the objections, he stated that there had been no antecedent history of joint trouble. At the time of the paralysis of the deltoid the patient could move the joint without pain. The suffering occurred in paroxysms, and with each paroxysm fresh paralyses developed. The circumflex was first affected, the paralysis of the deltoid occurring on the twelfth day, the supra-spinatus and infra-spinatus on the twenty-fourth day, and the triceps on the twenty-sixth day, after a very severe attack of pain which required a large dose of morphia and chloral for its relief. Later, the subscapularis and teres major had been affected, and last the biceps, upon the forty-fifth day of the disease. Repeated examinations of the condition of the joints had been made, and the possibility of joint-affection had been excluded.

Dr. Gibney had had the case under observation, and had been unable to arrive at any decision in regard to it.

Dr. M. Allen Starr presented a paper upon.

SYRINGOMYELIA, ITS PATHOLOGY AND CLINICAL FEATURES, WITH EXHIBITION OF SPECIMENS, A STUDY OF A CASE, AND REMARKS UPON ITS DIAGNOSIS.*

Syringomyelia is a condition of the spinal cord in which abnormal cavities are present within the organ. The cavities are of two kinds: First, a dilatation of the central canal—hydromyelia. Of this two specimens were shown, in each of which the cavity was easily seen by the naked eye and the complete epithelial lining was visible under the microscope. The tissue around the dilated canal was normal. Secondly, a destruction of elements of the spinal cord—syringomyelia. The first stage of its pathology is the infiltration of the spinal cord near the central canal with round cells, connective-tissue nuclei, or gliomatous cells. A specimen showing this condition was exhibited. The next stage is the production of a felt-like connective tissue with numerous nuclei through the spinal cord, but especially near the central gray substance, pushing aside or destroying the spinal elements. This gliomatous mass has a tend-

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*Published in full in the *Amer. Jour. Med. Science*, May, 1888.

ency to break down in its centre, leaving a fissure or cavity whose walls are formed by the felt-like connective-tissue mass. The formation of such a cavity is the third stage of the pathological process. Specimens were shown illustrating these three stages, the specimens being from Schultze's cases and obtained partly from Schultze and partly from his pupil, Dr. Van Giesen. In some specimens the cavity was wholly independent of the central canal, which was pushed forward. In another the new cavity opened into the central canal, which appeared to have been dilated. In still others, no trace of the central canal was to be seen. The gliomatous infiltration about the cavity was evident in all the specimens. The size of the cavity varied greatly, from a small fissure to a large cavity around which only a thin rim of spinal tissue remained. The cavity in some cases extended only through a few segments of the cord. In other cases it reached through its entire length. The above diagram was shown representing its extent

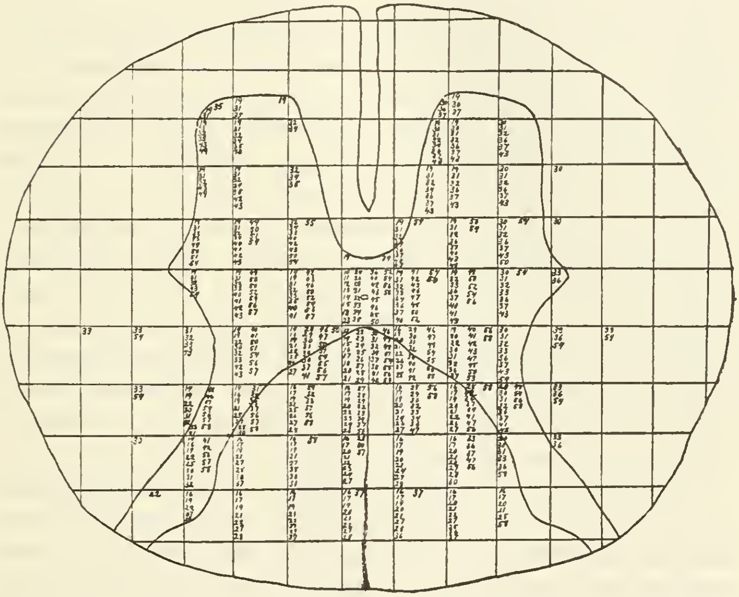

Diagram to show the extent of the cavity in the spinal cord in forty-eight cases. When the area represented by a square was invaded by disease, the number of the case is recorded in that square.

in a horizontal section of the spinal cord, the cord being divided into squares, and forty-eight cases being tabulated. The number of each case being placed in every square affected in the case, the greater number of squares numbered about the central area of the cord indicated the frequency with which this area was involved. The lower cervical and upper dorsal regions of the cord were the parts most frequently affected. The contents of the cavity is usually serous fluid, but may be bloody or thick and hyaline.

The clinical features of the disease were presented by means of a collection of typical cases accompanied by autopsies. They were found to be, first, progressive muscular atrophy with paralysis affecting some or all of the muscles of one limb, and usually extending to the opposite limb and to the trunk, sometimes attended by reaction of degeneration in the paretic muscles; secondly, vaso-motor and trophic disturbances in the affected limb, consisting of cyanosis, coldness, bullous eruptions, ulceration, and abscess, and even atrophy and fragility of the bones and a diminution in the excretion of sweat; thirdly, peculiar sensory disturbances, consisting of a loss of the sensations of pain and temperature in the atrophied part, while the senses of touch, pressure and location were preserved.

The distribution of these symptoms in accordance with the situation of the lesion in the cord was then fully discussed. In half the cases cited the anterior horns were involved and paralysis with atrophy was present. In two-thirds of the cases the posterior horns were involved, and the peculiar sensory disturbances were observed.

A case was then related in which the characteristic symptoms were present and in which the diagnosis of syringomyelia had been reached by exclusion, all other forms of spinal disease having been shown to be impossible. The differential diagnosis from amyotrophic lateral sclerosis, tabes, anterior polio-myelitis, meningitis, intraspinal tumor, and neuritis was considered in connection with this case. The paper closed with a reference to the etiology and treatment of the affection, regarding which little seems to be known.

DR. SHAW said that about five weeks previously he had been consulted by a man, a German stevedore, who by his occupation was much exposed to the cold. The patient's complaint had been that he was unable to feel and protect himself from anything hot. The anæsthesia was especially marked in the right hand, but was present to some extent in the other also. As in Dr. Starr's case, there had been a burn on the right arm near the wrist, for which the patient could not account. There was no ataxia, no atrophy. There were no pupillary symptoms, and the optic nerve was normal. It seemed to him that this might have been a case of cavity of the cord similar to those of the cases described. He inquired, however, whether the origin of the condition was invariable. Might we not, for instance, have a cavity in the cord from malacia secondary to caries of the spine.

DR. SACHS had been interested in the subject for several years. He had looked up the literature in connection with that of tumors of the cord, and he had also seen a number of living cases diagnosed by Schultze in Heidelberg. The clinical phenomena in Dr. Starr's case resembled those in Schultze's cases without doubt. He inquired, however, whether diagnosis between syringomyelia and intramedullary tumor was not difficult, especially at an early date. The duration of the condition would give a decisive indication at a later stage, as intramedullary tumors seldom lasted as long as two or three years. The difficulty of diagnosis was further increased by the fact that the symptoms so much resembled those of progressive muscular atrophy that the sensory manifestations might easily be neglected in favor of this diagnosis.

DR. BIRDSALL recalled from his own experience, three cases presenting motor symptoms of the atrophic type, vaso-motor and some sensory symptoms, which he thought might be due to cavity in the cervical portion of the cord; he had not yet arrived at an exact conclusion in the matter. There had been in these cases, too, cessations in the advancement of the disease. More careful observations would, he thought, lead to less frequent diagnoses of the typical or system diseases. There would be found many combinations of symptoms which did not correspond to these types. The system diseases, too, probably required to be divided up.

DR. BOOTH recalled a case of marked atrophy of the arm and hand with anæsthesis, so that burns were not felt.

DR. PUTNAM-JACOBI referred to the fact that in the cases cited a gliomatous tumor had served for the inception of the condition. She asked whether all cases of syringomyelia were referred to this origin. She also referred to the existence of vacuoles in the cord in pseudo-hypertrophic paralysis, in the medulla in tetanus, and in the condition of porencephalia in the brain. In five cases of pseudomuscular hypertrophy there had been found dilatation of the central cavity or cavities in the posterior horns, although the correlation of the lesion with the paralysis had not been established.

DR. PETERSON referred to a case at the Manhattan Hospital where, in 1878, Dr. Seguin had diagnosed cavity of the cervical enlargement of the cord. The patient had presented atrophy of the left hand and arm with anæsthesia of the left arm, thorax, and leg.

DR. DANA suggested that many cases diagnosed cervical pachymeningitis should probaly have been classed as cases of this disease, as in Wichman's case. Dr. Starr had assumed that all cases of syringomyelia were gliomatous in origin. Might we have cavity-building from some other cause : He showed a specimen in which similar cavities had resulted from a central myelitis. There had been two cavities in this case, one one and a half inch long and the other not so large.

DR. STARR closed the discussion. The stationary condition in his case could be explained by the fact of the glioma having broken down and advancement having ceased. The process had been arrested. It was not possible to diagnose between intramedullary tumors and syringomyelia except as had been suggested by the duration of the case. The lesion had been discovered accidentally in some of the cases cited. The same had been true of multiple sclerosis in its first recognition. Then it had been diagnosed by exclusion, all cases not presenting typical symptoms being referred to it. Now syringomyelia received consideration also in the diagnosis of the anomalous cases. It might be of assistance to bear in mind that 24 out of the 56 cases had presented cavity in the anterior, and 32 out of 56 cavity in the posterior part of the cord. The chances were therefore in favor of the symptoms of progressive muscular atrophy or analgesic areas being present. In the examination it was necessary to distinguish between analgesia with loss of the temperature-sense and general tactile anæsthesia. Intermissions were not uncommon in other conditions. He had seen ataxia disappear and return after several months. The reaction of degeneration did not appear to be an important diagnostic point. The majority of the cases had been diagnosed as progressive muscular atrophy, a condition in which this reaction was not found. The condition of porencephalia referred to was a condition of childhood and fœtal life ; he did not think that it was ever found developing in the adult. The condition found in pseudo-hypertrophic paralysis has been a congenital deformity of the cord with abnormal fissures, but without gliomatous infiltration, and hence was entirely different from that here described. The age of the patients with syringomyelia had been twenty-five to forty years. In all of the cases observed the previous state had been that of infiltration with gliomatous cells. Destruction did not occur during this stage ; the elements of the cord were simply displaced.

ELECTRICITY AND VITAL ENERGY.

Dr. T. W. Poole, in the *Medical Register*, Dec. 3d, 1887, makes comments on a paper by Dr. F. E. Stewart, who claims that heat and electricity are directly transmutable into vital energy, in accordance with the law of conservation of force; and also "that it is possible to change electricity into vital energy by passing the electric current from a battery through the living bodies of both animals and vegetables." A reason assigned in support of this conclusion is that plants grow under the influence of electric light. Electric *light* is not electricity. An established fact in relation to the conservation of energy is that a physical force *ceases to exist* when transmuted into another correlated force. Whenever work is done by heat, heat disappears (Tyndall). In accordance with the principle of conservation of force, light runs into heat, heat into electricity, electricity into magnetism, magnetism into mechanical force, mechanical force again into heat and light (Tyndall), or into chemical affinity, it is equally true that not one of these can possibly be converted into vital energy unless it be then and there present, as embodied in living protoplasm, "under the guidance of which the transformation of matter takes place" (Balfour). Vital force or energy can only be increased by appropriate food and nutritive processes. Electricity itself cannot be utilized as food even by plants, and when converted into chemical energy it becomes lost as electricity, just as it does when converted into light. Electricity may be made to promote growth and nutrition of the animal body or portions of it, not by any vitalizing quality but as is explained in "Medical and Surgical Electricity," p. 205, 300—"The muscular contractions that are produced by the current in its passage through the limbs or body cause increase of local processes of waste and repair, and accordingly the muscles increase in size, just as they naturally do under the influence of any other form of active or passive exercise." L. F. B.

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Original Articles.

ON SOME RESULTS OBTAINED BY THE ATROPHY METHOD.

BY E. C. SPITZKA AND R. MOLLENHAUER.

IN the sequel we shall have occasion to relate observations made by various methods of research employed on the brain axis of man and lower animals. But as the more novel and striking of these were derived from a case of artificially induced brain atrophy, and the latter constitutes as it were the text of this article, of which the other results collaterally obtained may be regarded as commentaries, we shall proceed to describe the former in its general features, before discussing special problems in cerebral anatomy.

The animal selected for operation was a kitten, one of a litter of six, and if anything, the largest and healthiest of these. A cataract needle was rapidly passed through the middle altitude of the lateral aspect of the skull, behind the left mastoid crest, obliquely inwards and downwards, to the floor of the skull, and then after a quick lateral motion, withdrawn. The time occupied by the operation, was at most three seconds, and but a drop of blood was lost. At the time of the operation the animal was two days old and the eyes were closed. It immediately showed *manége* movements, which were towards the right side ; it walked round and round in a circle with its head and tail appoaching each other on that side. This circle did not exceed the length of its body, and consequently, though continually moving—as it did at all times it was observed—it did not get further than a few inches from any given spot in which it might be placed. It being found that this imperative motion invariably defeated all attempts to obtain nourishment—for it circled past the mother's tit,—it was fed by hand, a task requiring considerable patience, and which was accomplished by one of the female members of the household. The date of the operation was June 22d, 1884. Four days later the eyes opened, that is a day later than those of the other kittens of the same litter. It was already then evident that it was not developing as well. The manège movements continued in undimenished intensity for two weeks ; then it was noted, that it occasionally made attempts to walk straight and would even in attempting to overcome the *manège* to the right, show a brief deviation to the left. At the age of one month it walked straight, as a rule ; the occasional deviations noted were to the left, it often tumbled over on this side. In judging this phenomenon, the defect of vision should be borne in mind. It at this age began to play with the other kittens, but did not possess one-third their range of motion, nor anything like the same degree of skill. It still had to be fed, the food being pushed into its mouth. The animal often assisted this procedure by pushing its mouth against the feeding receptacle so as to retain its hold on it.

When the eyes opened, they both were noticed to be abducted ; two weeks later the right appeared normal in direction, also showed normal pupillary reactions. To the end, the left eye remained strongly abducted and there was complete left iridoplegia and mydriasis.

Its gait was particularly deliberate and slow, as far as both hind extremities were concerned, and it wabbled from side to side as if paraparetic. In stepping out, it stretched its hind-legs overmuch. It is able to rest on its left fore-paw, raising the right in play, but holds its head in a peculiar position, as if to look with the left (abducted) eye on the floor. It evidently has the use of this eye, at least with a part of its visual field.

Cutaneous impressions appeared to be correctly localized, as far as reflex acts enabled us to determine. Except in so far as it was invalidated by defective nutrition, its muscular clumsiness and defective fields of vision, it appeared to have no defect of intelligence. It was exceedingly good-natured and far from being lethargic at this time. Some of the incidents noted in its subsequent career, are herewith detailed.

August 29th. Tried to get on the cross-piece of a chair in vain for over a quarter of an hour. The mother then called her kittens round her on a sofa. The others jumped up immediately—but the operated one after repeated failures was assisted by the little boy who had it in charge. When there it played with its mother. The other kittens evidently recognized its helplessness, and refrained from the rougher play which they indulged in with each other. In its manœuvres the defective one was often non-plussed, particularly in jumping at its antagonist, when, it either fell short or struck to one side, but it repeated the attempt again and again, evidently enjoying the sport as much as the uninjured ones.

September 5th. The animal has become more dull. It has retained the hair it was born with, not changing the fur as the healthy ones have done. It measures not fully two-thirds their average length, and does not equal one third (!) their average weight. Its head appears of the normal size, a slight narrowing at the temporal region is noted. The boy who had it in charge alone was capable of provoking it to action, when I exhibited it to my class. This he accomplished by imitating a fighting cat, it then ran towards him, bounding along in a line to the left, and "over-reaching" in raising the hind-quarters; being further provoked by the boy's imitation of a mewing and spitting cat, it mewed and spit in return.

This kitten was killed exactly ninety days after the operation, having become gradually more and more indifferent and passive. On examining the calvarium, it was seen that the sagittal suture, corresponded to the sulcus, separating the ectal gyrus from its fellow on the right cerebral hemisphere. In other words the right cerebral hemisphere protruded across the median line with a considerable portion of its mass. * On opening the skull cavity, it was found, that the right cerebral hemisphere occupied nearly the whole of it, there being but a shrunken membraneous sac to represent its fellow, only the olfactory lobe as well as the adjoining region, in the anterior perforated space, exhibited something by which the normal elements could be identified.

There was no trace of any active morbid process anywhere. There was a glairy rust-colored material in the antero-inferior part of the cranial cavity, it was connected with a trabeculated membrane, which evidently represented the residue of atrophied cortex fused with the leptomeninges. Possibly there had been a hemorrhage, but no distinct traces of such were found. Behind the larger cyst representing the left cerebral hemisphere, were smaller ones, included in a common investment which at the base of the brain merged with the shrunken crus of that side. These cysts corresponded to the left thalamus, which, as far as its nervous tissue is concerned had been almost entirely eliminated. Nothing beyond a ridge like thickening of the cyst wall at the region of the habena gave the slightest indication of the normal components of the thalamencephalon. The left optic lobe † was shortened in the cephalo-caudal direction to one-half and in the transverse direction to two-thirds of its fellow ; it was very flat, while the right was beautifully prominent, and encroached to the extent of fully one-third of its own diameter, beyond the ideal plane of the axis. The dividing line between the optic lobes corresponded to the mesal margin of the right cerebral hemisphere.

There was no gross asymmetry of the cerebellum. At first, owing the distortion of the cerebral axis, it seemed as if the right lobe were slightly less voluminous than the left. The subsequent study by sections showed that this asym-

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* The various measurements, made and recorded are omitted, except where they are of intrinsic interest.

† Anterior Pair of the Corpora Quadirgemina.

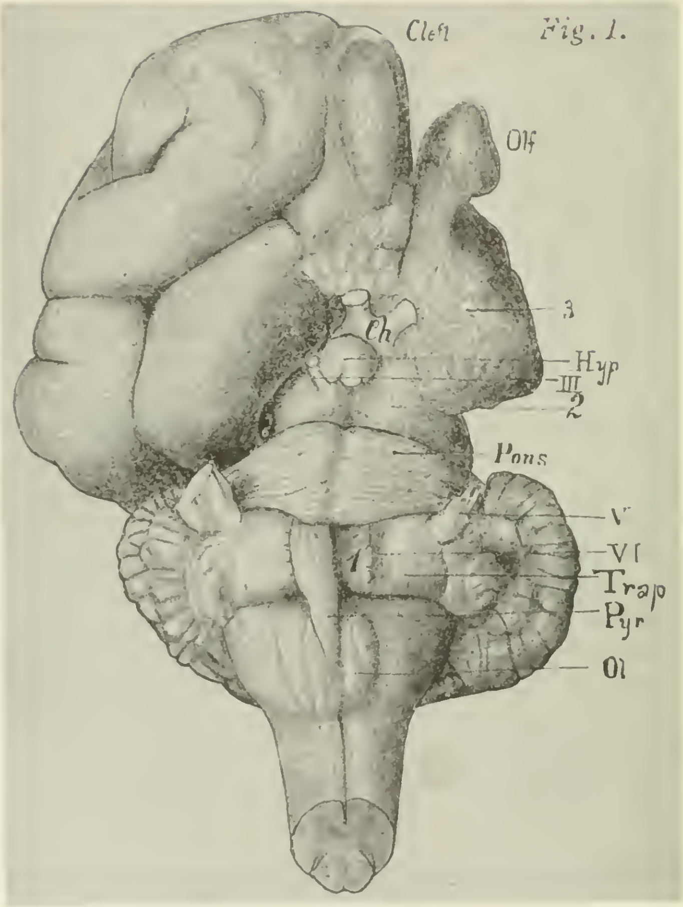

Fig. 1.

Cleft
Olf
3
Ch
Hyp
III
2
Pons
V
VI
Trap
Pyr
Ol
1

metry was apparent and not real in regard to bulk. What the right lobe lacked in width, it preponderated in, in regard to depth and fulness.

The distortion of the brain axis can be best pictured by a glance at the accompanying plate, (Figure 1). Beginning at the ethmoidal crest it ran obliquely caudad and to the right, in a rather direct line to the posterior border of the pons. Here it changed in direction sharply, the ventral furrow of the oblongata coinciding in direction with that of the cord. This asymmetry corresponded with that of the skull, the right half of which in front of the clivus greatly preponderated over the left, which was reduced in the three dimensions.

The optic nerves were both smaller than normal, the left was reduced to two-thirds the section-area of a corresponding healthy kitten, the right to about one-half, the latter was therefore the smaller.* There were no traces of the left optic tract; the right appeared normal. The hypophysis was symmetrical and normally developed in all its parts. The left oculo-motor nerve was entirely absent, the right entirely normal. The pons was as a whole smaller than in kittens of a corresponding age, but as subsequent examination showed equally so in all directions. The bulge in the right side of the basilar furrow was marked, in the left it was barely indicated. The left pyramid was entirely absent, and a depression marked the spot, where it is ordinarily located, the left olivary eminence consequently become entirely exposed, whereas the right was concealed in its cephalomesal part by the developed right pyramid. The distance between the left abducens radicles and the ventral-furrow was much less than that between the right abducens radicles and that furrow. A glance at the two figures (2 and 3) representing trans-sections respectively through the middle of the pons, and the trapezium shows the chief results produced by the elimination of the left thalamus and left cerebral hemisphere. The corresponding pyramid tract is entirely eliminated, not a trace of it can be discovered. The mesal division of the lemniscus (cortex-lemniscus of von Monakew, interolivary layer of Flechsig) is represented by atrophic relics.

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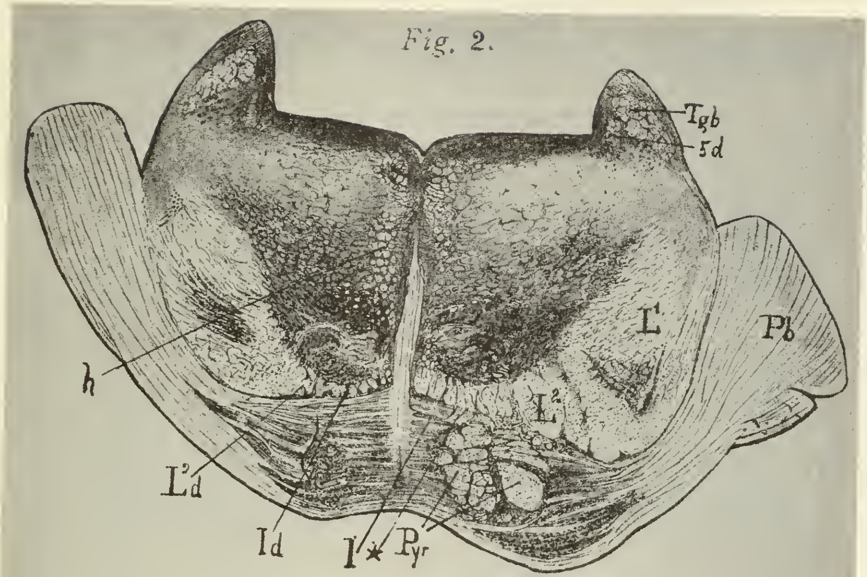
* The Figure does not show this, owing to the different direction of the dividing sections. The measurements were made near the entrance of the nerves in the eye ball.

The same is to be said of that intermediate division which is located between the mesal and lateral divisions : (Figure 2, L^2 on the right side, L^2 on the left side). The lateral division of the lemniscus is unaffected. The brachium conjunctivum (tegmenta-brachium, processus e cerebelli ad cerebrum) as well as the pontis-brachium also appears to be unaffected, or at least symmetrically developed. The posterior longitudinal fasciculus is much reduced on the left, as compared with the right side, while the tegmentum generally appears smaller on the left. A remarkable exception is noted in the field of large fibres situated near the raphe. These show a crossed atroply, and our curiosity is aroused, and leads us to search for a relationship between them and the fibres of the fountain decussation of Meynert, or perhaps of the posterior commissure. The further caudad we proceed, the less markedly do these various asymmetries influence the contour of the trans-sections, and the deeper topography of the brain axis.

To sum up, the experiment had consisted in the nicking of the left lateral aspect of the mesencephalon, dividing the various tracts which run to the higher centres, in a direction ventro-mesad, the left optic tract, and the left oculo-motor nerve. All other cranial nerves, as well as the larger blood-vessels escaped. There then resulted a passive atrophy of all the centres and tracts directly represented in the destroyed area. The nuclei of the left oculo-motor nerve, the left thalamus, the left cerebral hemisphere and all tracts directly depending on the latter two, were eliminated so completely, that not a trace of their nervous elements could be discovered. To derive useful results from the observation of so extensive an atrophy, it will be necessary to take up tract by tract as influenced by the operation, and compare it with its condition in experiments where less severe mutilation had been accomplished. As we shall find, the *plus* of tract atrophy found in our case, as compared with v. Mona-

Fig. 2.


Tgb
5d
L¹
Pb
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h
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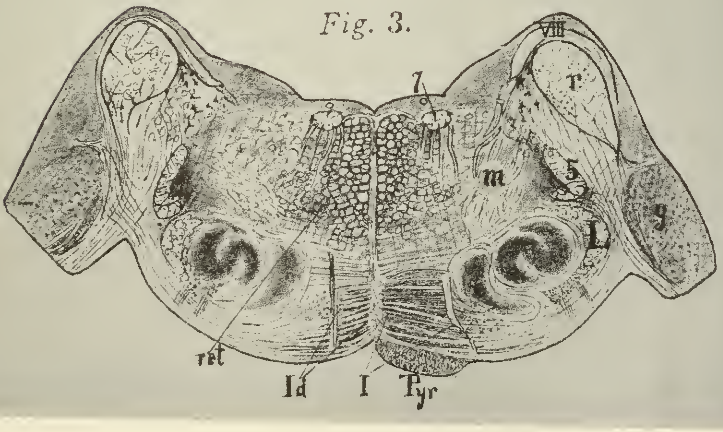
Fig. 3.


VIII
r
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kow and Forel's observations, is attributable to the destruction of the thalamus, and consequently we are enabled to determine the extent and course of certain of the tegmental fibre-systems.

(To be continued.)

FIGURE 1. Ventral view of the entire brain. *Cleft :* Median longitudinal fissure separating the two cerebral hemispheres ; *Olf*, Left Olfactory bult. 3. Left atrophic cerebral hemisphere, the pointer is on one of the obliterated vessels ; *Hyp* Pituitary body ; *Ch* Chiasm III. Right Oculo-motor nerve, the left being absent. 2. Cyst occupying the place of the left Thalamus, the pointer ends at the spot where the cyst terminates and the rudimentary crus begins ; *Pons* Pons Varolii V. Left Trigeminus, as the nerve is frayed out, it appears smaller than its fellow, in reality, they were symmetrically developed. 1. Depression corresponding to the eliminated left Pyramid bordered ectad by the (VI) Abducens roots. *Trap* Trapezium Pyr. Right Pyramid ; Ol Left Olive entirely uncovered.

FIGURE 2. Trans-section of Pons. Pb. Brachium Pontis (medipedunculus) I. Right interolivary layer. Id. Left ditto, atrophied : Pyr. Right Pyramid tract. L^1 L^2 various divisions of Right and normal lemniscus. L^2d. Left atrophied representation of the middle division. Tgb. Tegmentabrachium (Bindearm or prepedunculus); 5d. Descending radicle of Fifth Pair ; h crossed tegmental field.

FIGURE 3. Trans-section of Trapezium VIII. Auditory radicle. L part of Lemniscus, accumulating ectad of trapezial iridus (so-called superior olive) *ret* Reticular Field, *r* Restiform column ; *5* Ascending Radicle of Fifth Pair, *m* fibres. which in more cephalic levels become the lateral division of the Lemniscus, designated L 1 in Figure 2 ; 7 germ facialis ; *Pyr* Pyramid Tract of Right Side ; L. Right interolivary field, extent indicated by pointers *Id* atrophic Left interolivary field.

PARALYSIS AGITANS, AND A CONSIDERATION OF SOME CASES OF THIS DISEASE.

By LEONARD WEBER, M.D.

Read before the New York Neurological Society, May 1st, 1888.

FOR the first regular description of paralysis agitans we are indebted to Parkinson, 1817. Charcot was the first, I believe, to draw the line of distinction between paralysis agitans and disseminated sclerosis, showing that in the former the tremor is present while the muscles are in a state of rest, and that there is no spastic paralysis, although the progressive motor weakness may even amount to paresis; while in the latter there is tremor, only consecutive to motions which the patient wants to execute, and more or less spastic paralysis.

Paralysis agitans is, then, a complexity of symptoms, in which progressive tremor and motor weakness are most prominent. It is a neurosis in the sense that we cannot characterize it by a special lesion of its own in the brain or spine, or both, and it belongs to the second period of life.

General Symptomatology.—The tremor is at first limited to a single limb, gradually spreading to others, and overrunning the entire body, always respecting the head, I believe. Sooner or later we notice an apparent diminution of muscular force, the movements are slow and appear enfeebled, though no actual diminution of force may be demonstrated by the dynamometer. This motor weakness appears to be dependent in part upon a certain rigidity of the muscles. Among the curious symptoms, and sometimes even in the early stages of the disease, there is to be noted a loss of the faculty of maintaining the equilibrium in walking. When the patient starts, he runs; he is propelled forward.

In some cases, by tugging at the patient's clothes on the back, he can be made to run backward with a rapidly propelling motion, the same as he follows on attempting to go forward. This curious way of propelling himself, as if he had to run after his lost centre of gravitation, may be due to certain lesions in the cerebrum, but certainly also to the peculiar forward bent position which the patient generally maintains. This half bent and half contracted attitude of the body and limbs is peculiar and rather characteristic, and so also is the fixed stare and the immobility of the facial expression in the later stages of the disease.

Paralysis agitans is slowly progressive, of long duration, even up to thirty years. One of my own patients has had it for twenty-four years, and is still among the living. Death may be brought about by marasmus, bed-sores, and other complications, or an intercurrent disease.

Varieties.—1. In a great majority of cases, paralysis agitans has an insidious and rather benign beginning, the tremor being limited to one foot or hand, or even thumb; may remain so for a long time, having rather an innocent appearance. The oscillations of the different segments of the hand, for instance, toward each other are almost pathognomonic. The fingers move and rub toward the thumb, the wrist toward the forearm, and that toward the arm. At this period the tremor may be going and coming, sometimes remaining away for many months. The usual progress of the tremor is from the upper to the lower extremity on one side, then the same way on the other side. The cross form, according to Charcot, is rare, and the above hemiplegic form, if you please, much more common than the paraplegic form. The muscles of the head do not participate even in the most intense form of paralysis agitans—an important differential point from disseminated sclerosis, where the contrary is observed. There is an observation of Charcot which may be noted right here. He says that the tremor is not always the first phenomenon in the disease; that it may be preceded by a period of intense feeling of fatigue and neuralgic and rheumatoid pains in the limb which will be the seat of the tremor later on. Charcot's cases of this kind, as well as one of Romberg, have been of traumatic origin.

But the tremor may also come on suddenly after severe moral emotions or a great fright, attacking either one limb or all at once. After some days or weeks, there may be temporary or even permanent cessation of the trouble, but more often the disease takes a definite hold after a series of exacerbations and remissions.

In the further developed stage of the disease the tremor is of varying intensity, but incessant; the convulsive movements ceasing however, during natural sleep or that produced by anæsthetics. The facial muscles are almost immovable, the face itself wearing an expression of sadness or dullness. The speech is rather slow, sometimes even saccadé. The patient prefers using short words and sentences, as if speech was too great an exertion for him. Deglutition and respiration are not interfered with, although some complain of a sense of oppression about the chest. The bladder and rectum are seldom affected; but in the case of Mrs. T. there was vesical paresis and obstinate constipation of the bowels for two or three years before she died. There is a good deal of muscular rigidity, particularly in the flexor muscles of the upper and lower extremities, also in the muscles of the neck. The cramps and neuralgic pains are frequent, and often very annoying to the patient; but there are no permanent contractures. The hands, however, often show an attitude quite similar to the deformity seen in the various stages of arthritis deformans.

As to the urine of patients suffering from this disease, Topinard has seen glycosuria in one case, and so have I in the case of Mr. O. There is sluggishness of thought and action. The patient generally rises slowly, and hesitates a few seconds before he propels himself forward, and vertigo often attacks him in the attempt at doing so. (The cases of Mr. M. and Mr. O. show this symptom well marked.)

Besides the neuralgic pains, there is a feeling of tension and of traction in the muscles, and of great fatigue and prostration; also a desire to frequently change position, particularly after the patient has gone to bed. This latter symptom is due to cutaneous hyperæsthesia as well as muscular pains, and also to a disagreable sensation of excessive heat which some patients complain of, Mrs. T. among my patients. But this feeling of heat is subjective only, for dynamic convulsive movements do not affect the temperature of the body. The transmission of cutaneous sensations, likewise the sense of touch, etc., is generally normal.

In some cases there is marked melancholia, hallucinations, and even maniacal disturbances have been noted. I have not seen any such symptoms.

In the terminal stage of the disease there is an increased difficulty of motion, fatty degeneration of muscles, defective general nutrition, decrease of intellect and loss of memory. Bed-sores occur frequently, and the disease terminates by general marasmus or through some intercurrent affection, like pneumonia.

Visceral lesions, such as occur in tabes or sclerosis, have not been demonstrated in any cases of paralysis agitans.

Pathological Anatomy.—The true pathological anatomy of paralysis agitans has yet to be found. In looking over the reports of autopsies we find cases in which none or very trifling changes were present in the central nervous system (such are the cases of Ollivier, T. H. Saloman, Kühne, Jeffroy-Charcot); others with lesions of softening or lesions of sclerosis in the spinal cord only (cases of Lebert, Cohn, and Murchison), or the brain only (cases of Marshall Hall, Cohn, Rosenthal, Leyden, and Chvosteck), and still others with extensive changes in the brain and cord (cases of Parkinson, Opolzer, Skoda, Meschede).

With such contradictory post-mortem results it will require the utmost caution to advance a theory of the pathology of this disease; nor is it even possible yet to determine whether it is of cerebral or spinal, or of combined origin. To be sure, the pathognomonic tremor would be apt to point to lesions in the spinal cord. From the fact that there are some patients afflicted with this disease who present the symptom of falling forward on attempting to rise or to walk, and others who do not, Remak was led to distinguish a cerebral and a spinal form of paralysis agitans, according to the presence or absence of that symptom. Such differential diagnosis would strike one as somewhat arbitrary, but the cases of Mr. M. and Mrs. T. among my own would show a preponderance of the cerebral symptoms, while the conditions of Mr. O. and Mr. H. are such as to make me believe that the spinal cord is more or less exclusively affected in them. The cases of Cohn and Stoffele in which senile atrophy of the brain was found must be excluded from consideration, for the reason that senile atrophy of the brain occurs also without any symptoms of paralysis agitans.

The focal lesions thus far described have been variable: the thalamus, probably the most important, the pons, and the medulla oblongata have been found in a sclerotic or softened condition, but the variability of the seat of the lesions bars out positive conclusions.

Topinard's case with glycosuria and Mr. O.'s case among my own might add some value to the supposition that the medulla oblongata is frequently the seat of disease in paralysis agitans, if it were not for other autopsies in the same disease where the medulla oblongata was found entirely normal. There is finally a case reported by Larcher of isolated sclerosis of the pons with a course of symptoms during life not at all like those of paralysis agitans. Not a few cases in which sclerotic conditions in the brain and cord, or both, were found have been put on record as cases of paralysis agitans by older writers, but were without doubt cases of disseminated sclerosis.

Etiology.—Men are more liable to get the disease than women, and it occurs also more among the lower strata of society. It is by no means a frequent disease. It belongs to the later period of life, although Meschede reports one case of paralysis agitans in a boy only twelve years old, developing soon after he was kicked in the face by a horse, and Duchenne that of a young man of twenty. Sander and other writers make the statement that it is more frequent in England and America than elsewhere, but I do not know how much value may be attached to this assertion. Among the twelve cases that I have observed there are only two

Americans, but I have to take into consideration that my clientel is largely German or of direct German descent (probably three-fourths, and only one-fourth American).

Powerful emotional and moral influences, the action of prolonged damp cold, irritation and injury of peripheral nerves by traumatism, are mentioned among the chief causes by the different authors. I am inclined to believe that sexual excess may be considered as an etiological factor in some cases, and suspect it to be so in the cases of Mr. S. and Mr. H. I am glad to say that I have not read nor observed myself that syphilis for once has anything to do with the production of this disease as it does in so many other chronic ailments. The hereditary element does not seem to play any role here, although I must not forget to mention that in the case of J. S., his father and two brothers have been afflicted with the disease, and in that of L. H. one brother is also affected.

Treatment.—With a view of arresting or even retarding the progress of the disease, I have for some years tried principally arsenic, ergot, and nitrate of silver, and given them thoroughly and persistently, but the results having been entirely negative, I have for many years made no further attempts in the direction of so-called curative drugs. I have, however, always refrained from giving the patient an absolutely unfavorable prognosis of his case, for though we know the prognosis in fully developed cases to be always fatal, it has been my experience that putting forth such prognosis as a matter of fact to the afflicted almost always had a bad influence upon the progress of the disease. To be sure, nothing can be done to save the patient, but a great deal has to be done to alleviate his sufferings and give him such comfort as every poor sufferer is entitled to.

In some of my cases I have succeeded with Brown-Sequard's mixture of the bromides (the iodide of potassium being left out) in diminishing the tremor, allaying restlessness, and relieving neuralgias and cramps. The opiates generally act badly, and ought not to be given. Hyoscyamin is now being used by most observers, I believe ; but having found its continued use somewhat dangerous, I have not operated with it much in this disease. In the case of Mr. O., antipyrin, twenty grains given at bedtime, helped for a time to procure better nights for him; and so did chloral hydrate with bromide of potash in his case and that of Mrs. T.; but the action of all these remedies has been quite transitory and much inferior to another preparation which I have tried during the last eighteen months, and particularly in the cases of Mr. O. and Mrs. T., and that is paraldehyde. I have used Merck's preparation exclusively, and given from fifteen to thirty grains, made with gum acacia and ginger syrup into an emulsion, at bedtime for many days in succession. It is an excellent hypnotic, it assuages pain, and has done more to relieve the paræsthesias, restlessness, and distress at night in the cases of Mr. O. and Mrs. T. than any other remedy given before. It is perfectly safe, it does not molest the stomach, although its taste is not agreeable; it does not weaken the heart, it does not cause any collapse or other unpleasant symptoms, and it does not lose in its efficiency when continued for any length of time.

The galvanic current applied to the head and spine I have found of some value in some cases as to relieving the feeling of fatigue and prostration to some extent, also alleviating neuralgia and vertigo; but, on the whole, it has neither curative nor great palliative effects. The tepid half bath with cold affusions given two or three times a week acts in a similar way, but with more certainty and promptness than the galvanic current. In the early stages of the disease a course of sea-bathing, with all the necessary precautions, to be followed every year, would seem to be a reasonable and promising remedy.

Whatever remedy may be selected by the physician to relieve his patient's sufferings with, he must also bear in mind that paralysis agitans is debilitating and mostly concerns debilitated persons, and that nutritious food, stimulants and tonics are essential adjuvants to any special form of treatment.

About a dozen cases of paralysis agitans have come under my notice in private practice; five of them I have had under my charge long enough to study what there was of interest in them to me, and I have referred to them in this paper from time to time.

1. C. M——, now about seventy years old, Amer. mech., began with the usual tremor symptoms of upper and then lower extremities about twenty-four years ago. When I saw him first, about fifteen years ago, the cerebral symptoms of forward falling, associated with depression of spirits, diminution of mental powers, etc., were already marked, and his sleep badly disturbed by sensation of heat and cutaneous hyperæsthesia. He still lives, though quite unable to get about now, even with support. The prolonged influence of damp cold in his office was the only element of importance which I could find in the etiology of the case.

2. Mrs. L. T—— came under my care in 1884. She was then about seventy years, a woman of fine intellect and vast knowledge of men and things. To know and talk to her was a bit of education. She gave a very clear history of the origin of the tremor symptom, which came on in '78, very soon after receiving the news of the fatal ilness of the daughter she loved most. The disease having been upon her for six years, all the limbs were affected, her facial expression of marked sadness, and immobility of features; paresis of bladder, constipation, and increasing motory weakness, particularly of lower extremities, well marked, but no spastic paralysis and no contractures were present. While she suffered but little from neuralgias, and could sit with comfort in her easy chair during the day, her sleep was often disturbed by the peculiar restlessness common to all advanced cases; the sensation of heat and the frequent desire to have her position changed; vertigo frequently present. A tropho-neurotic symptom, which has not come under my observation in any other case, was present during the last three years of Mrs. T.'s life; an atrophy of the epiderma of both hands and forearms and feet and legs reducing it to the thinness of tissue paper and such transparency that the apparently hyperæmic cutis could be readily distingnished as a bright red membrane. The motor weakness of lower extremities developed to para-

paresis during the last six years of her life, and death occurred in consequence of pneumonia, in September, 1887. Her nightly sufferings were much relieved by paraldehyde.

3. Mr. H. O——, æt. fifty-three, German, inkeeper, has been much addicted to drinking and smoking, denies having had syphilis, but gives an uncertain history of rheumatism. He has the well-marked symptoms of the disease for seven years, is now in the fully developed stage of it, and also has glycosuria with considerably increased diuresis. How long he may have had this, I do not know; however, neither by it nor the plague of paralysis agitans has his original weight of 320 pounds been reduced to less than 280 pounds, his present weight. This patient suffers more from pains in the cutaneous branches of both sciatic and other neuralgias than any other of my cases, and neither by chloral, nor the bromides, nor hyoscyamin, have his nights been made comfortable in any way, until at last paraldehyde, in ʒss. doses at bedtime, appears to be of good service. Well-marked in his case are muscular rigidity in various parts and troublesome œdema of legs. He has been seen by various specialists of this city, also by my friend, Dr. Dana.

4. Mr. L. H——, æt sixty-three, German, market dealer; a brother of his has paralysis agitans also. No history except the rheumatic influences coincident to his business, and sexual excess. The tremor began two years ago in right hand, extending in the course of a year to arm and shoulder, and has affected the right leg during the last six months. Paræsthesias, but no neuralgias present. Vertigo frequent. Arsenic seems to do him some good.

5. Mr. J. S——, æt. seventy-two, German, butcher. His father and two brothers have the disease. Has been guilty of sexual excess until very recently, and acquired syphilis late in life. His trouble began three years ago and affects, up to the present, left upper and lower extremities only. He is, by the way, left-handed. The shaking of hand and arm becomes exceedingly violent when in the least excited. He has also had terrible headaches and vertigo, which may have been due to syphilis, as they got well by specific treatment. His sleep is good as yet.

CONCLUSIONS.

1. In the pathology of paralysis agitans we have not come much further than at the time when Parkinson first described the same.

2. The progressive tremor, while the patient is awake and the muscles are passive, and the progressive motor weakness, are as yet the pathognomic symptoms, while the absence of both the intention-tremor and the contractures of spastic paralysis distinguishes it from disseminated sclerosis.

3. In the etiology of two of my cases an hereditary element can be proved ; but emotional influences and long exposure to damp cold appear to be the most potent excitors of the disease.

4. In the fully developed stage of paralysis agitans the patients often suffer greatly by neuralgias and otherwise, and need our help as much as those who may be afflicted with more malignant disease. Opiates afford no relief, and are contra-indicated according to common experience. Hyoscyamin combined with tonics is praised highly by many authors. Antipyrin in 15 to 20 gr. doses, and particularly paraldehyde in ʒss. doses at bedtime, have proved quite efficacious in my hands to alleviate part of the sufferings of these invalids.

25 West 46th St., N. Y.

PARANOIA: SYSTEMATIZED DELUSIONS AND MENTAL DEGENERATIONS.

AN HISTORICAL AND CRITICAL REVIEW,

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[*Continued from last Number.*]

V.

IN France, since the works of Morel, we meet with this subject only in isolated memoirs, describing the forms of insanity which, in spite of their different names, seem to us to correspond to certain varieties of *paranoia* that we have passed in review.

We must notice principally in this connection the work M. Ach. Fovillé upon insanity with a predominance of the delusion of grandeur (1871); then that upon the delusion of persecution by Legrand du Saulle (1873), an amplification of the memoir of Lesègue upon the same subject. This delusion, as we have seen, is a type of *paranoia*.

The thesis of P. Garnier* (1877) on the same subject should also be remembered.

In 1876 M. Taguet† described the insane persecutors, which he separated from the group of persecuted. This form of insanity also enters into the domain of *paranoia*, for it corresponds to the *querulanten Wahnsinn* of the Germans, and to the *querelenti* and *litiganti* of the Italians,[*] and we have already seen that the most of the time it has been considered as a form having a degenerative basis.

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* P. E. Garnier.—*Des idees de grandeur dans le delirium des persecutions.* (Thèse de Paris, 1887.)

† Taguet.—*Les Aliénés persecuteurs.* (*Am. méd. psych., 1876.*)

This also appears to be the opinion of J. Falret, who discussed the question in 1878, and made the class of persecutors a form of the delusion of persecution developing in subjects with an hereditary taint. On several occasions since then he has returned to this subject and has developed his ideas in his clinical lessons and in the discussions on hereditary insanity before the Medico-psychological Society (1885–86). We may also refer to the ideas brought forward in the thesis of one of his pupils, Dr. Pottier[†].

In 1882 Cotard[‡] described under the name of the *delusion of negation* a psychopathic form that he distinguishes from the delusion of persecution with which it might be confounded by the systematization of the hypochondriacal ideas, and the ideas of persecution and grandeur. But, in spite of the particular characteristics that may distinguish the nature of these ideas, it should be said that the systematized delusion of negation is always secondary to melancholic conditions, and most frequently to anxious states, instead of being primary as in the delusion of persecution. It would be an example of the so-called secondary forms of *paranoia*. We have reported an example that we consider typical.[§]

But we can best see, by comparison, to what nosological forms *paranoia* corresponds when we turn to the works of Magnan.[‖] In fact we find it there complete; for although considering the facts from another point of view Magnan has described none the less perfectly the same forms that we have been examining. Resuming the views of Morel on hereditary insanity, he studies this in its different manifestations, which he seeks to classify. In his opinion the hereditary subjects, or rather the degenerate hereditary subjects, may be divided into four degrees according to their mental condition: first, idiocy; second, imbecility; third, mental debility; fourth, *the superior degenerates.* Now the mental condition of this last class, with its anomalies of character and of intelligence, corresponds absolutely to what other authors (Sander, Maudsley, Krafft-Ebing, Tanzi and Riva) have described under the name of the psychic constitution of *paranoia;* some of the cases would even be examples of the so-called indifferent or indeterminate *paranoia*, or *paranoia* without delusion. And in all these cases there is the soil favorable for the development of primary systematized insanity, and that even certain authors, admitting only the degenerative forms, regard as indispensable, the delusion being only the exaggeration of the particular character of these patients.

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[*] We have translated these words by *quarrelling insanity* (folie de la chicane). although they express more the idea of complaint. On this subject see also Liebmann—*Ueber querulanten Wahnsinn* (*Allg. Zeitsch. f. Psych.*, Bd. xxxv., p. 395); Brosius—*Ueber querulanten Wahnsinn* (*Alig. Zeitsch. f. Psych.*, Bd. xxxii., p. 770).

[†] Pottier.—*Etude sur les aliénés persecuteurs* (Thesis, Paris, 1886).

[‡] Cotard.—*Le dêlire des negations* (Arch. de neurolog., 1882).

[§] J. Seglas.—*Note sur un cas de melancholie anxieuse* (*délire des negations*). (*Arch. de neurolog.*, *1884*).

[‖] Magnan.—*Leçons sur la folie héreditaire, 1882–1883; Les délirants chroniques et les dégenéres* (*Gaz. des hopit.*, April, 1884. *De la folie héréditaire* (*Journ. des conn. med*, 1885, No. 48). *Ann, Med.-psych.*, 1885–1886; *Tribune medicale*, 1886, No. 954.

Among the superior degenerates Magnan makes the synthesis of a certain number of particular states that he designates under the name of episodical syndromes. These conditions, characterized by obstinancy and impulses, with mental anguish and clearness of mind, are what other alienists have designated under the name of fixed ideas, and thus represent that form of rudimentary *paranoia* that Arndt first described. In this connection we may mention that Magnan belongs to that group of physicians who consider that these psychical troubles are characteristic of a state of degeneration[*] (psychic stigmata).

Moreover, deliria may develop in these syndromes, and these may be of several kinds. Besides the deliria of the onset already noted by Morel, systematized deliria with a slow development may be met with; some are primary, that is to say, they fix themselves little by little without attracting attention; others may be consecutive to a delirium of the onset, which may prolong itself indefinitely; at other times again they may be seen to follow the simple delirious tendencies which seem to be the prodromal period, and of which they are only the exaggeration. Who will not recognize in this brief sketch of the slowly developed deliria of degenerates, those forms of delirious *paranoia* engrafted on degeneracy, as generally admitted, and of which the idiopathic (originäre) *paranoia* of Sander is the type.

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[*] Magnan.—*Leçons sur la dipsomania (Progres medical)*, *1884*. *De l'onomatomanie* (in collaboration with Charcot), *Arch. neur.*, *1885*.

We cannot enter here into Magnan's doctrine of hereditary insanity. We refer, for the details, to the thesis of Legrain, who gives in a very complete manner the ideas of his master on the different points we have noticed.

But in Morel's classification there is still another group of patients that seem to us to correspond also to certain forms of *paranoia*. These are the victims of *chronic delirium* (*les delirants chroniques*). From the symptomatological point of view the subject of chronic delirium is only the common persecuted patient taken in the different halting places of his delirium, as already partly seen by Morel, Snell, and others (period of disquiet, of persecution, of grandeur and of dementia), and representing the synthesis of certain old monomanias (hypochondria, demonomania, megalomania, theomania, etc.). It is then that the delirium presents a most marked systematization. Now, the comparison of observations on chronic delirium with those on delirious *paranoia* shows us in the majority of cases an identical description of one and the same form of insanity. There is the same symptomatology, the same course (as shown by the hallucinations, especially of hearing, the nature and evolution of deliria, and the reactions of the patient), just as other examples show us similar symptoms, and an evolution analagous to that of the deliria of degenerates of slow development (peculiar mental condition, insidious and progressive beginning or rapid appearance, hallucinations either numerous or absent, and the relations between these and the deliria).

In connection with this subject it should be recalled that with reference to the succession of the delusional ideas, the different alienists who have written on *paranoia* have observed that the ideas of persecution or of grandeur may exist in the isolated state, or if they are recognized in the same individual (*mixed paranoia*) they are seen to be contemporaneous or to succeed each other, the ambitious ideas being the consequence of the ideas of persecution. Now, when we turn to Magnan's classification we shall see in the last case a succession of ideas analogous to those that are found in chronic delirium, while the other varieties correspond to the deliria of the degenerates.

Finally, as regards the termination, we shall find again strong analogies between the forms of delusional *paranoia*, and the deliria of degenerates and chronic delirium. Their course, which is very long, rarely ends in a true dementia, and in the midst of the dissociation of the intellectual faculties there is often found a trace of the old systematized delusion. This period of dementia is rather a period of mental confusion.

In direct opposition to the authors that we have passed in review, and who nearly all unite together all the varieties of *paranoia* by attributing to them a common degenerative basis, Magnan makes a separate class of his subjects of chronic delirium, and while admitting that they are often hereditary subjects refuses to make them degenerates.

This opinion does not seem to be held by Gérente, who in his monograph on chronic delirium, says that this form of insanity is not met with in the earlier writers; it requires a long incubation, two or three generations preparing the ground, and *predisposition* is necessary.[*]

The author even went further when he said with relation to the breaking out of the delirium, that if he meets with some accident the patient succumbs, "being moreover *from his birth* what is called *a weakling* or being mentally enfeebled in the course of his life." He seems to us again to unite the chronic deliria with certain deliria of the degenerates of Magnan, when he says that of these insanities (the chronic deliria), those which have been most affected by direct insane hereditary influence will show themselves in their essentially intermittent delirium, and will recover or recover more easily. There are, moreover, among the observations that he reports examples of mental degeneration.

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[*] Gerente.—*Le délire chronique, son evolution* (*Thèse de Paris, 1883*). *Quelques considerations sur l'evolution de délire dans la vesanie* (*Arch. de neurolog.*, t. vi., 1883, p. 16).

Another pupil of Magnan, Legrain,* distinctly admits that degenerates may be affected with chronic delirium. This opinion, which we, for our part, shall be disposed to share, surprises us however in Legrain's book, for in our opinion it contradicts the classification that he adopts, and consequently renders useless the distinction that he makes between deliria of degenerates and chronic delirium, which would be only a form, at least in certain cases. In fact, whatever may be the basis on which it is admitted that the chronic delirium develops, its diagnosis from certain deliria of degenerates, which simulate it, even to being mistaken for it, is clinically the most difficult, not to say impossible.

Very interesting observations upon the question that is occupying us are found in the work of Legrain, who studies all the forms of deliria that are met with among the degenerates, their mental state, the episodical syndromes, and the deliria of the onset or of the chronic development. We must reproach him, however, for not giving us an historical review of the question, which, if it has not been considered under this aspect, has however been already treated in great part.

We may refer to an earlier work of Saury,† who has also studied the mental states of degenerates and the episodical syndromes, but has only described the deliria of the onset.

It remains to say a few words on the French works relating to *paranoia*, and we will close this review by citing the work of Régis,‡ where, under the name of partial insanity, he reproduces the ideas of Magnan on chronic delirium; and a lecture of Ball§ (1885) upon a particular form of distinct ambitious delusion, with ideas of the same nature as the weak subjects (*débiles*), those suffering from circular insanity, the persecuted and the general paralytics, and which he likens to Ach. Foville's insanity with predominance of delusions of grandeur.

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* Legrain.—*Du delire chez les degeneres* (*Thesis, Paris, 1886*).

† Saury.—*Etude clinique sur la folie hereditaire* (*les degeneres*), *1886*.

‡ Regis.—*Manuel practique de medicine mentale.* Paris, 1885.

§ Ball.—*Du delire ambitieux* (L'Encephale, 1885).

We have seen, in fact, that *paranoia* is no new thing in psychiatry, and we can recall its history by citing the numerous names under which the alienists of different epochs and different countries have designated it. We see, too, that born in France, the doctrine of primary systematized delusions has been especially developed in Germany, and since then in other countries and especially in Italy. Perhaps this study has even been pushed to the point of exaggeration, each one wishing to add his particular note, and bringing confusion from the multiplication of forms.

What is there especially to emphasize in a résumé of the different theories that we have sought to explain? One fact that stands out prominently from this historical review is that all authors admit a form of primary *paranoia* engrafted on a soil of degeneration, and the existence of which moreover is indisputable; but some admit only this form with its varieties; others restrict its domain more or less, and do not consider that the ground of mental degeneration is indispensable to the production of *paranoia*.

It is, then, in the scheme of this psychoneurotic *paranoia*, that we meet by the side of the chronic form that form of *paranoia* called acute, psychoneurotic, hallucinatory, and curable, admitted for the first time by Westphal. Here opinions are much divided; some follow the ideas of Westphal, as, for example, Meynert, Fristh, Mendel, Tiling, Amadei and Tonnini, etc. Others completely deny its existence, or at least do not describe it as a form of *paranoia*: these are Krafft-Ebing, Pelman, Mayser, Morselli, Tanzi and Riva, etc. For ourselves, we are fully inclined to adopt this latter opinion. The study of the observations on acute *paranoia* that we have met with in the course of our reading, has failed to show a single pathognomonic symptom which could in any way show a relationship between this acute *paranoia* and the chronic form, whether degenerative or primary.

On the contrary, it seems to us that this variety is very comparable sometimes to certain melancholic states more or less accentuated, often with stupor, but sometimes with depression or anxiety, and sometimes to states of simple or symptomatic maniacal excitement.

There is still much discussion on the subject of the form of insanity called rudimentary, described by Arndt, and the type of which is represented by the fixed ideas. The ground, as we have seen, upon which these ideas may develop is much contested; and on one side certain authors approach entirely the fixed ideas of *paranoia*, distinguishing them, however, because of the preservation of consciousness. Others admit them as a rudimentary form, others as a prodromal period, and still others as an episode in the course of *paranoia*.

Regarding the secondary form, its existence is indisputable; but it is only one form of *paranoia* properly so called, and it is only one form of systematized delusion simply secondary to some maniacal or especially melancholic states, of which it serves as the termination or as a bond of union between them and dementia. There remain still the pretended forms of hysterical, epileptic, and alcoholic *paranoia*.

For ourselves, we should wish with Krafft-Ebing to do justice and put them under the pathological state of which they form a part. It should be remembered, however, that certain of these patients are possibly true examples of *paranoia*, and that there may be found among them the co-existence of two delusions, that only an attentive observation is able to distinguish.*

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* In this connection see also Magnan, *Arch Neur.*, No. 1; Garnier, *Gaz. hebd.*, 1880; Dericq, Thesis, Paris, 1886; Krafft-Ebing, *loc. cit.* Among the works on the subject of *paranoia* that have come to our knowledge since the composition of this memoir we may cite: Poggi, *Riv. sp. di fren.*, anno x., fasc. 4; Guillardi and Tanzi, *ibid;* L. Bianchi, *La Psychiatrica*, anno iv., fasc. 3 and 4, p. 2; G. Zuno, *ibid.*, p. 220; Zenner, *The Medical Record*, 1887, p. 124; P. Garnier, J. Falret, Dagonet, Briand, and Cotard.—Discussion on chronic delusion (*Aun. Med.-psych.* and *Archives de Neur.*, 1887).

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INSANITY FROM BRIGHT'S DISEASE.

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(Read before the Missouri State Med. Association, April 17th, 1888.)

BRIGHT'S DISEASE as a factor in insanity is by no means a novelty, yet the literature on this subject is, on the whole, rather scanty. Besides, in many of the cases reported the connection between the renal and mental phenomena is not quite clear, and in others it seems hardly justifiable to qualify the manifestations of perverted mentality observed, as insanity.

Generally speaking the insanity of Bright's disease is that of uræmia, and "uræmic insanity" would perhaps be a more appropriate term. It is, indeed, generally observed only in the graver or fatal forms of nephritis, acute or chronic, in which the functions of the kidneys are impaired to such a degree that the greater part of the excrementitious substances normally eliminated by these glands is retained. Instead of the characteristic symptoms of uræmia, viz.: headache, vomiting and coma, a psychosis, either single or complicated, with one or the other uræmic manifestations is set up.

Whether in such cases, it is the retained urea alone that produces the morbid mental states, or whether there are any of the non-excreted leucomanies which are normally formed by the splitting up of the albuminoids, responsible for the disordered brain functions is, at present, an open question. It is held by some that in the common form of uræmia, ammonia, by others,* the potash salts cause the grave nerve symptoms. However this may be, it is in the highest degree probable that in uræmic insanity it is the urea that so peculiarly influences and morbidly affects the highest brain centres, although experimentally it has been demonstrated that this poison when injected into animals whose renal arteries have been ligated, gives rise only to convulsions and stupor.

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* N. Feltz and E. Ritter De l'uremie experimentale, Paris, 1881.

There is a certain similarity of chemical composition between urea and those poisonous albuminoid bodies, the ptomaines, which according to the present state of knowledge are looked upon as being at least in part instrumental in bringing about the delirium and mental aberations of many of the acute infectious diseases, *e.g.* typhoid fever, pneumonia, etc. It is true that generally the brain symptoms in those diseases keep in proportion to the rise and fall of the temperature, and that over-heating is the principal cause of disordered brain action, but there are cases of febrile disease in which the latter is entirely disproportionate to the moderately high temperature. Such discrepancy is, for instance, not infrequently seen in certain cases of typhoid fever, in which the sensorium is more deeply affected than the fever-curve would warrant.

Such cases are liable to be mistaken for insanity, the typhoid element, owing to the irregular or insignificant elevation of temperature, being entirely overlooked, and it may happen that patients of this class are sent to an insane asylum. I know of one instance in which a superintendent of a lunatic asylum, being in the first stage of typhoid, with strong predominance of cerebral symptoms, was committed by his assistants to his own institution.

In these cases as well as in uræmic insanity, it is a toxic akaloid like principle, a ptomaine in the former, and probably urea in the latter, that determines for some unknown reason the preponderance of the mental over the other usual disturbances.

So far only one case has come under my observation in which a causal connection between an exacerbation of renal disease, *i.e.* accumulation of urea in the blood, and *indubitable* insanity could be clearly proven.

CASE I.—M. C., a maiden lady, æt. 38, coming of healthy stock, had rheumatism followed by chorea at 14. Since this time she has periodically suffered from palpitation of the heart, sleeplessness and general nervousness. Four years ago she fell into ice-cold water and claims that from that time on her kidneys have been out of order, that she had to get up at night to urinate three or four times, and that the quantity voided has been very irregular, scanty at times, and excessive at others. Her urine had been examined by several physicians and a more or less considerable amount of albumen had been found. There never was any œdema. The present attack came on after exposure to cold and dampness, the secretion of the urine became exceedingly scanty ; after a few days she grew morose and fretful ; she did not sleep for several nights, her irritability and restlessness increasing constantly until a week after the exposure she had a maniacal attack. For a day and a night she shouted, gesticulated and sang religious songs, beating for hours the time with hands and feet. On the day following she began using obscene language, swore at the members of her family and tore her clothes. This lasted about two days when a period of depression set it, during which she wept a great deal, accused herself of base and mean actions, of having caused the death of her mother, and of having brought utter disgrace and ruin on her family and the whole neighborhood. During this melancholic stage there were twitchings of the muscles of the face and extremities. On the ninth day of her disease she attempted to cut her tongue out with a table knife ; a copious hemorrhage resulted, which was stopped with a great deal of difficulty after the patient had become almost exsanguinated. She then slept for nine hours uninterruptedly. When she awoke she was in a dazed condition, but took nourishment freely and gave rational answers to simple questions. In the course of two days her mind became perfectly clear ; she remembered dimly some of the events that had taken place, but had absolutely no recollection of her attempt at self mutilation. She made a rapid recovery.

From the third day of her sickness until sometime after her restoration to health I made daily examinations of her urine. In the mean its specific gravity was 1015; it was strongly acid, containing a large quantity of albumen, hyaline and epithelial casts, pus-cells and blood corpuscles in small number. The daily amount of urine voided could not be ascertained during the first nine days of her sickness, the patient passing it most of the time involuntarily, but it was far below the normal. The casts and the albumen, though diminished in amount, could be demonstrated for three weeks after she recovered her senses, when the urine seemed to become perfectly normal. During the maniacal excitement there had been a slight rise of temperature, after this it had been normal and even sub-normal. Neither vomiting nor headache had been complained of. Pilocarpine, elaterium, and later, digitaline had been administered without any apparent benefit.

I am strongly inclined to believe that the sudden termination of the attack is due to the copious hemorrhage. Some practitioners are still treating uræmia, especially when appearing under the form of puerperal eclampsia, with the lancet, and, apparently, with good results, especially in persons of full habit, and I am of the opinion that in some cases of insanity due to uræmia, bleeding is the proper remedy in spite of the fact that such a procedure would not be in harmony with the teachings of modern psychiatry, and that very likely it would be branded as a return to antiquated medical barbarism. But it appears to me rational and proper to remove as quickly as possible the retained products of metabolism, a powerful nerve poison, which by its continued action on the nerve centres, renders all remedies worthless.

Cases of real insanity of uræmic origin are, on the whole, of rare occurrence; as a rule the mental disorder consists of elementary deliria accompanied by hallucinations. The following case may serve as an example.

CASE 2.—C. B., æt. 26, a plumber, habitual, hard drinker, is suddenly taken with what appears to be inflammatory articular rheumatism; feet and ankles are swollen, red and painful; at the same time there is severe pain in the back; the left tonsil, parotid and submaxillary glands are swollen;

the gums look inflamed and are extremely turgid ; temperature 100°. His legs frequently twitch ; whenever he tries to walk there is a convulsive tremor through the whole body and a general spastic condition of all the muscles ; he walks on tip-toe, grating the floor with the ball of the foot ; can not bring down the heel ; the head is drawn backwards, the spine in a lordotic state. Several times a day he gets "rigid spells," even in bed ; there is opisthotonus, his legs and hands are stiff and extended, and he becomes temporarily aphasic. The superficial reflexes)plantar, cremasteric, and abdominal), normal ; knee jerk absent ; hyperæsthesia of epigastric region and of the lower third of thorax. The examination of the urine which is scanty at times and copious at others, reveals an enormous amount of albumen, of hyaline and blood casts and renal epithlia in a state of fatty degeneration, or impregnated with blood-pigment ; the color of the urine is of an intense reddish-brown ; it emits a fetid odor and abounds, even when freshly voided, in bacteria, the nature of which was, however, not determined. His mind wandered, although he could be easily roused to temporary consciousness, he was traveling constantly, driving horses, etc., he had hallucinations of sight and hearing; talked to imaginary persons ; could be kept in bed with the greatest difficulty ; when interfered with in his attempts to leave the bed he became violent. Six leeches were applied to the nape of the neck, and shortly afterwards a general oozing hemorrhage set in ; he bled from the gums, the nose and intestines ; the leech bites oozed for several days in spite of the efforts to stop the bleeding. His symptoms, however, grew better from day to day ; his mind became clear, the tremors and spastic states of the muscles disappeared ; the urine was free from albumen and contained only few casts and he made several successful attempts at walking. The improvement lasted three weeks when his mind again became clouded. Though his temperature was normal his mind was wandering ; he was constantly driving horses, but was always on the wrong road. At times he would be maniacal, try to break things, run off, etc. The urine contained an increased amount of casts and a greater quantity of albumen ; he died in coma eight weeks after the inception of the disease. Within the last four weeks the temperature had been normal and subnormal. Diagnosis ; acute parenchymatous nephritis. No post-mortem.

The clinical picture of this case was in the beginning, that of an infectious disease, perhaps rheumatic in origin, and complicated with some other morbid elements of bacterian nature, thus constituting a mixed infection.

I have reported this case at some length because it shows the usual type of mental disorder in uræmic conditions and is of pathogenous interest in so far as it exemplifies the truth of the assertion that Bright's disease is in its inception frequently a general vascular lesion. The general hemorrhage proved it. Had this patient recovered from the acute attack, the probability is that the remnant of the disease would have been localized in the kidney as one or the other form of Bright's disease.

The most insidious of all forms of nephritis and one that frequently remains unrecognized as a cause of mental disturbance, is the shrunken kidney. Being usually chronic in nature from the beginning, the symptoms are often vague and indistinct, and it runs its fatally progressive course under the guise of neurasthenia, neuralgia, etc., until, all at once, grave disorder of the mind sets in and the patient lands in the insane asylum.

Within the last six weeks three cases of this kind were received at the St. Vincent's Institution of St. Louis, all of which were declared insane by the attending physicians ; the urine had not been examined. I believe them of sufficient interest and importance to briefly report them as follows :

CASE 3.—Mrs. H., æt. 48, widow of a physician; of robust build. Early in life, when six or seven years old, she ran a piece of shingle into the right temporal bone, the wound healed and a slight tumor formed at the site of injury, which, about twenty years ago, gave rise to attacks of neuralgia. The tumor was removed about fifteen years back and two splinters were taken out. Fourteen years ago she had an attack of puerperal mania from which she recovered in the course of several weeks. Six years latter she suffered for weeks from rheumatism and intense insomnia. This was followed by an attack of outspoken melancholia lasting several months. Three weeks before her admission to the institution she went through a railroad accident with all of its excitement and exposure. For weeks she had been under great mental strain, owing to business affairs. Several days after the accident she was taken with a tonsillitis; great pain in the limbs, took (as is alleged) an overdose of morphine and chloral and became delirious without fever. She imagined she was on board a ship, saw fish coming towards her and talking to her, had visions of terrible faces and monsters threatening her; voided urine involuntarily, etc. A few days after admission to the hospital died of uræmic coma. The urine contained a moderate amount of albumen and great masses of hyaline and epithelial casts and a few pus corpuscles. Diagonis: shrunken kindey. Bright's disease had been "suspected" for a number of years by her husband, who was a physician. The probability is that her previous attacks of genuine insanity were due to the same cause as the last one, namely, to uræmia, and it may not be amiss to state at this place that in regard to puerperal mania, I have been twice in a position to corroborate and verify the statement of Scott Doncin, who maintains that there is a renal form of puerperal insanity. In two cases that came under my observation, albumen and casts could be demonstrated and the attack diminished in severity and disappeared together with the renal symptoms.

CASE 4 is similiar to the preceding one.

Mrs. F. S., æt. 55. No heredity. She is brought in a comatose condition to the institution. It is learned that for the last ten years she has been subject to spells of sciatica of great severity. About three weeks before her admission she was taken with an exceptionally severe one; strong doses of morphine were administered hypodermically and chloral given for about ten days to insure sleep, when her family began to think that she "talked funny" and that her mind was not quite right. She imagined that she was in a strange place, wanted to go home, although she was in her own room, called for her husband although he was constantly present, and gave other evidence which showed that she utterly failed to recognize her surroundings. Being very restless and becoming violent she was put on the train under a dose of chloral and shipped to St. Louis. She died in uræmic coma about one week after her admission to the institution, never having recovered consciousness although at times she could be roused and recognized her friends. On examination the urine (specific gravity 1030) was found to contain "mucous casts" (so-called), granular casts, epithelial cells in a stage of fatty degeneration, pus corpuscles and blood casts.

The interest in this case centres on the fact that the patient had been subject to sciatica. This is frequently one of the signs of shrunken kidney, and so prominent is it that often the original trouble, *i.e.* Bright's disease, is entirely overlooked. Among other similar cases I remember that of a St. Louis physician, who died several months ago with the symptoms of uræmia. He had for number of years been subject to occasional attacks of lumbago accompanied by sciatica. During the last of these he dosed himself with morphine to the extent of almost poisoning himself. From this time on grave cerebral symptoms developed, whilst the temperature remained most of the time normal. He became very violent, broke his bed, imagined to be on an ocean steamer, etc.

It was thought by the attending physician that there was some obscure form of brain and spinal disease. He lived in this deranged mental condition about eight weeks from the beginning of the sciatica. After death his brain was found to be slightly odematous, corresponding to the usual pathological condition of that organ in uræmia. Unfortunately the kidneys were not examined in this case, but in the light of my recent experience, I am morally certain that the urine as well as the kidneys would have revealed the true state of affairs, *i.e.*, uræmic poisoning.

CASE 5.—L., an alcoholic of long standing, becomes suddenly insane after the opening of an intra-muscular abscess situated about one and a half inches below the apex of the heart. He is admitted to the St. Vincent's on a certificate of insanity. There is cirrhosis of the liver, ascites and considerable exudation into the pleural cavity. His urine is loaded with albumen, hyaline and. epithelial casts, renal epithelia. Few pus corpuscles. He has ideas of persecution, believes that the attendants in the institution are after his money; that robbers are in the house and he continually attempts to bar the door of his room to keep them out, etc. At the hospital whence he came, he had become unmanageable. No fever. Death from coma. No post-mortem.

For the sake of completeness, I will briefly mention two cases which I had of late an opportunity of observing at the City Hospital of this city.

Case 6.—A negro about 35 years of age, an alcoholic and epileptic, had been time and again, at varying intervals, in the hospital for treatment of his epileptic seizures. It was known that he had Bright's disease. During the last six months he had had no epileptic attack. He was brought into the hospital in a stuporous condition, from which he could not be roused. He remained in this state for several days when, suddenly, one night he became unmanageable, tried to break the furniture, made speeches, declaring that he was a free born American citizen, entitled to all the rights and privileges of such, that he could whip any man in town, etc., in short, he was typical maniac. No fever. About one week after admission he died comatose. Post-mortem revealed as a principal lesions: cirrhosis of liver and kidneys.

Case 7.—S., a colored woman, æt. 50, had been admitted to the City Hospital without a history about one week previous to my examination; she was aphasic, could not pronounce a word nor understand the meaning of one; whether there was word-blindness or agraphia could not be made out, since the patient was illiterate. On being asked her name she invariably answered Til-lil-lil-lil. This was the only verbal expression that could be elicited to any question proposed. She had to be spoken to several times before she would try to answer. She was silly and had a giggling laugh without motive. No paralysis on right side. Temperature 97°. Urine specific gravity 1015. Pupils contracted and sluggish of reaction; urine and feces were passed involuntarily, slight tremor at times in hands and feet; casts, hyaline and epithelial, pus corpuscles, albumen, hypertrophy of left heart. A few days later she became more attentive, could pronounce her name (Martha Smith) though with difficulty, execute simple movements with her hands when told to do so, and gave other evidences of returning intelligence. It was now quite clear that she was suffering with motor aphasia, since she became impatient and irritated or laughed at herself whenever she tried to pronounce a word without success; after she had mastered the pronounciation of a word, she would repeat it several times; understood questions better. A few days later a hemiparesis of the right side supervened which did not last, however, very long. Finally she got well enough to help about the ward, which she did willingly. But the improvement did not last very long; she became destructive, uttered threats and seemed to have homicidal tendencies; everything she could get a hold of she would throw into the water-closet, etc. She was then transferred to the City Insane Asylum.

REMARKS.

There is no doubt as to the existence of renal affection in all the cases reported above, nor can there be any question as to the cause of the mental derangement observed in these patients, although there was in none of them the usually observed symptom-grouping of uræmia, etc., viz., headache, vomiting, and convulsions. The absence seems in a measure to be characteristic of uræmic insanity, and reminds one of the physical equivalent of the epileptic attack.

It might be questioned whether the morbid mental manifestations in all the cases detailed above can be legitimately classed with insanity. There is in a majority of them a close resemblance to the delirium of alcoholic intoxication. With the exception of cases I. and VII., this delirium is of an elementary character, and only in case V., barring case I., which is one of equivocal insanity, is there a feeble attempt at systematization of ideas begotten by delirium. Most doubtful is case VI., owing to the complication of alcoholism and possibly idiopathic epilepsy.

But although nobody would classify the drunken man or him that has an attack of delirium tremens with the insane, we know that etiologically, though not ontologically, there is such a thing as alcoholic insanity, as there is one from the continued abuse of drugs, morphine, hashish, and cocaine, for instance. Urea retained for a longer period in the blood seems to act in a manner similar to these substances on the highest brain-centres, producing perversion of thought, feeling, and action ; and the absence of fever and a degree of chronicity of the mental change will warrant the application of the term *insanity* to such cases.

But as in alcoholic or the other intoxication insanities, so in the uræmic variety, there is no type ; it may give rise to all kinds of mental abnormalities, from the most expansive forms down to imbecility.

That there must be a predisposition to mental disorder in the individual affected by uræmia would seem to be a postulate of common logic ; and it might be justly claimed that, as in ordinary cases of insanity, there must be in the uræmic kind, in addition to the exciting cause, a remote one which is of much greater pathogenic importance, namely, hereditary or acquired predisposition. With the exception of case III., which looks suspicious on account of the injury of the head, and case I. (chorea), no predisposition or heredity could be made out. (Owing to the absence of any record whatsoever, the cases VI. and VII. are not counted.)

Yet, judging from common experience in matters of insanity, and taking into consideration the, on the whole, deficient histories that were given, and the well-known tendency on the part of the relatives to deny insanity in the family, I am inclined to believe that my patients, if their and their families' histories had been known, would have been found to be tainted ; at all events, considering the different effect on different individuals of the same poison, there must be a preponderance of the insane over the convulsive temperament. Even the alcoholic intoxication, the prototype of toxic insanities, demonstrates clearly the different modes in which different persons are affected according to their organizations and idiosyncracies. One becomes maniacal, another melancholic, a third has convulsions, and a fourth one is at once rendered stuporous and even unconscious.

Besides these resemblances to the alcoholic delirium, the uræmic attacks reminded me often of post-epileptic insanity, even in those cases that were free from convulsions.

As regards the aphasia and transitory hemiparesis in case VII., there is a possibility of holding the uræmia alone responsible for such disturbance, although there is a possibility that it was produced by a coarse lesion, viz., circumscribed hæmorrhage, or thrombosis, the result of general vascular disease such as is common in Bright's disease.

Brieger (Klin. Beob. Charité Annalen, 1882, p. 237), saw a case of uræmia in which there were convulsions followed by a psychosis lasting eighteen hours. Amnesic aphasia terminated the attack. The patient got well.

I did not propose to write an exhaustive treatise on insanity from Bright's disease, and consequently refrain from enumerating and reviewing the literature on the subject. My purpose in publishing those cases that came under my observation was to urge the necessity of examining the urine of such patients as become suddenly insane, especially when the insanity partakes of a delirious nature and when alcoholism is to be excluded. I think that many a case of uræmia has been put down as mania without the correct diagnosis as to the cause having been made.

An exclusively chemical test is, of course, not sufficient; albuminuria is not Bright's disease. With the microscope alone rests the final decision. That even this instrument fails in some cases, for a time, to reveal the true state of affairs, notably in contracted kidney, is too well known to require discussion.

From a therapeutical point of view the importance of an early diagnosis is obvious. A timely regulation of the diet may turn the scales of the balance in favor of recovery, at least in the more acute forms of the disease.

From the clinical course of cases I. and II., I consider myself justified in concluding that in certain cases such incisive measure as blood-letting is indicated. That here the strictest individualization is required is self-evident.

Again, in case of death, it is of great import to the family to know of what form of insanity their relative died. As regards the social and business status and record of such a family in the community, it makes a great difference whether the death-certificate reads, "Mania," or whether the cause of death is given as "Uræmia."

Finally, a correct diagnosis will sometimes serve to keep a patient out of an insane asylum, and will cause him to be treated at home, on the same ground as a delirious typhoid fever patient receives home or general hospital, but not an asylum treatment. This remark does not imply that all patients suffering from uræmic insanity ought to be treated outside an asylum.

SIX CASES OF EPIDEMIC CEREBRO-SPINAL MENINGITIS.*

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OF PHILADELPHIA.

Of the six cases briefly detailed in this communication, five were observed at the Falls of Schuylkill, Philadelphia, and one in the southwestern portion of Philadelphia. At the Falls of Schuylkill several other cases have recently been observed, and it is interesting to recall that it was in this neighborhood, during 1864 and 1865, that the disease was widely prevalent and very virulent. In the first case an elaborate autopsy was made, which gives the case a distinct scientific value; in the other cases, certain special points of interest make them worthy of being recorded. They are reported briefly so as not to load the paper with unnecessary details. The first four cases occurred in the practice of Dr. Cahall, and two of them were seen by Dr. Mills in consultation; the fifth was a patient of Dr. J. Y. Kelley, of Manayunk, Philadelphia; the sixth was a patient of Dr. J. W. Dick, and was seen several times by Dr. Mills in consultation.

CASE I.—Mrs. W., aged thirty, the mother of three children, had had bad health since the birth of her last child, now two years old. She was greatly depressed in spirits and imagined she had all sorts of diseases, but for a few months she had regained, to a considerable degree, her former health and spirits.

February 21, 1888, she was taken with chills and headache, and on the morning of the 22d, when first seen by Dr.

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Cahall, she complained of acute pains down both legs, but more intense in the posterior aspect of the knee-joints, any movements of the limbs causing her to scream with pain. She already had headaches, contraction of the muscles of the neck; and ten to fifteen irregularly shaped spots of a color from pink to purple, and from the size of a pea to a silver dime, were scattered over the legs and abdomen. Diarrhœa, abdominal distention, and tenderness were present from the first. Delirium came on by the morning of the 23d, with lucid intervals, when she said she saw everything double. All the other symptoms were intensified.

On the 24th she was totally blind and partially deaf, but when aroused could give sensible answers to questions. She still complained of the headache, but not of the pain in the legs unless pressure was made along the course of the nerves, when she would cry out as though suffering great pain. The pulse was rapid, but the temperature was never over 103°. There was general hyperæsthesia.

On the 26th she sank into a stupor, from which it was gradually more difficult to arouse her. She died upon the morning of the 29th on the eighth day of her sickness.

Dr. Mills saw her in consultation the day before her death.

Autopsy.—Drs. A. H. P. Leuf and Judson Daland performed the autopsy, at which the writers were present, and examined the specimens. The following are the results of the megascopic examination.

Brain: The pericranium was very vascular. The veins of the dura mater were gorged with dark venous blood on the outside, and the inner side of the membrane showed marked arterial injection. It was adherent to the convexity of the right hemisphere at the upper border of the quadrate lobule. In front of this on both sides, were Pacchionian adhesions of the dura to the brain. The anterior lobes of the brain, especially on their convexity near the great median fissure, were œdematous, with a few slight opacities of the pia. At the base was seen liquid and semi-liquid pus about the optic chiasm, interpeduncular space, pons, and oblongata. The auditory and facial nerves were bathed in pus, as were, in fact, all the nerves at the base of the brain except the olfactories. All the cranial nerves were soft, especially the olfactory and optic. Softening of the crura cerébri, pons, crura cerebelli, and oblongata was also decided. The pia covering the isthmus was intensely injected, but this could only be seen by the removal of the pus that covered it. The arachnoid between the medulla and cerebellum was also covered by a thick layer of pus.

The puncta cruenta were well marked, numerous, and dark. The basal ganglia were normal, except some venous puncta. The fornix was softened. The fissure of Sylvius presented nothing abnormal on either side when opened for inspection; and all the cerebral vessels appeared normal.

Spinal cord: On opening the spinal canal, the cord lay flat and spread out in its membranes, instead of being narrow for the canal, and bulging with well marked convexity. The dural vessels were well injected externally and internally. On section of the dura pus exuded freely. The membranous coverings of the spinal nerves within the canals were almost ecchymotic. This was most noticeable in those given off opposite the lumbar enlargement, and especially on the left side. The whole cord was surrounded with yellow creamy pus. The lower half of the back of the cord was covered with a yellow gelatiniform pus layer, while the same covering was found on only the lower two inches in front. All the pus was situated between the dura and pia. The anterior and posterior spinal vessels were gorged with blood, and there was a fine injection of the arterioles of pia and dura. A few adhesions between the pia and dura were noticed behind, but many throughout the whole length of the cord in front.

Peripheral nerves: The lower end of the left sciatic in its two divisions (the internal and external popliteal) were removed; they were marked externally by several distended blood-vessels. On the right side this was not so noticeable as on the left. The second, third, and fourth digital nerves of the dorsum of the right foot presented nothing abnormal, but slight pressure at the end with forceps caused some blood to appear. The same was true of the second and third digitals of the dorsum of the left foot.

Muscles: A piece of muscle was excised from the lower end of each semi-membranosus. To the eye it presented nothing abnormal.

Thoracic and abdominal viscera: Careful examination showed the lungs to be crepitant throughout and apparently entirely normal. The pleural cavities were free from effusion. A few adhesions were present on the right side, and numerous adhesions on the left, many of which were evidently quite old.

So far as could be determined by careful naked-eye examination, the heart and aorta, intestines, liver, and kidneys were normal.

The stomach was normal in size, but here and there, under the mucous membrane, were seen small extravasations of blood. The spleen was twice its normal size, but of firm consistence, and its capsule normal. The uterus and its appendages were normal in size, shape, and position. The endometrium was somewhat thickened, and of a dark red color; there were considerable submucous extravasations of blood.

Marked evidence of recent moderate, adherent peritonitis were found. This was particularly noticeable on the surface and between the coils of the small intestine. At no place did the layer of lymph exceed the thickness of a sheet of paper, and the peritoneal cavity contained no liquid effusion.

A small drop of purulent-looking substance from the thick exudation covering the spinal cord was examined microscopically by Dr. Daland, and showed numerous leucocytes imbedded in a gelatinous-like substance, probably lymph, apparently there was not so much pus as would be expected from the marked yellow color.

CASE II.—N. W., aged eleven, daughter of Mrs. W. (Case I.), was of a nervous disposition inherited from her mother.

Three days before her mother's death the child was taken suddenly ill with pain in her left leg below the knee, particularly severe around the ankle-joint. Slight redness and swelling were perceived about this joint.

Tincture of opium and sodium salicylate were prescribed, and on the following morning the leg was much better, but her neck was painful and drawn to the left side, from contraction of the sterno-cleido-mastoid muscle. There was tenderness along the spine from between the shoulders to the occiput. She did not suffer with headache and had no delirium. The special senses remained normal. The bowels were constipated. In the evening a slight rise of temperature was noticed, but her pulse was slower than normal.

Under ordinary circumstances the case would probably have been dismissed as an ordinary one of rheumatic or neuralgic torticollis, but the patient's mother was lying up stairs with an undoubted attack of cerebro-spinal meningitis, which had commenced in a similar though more violent manner. This aroused suspicion as to a similar cause for the two attacks. An eruption was looked for, but did not appear until the fourth day, when five spots, two pink and three purple, made their appearance on the legs and lower part of the abdomen.

The tincture of opium and sodium salicylate were continued in moderately large doses. Friction and counter-irritation were used upon the neck, but the contraction and tenderness on pressure remained in a marked degree for several days, and did not finally disappear for several days longer.

CASE III.—M. M., aged twenty-four, a mill-girl, had a history both in herself and in the female portion of her family, of a highly nervous temperament. On the 16th of January, while enjoying her usual health, she had gone to her work. During a passionate debate among the employés as to the avisability of a strike, which she opposed, she proceeded, with the others, to the sidewalk, laboring under great mental excitement. The day was very cold, and shortly after reaching the windy street, she fell to the pavement screaming with a pain in her neck. She was taken to the house of a neighboring physician, who gave her two hypodermatic injections of morphia, after which she was brought home. At first she complained of nothing except the intense pain in the neck. The head was drawn backward. She had no fever; pulse 60. The day following her neck was about the same, while an intense headache of a constricting character was added. The mental faculties were clear and all the functions of the body were well performed. Fever was still absent, and pulse 60.

After the use for two or three days of morphia, bromide, and chloral, without the slightest improvement, hysteria was suspected, but later a genuine organic disease seemed manifest. At about the end of a week she complained of great pain in both legs, and especially upon pressure over the nerves. Dr. Mills saw her at this time in consultation. While the pain in the legs continued the headache and neckache were greatly lessened. Morphia, sodium salicylate, and oil of gaultheria were given, under which the pain in the legs disappeared. The headache never returned with its first intensity, but an active delirium took its place. Illusions and delusions kept her in a state of excitement for days with scarcely any sleep, in spite of large doses of anodynes. The drawn state of the neck was persistent. Fever come on, although never nigh. The pulse continued slow.

The patient drifted from bad to worse. The active delirium gave place to a heavy stupor, when it was difficult to administer either medicine or nourishment; but as nourishment was considered the most pressing need, it was regularly and persistently forced upon the patient. A flea-bite eruption appeared on the arms, face, and neck, but not until after the second week. The eruption would disappear and reappear, but never so distinctly as at first.

For three weeks longer, or to the fifth week of the sickness, the patient's mental condition remained practically unchanged. Iodide of potassium, 5 grains and bichloride of mercury $\frac{1}{45}$th grain were given three times a day. In three days the girl's mind was perfectly clear, and she remembered her sickness only as a disagreeable dream.

CASE IV.—R. L., aged nineteen, a mill girl, was taken suddenly ill while at her work upon the 1st of March. An intense headache followed by chills ushered in a high fever. Her temperature was 103.5° the first evening. She complained of pain in the limbs and neck, but there was no contraction of the muscles of the neck until March 3d. Nausea and vomiting were present during the first three days, and during the nights of the same days the patient was delirious.

Pills of opium and quinine, with an effervescing fever mixture, reduced the temperature by March 4th to 100° F., above which it never again rose. From the 4th to the 9th the case remained at a standstill. The headache was greatly better, but the contraction of the muscles of the neck was increased, fixing the head immovably backward. Hyperæthesia and deep-seated pain upon slight pressure were present over the whole posterior region of the neck. The patient complained of feebleness and exhaustion. On the 6th, the opium and quinine were given at longer intervals, and iodide of potassium 5 grains, and bichloride of mercury $\frac{1}{48}$th grain, four times a day.

In two days the patient was sitting up, with fever gone, neck greatly relieved, and appetite and sleep returned. Her convalescence was from this date forward rapid and complete. No spots were found in this case.

Case V.—Notes of the following case were furnished by Dr. J. V. Kelley, of Manayunk, Philadelphia :

The patient, a young man, was first seen February 18, 1888. He was in good health until the preceding day, when in the evening he was taken with a chill, vomited twice, and had two stools. He worked during the forenoon of the day he was taken sick, although feeling very badly. At 2 P. M., when first seen by Dr. Kelley, he was in bed and complained of great soreness. He complained of tenderness confined to the abdomen, so much so that the case was at first thought to be one of enteritis. He was very cold; pulse 134, and he seemed much prostrated. He was placed on brandy and tonics, and the next day in a manner revived.

February 20th, he was seen by Dr. Bruen, who thought the case to be one of cerebro-spinal fever. Herpes labialis was beginning to show. The next day he had some delirium ; his pulse was still 134.

On the 22d, he was seen by Dr. Pepper, who agreed that the case was one of cerebro-spinal fever. Herpes labialis was now profuse, some delirium was present, and hearing and sight both defective. He had two offensive stools, and complained of great general soreness, but had no characteristic spots, although there was a vesicular eruption on the chest. He had no retraction of the head, although his neck was somewhat stiff. His abdomen was tympanitic, and this persisted until death occurred, February 28th.

About the fifth day of his seizure the patient complained of blindness. On one occasion he bled from the nose and spat some blood. No heart or lung symptoms could be demonstrated by examination, and the man died from prostration on the eleventh day of the disorder. He showed typhoid signs, but the case was certainly not typhoid fever. The treatment was by stimulation and tonics, with full feeding. Brandy, quinine, digitalis, opium, and turpentine were given.

Case VI.—The following interesting case occurred in the practice of Dr. J. W. Dick, and was seen by Dr. Mills several times in consultation. The patient, R. A., a boy eight years old, in the latter part of October, 1887, had an attack of mild scarlet fever, followed by slight nephritis, which kept him in the house until the last of December. After this time he began running out of doors in all sorts of weather, in his play sometimes lying down in the snow. He seemed to be in the best of health until February 2d, when he was suddenly taken sick at school. He came home complaining of violent headache, and was taken down also with vomiting and high fever, with marked delirium. The delirium soon subsided, and he sank into a semi-conscious condition. He would not answer, as a rule, any question or demand, but sometimes asked for a drink, and he would give notice when he wished to evacuate the bowels or bladder. On the fourth day an eruption appeared over the entire body, more especially marked on the face, neck, and upper extremities. The eruption consisted of small pinpoint, fleabite-like spots, which did not disappear on pressure. It faded in about four days after its first appearance.

His temperature varied at first from 103° to 105° F., sometimes higher in the morning than in the evening, and sometimes the reverse. The following is the temperature record from February 17th to 26th :

February	Morning.	Evening.
17th	104.4°	104° F
18th	104.4	103.8
19th	103.6	103.6
20th	102.6	103.2
21st	102.4	102.6
22d	102.4	101.6
23d	102	102
24th	101	101
25th	101	101
26th	101	101

After this his temperature showed a tendency to fall, and came down to 95.5°.

His pulse was weak, but regular, varying usually from 112 to 120. His respirations ranged from about 30 to 40. The vomiting lasted only during the first day, after which he took and retained both food and medicine. His bowels were usually slightly constipated, but responded freely to enemata. After February 22d he did not attempt to speak, but lay in a comatose condition, with frequent sudden outcries, as if in pain. His abdomen became decidedly scaphoid. He was troubled with a cough throughout his entire sickness. There were abundant mucous râles, with a little dulness on percussion on the right side.

In the beginning of his sickness the slightest movement gave rise to intense pain. This gradually subsided ; but he continued throughout to have great tenderness on pressure in the region of the neck and in the legs. The pain in his lower extremities was a very marked symptom ; handling them, and pressure along the nerves or squeezing of the muscles, caused great suffering. No swelling of the joints was present. He exhibited some pain and tenderness in the upper extremities, but not nearly as marked as in the lower; the knee-jerks were retained. He could move the legs up and down freely, but the feet had a tendency to assume the equino-varus position ; this foot-drop was more marked on the right side. No paralysis of the cranial nerves was present. For three weeks before his death he was apparently both blind and deaf; for a short time he had a slight discharge from the left ear. He died on the thirty-sixth day.

REMARKS.—These cases are of considerable interest from various points of view, but we shall only be able to call attention to a few of the most important features. At a former meeting of the Society it had been suggested by Dr. Mills that neuritis was probably present as a complication or coincidence in some of the cases, and in others the infectious agent caused a multiple neuritis rather than a cerebro-spinal disease. As no microscopical examination has yet been made from the specimens of Case I., we are not able to say positively that neuritis was present, but the gross examination of the sciatic and other nerves led to the suggestion that either congestion of the nerves or neuritis was present.

A few words might be said with reference to treatment. Bromide of potassium, in half-drachm doses, failed to relieve the headache or produce sleep in the severe cases (the same result followed chloral). Nothing definite can be said as to the effect of quinine. Sodium salicylate and oil of gaultheria gave relief to the neuritic pains in the legs, but produced no appreciable benefit to the head and neck. Opium and morphia did positive good in every case, but after the more acute symptoms had passed the good effect seemed to be lost. In the two cases where iodide of potassium and bichloride of mercury were used by Dr. Cahall, the effect was surprising to patient and physician alike. The improvement was too rapid to be the result of the alterative properties of the drugs, but more like the action of specific remedies.

A CASE OF PARAMYOCLONUS MULTIPLEX.

Reported by FRANK R. FRY, A. M., M. D.

Clinical Lecturer on Diseases of the Nervous System, St. Louis Medical College, Consulting Physician to the St. Louis City Hospital on Diseases of the Nervous System.

Minnie R., æt. 30, single, sewing-machine girl, living at 2009 S. 12th St., St. Louis, applied to the Clinic of the St. Louis Medical College, January 9th, 1888, stating that she was troubled with a shaking and jerking of the extremities, especially of the lower.

As she sat in a chair the heels of her shoes kept up a great clatter on the floor. I grasped one of the knees, thinking to hold the foot to the floor and thus stop the shaking or tremor. I was surprised to find that all the force I could command was not sufficient to do so. I felt of the lower extremities under the clothing and found the muscles of the thighs in a condition of marked clonic spasm. With intervals of a few minutes, this peculiar spasm repeated itself a number of times during the short period she was before the class. I saw her again the same day, when she had a more severe attack. She made a determined effort, at my request, to restrain the movements. Not only was she unable to do so, but the effort caused her a very appreciable general fatigue. At this second interview there was an involvement of the muscles of the right shoulder and arm, consisting of occasional rapid abduction and adduction of the arm that disappeared after several jerks of some of the shoulder group of muscles.

Late in the afternoon of the following day, at her home, I witnessed her have an attack, of which the following is a description: The first intimation of it were several deep, sighing inspirations, immediately followed by a violent spasm of the flexors and extensors of the thighs, causing them to be thrown rapidly up and down, so that, as she sat in a chair, the feet tramped the floor with much force. Her mother and sister at once assisted her to a large reclining-chair, and placing a pillow on another chair, lifted her feet onto it, remarking as they did so that she hurt her heels in the hard attacks unless they were thus protected. She was immediately seized with another violent paroxysm. Her lower extremities were thrown up and down as she lay in a semi-reclining position so that the heels struck the pillow with much force and, except for the protection that it afforded, would certainly have been much bruised. Her body was jostled about in the chair by violent contractions of the gluteal and other muscles of the pelvis and thighs, and those of the back. With all the strength I could put forth, I was unable to hold either one or both of the extremities down on the chairs. During the attack I rapidly loosened and removed most of the clothing from the upper part of the body, and passing my hand over the muscles of the abdomen, back and shoulders, I felt them at different times in a condition of clonic spasm. There were, every few moments, violent movements of the respiratory muscles, causing the respiration to appear distressed ; but she at no time complained of difficulty in getting her breath. Occasionally the spasm of the arms, especially of the right, was quite as rhythmic and almost as violent as that of the thighs. The seizure lasted, in varying severity, for about ten minutes, passing away gradually with occasional jerks of some of the muscles of the thighs and arms. When it was gone she was much exhausted, saying that if we would let her alone she could drop off to sleep.

During the whole attack, I saw no spasm of either leg or foot, forearm or hand. I watched this point very carefully, having refreshed my mind on the characteristics of paramyoclonus multiplex by reading again, before I witnessed this attack, the article of Dr. M. Allen Starr, in the *Journal of Nervous and Mental Disease*, July, 1887. There was no distinct spasm of the muscles of the neck or face ; but toward the end of the attack there was a tremor of the jaw which I attributed to her exhausted condition. She had had during the day five or six such attacks as the one just described.

Dr. Henry W. Hermann, Professor of Diseases of the Nervous System in the St. Louis Post-Graduate School of Medicine, saw the case several times, and presented it to his class. I have asked him to furnish a description of one of the attacks, that I might present it along with my own. This he has kindly done as follows, it being a description of an attack of moderate severity: "While sitting in a chair the patient executed a tramping movement with her feet of considerable rapidity, bringing them down alternately, and toward the close of the attack, simultaneously. With a few slower kicks, then with a few jerks in the right arm and a few deep inspiratory sighs the attack ended, lasting a few minutes, to begin again in a little while. She evidently had no control over the movements, and felt very much exhausted after them. There were no symptoms of hysteria. The muscles implicated were those of the hips and thighs, those of the right shoulder and arm also participating slightly. Only once did I see a slight flexion in the hand. The left arm was quiet, and only rarely participated, I was told. There was no disturbance of sensibility. No paralysis except slight weakness on account of the exhaustion. The patellar tendon reflex was exaggerated. Co-ordination good, and the mind clear."

The patient's statement, recorded January 12th, 1888, is as follows: Her health has always been excellent. She never has suffered from headache or any other form of nervous trouble. Her menstruation has always been normal and regular. The family history, gained from the patient and her mother, is unimportant. She has been continously engaged in running a sewing-machine for the past twelve years, most of the time on heavy work, much of the time averaging ten and often twelve hours a day. Several years ago she formed a habit, which she has continued, of placing the left foot in front of the right on the treadle. On account of this position the left extremity did most of the work. (Until recently the attacks almost invariably began in the muscles of the left thigh). About October 1st, 1887, she had the first attack. While at work her lower extremities were suddenly seized with a jerking. Then followed an attack of the usual description, that lasted several hours, leaving her much fatigued. She arose on the following morning feeling very tired, but went to her place of employment and worked all day. She had no more attacks for two weeks, when she again had a hard one. From this time she had them occasionally, the intervals of time between them constantly lessening. She continued at work, losing an occasional day or two. About the 20th of December she had the most violent attack she has ever had. Since then she has not been able to work, except on portions of one or two days. The attacks have continued to come every day, and on many days frequently. Excepting a general lassitude she feels perfectly well and comfortable when free from attacks. On Christmas day a severe attack seized her when standing, and she almost fell before she could grasp a support. This is the only occasion on which she has come so near falling. She always feels a slightly distressing, drawing sensation at the pit of the stomach, and a general weak, slightly faint condition that prompts her to immediately sit down before the jerking begins. She says the attacks come harder if she is excited, annoyed, or hurried. After them she feels much exhausted, and at times in this condition, cannot resist a crying spell. In some instances there is an almost irresistible desire to sleep following a hard attack.

Present condition, April 6th, 1888. The severity and number of attacks have very gradually but almost uninterruptedly diminished. During the past week she has had three seizures of considerable severity, the only ones of the kind for five weeks. Prior to this she had gone three and four days without any attacks at all. Her general health is not as robust as before the attacks began ; but she has lost only slightly in weight, eats and sleeps well, and when free from attacks feels perfectly well.

She has taken hyoscyamine, chloral, bromides, morphine and antipyrin, separately and in various combinations.

Chloral seemed more effective than any other remedy used. Hyoscyamine evidently had some effect in arresting the attack when first employed. Antipyrin seemed to have an equal effect, that continued longer. The bromides seemed useless or nearly so. Morphine was used but little, and in combination with some of the other remedies. She has also received several courses of arsenic, each time continued until decided evidences of its constitutional effects were present.

The patient is of medium height, well nourished, with an unusually good muscular development, especially of the lower extremities. There is no evidence of organic disease of any description. There is an increase of galvanic and faradic excitability of the muscles of the extremities, especially of the lower; no qualitative changes. There is an exaggerated knee-jerk, and occasionally a decided ankle-clonus. There are no disturbances of sensation, or co-ordination. There are no evidences of hysteria.

The patient has been under my observation since January 9th, 1888. I have seen her have many attacks varying in severity from the one described above to a slight tremor of the lower extremities, with an occasional jerk of them and of the shoulders. Twice only I have seen slight spasm of some of the muscles of the right fore-arm. Dr. Hermann also observed this in one of the attacks that he saw. According to her own statement it has occurred very seldom. Until the present week I had never seen any spasm of the leg, when I found, in one attack, the muscles of the left calf in active clonic spasm. She called my attention to the fact and stated that it had happened for the first time two days before; and she was much distressed over the fact of the "jerking" coming in a new place. I have never seen any involvement of the neck or face. The attack always began with a rapid, rhythmic movement of the thighs. I have always been able to induce an attack by a sharp blow on the thighs, or on the patellar tendon, or often by several quick dorsal flexions of the foot. Twice she has had hard attacks immediately on getting into a cold bed.

Note, May 12th.—More than three weeks ago I began in this case a methodic course of galvanism, which prior to then had been impracticable. This has been continued to the present time. It has consisted of a séance of five to fifteen minutes every other day, with the anode (a 9x10 cm., sponge-covered electrode) on the neck and the mobile cathode (4x4 cm.) down the back and all the extremities, with a current of 10 to 15 ma. She has taken no medicine at all since the above treatment was begun. She has improved very rapidly during the last two weeks, occasional fibrillary contra ctions being, now, the only evidence of the presence of the trouble.

EDITORIAL NOTES AND MISCELLANY.

THE AMERICAN NEUROLOGICAL ASSOCIATION.

Preliminary Programme of Papers to be Read at the Special Meeting to be held in Washington, D. C., September 18th, 19th, and 20th, 1888.

Dr. Robert T. Edes, of Washington, D. C., will read a paper on "The Relation of Renal Diseases to Diseases of the Nervous System." This subject will be discussed by Dr. F. X. Dercum, Dr. Leonard Weber, Dr. E. C. Seguin, and Dr. L. C. Gray.

Dr. B. Sachs, of New York, will read a paper on "Muscular Dystrophies." This subject will be discussed by Dr. P. C. Knapp, Dr. G. W. Jacoby, Dr. W. R. Birdsall, Dr. L. C. Gray, and Dr. C. K. Mills.

Other members are cordially invited to participate in these discussions.

The Council requests that, so far as practical, the remarks offered in these discussions shall be in writing and shall not occupy more than ten minutes to read.

The Secretary will furnish, on application, a brief statement of the manner in which the readers intend to treat their subjects.

The following papers will also be read:

Post-hemiplegic Disturbances of Motion in Children. By Philip C. Knapp, M.D., of Boston.

Heat Centres in Man. By Isaac Ott, M.D., of Easton.

The "Ape-Fissure," so called, in Man. By Burt G. Wilder, M.D., of Ithica.

Clinical Report of Cases of Epilepsy following Cerebral Hemiplegia. By E. D. Fisher, M.D., of New York.

The Relation of Urinary Changes to Functional Nervous Diseases. By C. L. Dana, M.D., of New York.

The Differential Diagnosis between General Paresis, certain forms of Intra-cranial Syphilis, and the Cerebral Type of Disseminated Sclerosis. By Landon Carter Gray, M.D., of New York.

Report of a Case of Primary Lateral Sclerosis with late Cerebral Symptoms due to Cyst of the Floor of the Lateral Ventricle, with Microscopic Report. By Wharton Sinkler, M.D., of Philadelphia.

A Case of unusual form of Myxœdema, illustrated by photographs and sections of excised tissues. By F. X. Dercum, M.D., of Philadelphia.

A Case of Alcoholic Multiple Neuritis, with Sections. By James Hendrie Lloyd, M.D., of Philadelphia.

Observations and Experiments respecting the Pathology of Neuritis. By James J. Putnam, M.D., of Boston.

Dr. C. K. Mills and others will demonstrate specimens.

Myositis Subcuta Progressiva. By Geo. W. Jacoby, M.D., of New York.

The Differential Diagnosis between Peripheral Neuritis and those Cerebral Affections with which it is most likely to be confounded. By E. C. Spitzka, M.D., of New York.

Nervous Affections following Injury. By Philip C. Knapp, M.D., of Boston.

Hereditary Chorea. By Wharton Sinkler, M.D., of Philadelphia.

Aneurysm of an Anomalous Artery causing Antero-posterior Division of the Optic Chiasm and Bitemporal Hemianopsia. By S. Weir Mitchell, M.D., of Philadelphia. To be read by F. X. Dercum, M.D., of Philadelphia.

A Case of Focal Epilepsy, with Observations on Trephining. By James Hendrie Lloyd, M.D., of Philadelphia.

The preliminary report of the Committee on Encephalic Nomenclature will be read by Burt G. Wilder, M.D., Chairman, of Ithica.

GRÆME M. HAMMOND, M.D., *Secretary*,
58 West 45th St., New York.

JAMES J. PUTNAM, M.D., *President*,
106 Marlboro St., Boston, Mass.

In the February number of the JOURNAL, under the heading of "Electricity and Neuropathy in the Treatment of Cancer," Dr. Hughes is credited with being the first and only physician who has ever proposed to treat cancer with electricity. The subjoined letter from Dr. W. W. Wood, U. S. A., shows that we were in error.

FORT WALLA WALLA, WASH. TERR., June 3, 1888.

MY DEAR DR. HAMMOND:

The statements on pp. 155–6 in No. 2, Vol. XIII., JOUR. NERV. AND MENT. DIS., in regard to Dr. Hughes' proposition to use electricity in the treatment is calculated to mislead those who do not know the inaccuracy of "*to Dr. Hughes belongs the credit of having suggested the electric treatment*." So far as I am aware no one had, of record, treated a case of uterine cancer by electrolysis, properly so-called, when on Nov. 15, 1886, in a case reported *Dec. 27, 1886*, I used a current of 35 milliamperes for fifty minutes—current furnished by a McIntosh 18-cell battery, and measured by an Edelmann's Einheits galvanometre. I have since that time used electrolytic galvanic currents in cancers more than 225 times, with great benefit to my patients, in doses of from 15 to 225 milliamperes, with variable duration of seances.

I have no desire to detract from the well-earned reputation of Dr. Hughes, but "render unto Cæsar * * * *."

I trust that you will make the necessary correction.

Very truly yours,
W. W. WOOD,
Capt. and Asst. Surg'n U. S. Army.

DR. GRÆME M. HAMMOND.

PERISCOPE.

By DRS. G. W. JACOBY, N. E. BRILL, AND LOUISE FISKE-BRYSON.

PHYSIOLOGY OF THE NERVOUS SYSTEM.

BINET AND FÉRÉ ON THE PHYSIOLOGY OF MOVEMENTS AMONGST HYSTERICAL PERSONS.

The researches in this paper have been chiefly carried out on three points ;—1. Cataleptic plasticity of the waking state ; 2. Involuntary and unconscious movements ; 3. Voluntary movements.

1. *Cataleptic Plasticity.*—This phenomenon is produced in hysterical subjects, who present anæsthesia of the skin and muscular sense. The eyes are bandaged, the anæsthetic member is raised, the subject, who has lost the consciousness of passive movement, being unaware of it ; in the majority of cases member drops. In other cases, however, it remains in the position in which it has been placed. The chief characters of the phenomenon are as follows :—(1) The duration of the position is extremely lengthy ; in one instance the right arm was horizontally extended, and the fore-arm slightly flexed. They remained in this position for an hour and twenty minutes. (2) There is a complete absence of trembling in the member. (3) Fatigue is absent. (4) The plasticity of the members does not entirely suspend voluntary movements. (5) After the member has dropped there is no evidence of paralysis ; in fact, in one case, the dynamometer gave a higher figure after than before the experiment. When the conditions under which these phenomena take place are examined, it is found that they are always accompanied by superficial and deep

anæsthesia, or sometimes only by the superficial form. Anæsthesia to fatigue is also a necessary condition. Closure of the eyes is not essential. It is interesting to remark that subjects of this kind are able voluntarily to extend the anæsthetic member during a much longer period than the normal member. Temporary disappearance of the anæsthetic suppresses completely in that part of the body the power of preserving voluntarily a position during a considerable time.

2. *Unconscious Movements.*—These movements were observed in eight out of sixteen patients who were experimented upon. The seat of the movements is in those parts of the body which are anæsthetic. We may classify the results obtained under various headings.

Repetition of a Passive Movement.—The eyes of the subject are bandaged, and the anæsthetic arm is caused to execute rapidly or gently a regular movement. In the midst of its course the limb is suddenly abandoned, when the movement is seen to continue during a certain time, which varies in different individuals. The same result may be produced by *faradic* contraction, and also as the sequence of a *reflex* movement.

Repetition of a Graphic Movement.—In this case a pencil is placed in the hand of the patient, which is caused by the operator to execute some movement, such for instance, as one of a circular nature. Whilst this is being done the hand of the patient is observed not to be completely passive. The passive movement having been communicated, the hand of the patient is released, the point of the pencil being left on the paper. In one group of cases the hand at once falls to the side, in a second the hand still holds the pencil as if about to write, but nothing more than a slight trembling occurs. In a third group of cases, however, the hand continues to execute the movement which has been communicated to it for a period, which has been known to extend to a quarter of an hour. Complicated words, composed of five or six letters, are sometimes reproduced with exactitude.

Repetition of a Voluntary Movement.—This occurs notably in movements of writing. The patient, having voluntarily written with opened or closed eyes the same letter several times in succession. is told to stop. He believes that he has stopped, but the hand continues to write the same letter. A facsimile of a letter written by a patient is given, in which it is obvious that she was obliged, in spite of herself, through a kind of stammering of the hand, to write several times in succession the same letter.

Association of Unconscious Movements.—An unconscious movement determined by the observer may produce a second unconscious movement with which it has been generally associated; thus, for instance, when a perfectly anæsthetic subject, who is seated with closed eyes, is taken by the arm, he may be caused to rise by the associated movement in the lower extremities.

Unconscious Movements of Adaptation.—These may also be produced in hysteric subjects.

Unconscious Rendering of a State of Consciousness.—It often happens that when a patient whose eyes are closed is told to think of a figure or a word, the anæsthetic hand which holds a pencil traces unconsciously the figure or word which is in the mind.

Spontaneity of Unconscious Movements.—It has been a question as to whether the unconscious movements are simply the manifestations of an unconscious memory, or if other functions, reason, for example, may intervene sometimes in the operation. The experiment was made of causing a patient to write his own name misspelt. In some cases the unconscious repetition corrects the error; in others it is allowed to remain incorrect. The most striking effect in connection with all these manifestations is that the hysterical anæsthesia is nothing less than an anæsthesia. The patients on whom these observations were made have all lost the consciousness of the passive movements, but only the consciousness; the physiological process of sensation is preserved. The unconscious sensation is registered in the nervous centres of the patient, and thus the same movement is unconsciously reproduced. Without being infalible the unconscious perception of the hysterical person appears to be much more exact than the conscious perception of the normal individual. If the hand of the subject is touched during the writing it will be perceived to be extremely rigid, and any attempt to stop the movement meets with considerable resistance. The will of the patient has also no effect on the movement. Whilst the passive movement is in progress the patient may be permitted to open his eyes. He is then much surprised at seeing his members in movement, and his surprise is increased when he observes that he is incapable of stopping the movements whose cause he does not understand. When the patient, in the course of a passive movement, is asked to write a second word, a curious mixture of the two words is produced. The authors offer the following interpretation of the preceding observations. They seem to prove that there exists in certain hysterical persons in a waking state a doubling of the personality, which is not successive but simultaneous, both being *en rapport* with the operator, but in different ways. The prinpersonality, which is that which everybody knows, communicates by a word or by voluntary movements ; the second personality, which is more or less rudimentary, communitates with the experimenter principally by movements of the anæsthetic side.

Amongst certain hemi-anæsthetic or completely anæsthetic subjects, every motor phenomenon provoked in one-half of the body produces an analogous but feeble phenomenon localized in the corresponding portion of the opposite side. In the case of a contraction, for instance, a short interval exists between the movements of the two sides. The indirect contraction is generally more feeble, is sometimes incomplete, and occasionally a prolonged excitation of the one side is necessary to produce an action in the opposite. Faradic contractions, reflex, passive, and active movements, may also become unilateral. Paralyses are an exception to the rule which governs all these bilateral motor phenomena. Instead of a paralysis suddenly provoked being produced on the opposite side, an accession of force as shown by the dynamometer is the result.

3. *Voluntary Movements.*—The majority of hysterical subjects who have lost superficial and deep sensibility preserve the faculty of coördinating the movements writing with their eyes closed. Those whose insensibility is profound agreeing in saying that they do not feel themselves write. They almost all concur in stating that they see themselves writing. Some patients, and these form the majority, are incapable of writing exactly a letter any given number of times ; they either write it too often or too seldom. Others, on the contrary, obtain the exact number. It appears that both these classes and especially the former write chiefly under the guidance of their visual memory.—*London Medical Record.* L. F. B.

THERAPEUTICS OF THE NERVOUS SYSTEM.

CASCARA SAGRADA IN RHEUMATISM.

The effect of cascara sagrada in rheumatism I discovered by accident. About three months ago I was attacked with severe rheumatic pains in my shoulder, the slightest motion causing intense pain. The third day of the attack I commenced taking as a laxative ten drops of the cascara, t. i. d. The first morning after taking it the pains were so much less severe that I could move my arm freely. The day following I was entirely free of all discomfort.

Although, as I have intimated, I had not taken the cascara with any idea of relieving the rheumatism, it occurred to me a few days later that possibly the sudden subsidence of pain might have been due to the drug. There being a few cases of rheumatism in the wards, I determined to try to verify my suspicions. Discontinuing the salicylates, iodides, etc., which these patients were taking, I substituted ext. cascaræ sagradæ fl., 1 c. c., t. i. d. The result astonished me. Within twenty-four hours there was marked improvement in every case. One case is especially worthy of notice. The patient was a Swedish sailor who had been admitted three months previously. He suffered intensely, and, although almost everything had been given from which relief might be expected, his suffering was not allayed. For a day or two after admission he improved on large doses of salicylate of sodium, but subsequently the pains returned as badly as ever, and the salicylate had no further beneficial effect. Iodide of potassium was given several different times, but, owing to an idiosyncrasy, could be continued only two days at a time, a profuse rash making its appearance over the patient's entire body, the pains remaining as acute as ever. They were not confined to any two or three joints, but felt in all, being more severe, however, in the wrists, finger joints, and ankles, all of which sometimes became œdematous. On the evening of February 5th I commenced the exhibition of fifteen-drop doses of cascara sagrada three times daily. The following morning he was about the same ; the second day he was much better ; on the seventh he was so far recovered that he asked and obtained permission to walk out. From this on he continued to improve steadily, and on the 17th of February was discharged recovered.

I have since used the cascara in fully thirty cases, some ten of which were in out-patients, and, with the exception of three or four in which there was a syphilitic taint, I have obtained the most satisfactory results. I commenced with 1 c. c., t. i. d., and have so far never had to increase it beyond 1.5 c. c., and even to this extent in but two cases. I have seldom had to wait beyond twenty-four hours for beneficial effects. In two cases I had to stop it temporarily owing to its opening the bowels too freely. In such cases I would suggest that one of the preparations of iron be given (separately) at the same time. I usually combine it with syrup or glycerine in equal parts, and instruct the patient to take from thirty to forty drops in water. In one case in which neither it nor the salicylate of sodium appeared to give much benefit I combined the two with good effect. It is but seldom the bowels are opened too freely by it, the cases referred to being the only ones I have so far observed.

Among the out-patients upon whom I have used it were two intelligent officers of vessels. One was an old river pilot who had periodically suffered intensely for years. I gave him equal parts of the cascara and syrup, of which I instructed him to take 2 c. c., t. i. d., and requested him to see me again in three days. He returned a month later, and then only to get the medicine renewed. He reported that he had never before had anything relieve him so quickly. The pains began to abate within twenty-four hours after taking the first dose, and in three days after left him entirely. He had had no return, but, for fear of another attack, had come to ask for a bottle to keep with him.

The second case was that of Mr. R., first clerk on a large river steamer. He was suffering so much with pain in the hip-joint and thigh that he could scarcely get to the office. I put him on large doses of salicylate of sodium, with colchicum and iodide of potassium, and instructed him to return in a day or two. In a week he sent a friend to say that the pain, instead of lessening, was so severe that he could not get to the office. The salicylate, etc., were stopped and he was given cascara syrup, thirty-five drops, t. i. d. This was on Friday afternoon. On Sunday he came to the hospital and reported that he had commenced taking the second prescription Saturday morning, and that on Sunday he had felt decidedly better. He was ordered to continue the drops, and report on Wednesday. Tuesday he sent word that he should be unable to report, as he was sufficiently recovered to resume his usual place on the steamer.

I am not able to explain the action of the drug in relieving rheumatism ; I leave that to other observers. I write this in the hope of inducing other medical men to use the cascara, report their experience, and indicate, more particularly, in what class of cases they have found it of most benefit. (H. T. Goodwin, M.D., N. Y. Med. Jour., June 9, 1888.)

VOL. XIII. July. 1888. No. 7.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

THE OCULO-MOTOR CENTRES AND THEIR CO-ORDINATORS.

BY E. C. SPITZKA, M. D.

Address delivered before the Philadelphia Neurological Society.

I KNOW of no subject within the domain of neuro-anatomy, whose consideration is at the present moment so well timed, being replete with clinically suggestive facts, as that of the cell-nests which are connected with the third, fourth and sixth cranial nerves, together with the inter-nidal tracts which unite them in automatic co-ordination. Recent researches have brought our knowledge up to a point of almost ideal exactitude. Inasmuch as my collection of specimens enables me to illustrate several of these points, I have selected this subject in response to the kind invitation of your council.

The intrinsic anatomy of the cell nests of the fourth and sixth nerves is comparatively simple, and I shall refer to these only in so far as they are involved in the binocular mechanism. That of the third pair is more complex. There is strictly speaking no oculo-motor nucleus, in the sense in which Meynert and his followers use the term ; that is, no single undifferentiated cell nest, on each side of the median line, giving origin to the oculo-motor nerve of the corresponding side, and to nothing else. The first blow dealt this view was given by v. Gudden,[1] who found on destroying one third pair, that the ensuing nucleur elimination was not limited to one side, but involved both sides. He was thus able to distinguish *two* nidi for each oculo-motor ; one of these, representing a decussated origin, is situated meso-caudal (in the rabbit) the other, and main nest, is found lateral, and gives origin to those fibres of the third pair which remain on the same side. I have been able to confirm this result with some slight and immaterial modifications in the cat.[1] On examining more minutely, it is seen that it is the innermost and most posterior rootlets of the third pair, that cross the median line and have a decussated origin.

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[1] Ueber die Kerne des Augenbewegungsnerven. Tageblatt der 54ten Versammlung der Aerzte und Naturforscher in Salzburg, 1881, p. 186.

Now, pathological observations in the case of man[2] show that it is precisely these rootlets which carry the innervation of the rectus internus. The division of the third pair supplying that muscle must be therefore regarded as bearing a similar relation to the division supplying the levator palpebræ, rectus superior, inferior, obliquus inferior, sphincter iridis, and musculus ciliaris, which the decussated portion of the optic nerve bears to its non-decussated division. There is, however, this noteworthy inverse relation between the optic and oculo-motor nerves. The lower forms with total decussation of the optic nerves (and consequent non-identity of the retinal fields) appear to have no decussated origin for the nerve fibres supplying the rectus internus.[3] Those with a partial decussation of the optic nerves, do have such a decussated origin for the nerve fibres supplying the rectus internus. In other words, there is a parallelism in development between the decussated division of the third pair and the non-decussated division of the second pair, which is in harmony with physiological requirements, and, if I may use such a term, anatomical convenience.[1]

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[1] Neurologisches Centralblatt, 1885, p. 246. Vorlaufige Mittheilung uber einige durch die Atrophie Methode erzielte Resultate.

[2] Kahler-Pick. Archiv. f. Psychiatrie, X, p. 334.

Among the cases not generally cited which support this view, is one by Millingen, where a focus of disease situated to the right of the Aqueduct pressed close on the median line of the central tubular gray of that region. There had been during life spastic (irritative?) contracture of both internal recti. They would have to be situated near together, and mutually near the median plane, to account for this observation.

[3] I can find no indications thereof in the sea or fresh-water turtles (*Thallasochelys midas* and *Nannemys guttata*). The partial decussatian of the chiasm, the decussation of the rectus internus fibres, and the bilateral reflex reaction of the orbicularis palpebrarum go hand in hand.

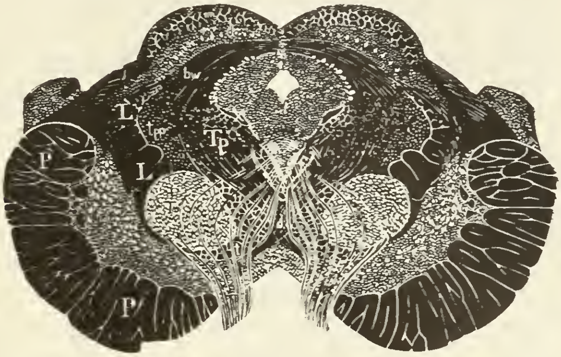

Figure I.—Transsection through human meso-cephalon, the clear spaces at the base of the aqueduct-gray represents the sub-nests of the venlo-motor nidus.

This is not the only differentiation of the nuclear masses connected with the third pair. Further cephalad near the median line there is in man, a closely crowded cell mass, with a dense molecular basis which is in most levels sharply demarcated, and in others (as far as I am able to determine individual differences exist) sends out a lateral process.[2] In caudal levels it is a vertical column parallel to the raphe; in cephalic levels the lateral extension preponderates, the vertical column shrinks and eventually it becomes a mere appendix to the transverse oval mass into which this extension becomes as it were inflated.

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[1] The philosophy of this mechanism will be considered in connection with the posterior longitudinal fasciculus of the tegmentum.

[2] Westphal, who is the first to accurately describe this sub-nidus, says that no indications can be found in any illustrations extant, of its prior recognition. In the accompanying wood-cut, which has seen service in two of the author's papers (Journal of Nervous and Mental Disease, 1879–1880, and *N. Y. Medical Record*, Oct. 25, 1884), it will, however, be seen that the distinction had been recognized, though less accurately, and without any physiological deduction, or reference in the text. See Westphal, "Archiv. f. Psychiatrie," *loc. cit.*

Westphal, who first sharply demarcated this sub-nucleus, adduces clinical and anatomical facts to prove that it is connected with the innervation of the inner muscles of the eye, that is, the ciliary or the iris sphincter, or both. Before proceeding to discuss this exceedingly plausible view, permit me to describe in detail the cell-nests, and their relations to root and other fibres, as derived from a study of three sets of trans-sections of the human isthmus, one of which was a complete one.

In order to simplify matters I will distinguish four trans-section levels, designating them respectively, *A*, *B*, *C* and *D* in the cephalo-caudal direction.

(*A*) The general contour of the aqueduct-gray is drawn ventrad into a very sharp apex. In its dorsal part it is materially encroached upon by the post-commissura. Powerful fibre bundles surround its margin,[1] occasionally breaking into its lateral contour, but mostly accumulating latero-ventrad to form the posterior longitudinal fasciculus, which in this level is extremely small as contrasted with the dimension it has in field *D*.

Cells.—The cells of this level are of intermediate dimensions, they are mainly accumulated in a single mass, which follows in contour the latero-ventral outline of the central tubular gray. They are parallel to this contour with their long axes. They do not stain as deeply in carmine as those of levels *B*, *C* and *D*. As we progress caudad they become more closely crowded, they appear smaller, but more distinctly stained, and the basis substance assumes more of the transparent gelatinous consistency, hence staining deeply in carmine.

Intra-nidal Fibres.—The medullated fibres are limited to the lateral angle of the aqueduct-gray, just ventrad of which they break into it. They appear to terminate in and around the dorso-lateral extension of the cell mass just described.

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[1] It is their existence which induced Darkschewitsch (Pfluger's Archiv. XXXVIII. and Neurol. Centralblatt, 1885, No. 5, p. 100), to predicate a connex between the post-commissura and the oculo-motor nuclei, particularly regarding it as a path for the light-reflex, a view which is opposed by the excellent development of this commissure in animals with rudimentary eyeballs, and the absence of any lesion in it, in cases where there was reflex iridoplegia. (Moeli, Arch. f. Psych., XVIII., p. 31.)

Posterior Longitudinal Fasciculus.—This important bundle can be distinctly seen to be built up by arched fibres coming from dorsad, circling round the lateral angle of the aqueduct-gray, ventro-mesad. They have the same derivation, apparently, as the intra-nidal fibres of this level.

Oculo-motor Rootlets.—These are as yet few, and in the cephalic sections of this level entirely absent. The most cephalic rootlet of the third pair, discoverable in a perfect series of sections from man is directly derived from the most mesal bundle of the posterior longitudinal fasciculus. It is the most cephalic and at the same time a mesal rootlet of that nerve, which has this origin. It is in caudal levels joined by fibers derived apparently from the nidus of cells above spoken of.

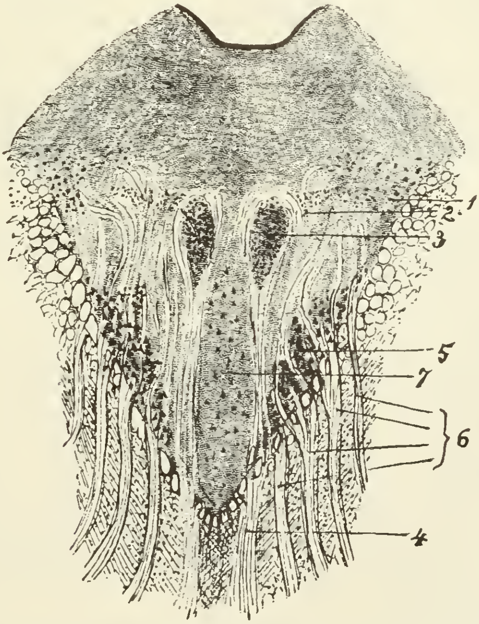

FIGURE II.– Transaction in second fifth of human oculo-motor nidus. 1. Lateral division of main nidus; probable center of levator palpebrae. 2. Fibres ectad of Westphal's nidus. 3. Westphal's nidus. 4. Emerging rootlets of III. pair, from fibres surrounding and entering that nidus. 5. Main portion of main nidus. 6. Rootlets of III. pair thence emerging. 7. Sagattil nidus.

(*B*) As we proceed caudad, the beautiful circular dorsal contour of the aqueduct-gray becomes established, the post-commissura no longer intruding. A lateral angle becomes more prominent, and the transverse diameter of the area becomes greater. At the same time the ventral prolongation becomes shortened. The demarcation is more distinct, the posterior longitudinal fasciculus forming its boundary even to and through the raphe.

Cells.—Two distinct nests can be recognized; one bordering directly on the posterior fascicle. The other, as yet the larger, corresponding to Westphal's nidus, is more mesal and slightly dorsal. Its cells are smaller, closely crowded, and become more so the more caudal we pass. The lateral mass is of larger cells. It grows larger as we pass caudad. I shall designate them respectively as Westphal's and the main nidus.

(*a*) *Westphal's nidus.*—From being parallel to the lateral contour of the ventral half of the aqueduct-gray, and separated by a considerable interval from its fellow, which it approaches only with its ventro-mesal extension, it becomes shaped like an inverted letter L, the two together occupying this relation ˥Γ. It is the lateral extension which is occasionaly cut off from the main mass and which Westphal designates as a separate nidus, (his lateral cell-group). Histologically it is exactly the same as the remainder of Westphal's nidus.

(*b*) *Main nidus.*—Closely applied to the contour of the posterior fasciculus, extending into the peninsulas of gray matter which the aqueduct-gray detaches betwixt its bundles, this cell-nest gradually increases caudad,as Westphal's nest decreases. It extends nearer and nearer to the median line, but never quite reaches it, at least not as a compact cell-mass. It is everywhere demarcated from Westphal's nest, and all other cell-groups by the powerful fibre-arch to be described.

Intra-nidal Fibers.—The large intrusion of fibres noted in level *A* increases and arches completely round the main nidus. It sends off numerous detachments into it. On the whole it may be compared to a horse-shoe joined at either end, apparently, to the most ectal and mesal rootlets of the third pair, its arch resting on the main nucleus, its fibres fielding off the latter into various irregular columns and groups, resembling the sub-nests of the hypoglossal, the facial and other motor nerve origins. I shall henceforth designate this as the *great intra-nidal fibre arch.*

In and bordering on the middle line of the aqueduct-gray, are straight fibres which take the following three courses, dorsal: 1. Parallel to the middle line, coursing along the mesal aspect of Westphal's nest and arching ectal round its dorsal aspect. 2. Parallel to the middle line, ectal of those just described, and directly abutting on that nest. 3. Starts parallel to median line, then arch over between the ectal aspect of Westphal's nest and the great intra-nidal fibre-arch, with which it is confounded, to arch round the ectal prolongation of Westphal's nest or even to break into it, so as to cut off one, sometimes two, islands.

Posterior longitudinal fasciculus.—This bundle has nearly attained its full dimensions, it extends to the middle line, across which, but for interruption by connective tissue septa, it is continuous.

Rootlets.—The innermost rootlets of the third pair are in part, directly continuous with the fibers abutting on and encircling Westphal's cell-nest. The middle and ectal radicles are not necessarily derived from those divisions of the main cell-nest opposite which they appear to emerge, for individual fibres can be seen to meander quite a distance amongst and over the sub-nests before becoming lost. Some fibres appear—but it is impossible to establish this clearly—to emerge from the great intra-nidal fiber-arch.

(*C*) The aqueduct gray continues to becomes wider, and lower, the meso-caudal prolongation shrinking upwards; still there is a considerable prolongation between the two main masses of the posterior fasciculus, abutting the junction piece of the latter. At the lateral angle, where the "arched" (dorsal) and angular (ventral) half of the aqueduct joins, fibres appear to originate in a lateral mass of cells shaped like those of the oculo-motor nidus,[1] and running lateral and slightly caudal, after a direct course, become lost. Of their significance nothing is known, and they are excluded from consideration here.

Cell nests.—There are three, the main nest, attaining its full development, a sagittal nest, between the two main nests, and the last residue of the disappearing nests of Westphal.

(*a*) *Westphal's nest* is represented by an oval mass, whose long axis is parallel to its fellow from which it is separated by a considerable interval.

Main nest—same character as in level B, the peninsular extensions into the posterior fasciculus are in some individuals very large.

Sagittal nest.—The ventral extension of the gray matter contains cells shaped like those of the main nest, and demarcated from it by fibres passing on either side ventrodorsal to the relics of Westphal's nest.

In addition to these cell nests, the small scattered angular nerve cells which are found in all levels in the caudal or angular half of the aqueduct-gray, become larger, and near the floor of the aqueduct present an accumulation which is quite distinct, and exactly in the median line.

Intra-nidal fibres.—(*a*) The *great intra-nidal fibre arch* becomes more and more individualized as we proceed caudal, and the main nucleus becomes distinctly isolated and often driven out of the contour of the central tubular gray by it. It is plexiform in arrangement, its connex with the great rootlets of the third pair is very distinct.

b Westphal's fibre field is represented by a vertical column of fibres, to whose dorsal end Westphal's nest is attached like a stone enclosed in a sling. It is very compact, but ventrad becomes less so—probably by gradual passage to cephalic levels. The symmetrical columns form the lateral boundary of the sagittal cell-nest.

(*c*) *The decussated fibre field.*—There is a distinct decussation of fibres which appear to originate in the cells of the sagittal nest. This decussation is not symmetrical. In one level, the bundle from left to right, in the next, that from right to left preponderates. They extend but a short distance across the median line with their cell origins ; the crossed part becomes lost in the great arched intra-nidal field.

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[1] They might be confounded with the lateral group of Westphal's nidus, but they do not appear in the same level, are not continuous therewith, and are larger and less crowded.

(*D*) The angular part of the aqueduct gray has contracted so much that but a small part falls below a line connecting the "angles." The ventral apex is still present, but the lateral contour has become gradually tilted, so as to be nearly horizontal.

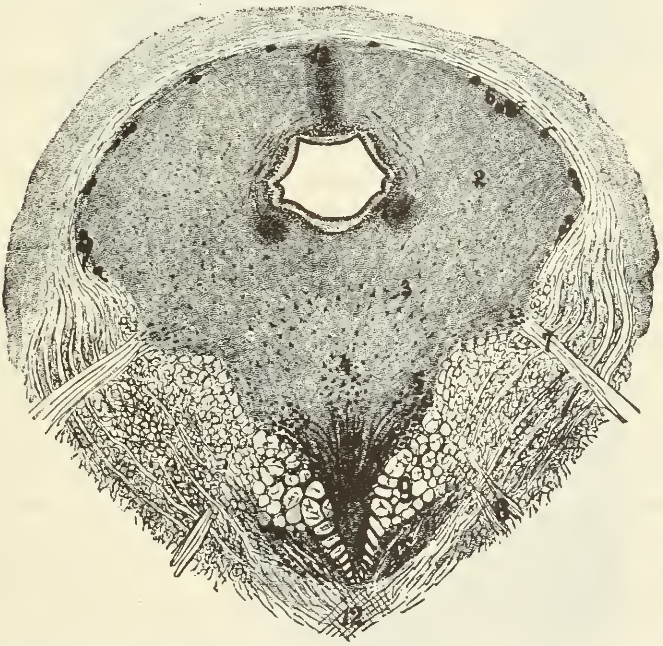

FIGURE III.—Section through more caudal level of human oculo-motor nidus. 7. Rootlets from the subdivision of main nidus, probably representing levator palpebra. 12. Fountain-like decussation of tegmentum.

1. Molecular mass of raphe. 6. Cells of descending trigeminal origin. 3. Scattered cells. 5. Main nidus. 9. Posterior longitudinal fasciculus. 10. Sagittal nidus. 11. Great intra nidal fibre arch. The molecular masses near either angle of the floor of the aqueduct may represent the interoptic ganglia of the reptilia.

Cell nests.—Aside from the diffuse small cells, there is but one, and that is the main nest. It has become almost isolated from the aqueduct gray by the great intra-nidal fibre arch, which here shows a diminution, to eventually disappear, before the nucleus does so. The distinction between the main nucleus and the common gray of the aqueduct can however, easily be made. It is less gelatinous, and its basic substance stains less deeply in carmine. Its cells are the largest in the oculo-motor nidus. It is intimately connected with the septa of the posterior fasciculus, *accessory bundles of which appear in the cross-section of this nidus, and in sagittal sections appear to proceed directly out of it.*

Rootlets.—There are no rootlets in these levels, although the large nuclear mass above spoken of extends a considerable distance caudad.

Intra-nidal fibre fields.—The great arched field disappears at the cephalic part of the insulated division of the main nest just described. There are numerous fibres apparently passing from one main nest to the other.

Summing up the foregoing, the topography of the cell nests in the angular division of the aqueduct gray may be stated to be as follows: First, a diffuse formation of small angular cells, loosely scattered, or as in level *c*, showing an accumulation ventrad of the aqueduct. Second, a distinct cell nest, successively laurel leaf, inverted L, and oval shaped, with small crowded cells imbedded in dense molecular substance, which is at once a dorso-mesal and the most cephalic. This is the one discovered by Westphal. Third, a large complex nucleus, beginning far cephalad, but yet caudad of the cephalic end of Westphal's nucleus, closely applied to the posterior fasciculus,increasing caudad, finally almost isolated from its parent gray, and ultimately lost among the septa of the posterior fasciculus. This is the main or proper cell-nest. Fourth, a sagittal cell-nest, not separable into symmetrical halves, which begins in the third fifth and ends before the last fifth of the cephalo-caudal extent of the main nest.

The main part of the oculo-motor, namely, all except its most cephalo-mesal and caudo-mesal fibres, originates from the *main nidus.* Its most cephalo-mesal fibres originate from the *nidus of Westphal.* As to its caudo-mesal fibres there is some doubt, though the probability is that they originate from the *sagittal nidus.* It is noteworthy that no oculo-motor rootlets originate from the posterior (semi-isolated) division of the main nest, imbedded in the posterior fasciculus. I believe this mass can be identified with those cells to which Mendel traced the orbicularis oculi fibers of the facial nerve, and will in the sequel advance reasons for this view.

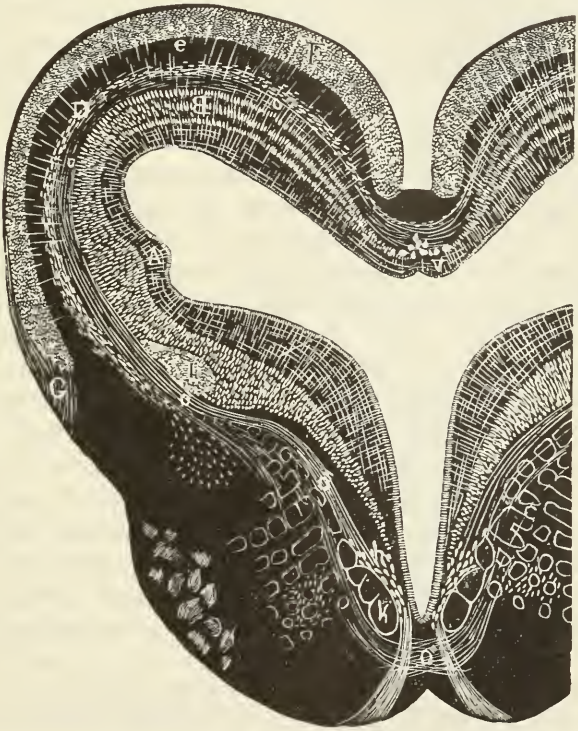

Figure IV.—Transsection of mesen-cephalon of fresh-water turtle.

COMPARATIVE DEVELOPMENT OF THE OCULO-MOTOR NUCLEI AND FIBRE TRACTS.

In the reptiles generally,[1] there is but little, if any, indication of a differentiation of these cell-nests of the third pair. The ventral part of the aqueduct-gray, which in these animals is drawn out in a long attenuated area on either side of the mesocœlian slit, contains a number of scattered elements shaped like typical motor cells, and from which the fibres of the third pair can be traced with a distinctness seldom found in mammals. Some indication of individualization is found in a group lying most ventral, where the aqueduct-gray often shows a slight swelling on it. Dorsal the cells become smaller and are followed by the ordinary minute elements characterizing the outer part of the aqueduct-gray of these animals. In some Saurians like the Anolis and Iguana, the nuclei are better developed, and it is particularly the cell-nest of the trochlearis that is beautifully individualized.[1]

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[1] The sea-turtle, fresh-water turtles, snakes and Sheltopazik which I have examined.

In birds the nucleus of the third pair is very large. In a series of sections (sagittal) from the ostrich, I can distinguish two superimposed strata of cells, which are probably the expression of as many lateral extensions of the central tubular gray; one of these is interradicular and ventrad of the posterior fasciculus. In all the sauropsida the posterior longitudinal fasciculus is very distinct, and in some (chelydra) an uninterrupted course of single axis cylinders can be traced to the fundamental anterior column, of the spinal cord.[2] In all, its connection with the nucleus of the third, fourth and fifth pairs is obvious. In none can its origin be traced much further forward than the anterior end of the oculo-motor nucleus. The fibres of most cephalic extent appear to be derived from the deep gray of the optici[3] (anterior pair of the corpora quadrigmina); succeeding fibers originate or terminate in the nuclei of the oculo-motor system, and the remainder run into the spinal cord, as the deepest fibres of the anterior column.[1] These relations appear to be maintained in all higher vertebrates, including man. In the dog, cat, sea-lion and lion, the nuclei are disposed similarly to the plan which will be detailed when discussing my atrophy experiment. In a beautiful section parallel with the base of the brain of a dog, two distinct cell-nests can be readily distinguished. One of these is a slender column near the median line, and nearly fusing across it ; it extends further cephalad and ceases first caudad. Its posterior extremity is grasped, as it were, by the other large celled cell-nest. In other words, the main nidus opens out like the petals of a flower to enclose the root of its pistils, represented by the former small celled, or Westphal's cell-nest.[2] In the sea-lion the sagittal nuclei described by myself, is well-developed. In the dog a remarkable small celled, thickly crowded sagittal cell mass bisects the central tubular gray in the level of the posterior pair of the quadrigemina, and extends into the level of the trochlearis origin. I cannot establish its existence in man.

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[1] The cell-nests in the chamaleon would prove an interesting study. It is not impossible that the nest connected with the nictitating membrane may be discovered in some of the sauropsida.

[2] Osborn has shown me drawings from an amphibian brain in which this bundle is represented as giving off from its caudal extent a fibre to the horseshoe shaped root of the facial nerve, a noteworthy observation stated to be based on unmistakable appearances.

[3] Owing to the peculiar distortion of the mesencephalon in birds the optici are crowded latero-ventral. It is probably connected with this fact that the anterior end of the posterior fasciculus undergoes a marked curve ventrad.

In the crepuscular bats the cell-nests of the oculo-motor system are very small, and the posterior fasciculus is attenuated. In the mole the nuclei are almost absent, and the fascicular is atrophic.[3] In the anthropoid apes alone, of all animals examined, is the anatomy of the oculo-motor cell-nests and their intranidal tracts approximately as complex as in man. This fact adds another to the numerous observations, which induced the writer, years ago,[4] to point out the erroneous nature of the view held by many, that in higher development the "intellectual" centres are developed at the expense of the reflex centres, or that the more perfect reflex mechanisms are to be sought for in lower forms. The nuclear anatomy of the spinal centres of the hypoglossal and facial nerves, and even if so blindly automatic a system as that of the ocular-motor mechanism, prove at every step that man is at the head of the vertebrata, not alone in regard to his central development, but also in the extent, complexity and intricacy of the reflex centres and their uniting fibre tracts.

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[1] Those nearest the gray substance of the base of the anterior horn, included between it, the anterior spinal commissura and the ventral fissure.

[2] The reader may be reminded at this point that in the dog, the accommodation pupil reaction is the reverse of the human.

[3] Ford, Archiv. f. Psychiatrie, vii., p. 421.

[4] Architecture and Mechanism of the Brain, JOURNAL OF NERVOUS AND MENTAL DISEASE, 1879–1880, pp. 45 and 67 of reprint.

The atrophy experiment I have referred to has been detailed in its general features elsewhere.[1] Suffice it to say that the left third pair was divided at its emergence from the crus, and that all the other oculo-motor nerves were normal. The left optic tract had been also divided. In the sequel I shall attempt to homologize the atrophied developed sub-nuclei with the human by giving the same names used in the above description of the human sub-nuclei.

(*a*) *Main nucleus.*—Normally developed in all levels on the right side. Present only in its most posterior division on the left side.

(*b*) *Sagittal nucleus.*—On the left side it is normally present, but on the right side of a line drawn from the ventral apex of the aqueduct to the raphe, few, and those very small, cells are found.

(*c*) *Westphal's nucleus.*—As I am uncertain about the homologies of this nucleus in the cat, though well developed in the dog, I reserve any opinion for the present.

(*d*) *Nucleus under aqueduct* symmetrically developed.

(*e*) *Intranidal fibres.*—These in my specimens of normal carnivora, as well as the experimental kitten, are more interlaced and less distinctly grouped in bundles than in man. In the level where the left sagittal and right main nucleus are in their main development, they are seen in large numbers extending dorsad and mesad of the right main, and across the median line into the left sagittal nucleus. Both classes of fibres enter the emerging rootlets of the right third pair.

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[1] JOURNAL OF NERVOUS AND MENTAL DISEASE, June, 1888, vol. xiii., No. 6, p. 349.

(*f*) *Posterior longitudinal fasciculus.*—In all higher levels a marked asymmetry is observed. In the upper level of the main nucleus it is absent on the operated side. With the appearance of the sagittal nidus, it is found on both sides, about half as large on the operated as on the unoperated side. At the lower end of the oculo-motor nidus, whose posterior division as stated is present on both sides, it is about two-thirds the area on the operated side, as compared with the other. It thus continues to the level of the abducens nidus caudad, of which a marked difference is not noticeable.

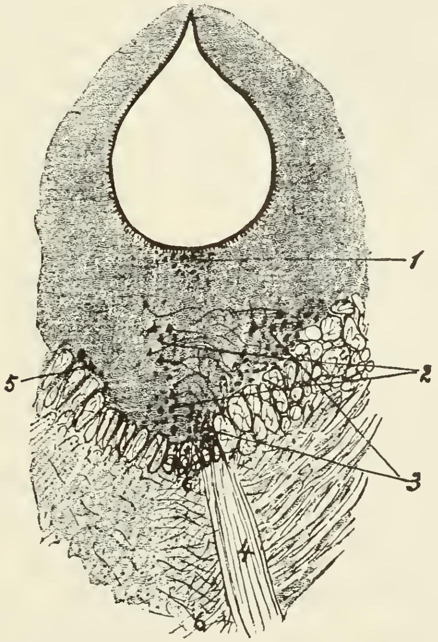

FIGURE V.—Transsection through oculo-motor nidus of cat, whose left third pair had been destroyed. 1. Subendymal cell mass. 2. Crossed nidus of third pair, (sagittal nidus). 3. Main or uncrossed nidus. 4. Intact right root. 5. Nidus symmetrically developed notwithstanding the unilateral destruction of the third pair, and identical with the nidal centre, eliminated by Mendel through peripherel destruction of the orbicularis palpebraerum. 6. Raphe.

In addition to these observations in normal human and comparative anatomy, as well as those derived from the atrophy method, I had the specimens of a case of organic central ophthalmoplegia partialis externa at my disposal, a complete series of sections from which had been prepared jointly with Dr. N. E. Brill. My conclusions from these observations and the observations of others are subjoined under appropriate headings.

THE INTRA-OCULAR INNERVATIONS.

The rootlets for the pupillary and ciliary muscles.—Pathological observations show that lesions of the crus, involving those rootlets of the third pair which traverse the crus, produce paralysis of the outer eye muscles, but leave the inner ones intact. It is to be assumed from this that those anterior-most fibres which do not traverse the crus, or only reach it shortly before their exit, are related to pupillary and ciliary projection. Experimentally the complementary observation has been established. Dividing the anterior-most fibres of one third pair, eliminates accommodative power on the corresponding eye, leaving that of the other eye intact.[2]

The cell-nests from which rootlets for the pupil and ciliary muscles arise.—Pathological destruction of the central tubular gray of the posterior division of the third ventricle,[3] sclerotic induration with contiguous endymal hypertrophy,[4] and experimental interference[5] with the same region will destroy the light reflex of the pupil. In case the affection be unilateral, the loss of the light reflex is homolateral. Inasmuch as the retinal fibres mediating the light reflex do not run in the optic tract, but leave the optic nerve at the chiasm to plunge into the ventricular gray,[6] it appears reasonable to assume that the sub-nest of the oculo-motor nucleus, related to the pupillary movements must be situated far cephalad, in order to be most conveniently situated for the reception of the nidal end of the pupillary reflex arch. This view is strengthened by the observations just

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[1] Oyon, cited from Gazette Medical de Paris, 1870, No. 47, by Wernicke. Gehunkrankheiten.

[2] Heuser-Volckers, Archiv. f. Ophthalmologic, xxiv., pp. 1–26.

[3] W. Sander, Archiv. f. Psychiatrie und neuenkrankheiten, p. 287.

[4] Our observation, unpublished.

[5] Bechtherew, Pfluger's Archiv., xxxi., 1883.

[6] Bechtherew, Neurologisches Centralblatt, 1883, No. 12.

cited, demonstrating the rootlets related to the intra-ocular movements to be most cephalic. There is one observation which enables us to limit the cephalic extent of the pupillary nidus more narrowly by exclusion. It is not situated much, if at all, in front of the trans-section level of the post-commissura, because lesion of the region in front when unilateral abolishes the light reflex of the corresponding eye, but it does not abolish consensual reaction on that eye.[1] We are also able to say that the pupillary nidus is homolatateral for each eye, as the lesions of the central, tubular gray,[2] or special rootlets, producing limited pupillary paresis, when unilateral were always on the same side as the symptom. But the strongest evidence in favor of the location of the sub-nest for the intra-ocular muscles is furnished by two cases, one in which there was practically total external ophthalmoplegia with preserved accommodation,[3] and the other in which there were gross disturbances in the innervation of all the external muscles of the eye except the right abducens and both superior obliques, also with preserved accommodation,[4] and in both of which the only unquestionably healthy cell-nests were Westphal's nidi, one on either side of the median line. At present we are only able to affirm with confidence that it is the nidal centre of accommodation. Its freedom from disease with preserved accommodation in the midst of ophthalmoplegia, its cephalic location, in topographical harmony with the results of electrical irritation,[5] and with the situation of the accommodative nerve-rootlets, all strongly support this view. We are unable to differentiate between such parts of this cell-nest, if any, which may be separately consigned to the ciliary and pupillary muscles.

THE EXTRA-OCULAR NIDAL CENTRES.

The rootlets related to the innervatiou of the internal rectus.—In a case where the most meso-caudal rootlets of the third pair were destroyed in man, the paralysis was limited to the internal rectus muscle. The pupils were entirely unaffected.[1] This proves that the rootlets for the two are distinct. Indirectly it suggests that the respective nidi must be distinct, because the nest for pupillary innervation is homolateral, and that for the internal rectus is contralateral.

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[1] Bechtherew, Pfluger's Arch. loc. cit.

[2] Sander, Arch. f. Psych. loc. cit.

[3] The case of Westphal already cited.

[4] The unpublished case of my own, elsewhere referred to.

[5] Hensen and Volckers, Graefé's Archiv., 1874, p. 1.

The nidal centre for the internal rectus.—The atrophy experiment of von Gudden in the rabbit, and the same experiment repeated in the case of the cat by the present writer, conclusively prove that there is a decussated origin for a part of the oculo-motor nerve. By exclusion, and by physiological deduction, we can prove that this cannot be the nidus of any other muscle than the internal rectus.

The nidal centre for the levator palpebræ.—All gross lesions of the main nidus produce ptosis. There is one observation of a limited lesion, a hemorrhage destroying the most lateral part of one main nucleus, with homolateral ptosis.[2] In another case of multiple paresis of the oculomotor apparatus, marked left ptosis was associated with a macroscopic hemorrhagic focus,[3] destroying the most external sub-group of the main nest, in varying extent in its upper and middle levels. We may therefore assume the levator palpebræ to be represented in at least the middle altitude of the main nest in its most external division.

The nidal centre for the orbicularis palpebrarum.—The eyelid sphincter, under the reflex dominion of retinal impressions as it is, and intimately bound in associate innervation with certain extra-ocular muscles of the oculo-motor system,[4] is not, as has been heretofore thought, under the dominion of the *nidus facialis.* Mendel, by destroying the orbicular muscle in the new-born, produced atrophy in the most posterior division of the oculo-motor cell-nest. It is precisely this cell-nest which remains uninfluenced by destruction of the trunk of the third pair. The inference is obvious. There must be contained in the oculo-motor cell-nests one sub-nest whose emerging fibres are not included in the third pair. We know from other facts that there is one muscle represented in the seventh pair which is not represented in the nidus facialis.[1] The conclusion follows that an aberrant fasciculus from the oculo-motor nidus must join the emerging facial root. There is such a bundle from

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[1] Kahler-Pick, Archiv. f. Psych. in Nokikhtn., x., p. 334. Wallenbergh's case, if the accompanying drawings be correct, is opposed to the deductions which appear to follow so easily from Kahler-Pick's (Archiv. f. Psych., xix., p. 298, et seq).

[2] Leube, Deutsches Archiv. f. Klinische Medizin, 1887, p. 219.

[3] My own unpublished case, herein repeatedly referred to.

[4] As evinced in the associated rolling motion of the eyeballs accompanying eye-closure.

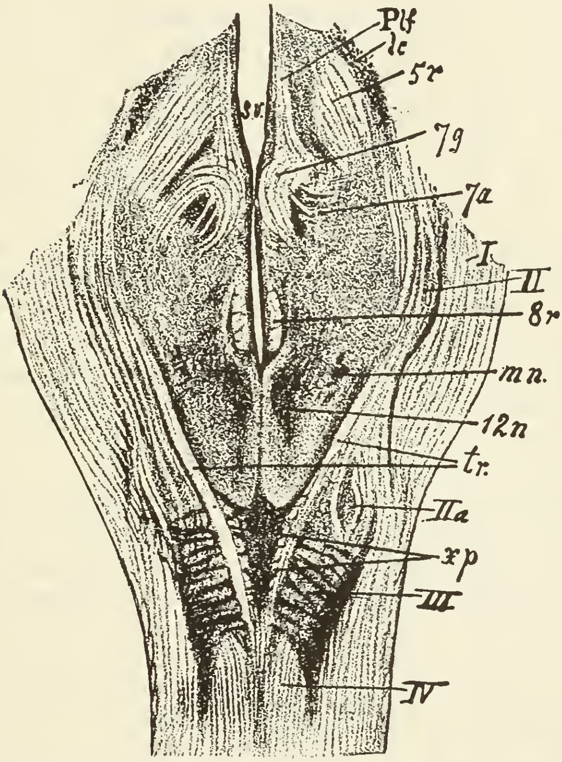

Figure VI.—Flatwise section of human isthmus. 7. g. Genu facialis. P. lf. Posterior longitudinal fasciculus. On the lettered side the transition of fibres from the latter to the former can be clearly seen.

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[1] Detailed by Mendel, Neurologisches Centralblatt, 1887, No. 23.

the posterior longitudinal fasciculus to the genu facialis.

THE POSTERIOR LONGITUDINAL FASCICULUS.

This bundle contains a number of elements. (*a*) The bundle connecting the abducens nidus with the nidus of the internal rectus, and through which lateral associated movement of both eyeballs is effected. It was formerly thought necessary to assume a decussated course for this bundle.[2] But since it is now known that the nidus of the right internal rectus is on the same side as the nidus of the left external rectus, such a decussated course is no longer plausible.[3] The decussation is in the root-fibres of the third pair. (*b*) This fasciculus includes that part of the root of the facial nerve which represents the orbicularis palpebrarum. (*c*) It contains other fibres, which from their (probable) origin in the deep gray of the optic lobes and (almost certain) caudal termination in the nidal centres of the cervical muscles (head-rotation, etc.,) may be regarded as mediating that element in conjugated deviation of the eyes and head which is not mediated by the portion described under the caption *a*.

The oculo-motor mechanism is thus shown to have as complex an anatomical basis as the physiological study of its peripheral reactions would lead us to anticipate. And we are approaching a degree of accurate knowledge regarding the central seat of these reactions which approaches in exactitude and certainty the physiological observation itself.

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[1] The value of this observation is not diminished by the fact that the drawing was made and published before its significance was appreciated. It is simply an unbiassed record,

[2] Duval and Laborde.

[3] And is contradicted by the observations of J. Nussbaum, Wiener Medizinische Yahrbucher, 1887.

DEGENERATION OF THE PERIPHERAL NERVES IN LOCOMOTOR ATAXIA.

By Dr. JOHN C. SHAW, of Brooklyn, N. Y.

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A paper read before the N. Y. Neurological Society, June, 1888.

THE opportunity having been given me to examine some of the peripheral nerves in a case of locomotor ataxia, I believe a presentation of the specimens may be of interest to the society.

It appears that Türck was the first to investigate into the condition of the peripheral nerves in locomotor ataxia. In 1858 he examined the nerves of the skin in two cases (Dejerine), but found no alterations. This may have been owing to the then imperfect methods of examination. In 1863 Frederich found alterations in the peripheral nerves in some cases in which he examined the sciatic, crural, and radial (Dejerine). In 1868 Vulpian, in a study of the sensitive nerves, the spinal ganglia, and the sympathetic in sclerosis of the posterior columns, found no change in the cutaneous nerves.

Westphal, in 1878, in the study of a case of combined sclerosis (posterior and lateral columns), in which the sciatic, tibial, and posterior cutaneous nerves were examined, found atrophy of the nerve fibres, increase of the connective tissue, and it is especially noted that in the specimens the axis cylinders can be seen devoid of myeline; these nerves were examined only after hardening, staining with carmine, and on sections.

In 1880 Pierret (Thesis of Robin) says that, in cases of locomotor ataxia in which there are lightning pains, anæsthesia, and pemphigoid eruptions, if the terminal expansions of the nerves supplying these parts be examined, a neuritis will be discovered comparable to that of optic neuritis.

In 1883 Dejerine reported two cases of locomotor ataxia; in both were found the changes in the posterior columns; the posterior roots were atrophied. Examination of the cutaneous nerves in both cases showed alterations which he considers to be like those found after section of nerves.

In 1883 Pitres and Vaillard report a case of locomotor ataxia with œdema of the left lower extremity, arthropathy of the left knee. Microscopic examination of the peripheral nerves showed the right sciatic to be normal, while the left sciatic presented evidences of alterations similar to those found in the Wallerian degeneration.

In 1884 Sakaky reports a case of tabes from the clinic of Professor Westphal, in which some of the peripheral nerves were examined and marked atrophy of the nerve tubes found. This study was made on sections.

In 1886 Pitres and Vaillard report five new cases, in all of which changes were found in the peripheral nerves. The nerves examined were the sciatic, anterior and posterior tibial, etc.

My own case, in which the spinal cord and some of the peripheral nerves have been examined, I believe is the first case reported in this country, and, although the clinical history is imperfect, the interest attaching to the changes in the peripheral nerves justify me in presenting it to you.

A man, forty-seven years old, was seen by me six years ago; examination showed him to be suffering from typical locomotor ataxia. The notes dictated to the house physician of the hospital at that time have been mislaid. In December, 1886, I saw this patient for the second time, and made a rapid examination of him. His ataxia is extreme, he throws his legs about wildly in attempting to walk; he cannot stand without being supported on each side; muscular power is good; the tendon reflex is absent; the pupils are quite small, and do not react to light or accommodation; he has extravagant ideas; mentally he is weak, memory very poor.

A history is obtained from his sister at this time. He has always been temperate; his mother, a nephew, a maternal cousin, and an aunt are insane. His first symptoms began nine years ago; he had sharp pains in his legs and knees, and when he walked his legs "locked together;" he soon began to have pains in his abdomen, and spells of vomiting (gastric crisis); he became very irritable. Six years ago he went to the hospital, where I saw him the first time; the ataxia was extreme then. In October, 1885, he had a number of convulsions. In April, 1886, she first noticed that his mind was affected, he appeared to be absent-minded; and by August he was quite childish. In November he had some more convulsions, and developed extravagant ideas, thought he had plenty of money, and was about to marry the queen of England, etc. At present is demented, memory quite poor; thinks he is pregnant.

He becomes gradually more demented, and noisy at night. Insists upon saying he is pregnant. Is growing more and more feeble.

April 3, 1887.—During the night he had an attack of hæmaturia, passing about a pint of blood; he becomes very feeble and comatose. A large discolored patch is observed in left inguinal region. The bleeding from penis ceases, and he appeared somewhat improved at night; but on the morning of April 4th he had two epileptiform seizures, becomes comatose, and dies early on the morning of April 5th. An autopsy was made on the afternoon of the same day. The body is emaciated; there is a very extensive ecchymosis over the left lower half of the abdomen and some slight oozing of blood from the penis. Section through the abdominal muscles shows that under the discolored spot they are infiltrated with blood; all the muscles bleed freely when cut into; there is no evidence of injury. The bladder contains a large quantity of bloody fluid. The intestines are all more or less discolored, of a darkish brown. The right kidney is much congested; the capsule tears off easily; one of the calicis at the lower part of the kidney has projecting from it clots of blood, while the others present no similar condition; the pelvis of kidney contains a small quantity of slightly bloody fluid; sections through the organ showed extravazations of blood in several places in its substance. From this kidney evidently came the hæmorrhage which had occurred a day or two before and continued somewhat ever since. The left kidney was only congested. The lungs were the seat of hypostatic congestion, otherwise healthy. The heart is flabby and the aorta contains atheromatous plates.

Portions of the peripheral nerves were removed from the right leg, placed in osmic acid, and other pieces in bichromate potass. solution. The cord was also removed and placed in solution of bicromate potass.

The spinal cord shows the usual gray degeneration of the posterior columns. The portions of nerve examined are taken from the sciatic, popliteal, and external plantar. Of those pieces placed in osmic acid, the first thing observed was that they were stained by the osmic acid with great difficulty, and this lead to their being kept in the solution a longer time than is usual; even then it was found, on teasing them, that there were portions of the nerve which had not taken the black color usual in this method of preparation. The changes observed can be found in the sciatic, popliteal, and plantar; it is more extensive, however, in the popliteal and plantar; the number of healthy tubes is less great than in the sciatic

The preparations are stained in carmine and mounted in glycerine. In every preparation made very few normal tubes are to be seen; the degree of change varies very much in the different tubes; many tubes can be found in each specimen presenting the condition of myeline which is observed after section of nerves; that is, the breaking up of the myeline into more or less irregular pieces, scattered about in the sheath, and in various stages of disintegration. Some stained very black with the osmic acid, and presenting a dense, compact appearance; other pieces only stained lightly brown, and presenting a granular appearance. In another portion of the tube may be found a still more advanced process, where the myeline is completely broken up into a fine granular mass, hardly stained by the osmic acid and the tube is smaller. A still further process may be seen in the complete absorption of this granular myeline and shrinking of the sheath. There is commonly observed in the preparation entire nerve tubes presenting this light, granular appearance, and which has hardly been stained, if at all, by the osmic acid; not a complete block of well-stained myeline is to be seen in the tube, and this explains the lack of black staining which the nerve tubes present at various places; the change has been so great in that portion of the bundle that there is no myeline to stain. In those portions of the nerve where the myeline has undergone this extensive change, the sheath appears to have taken up the carmine a little more freely than it usually does; and when a large number of fibres together has undergone this process, they present the deep carmine stain, which is in marked contrast with the adjacent portions of the fibre which are stained more or less strongly black by the osmic acid.

Very frequently portions of a fibre or fibres appear to have undergone such rapid change in its myeline that the sheath appears to be filled with a granular-looking mass which must have been more or less liquid. The process of liquefaction was too rapid for absorption, and collapse of the sheath to occur, so that the tube appears to have nearly its normal dimensions. The interannular nucleus, with its protoplasm, appears not to have taken any active part in this process of disintegration of the myeline, as they are not enlarged, in this differing from that found after section of nerves. The nucleus is sharply stained by the carmine. In all the preparations there are to be observed numerous rolled-up, collapsed sheaths of Schwann. With a high power one can observe the vestige of myeline drops as fine granules in some of these shrunken sheaths.

In many of the tubes this change in the myeline can be observed beginning at each end of an interannular constriction, near the constriction of Ranvier, while the intermediate portions are apparently normal. Numerous tubes can be seen in which these changes in the myeline occupy only a portion of the tube, so that the parts above and below it are apparently normal.

In these tubes in which the myeline has been absorbed there appears to be an increase in the number and size of the nuclei in the sheath of Schwann. A peculiarity of this process is the persistence of the axis-cylinder. In those fibres in which the myeline is broken up into blocks, I have been unable to discover the persistence of the axis-cylinder, but these fibres are not as numerous as those which present the granular myeline alone; in these fibres the persistence of the axis-cylinder is shown beautifully, as it is stained sharply with the carmine, and can be clearly seen, especially in those fibres which have undergone that process which I have considered as a rapid liquefaction of the myeline. Often the axis-cylinder can be seen pushed to one side by this fluid, so that it rests against the sheath. I have not observed any change in the axis-cylinder.

Many tubes are to be seen, which, I suppose, are tubes in process of regeneration, at least they have been so described by writers on this subject; they are small fibres with a narrow, dark-stained band of myeline. I have not observed any axis-cylinder in them.

If sections are examined after hardening in bichromate potass., and mounted after staining with carmine in Canada balsam, a great many nerve tubes are found atrophied in each bundle. The changes are not made out as clearly, however, on sections as they are in preparations stained with osmic acid, carmine, and teased.

The condition observed in these nerves, and which I have described, does not correspond exactly to that given by most writers on the changes in the peripheral nerves in locomotor ataxia, many of them stating that it is like the changes observed in the Wallerian degeneration. The changes observed in these nerves which I present to you are, it appears to me, a different process from that observed in the Wallerian degeneration. The absence of increase in the interannular nucleus, the persistence of the axis-cylinder, the appearance of changes in certain portions of the nerve, while that portion above and below remains comparatively healthy. The greater number of tubes in which the myeline undergoes evidently a rapid granular liquefaction rather than breaking up into blocks, are all points of marked difference in the two processes.

There can be seen in these specimens, nerve tubes of considerable length, in which the changes appear exactly like those in the Wallerian degeneration, that is, as far as the myeline is concerned. The interannular, nucleus however, does not present the marked increase in size that is so common after section of nerves. The fibres presenting this appearance are, however, not nearly so numerous as those with the very fine granular myeline and the persistent axis-cylinder.

The condition here is similar to that which has been described by Gombault in 1880, in an article on Parenchymatous Neuritis or Segmentary Periaxillary Neuritis. The observations were made on the sciatic of fowls which had been slowly poisoned with lead. He especially calls attention to the persistence of the axis-cylinder, the segmentary nature of the neuritis, and the difference in the process from that found in the Wallerian degeneration.

Since that time Pitres and Vaillard, in 1886, in an article on the Alterations of Nerves in Diphtheritic Paralysis, have recognized the condition described by Gombault. Still they do not agree fully with his description. They admit the segmentary nature of the neuritis, but deny the persistence of the axis-cylinder. This peculiar change of the myeline in segments was observed by Gombault in the peripheral nerves of man suffering from various conditions, such as protopathic muscular atrophy, lateral amyotrophic sclerosis, traumatic neuritis, and diphtheritic paralysis.

In 1882 P. Meyer observed the same condition in a case of diphtheritic paralysis. The presence of these two kinds of disease fibres in the specimens, the one having the blocks of myeline scattered through it and the other with only a fine granular, liquid-looking contents, have been observed by Gombault, Meyer, Pitres and Vaillard, and myself, in all the specimens examined. The beginning of the change in the myeline near the construction of Ranvier has also been pointed out by each observer. Gombault lays considerable stress upon this; in my own specimens it can be observed if carefully looked for.

In view of the presence of these apparently different processes in the same nerve bundle, one asks is this a peculiar form of neuritis parenchymatous in origin, or is it a process similar to that found in nerves after section. I would not venture any positive statement from an examination of one case, but it appears to me that the process is quite a different one from that observed in nerves after section. I look upon it as a parenchymatous neuritis, the expression of a more or less general degenerative change in the entire nervous system, a kind of slow breaking up of the organism.

Gombault and Pitres and Vaillard have taken opposite sides on this question. Gombault looking upon it as a special form of neuritis quite distinct from the Wallerian degeneration and to explain the presence of these fibres with the large blocks of myeline in them which are so characteristic of the Wallerian degeneration, he assumes that at some point above where this segmentary neuritis has been going on the nerve fibre has been entirely cut off by the extensiveness of the process, the other portion of the fibre then degenerates, following the course of that found in the Wallerian change.

Pitres and Vaillard look upon the changes as one and the same thing, and the difference in appearance is due to the various stages of the process.

The view advanced by Gombault is ingenious, and appears to me the most likely to be correct.

These peculiar changes in the peripheral nerves which I have been describing, have not, as far as I know, been observed in cases of locomotor ataxia by any one but myself; it is a change which is evidently not peculiar to cases of posterior spinal sclerosis, as it has been seen by others in a variety of conditions, especially in the cases of diphtheritic paralysis and the experimental production of it by Gombault, which has been previously alluded to, and it is very probable that further studies may show it to be present in a large number of the changes occurring in peripheral nerves.

Fig 1

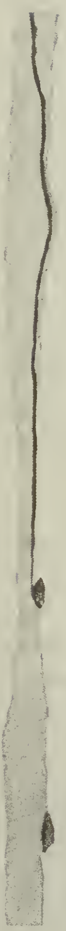


Fig 2

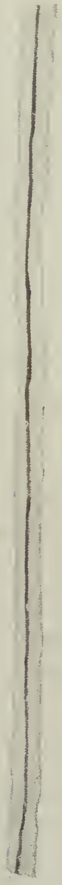


Fig 3

Fig 5

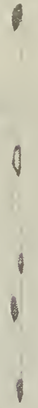


Fig 4

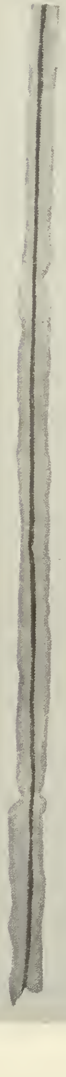
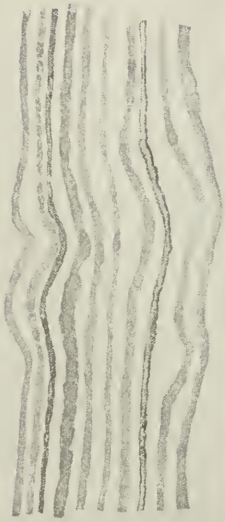


Fig 6



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EXPLANATION OF PLATE.

FIG. 1.—Nerve fibre which has undergone rapid granular disintegration with persistence of the axis cylinder and which is pushed to one side.

FIG. 2.—Tube in which the myeline has disappeared, leaving the axis cylinder sharply stained by the carmine.

FIG. 3.—Nerve tube, showing the typical changes as seen in the Wallerian degeneration, the myeline broken up into blocks and floating about in the more granular myeline.

FIG. 4.—Fibre, showing the beginning change in the myeline at each end of an interannular segment persistence of the axis cylinder.

FIG. 5.—Tube in which the myeline has entirely disappeared, the sheath of Schwann has collapsed, and an increase in the nuclei of the sheath.

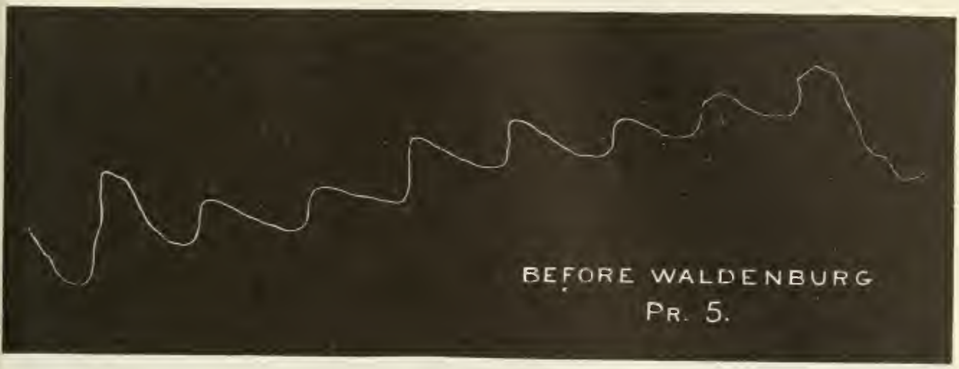
FIG. 6.—Showing a small portion of a bundle of fibres with nerve tubes in various stages of degeneration.

CASE OF POST EPILEPTIC HYSTERIA. EFFECT OF INHALATION OF COMPRESSED AIR. PHENOMENON OF TRANSFER.

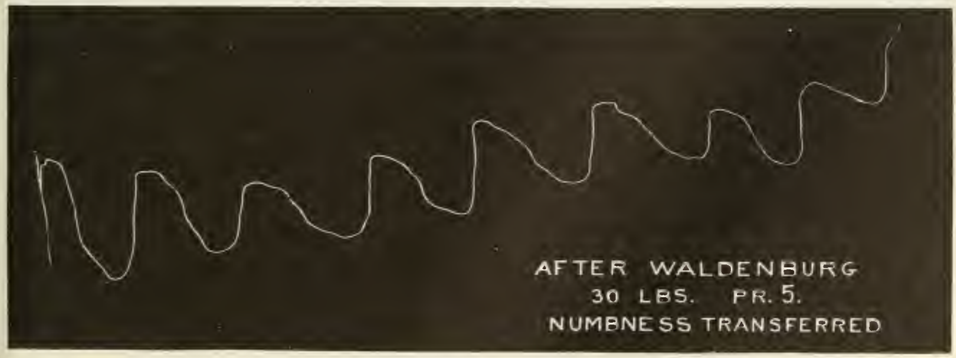
BY DR. MARY PUTNAM JACOBI, M. D.

MISS R. K., æt. 35, suffering from epilepsy for about twenty years. Violent attacks infrequent, and patient often enjoys several months of quite good health: at other times, liable to frequent epileptical seizures, mingled with hysteriform attacks. Both are usually controllable by nitrite of amyl. Patient takes habitually 60 grains of bromide a day: often has taken three and four drachms, and with no marked benefit.

In October, Miss K. suffered for a fortnight from a severe attack of gastric catarrh. Towards the close of this attack, which had been treated carefully, and without suspending the bromides, the patient had an epileptic fit of extreme violence, such as she was said not to have experienced in five or six years. After this fit, she suffered from marked nervous exhaustion, claimed to feel extremly "queer" in the head, to tremble all the time, to have sensation of numbness throughout the body, slight on the right, but very pronounced on the left side. There was no objective anaesthesia. The symptoms all pointed to a persistent effect in the cortical centres of the shock sustained during the epileptic fit. It was a question, whether, on account of this, the cortex had not yet regained its normal control over the subcortical vaso motor centres, and hence that some degree of spasm persisted in the cortical blood vessels, maintaining the anæmia, or dyspnœa of the cortical nerve tissue. I decided to try the effect of the inhalation of compressed air from the Waldenburg apparatus, and anticipated from this a double effect: 1st. The introduction of some more oxygen into the blood, which might serve to feed the dyspnœic cortical centres of the brain. 2d. An increased force of the heart, indirectly determined by the greater amount of blood in the systemic circulation, when blood should have been expelled from the lungs by the increased pressure of air within the air cells.

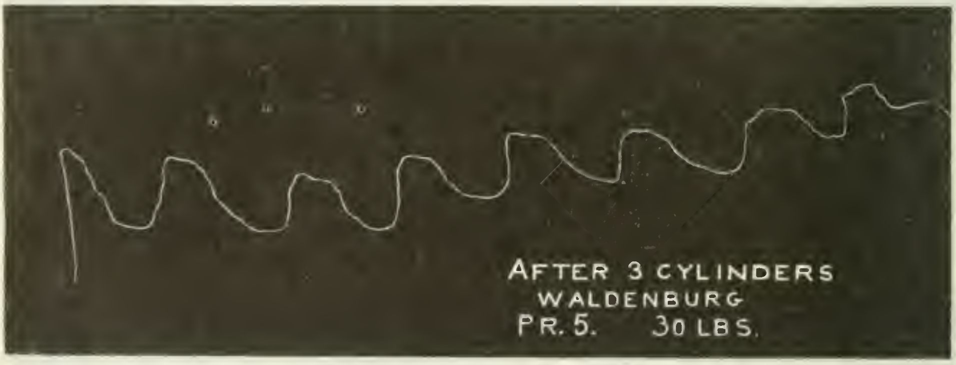


The accompanying pulse traces show the condition of the radical pulse before and after the inhalation. The trace No. I, taken before inhalation, is remarkable for its vertical and high percussion stroke, relatively rapid collapse, absence of dicrotism, very small tidal wave. These characters indicate a low arterial tension, against which the heart works with energy, but without much benefit.



Condition of the Heart.—Immediately after inhalation of one cylinder of air under pressure of 30 lbs. or $\frac{1}{40}$ th of an atmosphere, trace No. II was taken. In this the percussion stroke is considerably increased in height, but the tidal wave is also increased, the collapse of the artery is prolonged, and there is an approach to a dicrotic notch. The respiratory base line rises to about the same extent in both tracings.

No. III was taken after three cylinders had been inhaled. The percussion stroke is lower than in the second trace, though higher than in the first, but the tidal wave is still more developed. This seems to indicate that, with an increased force of cardiac contraction, an increased resistance was now offered in the capillaries and arterioles. This might be owing to the greater amount of blood retained in them in a unity of time ; or else, to an improved tone of the vaso motor centre, under increased respiratory stimulus and oxygenation.



Coincidently with these modifications of the pulse, the patient noticed that the left side of the body was now free from numbness, while this was intensified on the right side, where, hitherto it had been scarcely perceptible. For several subsequent hours, the patient felt a great deal better, and continued to do so during four or five days of daily inhalation. She then ceased attendance, but in forty-eight hours symptoms returned : consisting now in clutching sensation at epigastrium, pains all through the limbs, ravenous hunger and profound mental depression ; menstruation was then over-due five days. The patient was then ordered wine of cocoa, to each dose of which was added a grain of cocaine. This at once made her feel, (to use her own expression), "splendidly;" menstruation came on, and the nervous symptoms all disappeared.

The condition which persisted after the violent epileptic attack in this case was probably the hysteroid state, so well described by Gowers, and which that author interprets as indicating commencing nutritive degeneration of the brain.

The point of interest in this special attack, lies I think, in the phenomenon of transfer, which was effected by the compressed air inhalation as promptly as it is some times observed after the use of the metallic disks.

This transfer began immediately, and when the tracing showed no sign of an effect on the vaso motor system, but only of increased energy in the cardiac contraction.

If the numbness be attributed to temporary impairment of the nutrition of the cortical sensory centres, and of their circulation, two conditions may be implied. The chemical processes of synthesis and of deoxidation, resulting respectively in storage and in circulation of nerve force, must be slackened or nearly arrested, while the process of supplying nutritive material, and of eliminating of waste products must both be similarly reduced. The increased force of circulation determined by the compressed air inhalation, as well as the presumable, though slight increase in the oxygen carried by the blood, should both act in the same way upon the dyspnœic nerve tissues. The greater supply of oxygen should stimulate the slackened chemical processes sustained in them; and by the greater rapidity of the blood stream, their lymphatic sheaths should be swept clear of accumulated waste products. In this way may be explained the relief afforded to the various paræsthesias complained of. But the transfer of the numbness from the left to the right side of the body remains unexplained. We may ask the following question: When in one portion of the cortex, depression of function is relieved, and an active circulation re-established, must there be a temporary recession of blood supply and nervous activity from other portions, until, after a succession of oscillations, complete equilibrium shall have been restored?

SKIN PRODUCTS.

By WALLACE WOOD, M.D.,

Professor in the University of the City of New York.

IT requires little more than simple inspection to reveal the fact that the human body, like that of bird or quadruped, consists of a frame covered by a kind of double sack, the skin and the intestines. Take a rough example: When a butcher dresses a sheep, he pulls off the one and pulls out the other. One is the tegument or outer covering, the other the intestinal or inner lining. Stripped of these two skin tissues, the lining and the covering, there remain the flesh and blood and bone constituting the animal frame. One of these sacks is the cutaneous tissue with its epidermis, the other the mucous membrane with its epithelium. Apart from its fibrous base the double sack, between the two portions of which the organism grows, consists of epidermis without and epithelium within, called by morphologists the skin sensory layer and the intestinal glandular layer;* between these two is the double fibrous layer of flesh and blood.

In lowest organisms the covering and lining, ectoderm and endoderm, constitute the whole being. In embryology the covering and lining are called epiblast and hypoblast. Between the sack covering and the sack lining, ectoderm and endoderm, lies the mesoderm, which is the gross construction of the body, the skeleton with its muscular tissue about it, and its sanguiniferous trunk within. It is plain to be seen how perfectly the vertebral column with its enclosing ribs embraces and holds that "light ramified tree," the circulatory system, and how upon its exterior its bears the powerful muscular masses and long limbs.

° Haeckel, "Anthropogenie."

As the organism develops, and while the mesoderm or gross construction is differentiating into skeleton, vascular system and muscular system, what progress is taking place in the covering and lining? Both embryology and comparative anatomy* show that cutaneous tissue and mucous membrane, ectoderm and endoderm, are the seat of extraordinary operations; that here are exhibited extremely singular processes, such processes being the various forms of inversion which these membranes undergo. Over the surface small depressions appear, which, sinking deeper, become pits, crypts or follicles, become inversions or invaginations or pouches. At first one would hardly think of identifying them with glands, much less with ganglions.

A kind of pouch-making process seems to prevail all over the body, inside and out, wherever the skin covering or lining with its extraordinary epidermis or epithelium is found. We know that in vertebrates the lungs and the liver and the urinary bladder are such pouches, inversions of the mucous membrane (endoderm, intestinal glandular layer). We know that the trachea of insects are inversions of the outer skin and their nitrogenous excretory organs are inversions of the intestine.† We know that in man the sweat glands, the lacrymal glands, the sebaceous glands, the cerumenous glands, and the mammæ, are all pouches or inversions of the outer skin.

Now comes the curious part of the teaching. That the liver, the lungs, or even the ovarium, should prove to be intestinal inversions seems not surprising; but when we find also that upon the outer skin appear depressions that are called eye spots and retina and ear vesicles and brain bladders, and that these are simply cutaneous inversions, amazement begins; yet such is the fact. The single urinary bladder of the body is an intestinal inversion, the double air bladders or pair of air bladders which become lungs are intestinal inversions lined with epithelium; the five brain bladders are simply cutaneous inversions lined with their epithelium.*

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* Balfour, Comparative Embryology; Wiedersheim, Comparative Anatomy of Vertebrates—two invaluable works.

† Jeffry Bell, Comparative Anatomy and Physiology. Also Balfour, Comp. Embryol.

This then is the truth that science has forced upon us; that the eye and the ear in their essential parts and the brain itself are pouch-like inversions of the ectoderm, as the lungs, the liver and the ovarum, etc., are all pouches or inversions of the endoderm. The kidneys and testes are of precisely similar origin, dermal inversions or pouches, either in lining or covering.†

While kidneys, testes and ovaries at one extremity of the organism are lined or made up of secreting and germinating epithelium, the retina, the internal ear, and the fore brain at the other extremity are lined with sensory and reflecting epithelium. The skin follicles, the organs of sense and the brain, are part of the outer skin series of inversions; the mucous follicles, the great glands and possibly the ovarum, part of the inner skin series.

Epidermis and epithelium, outer covering and inner lining are united in one. The inner skin is but a continuation of the outer, subject to different conditions, darkness and moisture instead of light and the shocks of the environment. From recent anatomy and embryology it may be laid down that the human organism, and every other consists of two parts, a frame and a skin. Even a tree or plant consists of these two essential parts, the wood or trunk, fibre, and the outer bark or parenchyme; an animal being double, tube formed instead of rod formed, has its frame or gross construction compound, and its bark or skin is continued down into the tube, forming a lining.

In both organisms is seen a kind of unit or life of the frame, a gross or crude life, and a unit or life of the skin—a culminating or refined life. The skin of a small plant, if examined, seems to culminate in leaves and flowers; the skin of the vegetative or internal part of a small animal,

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° Haeckel, "Anthropogenie." Balfour, "Comparative Embryology."

† The kidneys certainly of cutaneous origin; that of the testes disputed. Balfour gives bibliography. In the vertebrate embryo testes and ovaries seem to be connected with the mesoderm. The germ glands cannot be called inversions.

i. e., the mucous membrane, seems to culminate in these inversions—lungs, kidneys, and genitals, glandular inversions giving rise to gaseous and liquid products and ponderable forms or germs; while the skin of animal life seems to culminate in the organs of special sense and the brain bladders, ganglionic inversions producing color and vibration, light and shadow and phosphorescence, imponderable forms or spectra.

This greater skin, cutaneous and mucous, with all its involutions, constitutes a vast field of varied and wonderful products. The inner tract is covered with thick-set columnar epithelium, living and moving like a field of waving grain. This membrane produces first a protective mucus and various juices—œsophageal juice, gastric juice, intestinal juice; while the outer tract produces sweat and oil, its epithelium perpetually exuviating, and bringing forth no longer soft mucus but hardened horn and hair. Both the inner and outer field invert into glands producing strange substances —the ferment of saliva, the brine of tears, the bitter of gall, the nourishment of milk, and the formed eggs and semen, the highest inner product being these material germs.

On the outer and partly the inner field are the inversions of the taste bulbs, the olfactory epithelium of the nasal membrane, the retinal rod and cone epithelium, the vibrating epithelium of the ear, and the pyramidal epithelium of the cerebral cortex* where etherial or phosphorescent germs are found, shadows or spectra gathering in clusters and forming not ponderable embryos but imponderable spectra or "ideals." Cerebral shadows or spectra are the highest outer-skin products, as the material germs are the highest inner-skin products. The embryo created on the interior is semi-fluid and ponderable like the organism itself; the ideal or spectra created on the exterior is flitting and imponderable, it is, as it were, the shadow of the organism.

Of an indifferent sculptor with very handsome children, Michael Angelo said that he made better work by night than he did by day. The inner-skin products were better than the outer-skin products.

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* The gray matter of the brain is the original epithelium lining the cutaneous inversion. See paper by George Jacoby in the Medical Journal, May, 1888.

Man consists of a frame and a skin. The frame is the skeleton with the muscular and sanguiniferous systems; the skin is the highly involuted covering and lining of the frame. The function of the frame is to support and to nourish; the function of the skin throughout is production. Physicians and surgeons in ordinary deal with the human frame; gynæcologists, dermatologists, alienists, and neurologists deal with the human skin and its products. In this view, everything about the organism that is not muscle or bone or blood or aliment is skin or skin product, and it is the greater skin, the whole skin—cutaneous, mucous, and serous,—which is the common basis upon which these four specialists may be said to stand.

Skin products are the results of the thousand epithelial inversions and exuviations, and are solid or liquid or gaseous or viscid, are vaporous or etherial, are formed or unformed. Breath and the cutaneous exhalations are gaseous, or gaseous and vaporous; tears, urine, bile, and milk are fluid; mucus saliva, sebum, cerumen, are viscid or semisolid; hair and horn are solid and formed products; eggs and ova are viscid and formed; tastes and smells, sounds and spectra, are gaseous, vaporous, or etherial, and tend to be formed products.

Important products of the skin of the vegetative life in man are gaseous or viscid—the breath from the lungs, the ova and sperm from the genital glands. Important products from the skin of animal life in man are solid or etherial—hair and horn on the one hand, sensations and reflections from the sensory and ganglionic inversions on the other. The products of the skin of vegetative life are nearly as mysterious as those from the skin of animal life; an ovum or a spermatozoön is visible and tangible, can be measured and weighed; a breath cannot ordinarily be seen, but may be smelt and felt, can with difficulty be measured and weighed, and is the theme of the poet. Of the products of the animal life, a horn, a hair, a tooth, or a nail is solidly visible and tangible, measurable and ponderable; a sound, an odor, a taste, or a color or spectrum* can] hardly be weighed, but may be measured.—These are only one kind, and the highest kind, of skin products.

When, a hundred years ago, it was announced that the brain secreted thought as the liver secreted bile, the crudeness of the doctrine produced a shock; but it seems proved by the comparative anatomy and the comparative embryology of to-day that the skin, by its ectodermal inversions, produces, with its ganglionic epithelium the medullary-plate, the elements of thought, vibrations, and spectra, or sensations and reflections, in a way analagous to that in which the more superficial horn-plate brings forth hair, nails, and teeth, and in which the glandular inversions of the inner skin produce mucus and exhalations, and in which the terminal germ tissue brings forth germs.

Concentrate our attention for a moment on the two foci of the organism. The spermatic and ovarian germs are the elements of a new organism, that new and mysterious, but material man, the embryo. Sensations are the elements of the image, or ideal, the cerebral born man. The optic spectrum gives this shadow man a form, and the auricular vibration gives it a voice; taste, smell, and touch, and voluptuousness may all add their quota to make the sweet (or terrible) illusion, the shadow man or homunculus more real. That the shadow man can come out of its cerebral womb, and materialize and talk, is a matter of every-day observation. It is materialized in marble or paint; it talks in a book or periodical. No greater wonder need be sought; the truly wonderful lies in nature. Science with its research will show still greater wonders when it shall prove, as seems now likely, that sensations, emotions, and reflections are produced by the inverted epithelium—when it shows that ideals and phantasms, spectres and goblins and faries, spirits of good and spirits of evil, angels and demons, the godesses and the gods, are like teeth and hair, like breath and saliva, like tears and like sweat, like the egg and like seed, and like the embryo, pure, simple, and natural skin products.

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* To see a spectrum, look steadily at the sun an instant, then close the eyes tightly.

CLINIC ON NERVOUS DISEASES.

By M. A. STARR, M.D., Ph.D.,

Held at the Vanderbilt Clinic, March 24, 1888.

LEAD PARALYSIS.

GENTLEMEN:—The demonstration of a thing is worth all the descriptions in the world. You will remember that at our last two or three clinics we were talking about the reaction of degeneration, and you will further remember what we considered that reaction to be. I have here a man regarding whose condition I will say but little, as his complaint is one with which you are familiar, but I will proceed at once to demonstrate it. The patient is about thirty years of age, and states that he has had his present trouble about seven weeks; that it began with weakness in the shoulder; that he was unable to lift his arm, or to straighten his fingers. He says he has had no pain, no numbness, no tingling in the affected parts; he complains only of motor weakness. He also tells us that his condition is better than it was seven weeks ago. His occupation is that of carriage-painter. He had colic preceding the paralysis. His bowels are now free.

Gentlemen:—This man, as you hear, gives a straightforward history. He is a painter, works in varnish, and is engaged indoors. You know that painters who work within buildings, and especially those who work in close rooms, are more liable to develop lead poisoning than others. When you look at this man I think you will see at once that he has the appearance of being well-nourished, but is anæmic and pale. He has no apparent atrophy in any of the muscles, but if we ask him to go through certain movements, you will see he has deficiencies. When told to lift the right arm to the head, he is unable to do so. There is inability to abduct the arm. On the left side he succeeds in abducting the arm to a certain extent, but it is limited. When we look at him from behind and ask him to lift the arms, we see the degree of abduction made by the right arm is due to rotation of the scapula by the serratus magnus muscle. We know that complete abduction of the arm involves a complex process. It is performed partly by rotation of the scapula by means of the serratus magnus, and partly by the action of the deltoid. This man's scapula rotates normally, therefore the trouble with abduction is due to disturbance in the deltoid muscle. Now, besides the trouble in his shoulder he complains also of trouble in his arms. When I hold up his arm you will plainly see the condition known as drop-wrist. This is not as marked on the left as on the right side in this case. You will also notice very marked tremor when he attempts to hold the arms out by his own efforts. You will remember that at a previous clinic, when I spoke to you about tremor, I said it was a symptom of lead poisoning.

When I place my hand in his and ask him to grasp it, he does so with great feebleness; but when I correct the paralysis of the extensor muscles, he squeezes my hand so tightly as to cause pain. You see, therefore, although flexion is apparently impaired, it is not actually impaired in this case. The condition of paralysis is less marked on the left side than it is on the right.

No examination of a case of paralysis is complete until the electrical reaction has been tested. We will now proceed to test the electrical reaction with the Faradic current. We find that a moderately strong current causes the biceps to contract. The same strength of current applied over the deltoid produces no effect, and when I carry the strength of the current up to quite a high degree there is still no contraction. On the left side, on the contrary, there is a slight movement in the deltoid produced by the strong Faradic current.

We will now try the galvanic current, using this small portable chloride of silver battery. The normal reaction to galvanism is easily elicited. This we prove by first applying the negative pole over the patient's biceps, in which it causes at once perceptible contraction when the circuit is closed. That then gives us the cathodal closure contraction. Now we will apply the positive pole. The anodal closure contraction in the biceps muscle is less than the cathodal closure contraction. This man's biceps is not paralyzed, and we have in it the normal reaction to the electrical currents. Now we will try the deltoid which is paralyzed. Cathodal closure, as you see, gives but very slight contraction. Anodal closure gives very much greater contraction. Thus we find that the anodal closure contraction in the deltoid is greater than the cathodal closure contraction, which, as I have told you before, constitutes a reaction of degeneration. On the left side the difference between the cathodal and the anodal closure contraction is scarcely perceptible. It is difficult to say whether the one is greater than the other; at any rate the reaction cannot be normal because the cathode should produce distinctly greater contraction than the anode. We have then on the left side a condition known as partial reaction of degeneration, while in the right deltoid we have the condition known as total reaction of degeneration. This you see is the result of the electrical examination. It demonstrates at once that the condition in the deltoids is entirely different from that in the biceps.

You well know that the reaction of degeneration is due to disturbance either in the gray anterior horns of the spinal cord, or in the nerve outward from the cord to the muscle. In this case, which is distinctly one of lead palsy, we know that the lesion lies in the nerves and not in the cord. From the electrical reaction of the muscles we could not tell whether the lesion lay in the spinal cord or in the nerve; we could only tell by such a test that it did not lie in the brain or in the motor tract from the brain to the anterior gray horn of the cord. Through the demonstrations of pathology we have learned that the lesion in lead palsy is a neuritis; a neuritis which is limited to certain nerves going to the extensors of the arm and to the deltoid.

Now, what is the prognosis? The man has stopped his work, he has been ill only seven weeks, he has improved rapidly; therefore I think we may safely say that he will be completely well within three months of the time when his symptoms first manifested themselves. It is a light case of lead poisoning. Severe cases sometimes last longer. I have known them to be under treatment two years and then recover.

What is the treatment? The indications are first to eliminate the lead from the system so far as possible. That can be done by means of cathartics, especially by epsom salts, and by diuretics, especially iodide of potassium. Iodide of potash hastens the elimination of the lead through the kidneys. The second indication in treatment is to increase, if possible, the nutrition of the nerves which are degenerated. That can be accomplished by tonic treatment in general, especially by the use of strychnine in doses of one-sixtieth to one-forty-eighth of a grain, administered three times a day, or of fifteen to twenty minims of the tincture of nux vomica. The patient is taking nux vomica. The third indication is to hasten regeneration of the affected nerves by means of electricity. This is applied in the form of a constant galvanic current of moderate strength passed through the nerve. Now, the treatment which this patient has received is just that which I have mentioned. He has received the galvanic current passed from the neck down over the muscles, and in that way the current passes the entire length of the nerves affected with degeneration. But there is still another indication. You not only want to eliminate the lead and increase the regeneration of the nerve, but you should keep the muscles in good condition. When the nerve is degenerated the muscle to which it goes atrophies. If we do not do anything for these muscles they will go on atrophying, and then when the man's nerves have been regenerated and enable him to move the affected parts, he will find the muscles so weak that he will be disinclined to use them. Therefore the paralyzed muscles should be exercised by the aid of the interrupted galvanic current (not the constant galvanic current). You will see, therefore, that galvanism is used in this case in two different ways. First, the constant galvanic current is used to regenerate the nerve. Secondly, the interrupted galvanic current is applied to the affected muscles in order to give them exercise. Besides this we tell the patient to rub his muscles thoroughly with alcohol, salt water, or liniment of any kind. This patient has been usuing salt water. The benefit does not come so much from what we put on the limb as from the rubbing which the muscles receive. There is another little practical point in the treatment of cases of lead palsy which is of some interest. That is, that the atrophy of the muscles seems to progress more rapidly when the paralyzed muscles are put on the stretch. Now, when a man has lead poisoning, he is apt to go around with his wrists dropped, and you see at once that the paralyzed extensor muscles are those put on the stretch. You should, therefore, instruct such patients to carry their hands up against the chest in this way, so that the hand shall be kept in the position of extension.

CONFINEMENT PARALYLIS.

As there are two other cases which I wish to show you in addition to the one now present, I will hasten over the history. This little girl is eleven months old. The mother says that her right arm has been practically useless since she was born. I saw the child two weeks after its birth, when its condition was the same as to-day. She is unable to move the right arm as she does the left. The arm, you will observe, is held in a natural position, and when I take hold of it she attempts to pull it away. She moves it voluntarily. The right arm, on the contrary, hangs extended at the side, and apparently is not moved voluntarily at all. When I lift it and attempt to make her lift the arm, it drops heavily at her side when released. There is at least no voluntary movement at the shoulder. When I turn the hand up, supinate it, and then let it fall, we can see that it returns at once to the position of pronation. You can see that the child is unable to move the arm into the position of flexion at the elbow or into the position of supination. There is, therefore, not only paralysis at the shoulder, but also paralysis of flexion at the elbow and of supination. But when I hold the hand out you can see that the fingers move freely; when I place my finger into the palm of the child's hand it is clasped. There is, then, no trouble in either flexion or extension of the fingers and wrist. Now, I have made a careful electrical examination of the muscles (which I will not now repeat for want of time and because of the difficulty of such an examination in a child), and found the reaction of degeneration in the deltoid, biceps, coraco brachialis, brachialis anticus, and in the supinator longus muscles. I could not get at the supinator brevis because the arm was too fat. We have, then, paralysis of a peculiar group of muscles which I have just named. Now, this form of paralysis is known as Erb's paralysis, because Erb, of Heidelberg, was the first to describe it. It is, as you see, a paralysis of a group of muscles, while the other muscles of the arm escape.

To what may that be due? To one or two causes. It may be due to a lesion of the spinal cord at the level of the fifth cervical segment. As I have shown you by certain diagrams, that locality corresponds to the centres in the spinal cord which govern the group of muscles paralyzed in this case. The lesion at that point might, then, have caused this trouble; but there is also another lesion which sometimes produces it, namely, pressure exerted upon the fifth and sixth cervical nerve roots of the brachial plexus. How can we distinguish between the two? It can be done by the test of sensibility. If the lesion is in the anterior horns of the spinal cord; that is, if there is infantile paralysis, there will be no disturbance of sensation. If, however, the lesion is in the nerves containing sensory fibres there will be disturbance of sensation. Now, it is difficult to test the sensation of a child, and sometimes it can be done accurately only after repeated trials, but I think you can see in this case that when I scratch the right shoulder with a sharp instrument the child, although it looks around at what I am doing, pays but little attention, yet I have caused the skin to become quite red. When, on the contrary, I scratch the left shoulder, its attention is attracted at once, and on going a little further with the test the patient makes a face as if about to cry. I think we can fairly conclude from this test that the child has lost sensation on the outer side of the right arm. I have further proven this fact at former sittings by the use of electricity. The child will stand a very much stronger current without crying when it is applied to the outer side of the right arm than when it is applied to the left. There is disturbance of sensibility in the right arm. The trouble is not in the child's spinal cord, but it is in the fifth and sixth cervical nerves.

What is the cause of such paralysis? Usually the cause is a forcible delivery in the breach presentation. Now, this child presented by the breach and the labor went on until it was time for the head to be delivered. The midwife who was present extracted the child manually. How did she do it? In all probability just as you would have done, by introducing two fingers of the left hand and seizing the child on either side of the nose upon the superior maxillary bones. The next step is to extend the fingers of the right hand over the back of the child's neck and to make traction downward and backward. You will see as I put my fingers here on the side of the child's neck in this manner, as it would be done in the course of labor, I make pressure inward and downward above the clavicles, especially with the tips of my fingers; the pressure coming about the point of exit of the fifth and sixth cervical nerves. It is an interesting fact that in all the cases that I have seen of this disease (and I have seen quite a number) the paralysis has been on the right side; and you will notice that in this position the middle finger falls upon the right side of the patient's neck, and the index finger on the left side. That is, the longer finger is upon the right side, and is more likely to make downward pressure so as to involve these nerves. I think this may be the true explanation of the greater frequency of the accident on the right side of the body.

This form of paralysis was first described by Duchenne as confinement paralysis, and it has been recognized as such by all writers on paralysis of the nerves since that date. It is a paralysis due to pressure at the time of delivery upon the fifth and sixth cervical nerves. Those of you who have attended the surgical clinics will remember a similar condition in an adult at the Roosevelt Hospital. He had paralysis of the deltoid, of the biceps, of the coraco-brachialis, of the brachialis anticus and supinator longus muscles. The other muscles of the arm were somewhat weak, but they were not paralyzed. The muscles I have just named exhibited the reaction of degeneration, and from that fact and the further history, the man having received a stab-wound, Drs. Sands and Hartley made a diagnosis of division of the fifth and sixth cervical nerves. That diagnosis was established by the operation. They cut down and found the divided nerves, united them, and the man recovered. That case is worthy of association in your minds with this one, which is a typical case of confinement or Erb's paralysis.

What is the prognosis in this case? It is somewhat doubtful. I recall three cases in which recovery was spontaneous. I have seen two others in which recovery took place under electrical treatment. The electrical treatment has been carried on somewhat irregularly in the case of this child, and we cannot say that there has been any change for the better. I should say, therefore, that it is a somewhat unfavorable case, yet there is one thing which is favorable about it. You will notice in comparing one shoulder with the other that there is not a great amount of difference as to size; in other words, the right deltoid is not atrophied to any extent. This is a favorable point in prognosis. If the nerve were absolutely degenerated and severed, the deltoid would be thoroughly atrophied. When the nerve is injured in only a moderate degree, atrophy is less extreme. I suppose the atrophy in a well-nourished child like this one would be somewhat concealed by the amount of fat. Nevertheless, I think the fact that these muscles which are paralyzed have not atrophied wholly, but have grown to some extent, would make the prognosis in this case good. It may be two years before the child will get the use of its arm, but I think that eventually she will get it. The electrical treatment in a case of this kind is the same as in the one of lead palsy, just described. Give the constant current through the nerve and the interrupted current over the muscle so as to produce contraction.

EPILEPTIC INSANITY.

I spoke tc you, in my lecture on Thursday, of mental conditions arising in connection with epilepsy. You will have observed that the case, the history of which we have just obtained, illustrates two of those conditions very well. In the first place, it illustrates the condition of post-epileptic insanity. Post-epileptic insanity may take the form either of mania, or of melancholia, or of simple dementia, for they are all examples of psychical neuroses. In this man it evidently takes the form, to a certain extent, of melancholia. He is now thirty-two years old ; he says he has been sick at least twenty years. His mother, who has come with him, tells us that he has convulsions, which we at once recognize from her description as those of epilepsy, and that he has after each convulsion a short period of melancholia, is alarmed, is troubled, and makes exclamations at the times of his attacks in the form of prayer, and often cries, but is never violent. During the attacks and shortly afterwards he is perfectly unconscious, and later has no recollection of what had taken place. You will find other forms of psychical disturbance which are not so harmless, forms in which the individual after the fit passes into a state of acute mania, has hallucinations which terrify him, which make him fear that he is going to be injured, and which lead him to try to get away from those around him. If he is opposed in his attempts at escape, he will kill those in his way. The most violent of all lunatics are those who have temporary attacks of mania after epilepsy. They are perfectly ungovernable. That is a form of insanity in which restraint is absolutely neccessary ; you cannot get along without it. The state of mania, melancholia, or simple dementia, which occurs in these cases, lasts for a variable time. In this man it lasts ten minutes, in other cases fifteen or twenty minutes, and in still other cases four to five hours. During that time the individual is not at all legally responsible for what he does. He may go through an apparently well-planned attempt at murder, or a well-planned attempt at theft, or an attempt at any crime, and yet when he awakes from his sleep and comes out of this abnormal mental condition he has no recollection of anything he has done during that time. Now, that is a condition of post-epileptic insanity.

But there is another form of mental disturbance which develops in epilepsy. It is a gradual development of chronic dementia, which this man also shows. You can see from his appearance and judge from his mother's statements that this man's mind is not right. His memory is very poor. He cannot do anything in the way of work, evidently because no one will employ so weak-minded a person. This condition of weak-mindedness develops at a variable period in the course of epilepsy. If the epilepsy develops in early life, the child does not acquire knowledge readily, it grows up somewhat stupid; if the disease develops in the adult and continues for a long period, at the end of some twenty years you will find mental deterioration which goes on frequently to actual dementia.

I will not dwell upon the epileptic features of the case. You have heard the history; that the man has two kinds of attacks—grand-mal type, in which he has severe convulsions with biting of the tongue and convulsive movements all over the body; and the petit-mal type, in which he has simply temporary loss of consciousness, and does not go on to have a convulsion. These occur several times a week. The case is, therefore, a typical one of epilepsy. The aura which he has before the fit consists of dizziness, and is a very common aura, one which does not give us any indication as to the cause of the attack. The aura is a very important thing to investigate, for it is really an hallucination, and an hallucination is always produced by an irritation of the brain somewhere. If the aura is a constant one, we have an indication that irritation in the brain is localized at a definite point. If the aura consists in numbness beginning in the thumb and gradually going up the arm, and after the numbness has reached the body or head the patient goes into a fit, we know that there is an irritation in the brain which begins in the sensory area related to the arm. If the man has an aura of an epileptic fit consisting of a ball of fire, or of blue light, or of the appearance of certain figures moving around from one side of him until they reach the middle line, you may be sure that that man has an irritation in the posterior lobe in the visual area of the brain. If the man has an aura beginning as a very loud sound in the ear, you may be sure the irritation in the brain begins in the auditory area. So you see that the aura of an epileptic fit gives us information of the seat of the irritation, and it is an interesting fact that most of these auras occur in cases where organic disease of the cortex of the brain is to be suspected. But in a case like this one, in which the aura is only an indefinite sense of vertigo, which may occur in idiopathic epilepsy, the lesion is one which we do not know anything about. The treatment of such a case as this is similar to that of any case of epilepsy, and he is taking a mixture of the bromides of potassium, sodium, and ammonium, thirty grains three times a day. Under this line of treatment the number of attacks is being reduced. The treatment does not, however, appear to effect any change in the occurrence or duration of the melancholia period following the fit. When such a patient has maniacal attacks he requires constant watching.

PARANOIA.

I have still another case which illustrates an interesting mental condition, and one which those of you who have attended the lectures regularly will at once recognize as the history is called out. We have elicited from the man himself as distinct a history of his condition as we could desire. His age is about forty-five years. His sister, in reply to our questions, gives us no history of hereditary trouble. She says he has always been a somewhat simple-minded man. Simple in the Irish vocabulary means something a little different from what we naturally think of when that term is used, for in Ireland an imbecile is called a simple man, and it is probable that by the term simple she meant to say in as kind a word as possible that her brother has not been quite as strong mentally as other men. In his description of his case you will notice that he seems not to have the strength of character which most men have. It is possible that he has always been somewhat weak mentally, but his present mental trouble has come on recently, and is characterized by a delusion of persecution, a delusion which has increased rapidly in the course of three months, and which now, as you see, dominates his views of things, and which he has told us freely. He not only thinks certain men with whom he has worked have talked against him in his shop, but he thinks they have spread a malicious report around the city that he has seduced a young girl, and is the father of her child. He not only thinks that they have spread the report around the city, but that they have put it in the papers. He cannot find it in the English papers, he cannot read the German papers, but he thinks it is in the papers and therefore that it must be in the German papers, where he cannot detect it. It leads him not only to believe that everybody in the streets know of it, but that they single him out and look at him as he goes by. It is not probable, although he thinks it is so, that a lady walking along the street with her husband, would say anything about his "first-born," addressing him, a stranger, in that way. Therefore, it is not unlikely that he has hallucinations of sound. I could not get a history of hallucinations from his sister, but it is not impossible that he may have such. Hallucinations of sound are not at all uncommon in the course of paranoia. That is a form of degenerative insanity which is chronic and which is bad in its prognosis. The man is apparently harmless as yet, but his sister admits that he gets excited, that this delusion excites him at times, and on one occasion she had to lock him in. The only thing to do for a condition of mind of that kind is to put the man into a well-regulated asylum. There he will be prevented from doing any harm, which elsewhere he might take it into his head to do. As far as any active treatment for his condition goes, we know of nothing which will in any way alleviate it. We have to deal with the symptoms. You have heard his sister say that since he has been taking medicine the insomnia has subsided. Insomnia is a symptom of this form of insanity. We gave him bromides, but they failed, and we then gave him a mixture of bromides and chloral, which enabled him to sleep. He has also some slight amount of gastric catarrh. He has chronic dyspepsia, and he is in some degree constipated. When I examined him at a time previous to this he volunteered the information that the trouble in his stomach was due to something which his enemies had put into his food. You see, therefore, how his sensations are brought into relation with his dominant idea. He has symptoms of pain in the chest, of disturbance in the stomach, of insomnia, all of which he ascribes to the influence of outside enemies. His case illustrates what I told you in the course of lectures, that in the examination of a lunatic it is better to get at his history from a standpoint of a physician asking him medical questions, which will not direct his attention particularly to his delusion; it may be his desire to conceal that; but through his physical symptoms you can often get at his mental smyptoms. You saw that fact illustrated in this case. My first question was with regard to his health, and his first reply was with regard to the pain, and it was accompanied with the statement that "they" gave him the pain. That simple remark gave his whole story away. It is an interesting fact that in the examination of a lunatic you can often get at his delusion much better indirectly than directly. The characteristic feature of this case is the existence of a systematized delusion of persecution which dominates, but does impair his mental processes; and this is the characteristic of paranoia.

Reviews.

HYSTERIA AND BRAIN-TUMOR. By Mary Putnam Jacobi, M.D. pp. 213. G. P. Putnam's Sons, 1888.

In this volume the author presents seven essays which have previously appeared separately in numerous publications or have been read at society meetings.

These essays are entitled: Some Considerations of Hysteria; Tumors of the Brain; Note on the Special Liability to Loss of Nouns in Aphasia; Case of Nocturnal Rotary Spasm; The Prophylaxis of Insanity; Antagonism between Medicines and between Remedies and Diseases; and Hysterical Locomotor Ataxia.

Of these, the first two deserve more than a passing notice. The author's depth of thought and closeness of observation are nowhere made more apparent than in the opening essay on Hysteria. Many original and plausible theories are advanced and numerous cases are cited to bear out the author's views.

After a few remarks on the conditions fundamental to hysteria, the writer goes on to discuss the origin of the sensory and motor symptoms. The sensory symptoms—anæsthesia and pain—are attributed respectively to privation of the blood supply of the sensory centres, and to excessive centripetal irritations which are distributed throughout all the receiving stations of the cerebro-spinal axis. The motor symptoms—paralysis and convulsion—are attributed respectively to depression, or inhibition of the function of the cortical motor centres in liberating energy in motor tracts in response to intracerebral stimulus, and to a diminished control over the subcortical motor centres by the cortical centres which normally inhibit them in part. The causes which lead to these abnormal conditions are fully discussed and explained, and the various forms of pain, paralysis and convulsions are entered into with a minuteness characteristic of the author. The psychical phenomena of hysteria are carefully studied, and the difference between hysterical neuroses and neuroses originating in medullary spinal centres is made apparent. The latter part of the essay is devoted to the diagnosis and treatment of hysteria. In regard to the latter, electricity, massage, gymnastics, the health lift, together with the usual medicinal remedies, are recommended. The removal of the ovaries for intractable cases of hysteria is advised in two classes of cases. One, where the ovaries are diseased; the other, where the ovaries are normal, but in which normal menstruation causes intolerable irritation.

It cannot be denied that the removal of diseased ovaries is often beneficial in the treatment of hysteria, but where the ovaries are normal even our most ardent ovariotomists would be inclined to respect them. The author admits that the operation is not often immediately successful, either because menstruation persists, or because the nervous phenomena persist, and claims that the operation can only be considered unsuccessful after the lapse of two years.

The article on Brain Tumor, though not containing as much original thought, is of as great, if not of greater, importance than the preceding essay. It is almost a complete analysis of the subject. The symptoms are exhaustively considered in relation to the location of the lesions, and the results are carefully tabulated, so that a complete understanding of the subject may be obtained almost at a glance.

The remaining essays are treated with that careful consideration characteristic of the author.

As a whole, the work is a valuable addition to neurological science.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, May 1st, 1888.

The President, GEORGE W. JACOBY, M.D., in the Chair.

DR. LEONARD WEBER presented a paper upon

PARALYSIS AGITANS, WITH CASES.

The disease had been first described by Parkinson in 1817. Charcot had differentiated it from disseminated sclerosis. Progressive tremor and muscular weakness affecting first one limb and subsequently spreading to other limbs were its chief symptoms. According to Charcot, the head was always respected. There was apparent diminution of muscular force, and in well-advanced cases movements were slow and uncertain. There was loss of faculty of equilibrium. In trying to walk the patient would run, appearing to be running after his lost centre of gravity. There was bending of the body to a stooping posture and an immobile facial expression with a fixed stare. Death occurs from marasmus or intercurrent disease. The onset was insidious, a hand, a foot, or even a thumb had been first affected. The crossed form is rare, and the hemiplegic form was more common than the paraplegic. Charcot referred to cases in which pain had been the first symptom, tremor following later on. Deglutition was not interfered with, the bladder and rectum but seldom. There might be annoying cramps, but contractures did not occur. Deformity might be present, giving to the hands the aspect of arthritis deformans. Glycosuria had been noted. This complication had been present in a case of his own. In an advanced stage there was disturbance both of thought and action. Neuralgias were present with a sense of fatigue and restlessness, requiring frequent change in position. There was a subjective feeling of heat.

Marked melancholia or general hallucinations may be present with even maniacal attacks. Remak had differentiated a cerebral and spinal form. In two of his own cases cerebral, and in two others spinal symptoms had predominated. Nothing definite to account for the tremor had been found in either the brain or the cord. It is a disease which affects the lower strata of society particularly, and men rather than women. It is a disease of advanced life, though Meschede had reported a case in a boy of twelve years, where it had developed after a kick in the face by a horse; and Duchenne one in a man of twenty. It is said to be more frequent among English and American people than among those of other nations. Powerful emotional and moral shocks have produced it, also irritation of the peripheral nerves by traumatism. Prolonged damp cold is a cause; also, in the reader's opinion, sexual excess. The reader had not observed that syphilis had anything to do with its production. Heredity is said not to be a factor, but in one of his own cases the patient's father and two brothers, and in another one brother, had been affected.

Arsenic, ergot, and nitrate of silver had no retarding action. The comfort of the patient may be increased by using Brown-Séquard's bromide mixture, leaving out the iodide of potassium. Hyoscyamine had not been of any value in the reader's experience, producing head symptoms and even maniacal attacks. He had used the amorphous preparation in one-tenth grain doses. Antipyrin in fifteen to twenty grain doses gave rest at night in some cases; chloral, too, had been useful, but the effect of both had been transitory. He had found paraldehyde superior to either as a hypnotic in these cases. It was given in emulsion, fifteen to thirty grains to a dose. It did not affect the stomach, did not weaken the heart, and did not lose its efficiency by use. The galvanic current relieves the feeling of fatigue.

Tepid half baths, with cold affusions to the head and spine, are even more effective and prompt. The reader has had twelve cases, five of whom had been under his care sufficiently long for study. He briefly referred to these cases.

Case I. was a man of seventy years, in whom the disease had existed for twenty-four years. The patient is still able to get about. The disease was produced by cold.

Case II. was a woman of seventy years, first seen by the reader in 1884. This patient was a very cultivated lady, with fine intellect, and a great knowledge of men and affairs. The disease dated from 1876, and an occasion upon which she had received news of the illness of a loved daughter. The expression of face in this case was markedly sad and immobile. There was little neuralgia, no spastic paralysis, and no contractures. A peculiar feature was atrophy of the epidermis of the hands, forearm, feet, and legs. Death occurred in 1887 from pneumonia. Nightly suffering had been in this case relieved by paraldehyde during six months.

Case III. was a man of fifty-three years, a German, addicted to drinking and smoking in excess. The disease had existed for seven years and glycosuria was present. The weight, which had been three hundred and twenty pounds, had been reduced, but not below two hundred and eighty pounds. Œdema of the legs had been present in this case for six months. Paraldehyde in a dose of thirty grains at bedtime gave a good night's rest.

Case IV., a German, aged sixty-three years, was a market dealer, presenting as causes for his illness exposure to rheumatic influences connected with his business, and sexual excess. The right hand had been first affected, and in the course of the following year the arm and shoulder. The right leg had been seized during the last six months. There was paræsthesia but no neuralgia in this case.

Case V. was a German, aged seventy-two years, whose father and two brothers also suffered from the disease. The left upper and lower extremities only were affected, thus differing from the usual hemiplegic form, which affects the right side. It was to be remarked, however, that this patient was left-handed. The shaking was violent in this case. The patient was easily excited, and suffered with vertigo and headache.

Dr. C. L. Dana felt that the obscure points of pathology and treatment should receive consideration. We do not know the nature of the disease nor how to cure it. It was his opinion that paralysis agitans is a degenerative disorder of the voluntary motor system. The sensory symptoms and the mental inquietude are explained by the close relations between the motor and sensory centres. The speaker had had twenty cases under his own observation, and had seen thirteen others in the Bellevue out-door department, 2 had been between thirty and forty years of age, 5 between forty and fifty, 13 between fifty and sixty, 9 between sixty and seventy, and 4 between seventy and eighty. Of 31, 20 had been males and 11 females. Of 34 in whom the race had been mentioned, 26 had been Irish, 6 German, 1 Russian, and 1 born in the United States. The Celtic element had thus greatly preponderated, a fact not entirely explained by the large percentage of Irish attendants at this dispensary. The disease seemed increasing in frequency. From 1876 to 1879 the Bellevue dispensary had presented but 7 cases of paralysis agitans in a total of 2,300 cases; while from 1885 to 1888, 11 were recorded out of a total of 2,200.

He could corroborate the statement that rheumatic influences are active in its production. He too had had a case complicated with diabetes, and he had had four cases in which the head had been affected. He thought this not uncommon. It might be shown in the muscles of the angles of the mouth and tongue. In treatment, hyoscyamine had given the best results. He had used, however, not the amorphous but the crystalline form in $\frac{1}{100}$ to $\frac{1}{60}$ grain doses. Given thus, it has never failed to exert a palliative action.

Dr. M. A. Starr called attention to a monograph upon paralysis agitans, by Anton Heimann, published in Berlin six weeks ago, and only last week received here, in which the theory of cortical origin referred to by Dr. Dana was supported by the report of a case. In this case, which had developed under the writer's observation, the four limbs were affected. The lesion could be located as hemorrhage into the internal capsule. Coincidently the paralysis agitans had ceased upon the hemiplegic side. The inference was that the paralysis agitans was due to impulses from the cortex through the internal capsule, and that the irritated cortical centres were cut off from the muscles by the injury. Gowers had demonstrated, too, that the rate of vibration in paralysis agitans was eight per second. It is known that irritation of the cortex, of a moderate degree, in monkeys, produced tremor of the muscles characterized by eight vibrations per second.

In regard to the extension of the tremor to the head: while Charcot had stated that the head never was affected, the majority of German observers among whom were Seeligmüller, Strumpell, and Heimann, added to Gowers, in his new book, had found the head affected in a considerable proportion of cases. The speaker had had seven cases of paralysis agitans under his care during the last two years. The tremor had affected the head in two of these cases. He could join Dr. Dana in his endorsement of hyoscyamine, given in the crystalline form and $\frac{1}{100}$ grain doses. This remedy had had a good effect in all of the cases which he had seen.

Dr. L. C. GRAY considered the origin of the tremor very obscure. He recalled a case presenting the typical symptoms of disseminated sclerosis. The patient was under observation in the hospital for a year and a half, and the post-mortem showed nothing but a cortical meningitis, no lesion in the brain or cord. The speaker did not agree with Dr. Weber that the diagnosis between paralysis agitans and disseminated sclerosis was in all cases easy to make. Voluntary tremor might be present in both, and in the earlier stages diagnosis might be impossible. One of the most typical cases which he had known had had movements of the head. This case was that of a woman of seventy-five years, who had had paralysis agitans for thirty years. It had started one day, when on a ferry-boat, upon seeing a baby fall into the water, and buoyed up by its long clothes float down the stream. Upon getting home she had found her upper lip quivering like that of a rabbit. This had been the commencement of a widespread affection.

Dr. GRÆME HAMMOND had seen three or four typical cases of face tremor. In a case of which he had the brain, the paralysis agitans had been present for eight years. There had been no change in the voice, but during the last year there had been paralysis of the tongue, lips, throat, and heart, which finally terminated life. Post-mortem there was absolutely nothing found ; no softening in the medulla and no change in the cortex. A few years ago, the speaker had collected all the reports of autopsies which he could find of cases of athetosis, chorea, ataxic tremor, and paralysis agitans.

Where any lesion was found it occupied the position of the gray nerve cells of the optic thalamus, corpus striatum or cortex. Where spastic spasm was present, either combined with the tremor, or alone, the white fibres of the internal capsule were affected as well. Where spastic spasm was not present, the lesion did not affect these tracts.

Dr. ROCKWELL had had two or three cases under forty years. He thought that the younger cases responded best to palliative treatment. He recalled a case from Peru which he had treated with hyoscyamine $\frac{1}{100}$ grain dose and the galvanic current. This patient had returned to his home apparently cured. The symptoms returned, he came back, was again subjected to the treatment, was again cured and had remained well for a year.

Dr. SACHS regarded Dr. Heiman's argument as ingenious but not conclusive. Hemorrhage into the internal capsule is the commonest cause of hemiplegia in middle life. Hemiplegia is not very uncommon in the course of paralysis agitans and it does not interfere with the tremor in the usual case. In regard to the involvement of the head, he had a case now under treatment in which the head was affected. He used hyoscyamine in the crystalline form, and had found it so reliable as a palliative that no other drug was required in his experience. Even in severe cases the tremor was lessened by means of it.

Dr. Petersen referred to the fact that when in the Poughkeepsie asylum, while administering the hydrobromate of hyoscyamus for headache, he had incidentally relieved the tremor of paralysis agitans.

Dr. Weber thought that Charcot's rule for the involvement of the head would be found in the main correct. General shaking would move the head, but tremor of the intrinsic muscles he thought rare. In regard to Dr. Rockwell's case, he would hesitate about pronouncing a cure. Paralysis not infrequently presented intermissions of a year or two, but would ultimately return. In regard to the hyoscyamine, he had tried it some years ago; possibly the quality had not been as good as that now obtained. He would again give it a trial in the crystalline form.

NEW YORK NEUROLOGICAL SOCIETY.

Meeting of June 5th, 1888.

The President, Dr. George W. Jacoby, in the Chair.

NOTES ON THE PRINCIPLES OF CRANIOMETRY.

Dr. Frederick Petersen read a paper thus entitled. After a review of craniometric nomenclature, the reader stated that, while individual convolutions exerted no specific influence upon the bones of the head, the shape of the skull was modified in correspondence with the gross divisions of the brain beneath it. The left temporal bone was said to be depressed in congenital aphasia. In infantile spastic hemiplegia there was flattening of the side of the skull oppothe paralyzed part. Cerebral localization had been concerned mainly with motor and sensory functions. Ideational localization had yet to be developed. In his own opinion, the tempero-sphenoidal lobes, and perhaps the occipital, contained cortical centres for depressing emotions. Musical ideas and auditory memories had their origin in the tempero-sphenoidal lobes. Benedikt had reduced craniometry to a science, showing that the skull was built upon crystallographic principles. The measurements taken should be sufficient to reconstruct the skull. Triangulation of the skull should be required in asylums in the case of every patient, and in prisons in the case of every criminal. We were behind European countries in this matter. Even in Italy, fourteen measurements were required for asylum records. The reader thought that eleven measurements at least should be made: 1. The circumference of the skull. 2. The naso-occipital arc. 3. The naso-bregmatic arc. 4. The bregmatic-lambdoid. 5. The binauricular. 6. The antero-posterior diameter, taken from the glabella to the maximal occipital point. 7. The greatest transverse diameter. 8. The binauricular diameter. 9. The two auricular-bregmatic radii. 10. The facial length. 11. The greatest height of the skull. Only a pair of calipers, a tape-measure, and a strip of lead two feet long were required. For more detailed measurements other instruments were necessary. Benedikt's calipers were recommended. The pathological and forensic importance of such measurements was shown by the fact that minimal and maximal dimensions were more common among the insane and criminal classes than among other people. The bregmatic-lambdoid arc was said to be shortened in epilepsy. The reader referred to a hundred cases of his own observed at Hudson River State Hospital, at Ponghkeepsie, in which asymetry had been observed.

PROGRESSIVE MUSCULAR ATROPHY IN ANÆSTHESIA.

Dr. J. A. BOOTH reported the case of a man, forty-two years of age, a shoemaker by trade, who was still under observation. There was no family history of nervous disorders, nor any history of alcoholism or syphilis in the case. The patient had been married eighteen years, and had had two children; one, a girl of five, had never walked.The affection had commenced in October, 1878, with general weakness and weakness in the arms and hands. In January, 1879, the patient's voice had commenced to be husky. Six months later he complained of a feeling of cold and numbness in the left shoulder and side of the neck, with subsequent decrease in size. The atrophy, commencing in the deltoid, had spread to other muscles of the trunk and upper extremities. At the present time the patient weighed 155 pounds. There was marked sinking in of both shoulders, also weakness of the upper extremities, with marked atrophy of the interossei. There were scars and abrasions about the hands and a scar on the neck. The patient stated that he did not know where these injuries had been received, that he had not felt them. There was no ataxia of the gait or upon standing. The voice was harsh, and the left side of the palate was paretic, the uvula being drawn to the right. The larynx had been examined by Dr. A. H. Smith. The left superior constrictor, the left palato-pharyngeus, and the adductors of the left vocal band were paralyzed. There was slight deviation of the tongue to the right. There were marked fibrillary contractions in the atrophied muscles. The patellar reflexes were exaggerated. There was sluggishness of the accommodation, but no change in the visual field and no diplopia. Taste, smell, and hearing were not impaired. It was apparently a case of progressive muscular atrophy with bulbar symptoms. The reader called attention to the sensory impairment as an unusual complication, and suggested, to account for the anæsthesia and analgesia, a lesion in the peduncle or pons on the right side.

Dr. STARR remarked that the anomaly mentioned had been recorded by Ross and by Gowers in their text-books. In cases of this character, post-mortem examinations had shown abnormal cavities in the cord, due chiefly to the degeneration of gliomatous tumors. Schultze had described cases, Bäumler also in her article upon syringomyelia. The case reported by Dr. Booth was, in the speaker's opinion, a case of this kind. The fact that the senses of touch, pain, and temperature were all abolished would support this view. The sense of touch sometimes escaped in syringomyelia, but not always. The three tracts were found in the formatio-reticularis of the medulla and pons, and extended through at least one-fourth of its extent. A lesion affecting them all would involve also the cranial nerves passing through this part. The symptoms reported could be more satisfactorily explained by a lesion in the cord and by considering the case as one of syringomyelia.

DEGENERATION OF THE PERIPHERAL NERVES IN LOCOMOTOR ATAXIA.*

Dr. J. C. **SHAW** reported the case of a man, forty-seven years old, who had a typical locomotor ataxia. Following an attack of hæmaturia, he had two epileptic seizures, and had died the following day. At the post-mortem examination the hæmorrhage was found to have come from the right kidney. Pieces of the sciatic, plantar, and popliteal nerves had been removed for examination, part of the specimens being stained with osmic acid and part with bichromate of potassium solution. Changes were found which the reader considered distinct from the Wallerian degeneration. These changes consisted in granular degeneration, liquefaction, and even absorption of the myeline sheath, with persistence of the axis-cylinder and in some places a collapsed sheath of Schwann.

° For full report of the case see page 433.

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Original Articles

A CONTRIBUTION TO THE PATHOLOGY OF TROPHIC DISORDERS OF THE MUSCULAR SYSTEM.*

By DAVID INGLIS, M.D.,

PROFESSOR OF NERVOUS AND MENTAL DISEASES IN THE DETROIT COLLEGE OF MEDICINE.

THE patients whom you have just seen present the following history:

Their grandparents lived to old age and, as far as can be learned, both were healthy themselves, and had brothers and sisters of excellent physique. They had a family consisting of one son and four daughters. This son, who died of accidental injury at the age of 45, was healthy and left a family of thirteen children, all healthy. The four daughters are themselves in good health, are all married and have families of boys and girls. The family of one daughter has as yet shown none of the hereditary tendencies now to be related. Of the remaining daughters, one, Eliza, now living in England, has been twice married and has had four sons and ten daughters; her daughters are healthy, but her oldest son from his earliest infancy showed marked muscular atrophy; he has however grown up and married, but is, at the present time, an entirely helpless cripple from the gradual increase of the disease. A younger son by her second husband has also been affected, and is now helpless; he is said to be apparently very muscular.

Of Eliza's daughters several are married; the eldest daughter has a son who is similarly affected.

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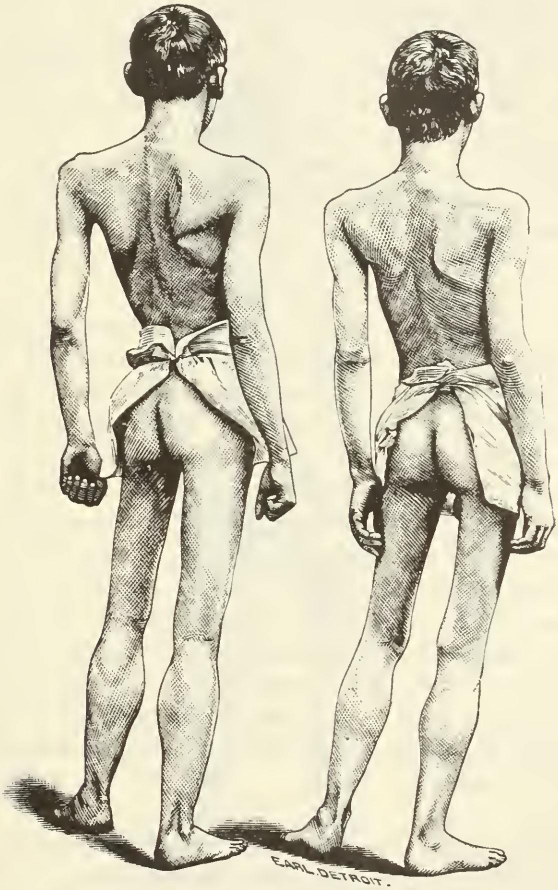
* Read before the Detroit Medical and Library Association.

The next sister of Eliza, Mrs. K., has a family of four sons and one daughter, the latter healthy; of the sons, Herbert, the oldest, now aged 20 years, showed, from the time he began to walk, the first evidences of his disease. The parents noticed that the muscles of his arms instead of increasing seemed rather to grow smaller. When he had learned to walk, the attitude was with the shoulders held well back, and the shoulder blades were unusually prominent. Close questioning reveals no history either in this or in the two following of any sudden paralysis or other evidence of attacks of poliomyelitis anterior. The lad learned to walk at about the usual age, grew at the usual rate, and is now a tall young fellow. It is to be remarked that coincidently with the fact of extensive muscular atrophy there has taken place a certain amount of natural muscular growth. There is no defect in the bony development; and the muscles of the arms, while they have been atrophied since infancy, have yet grown both in length and size. The patient now presents the characteristic attitude which you have seen; his shoulders are thrown far back when standing, the spine curved with the convexity forward, the feet spread apart, one always in advance of the other, and one heel always off the ground. He rises from the sitting position by lifting as much of his weight as possible by the arms braced upon his knees; then, with feet widely extended and an extraordinary curving of the spine, he alternately braces his hands further and further up his thighs, and so climbs up his thighs. From lying on the floor, the process of standing is attained with difficulty, and in a similar manner. He first gets on his feet, legs extended on the thighs; then, from resting on feet and hands, goes through the same process of climbing.

Physical examination shows very extensive muscular atrophy, symmetrical in its distribution. The erector muscles of the back are most involved; those of the hand, forearm, arm, and shoulder are all involved; in grasping firmly, the pronators act to excess. The muscles of the buttocks and thighs are perhaps as well developed as could be expected, and the calves are developed out of all proportion.

They stand out rounded and hard, but the appearance of strength is fallacious; the muscles are nearly as hard and firm when at ease as when in contraction, and offer but feeble resistance.



HERBERT. FRED.

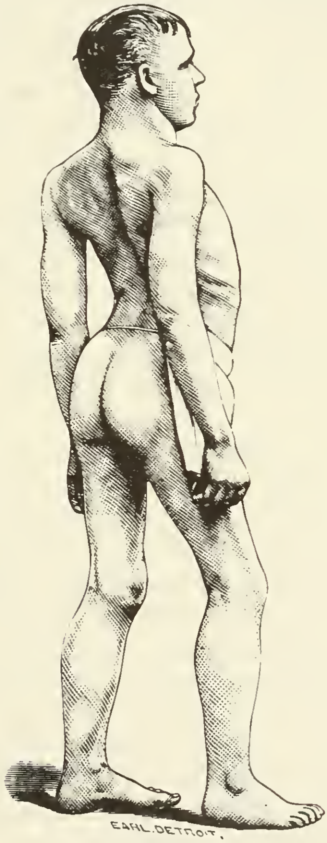
Owing to relative shortening, the feet are habitually in a position of slight talipes equinus, thus preventing his standing with both heels touching the floor. The picture, then, is complete of pseudohypertrophy of the muscles of the legs, with general muscular atrophy of the muscles of the back and upper extremities. Before leaving this case, I should state that the boy's general health has been good; he has had no symptoms indicating any disorders of sensation or of voluntary motion save the weakness clearly caused by the state of the muscles themselves; his intelligence is good. Tendon reflexes absent.

The next brother, Fred, now aged 18, presents, as you see, as nearly as possible, the same appearance, the same gait and attitude, the same distribution of atrophied muscles, the same pseudohypertrophy of the calves. It would be impossible to more closely duplicate the entire group of symptoms, both as regards their character and extent; yet the history of the case presents this curious feature that in the case of Fred the *atrophic* changes have all taken place since the age of twelve years.

I am inclined to believe that the pseudohypertrophy was going on for a long period before that, for the boy states that he was always easily knocked down, but he was considered by his parents perfectly sound up to the age of twelve. He was vigorous and active as any young lad and with the full use of his arms and hands. Without any apparent illness, there gradually came on a weakness and wasting of the hands, arms and back. The order in which the muscles were affected, as far as can be learned, was first those of the back, then the forearm, the flexors of the arm, and last the shoulder muscles. It would be possible to claim that, in the elder brother, the apparent atrophy is simply the result of lack of development, but in this case the process has been one of distinct atrophy, ending in an identical condition; here, too, the tendon reflexes are absent.

Turning now to the third married sister, Mrs. King, we find that she has three sons and four daughters; her daughters are robust and healthy. Of her sons, George, now aged 19 years, is here present, and presents an appearance even more striking than the other two. His height is 5 feet 4$\frac{1}{2}$ inches; his weight 130 pounds; but the first impression of him is that of an extraordinarily muscular young fellow. On physical examination we find an enormous pseudohypertrophy of the calves, buttocks, arms and forearms—

symmetrical; the erectors of the spine, on the contrary, are atrophied. The tendon reflexes and electrical reactions are the same as in his two cousins. Strikingly different as is his appearance the net result is the same. The same gait and attitude, the same method of attaining the erect position, the same muscular weakness. The history of this case also dates back to early infancy. A fat baby, he was supposed to be slow in learning to walk from his weight. When he did walk the gait was peculiar and unsteady. Hypertrophy of the calves was followed by that of the buttocks, then the triceps, and lastly the forearms.



GEORGE.

Some ten years ago this lad developed a bronchocele.

which disappeared under treatment, but began to return two years ago and has now reached a large size, bilateral and soft. The oldest brother of George I also present, showing the malformation of his foot. He is a hard-working young man, aged 28, with no history of pseudo-hypertrophy or atrophy save that the deformity is the result of a stroke of paralysis occurring when he was about about a year old. His mother states that he could walk well at the early age of ten months, but shortly after was noticed to lie wherever he was put. By degrees it was noticed that he had lost the use of his right leg entirely, from the hip down. Later the control returned, and at seventeen months he began to walk again. Recognizing the difficulty of making a positive diagnosis at this late date, it yet seems fair to believe that this insidious paralysis of the leg, evidently of rapid onset followed by recovery of control in the main, but also by contracture and permanent deformity, was an attack of poliomyelitis anterior.

In none of the cases fibrillary twitching or paræsthesiæ were manifested.

Having thus presented the patient with this interesting family history, several questions present themselves. Notably the extraordinary uniformity of hereditary transmission through the female line to the male children. I am in correspondence with the members of the family in England, and hope soon to be able to elicit information upon other points, *e. g.*, whether the married cripple has children, and if so, what hereditary peculiarities may have occurred. It is interesting to note the fact that the mother, Eliza, being twice married, has children by both husbands, presenting the same inherited tendencies. The disease has now begun to show itself in the children's children, and the mysterious question awaits solution: what was the cause of the fatal gift of the mother to her daughters, and wherein lies the cause that only the daughters should transmit the inheritance which could only develop itself in the sons. It is evident that the explanation so frequently given of greater frequency of certain diseases in males, to wit, greater exposure to hardships, does not here apply. An interesting series of cases of hereditary hæmophilia, showing an almost identical order of transmission, and involving five generations, is reported in the *London Lancet* of November, 1886.

Leaving the question of heredity, the problem at once presents itself: What is the bond of union between these varied cases—infantile atrophy, pseudohypertrophic paralysis, progressive muscular atrophy and bronchocele? The question has additional interest from the fact that there is at present a strong tendency to separate pseudohypertrophy from the atrophies of spinal origin, and to regard it as a purely muscular affection. Such a series of cases as that now presented seems to me to possess a distinct value in the elucidation of this question, which is one of vital importance if we ever hope to solve the problem of the successful treatment of these intractable cases.

The group embracing infantile atrophy, progressive muscular atrophy and pseudohypertrophic paralysis possess one common factor. Various as their symptoms, they are all affections of the motor apparatus, involving sooner or later the atrophy of groups of muscles with consequent loss of function. Before entering upon the bearing of this series of cases, let us recall the facts which have been established concerning the pathological changes in these affections. Beginning with infantile paralysis: The frequency of the occurrence of poliomyelitis anterior has enabled us to obtain very conclusive proof that the disease is distinctly an acute process by which a rapid atrophy of the multipolar cells of the anterior cornua is induced, which is accompanied by an immediate loss of function and followed by atrophy of certain groups of muscles. The acuteness of onset, the sudden comparatively widespread paralysis, the fact that the atrophy does not involve all of the muscles first paralyzed, and the fact that the atrophy follows the paralysis at a comparatively late date, proves to demonstration that the atrophic change has its starting point in the cord, that the muscular degeneration is secondary. While the atrophied muscles present a constant pathological appearance, the gray matter of the cord presents just as constant evidences of morbid process.

In progressive muscular atrophy the pathology is not disposed of so summarily. The course of the disease being slow it becomes difficult to state which post-mortem appearances are primary and which secondary.

All authors are agreed that the atrophied muscles give evidence of a chronic interstitial change, ordinarily termed a myositis. It is indeed questionable whether the process deserves to be thus considered as in any way inflammatory, being rather a substitution of a less highly organized tissue, but at any rate the process ends in a cirrhosis—muscle fibre disappears, connective tissue remains. The question hinges upon the lesions of the nerves and cord. A large number of observations have been made in which lesions of the sympathetic and of the spinal roots, as well as of the antero-lateral columns, have occasionally been noted, but the preponderance of evidence is clearly that the anterior cornua are in the greater number of cases the seat of degenerative changes. Ross believes that the diseased process begins in the gray matter about the central canal and spreads especially toward the anterior cornua and also perpendicularly, a theory which plausibly explains the erratic manner in which groups of muscles are involved as well as occasional sensory disturbances. The atrophy of the cells of the anterior cornua in greater or less number is the common, but it is to be borne in mind that, leaving out of account observations made many years ago, at a time when methods of examination were imperfect, there still remain cases in which observers of recognized ability have found the cord intact. Such cases demand consideration in formulating a theory of the disease.

The pathology of pseudohypertrophic paralysis presents still greater difficulty. The remarkable gross appearance of the muscular masses very naturally attracts attention. Frequent examination has shown that the process is, in this case as in the last, one of substitution. The normal connective tissue of the muscular substance is enormously increased, and amid the mass of connective tissue the muscular elements waste away and at last disappear. In the newly formed connective tissue occurs a more or less marked formation of fatty tissue. These newer and more lowly organized tissues give the bulk to the muscle, but the atrophy of the true muscular substance is the same as in the progressive muscular atrophy. At a later period the adipose tissue, and even a considerable part of the connective tissue, may in time waste away, leaving the gross appearance, as well as the microscopical, indistinguishable from that resulting from progressive muscular atrophy. The pathological changes in the nervous apparatus have been examined in a much smaller number of cases than those of progressive muscular atrophy, and vary widely. In the greater number of cases lesions of the cord have been observed; but while in some atrophy of the anterior cornua has been present, in several the lesion has consisted in changes in the form and place of the central canal, while in others the cord has seemed intact.

The problem to be solved is, What is the relation between the changes in the muscle and those in the cord? Are we, with our present knowledge, justified in believing that progressive muscular atrophy is due to a primary lesion of the gray matter of the cord, to which the muscle changes are secondary? and, if so, on what ground are we to separate pseudohypertrophy from it, and consider the muscular change primary, those in the cord as non-essential?

I hold that, at whatever conclusion we arrive, the two diseases should be classed together. Clearly pseudohypertrophic paralysis is allied in the most intimate manner with lesions undoubtedly spinal; for, in cases of infantile paralysis, instead of the usual process of simple atrophy, this same pseudohypertrophy at times occurs.

It is evident, then, that an atrophic lesion in the cord can cause just that form of muscular change which does occur in pseudohypertrophied paralysis. This same process of lifomatosis also occurs in the progressive muscular atrophy of adults.

In order to throw some further light upon the enigma thus presented, it will be well to recall the pathological changes which occur in certain atrophic disorders due to toxic agents. In the case of alcoholic paraplegia, the symptoms are those of combined ataxia and progressive muscular atrophy, both of which groups may reach an extreme grade, so that the atrophy may be as complete as in a far advanced case of progressive muscular atrophy. When we seek for the seat of the pathological changes thus developed, we find the brunt of the affection has fallen upon the *conducting* nerves ; while the muscle shows the usual atrophic changes, the nerves show evidences of widespread neuritis ; but the cord in this case also seems to be intact.

In lead paralysis, likewise, the pathological changes seem usually to prove the existence of a neuritis ; the atrophic muscular changes are present.

We have before us the enigma of atrophy of the muscle (varied in some cases by an apparent but fictitious hypertrophy, which does not alter the fact of a real atrophy). The atrophy is constant, and the connected nervous lesions have an inconstant location. Yet inconstant as are the lesions they still have this in common ; they are all to be found either in the cells of the anterior cornua, in the course of the axis cylinders or in the muscular elements.

We speak of these as dissimilar elements, as if the cell were one thing, the nerve fibres another, and the muscle something quite different. A moment's reflection is sufficient to recall that from the ganglionic cell its protoplasm is continued by its axis cylinder process to the axis cylinder, and this, always unbroken, to the motor end plate, from which there seems to be a direct communication with the nuclei of the muscular elements ; in other words, ganglionic cell, axis cylinder, and motor end plate are not three things, but one continuous mass of protoplasm.

Granting that excitory impulses travel uniformly over this route from central cell to muscular fibre, there yet remains the fact that the nutritive condition of this contiuous mass of protoplasm tends to uniformity. We have undoubted evidence of this in the well-known fact of descending degeneration. Once the axis cylinder is cut off from its connection with the motor cell, it dies ; and it is the disconnected part which dies. Evidently the central point for the regulation of the life of the string is at the gray cell; but the problem is not so simple. Granting that the cutting of the communications causes the death of the distal fragment by no means proves that while the communication remains a change in the nutritive condition of the distal extremity may not continuously influence the condition of the central end. If, then, the lesion in infantile paralysis be such as to immediately destroy the gray cells, we can conceive of the entire mass of protoplasm dying downwards. The cell being destroyed, the process is rapid; the disease has struck at the nutritive centre of the protoplasmic system. In multiple neuritis the break occurs lower down, but the degenerative process goes on to exert its influence on the muscular fibre. In progressive muscular atrophy it is still questionable whether the atrophy of the motor cells is primary or secondary, but in pseudohypertrophic muscular paralysis there would seem to be much reason to believe that the process makes its first appear- in the muscular substance. I submit that in diseases as slow in their progress as pseudohypertrophy and progressive muscular atrophy, it is reasonable to conceive that a process of degeneration slowly going on in the motor end plates might well influence the nutrition of the long-drawn-out strand of protoplasm of which the motor end plate is merely one portion. That such an origin for the degenerative process explains better than any other the anomaly of constant atrophic changes occurring in connection with nerve lesions of such varied location. We have not yet touched upon another question, viz., the increase of connective tissue and fatty deposit. To enter upon the consideration of this in detail would lead too far for our limits. Suffice that this fibrosis seems to be the customary endeavor of the organism to repair damages, that the irritative processes which lead to the atrophy of the muscular and nerve structures according to common experience might well be expected to cause an increase of the more lowly organized connective tissue.

The value of the series of cases presented lies in this, that they give evidence that the same inherited defect is shown to give rise in one case to an attack of poliomyelitis anterior in several others of the family to either extraordinary muscular atrophy or extreme pseudohypertrophy. I have endeavored in this paper to show that the three diseases ought to be grouped together; the inherited defect groups them together, as we see here to-night. The common inheritance of these young men has been a tendency to degeneration of those protoplasmic strands which begin as motor cells and end as motor end plates or muscle nuclei.

To sum up, I would claim that muscular dystrophies can be properly divided into two classes: first, those in which a true paralysis occurs, in which there is a break in the motor conduction, under which are to be included acute and chronic myelitis anterior, amyotrophic lateral sclerosis, primary or secondary, and atrophies, due to neuritis or section of nerves; and a second class, in which no true paralysis exists, but an impairment of function proportionate to and dependent upon the muscular atrophy—in this class are progressive muscular atrophy and pseudohypertrophic paralysis.

I would also claim that there is a distinction to be made between the relation of the central and distal ends of a nerve fibre in the case of an entire severance of connection, and in the other case of maintenance of continuity with slow changes of the nutritive condition.

I would claim that both progressive muscular atrophy and pseudohypertrophy are essentially of spinal origin; that the cases in which the post-mortem examination shows the cord visibly intact do not invalidate this idea, but that the defect in the distal ends of the motor fibres, while not in every case accompanied by atrophy of the central cells, is yet the indication of an impaired activity of those cells.

The not unfrequent occurrence, in connection with pseudohypertrophy, of bronchocele, and, in other cases, of forms of mental disturbance, cannot be explained by a primary muscular disease, but admit of explanation as due to lesions of the sympathetic ganglia propagated from the cord.

IS BELIEF IN SPIRITUALISM EVER EVIDENCE OF INSANITY PER SE?

By **MATTHEW D. FIELD, M.D.**

Read at the June Meeting of the Medico-Legal Society.

MUCH interest has been recently shown by the public in this question. The developments that resulted in placing certain persons in the Tombs, and their indictment, have led people to ask what is the mental condition of one of their prominent believers, who had given a large amount of property to place the "Science" on a sure foundation. Is this gentleman capable of filling a position of trust, requiring skill and judgment? Was his firm belief in the reality of the manifestations that he saw evidence, in itself, of mental degeneration, of defective judgment sufficient to indicate insanity?

A will contest is now going on in an adjoining State, where it is claimed that the testator was influenced by spirits, and acted in accordance with information that he received from the unseen world.

Last year I was a witness in a case where the the testamentary capacity of a gentleman, who died leaving a large fortune, was attacked before the Supreme Court in this State. Besides other evidences of insanity, it was shown, during the course of the trial, that this gentleman had for some years previous to the execution of the will been in the habit of receiving communications from the dead, and from the living whom he knew to be many miles distant at the time; that he conferred and advised with these spirits upon matters of business; and also that his actions were governed, in certain instances, by these spirit communications. It was also shown that this gentleman's second wife was a spiritualist, and had written quite extensively upon that subject. The lawyers for the defence attempted to ignore all other evidences of insanity except those of his conversing with the spirits, and, of course, held that belief in spiritualism was no proof of insanity.

Examinations of medical literature show very little that has a direct bearing upon this question.

In this case I held that it was necessary to divide the question, or, rather, to classify the believers in spiritualism.

Those who nave an abstract belief in the communion of spirits I did not consider at all; for no abstract belief is evidence of insanity *per se*, no matter how absurd it may be. And again, as most religions treat of a future life, and of the participation of the soul or of the spirit in the enjoyments or miseries of the hereafter, and that spirits have communion one with another, it is but a step to believe that spirits may return to this earth. As is related in the Bible, Elias and Moses appeared unto Christ when he was accompanied by Peter and James and John. It is only when the individual himself participates that insanity may be suspected. In insanity the ego is always involved. People may believe that God can talk to us; this may be, to some, the most reasonable belief, or, to others, the most absurd. The belief that He can, or cannot, speak to us here assembled has naught to do with insanity; but, if an individual states to you, in sober earnest, that he hears God speaking to him, and his actions show beyond peradventure that he does believe this, then we question his sanity. For, even though we believe God may talk to us, and that He did talk to Moses and many others in the Bible times, yet this introduction of the ego convinces us of mental alienation. We may believe that the ass spoke to Balaam, and assume that it is so simple because the Bible says so, and accept the Bible as sufficient authority for our belief, and we may believe that God can make any beast speak; but, at the present time, if a person says, and evidently believes, that a beast was talking to him, we think he is insane, and we think this because the ego participates. Therefore, leaving the belief in spiritualism in the abstract out of the question, we come to the consideration of the so-called spiritualist, and of these I make three classes:

First, those who make it a business to delude and mystify, i. e., the so-called mediums.

Second, those who attend seances, and are deluded and mystified; being caused to see curious things, as hands and faces ot the dead; or faces produced on virgin canvas, apparently by unseen agencies; or hear rappings and voices, and receive written communications in the same inexplicable manner; and things are told them that they supposed nobody else knew but themselves. By these things are they so astonished, and so incapable of understanding how they could be accomplished except by supernatural agency, they believe; but this class never receive these manifestations, nor see the dead, except through the instrumentality of members of the first class.

This class embraces a large number who are, undoubtedly, of weak mind; those who are superstitious, and of an unstable and neurotic organization; and those who require but a slight cause to render them insane. Yet many persons of fine intelligence and of brilliant mind are found in this class. There would not be sufficient in this belief alone upon which to base an opinion of mental incapacity.

In the third class I would place those who actually believe that they see the dead, and those at a distance, face to face in the material form, and that they communicate with them, hearing their voices distinctly and clearly. All of this class I believe to be insane; at least, of the large number that have come under my observation, I never saw one who did not demonstrate his insanity in other directions as well.

It may be a very difficult matter, in some instances, to distinguish between the first and third classes; but I think the rule would hold good in every case. The difficulty would be to determine what individuals actually *believed*, and what ones only assumed and claimed to believe for the purpose of deception, gain, and self-glorification.

To distinguish between these two groups is very important, for one set is deserving of pity and kind care, and the other of reproach and punishment. This distinction once made, it is an easy matter to determine the treatment each class deserves.

In the middle class, or those who, after attending seances and being mystified, believe, many will be found who are insane, and those who are of an unstable and neurotic organization. Yet I am sure no one would consider that belief, under such circumstances, would be evidence of insanity *per se*. The communications, materializations, and other manifestations, are always received through the instrumentality of members of our first class. The perceptions, under such circumstances, are real; there is an actual external object produced in some manner by the so-called medium. The belief in supernatural production, and that the communications received are actually from the dead or those at a distance, is delusion, beyond doubt; yet this false belief cannot be justly considered an insane delusion. However,such belief, taking strong possession of an individual of mature years, of acknowledged good judgment, whose intelligence and will had always dominated his emotions, would arouse strong suspicion of mental deterioration. Whenever we discover *alteration* in an individual's mode of thought, actions, and emotions, we are sure of some mental change as well; yet it may be only the beginning, and proper care and treatment may arrest insanity; still, such alteration is always a grave symptom.

This belief, held by persons who we know have always been emotional, superstitious, and fanatical, would be of slight significance, as it would be in harmony with the usual mode of thought of such a one. We have already mentioned that among the middle class are found many unstable and neurotic organizations; these individuals are more easily upset, and become insane from causes that would not affect those with strong and healthy nervous systems. These people are always drawn to everything mysterious and all that appeals to the emotional side of their nature; many minds of this class are unbalanced and destroyed by every public excitement, where the feelings and emotions are thoroughly aroused.

What could more strongly excite the emotions, at the expense of the intellect and will, than a spiritualistic seance, with its dim and ghastly light, the expectation of supernatural communication, being often startled and astonished by what is seen and heard? Much insanity is unquestionably caused by this means; and, I believe, great misery and distress results from every outbreak that brings this subject prominently before the public.

I must, in justice, say that the delusions of many insane take the direction of spiritualism, where spiritualism itself had really nothing to do with the production of insanity An insane person may believe that the spirit of Abraham told him to sacrifice his child, and he acts in accordance with this command. Another is told by the spirit of his dead child to reward people in this world for kindness done him while living, and he does as requested. A third hears the voice of God, proclaiming him to be the second Christ.

The insanity, in each of these causes, may have come from the same cause; and that cause may have been masturbation. The false belief following, and being dependent upon, false perceptions; that is, an individual of diseased brain has an hallucination; by this I mean a sensory hallucination; an involuntary preception, without corresponding external object. If the false perception be, as in the cases cited, that of hearing, the insane individual does, as a sane person would do, tries to explain how this voice reaches him. He fails to do one thing that a sane man would do, namely, correct the false perception by the other senses, and by his intelligence. But, notwithstanding that he fails to correct the false perception, he nevertheless tries to explain, and does explain to his own satisfaction. He does not see the individual who is speaking, and he looks to some mysterious agency. One satisfies himself that it is the spirit of Abraham; the second, that it is the spirit of his dead child; and the third, that it is the voice of God. A fourth might believe the voice was that of a witch; and a fifth, that it was a telephone. Had there been no spirit, God, witch or telephone known to the world, these people would all have become insane, had hallucinations of hearing, but would have explained them in some different way, and have built up some other delusion, in accordance with the other explanation. It is quite probable that the larger number of persons, who I place in my third group, and who I would consider insane, may never have been believers in spiritualism, and never have attended a seance in their lives. They first become victims of hallucinations of the senses, and these false preceptions become fixed beliefs, and the delusions were founded upon these; the spiritualism being only the means of explanation to their own minds. After they have once turned their thoughts to the subject, they dwell thereon, and their disordered brains build up new and more elaborate delusions in that direction. Whatever subject there may be most prominent in the community at a given time, which has about it the greatest element of mystery, will most likely shape the direction of insane delusions at that particular time. A few years ago, and very often now, the telegraph, telephone, and electricity played a large part in the delusions of the insane, and spiritualism has been correspondingly less prominent, and witchcraft insignificant. To illustrate how easily delusions may be built up from sensory hallucinations, I can state that I have seen at least a score of insane people who believed that Mr. Jay Gould was persecuting them; the steps in the foundation of this delusion in these cases were as follows: First, the hallucination of hearing; second, explanation must come by telephone; third, Mr. Gould controls all the telegraphs and telephones, and it must be he who is persecuting them.

The eminent editor of the *Alienist and Neurologist*, in the latest number of that periodical, after quoting freely from a recent sermon of the Rev. Dr. Talmadge on "Spiritualism and Insanity," observes: "The superintendents of American and foreign asylumns for the Insane will bear out this theologian's statements that spiritualism makes many lunatics, and the counter-statement that lunacy makes spiritualists. . . . . All alienists must concede from observation that spiritualism has destroyed some of the brightest intellects."

It hardly seems necessary to devote much time to the consideration of my reasons for considering all of those insane who would come under my third class. I restricted this class to those who actually believe that they see the dead, and those at a distance, face to face, in the material form; and that they communicate with them, hearing their voices distinctly and clearly. Here I would emphasize the actual belief in the reality, and the fact that this class see and hear by themselves when not aided by any medium or second person. These individuals are the victims of well-defined sensory hallucinations; and that, as they actually believe in their reality, it is evident that they do not correct their false perceptions by other senses, or by their intelligence, but rather build up a distinct false belief. I can imagine that my legal friends are running over in their minds many questions that they would like to ask on cross-examination of one expressing these views upon the witness stand, as they have in their minds so many examples of hallucinations occurring in the illustrious men of great intellect—as Martin Luther, when he threw the ink-stand at the devil; Goethe, when he saw his own shadow walking before him; Sam Johnson, when he heard his mother's voice calling him "Sam," when she was miles away. These examples might be greatly multiplied; but we have only to reply to this that, while certain illustrious men have become insane with sensory hallucinations as among the most marked manifestations of their insanity, others being subject to hallucinations have been able to correct these false perceptions, in the reality of which they never had a fixed or permanent belief.

A CLINICAL LECTURE ON THE DIFFERENTIAL DIAGNOSIS OF ANTERO-LATERAL SCLEROSIS AND POSTERIOR SCLEROSIS OF THE SPINAL CORD.*

BY PROFESSOR WILLIAM A. HAMMOND.

THERE are two patients present to-day who exhibit very opposite manifestations of spinal disease, and yet they are very frequently confounded one with the other. I think, however, that when we come to examine them, we shall find that the symptoms are almost as different as those of any other two diseases with which the human body can become afflicted.

Before proceeding to examine those patients, I shall say a few words regarding the spinal cord, as the basis of the remarks I shall have to make. The spinal cord is not a simple organ. On the contrary, it is a compound organ, anatomically and physiologically. It has distinct anatomical features, and these, as a matter of course, have distinct physiological manifestations. As a consequence, we find that in spinal disease the symptoms exhibited bear an exact relation with the physiology of the part of the cord affected.

As you will see by the diagram, the spinal cord consists essentially of two masses of tissue. All that portion on the periphery is called the white substance, while that located centrally is called the gray substance.

The external portion is divided into various sections; but not to enter into details of the divisions, it will serve our purpose this afternoon to speak of this external portion as the antero-lateral and posterior columns.

The antero-lateral columns include all that part of the white matter on each side lying between the posterior horns of gray matter and the anterior median fissure. The posterior columns embrace that portion of each side lying between the posterior horns of gray matter and the posterior median fissure. This latter is divided into two parts, that lying nearer the median fissure being called the column of Goll, and that contiguous to the posterior horn of gray matter being called the column of Burdach. It is with this latter of the two portions of the spinal cord that we have to deal with in one of these cases, while the other, as I think I shall be able to show you, is affected with a disease of the antero-lateral column.

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*Delivered at the New York Post-Graduate Medical School, March 23, 1888.

The posterior columns of the spinal cord have certain distinct functions; they relate to sensibility and co-ordination.

The column of Goll is primarily so seldom the seat of disease, and a post-mortem examination has been made on so few of the patients in whom it has been affected, that the diseased manifestations to which it gives rise are not definitely known. It has been pretty reasonably established, however, that the column of Goll is concerned, like the column of Burdach, with sensibility and to a certain extent with co-ordination. This column of Burdach, with which we are particularly concerned to-day, is sometimes called the posterior root zone, because the radicals which come from the posterior horns of gray matter seem to start from them. It is owing to pressure upon the radicals, due to the abnormal process characteristic to locomotor ataxia, that we have the sharp, shooting, lightning like pains which are almost invariably met with in cases of that disease.

It used to be supposed that the co-ordinating faculty resided in the cerebellum. Probably it does to some extent. The cerebellum certainly has something to do in maintaining the equilibrium of the body, and so have the semicircular canals of the auditory apparatus. But that function seems to be a little different from co-ordination. A person suffering from a disease of the cerebellum cannot, it is true, walk well, but his difficulty in walking is not due to his inability to co-ordinate well, but to vertigo.

You probably, in the course of your attendance upon physiological lectures, witnessed the removal of the cerebellum from pigeons; you have then noticed that the bird lodged upon the table unable to stand, and every attitude, every expression, seems to show that it suffers from vertigo; its eyes roll, and its head partakes of the same motion. When thrown into the air, it flies in a way which shows that it is subject to a peculiar sensation that causes it to act in a manner similar to that of a child that has turned around many times; it staggers in fact in the air, at the same time it does not appear to be deprived of the ability to co-ordinate its limbs when it desires to move them. That is, if you disturb its limbs individually, it will move them in a perfectly co-ordinate manner. I judge from these phenomena, and from the symptoms of patients suffering from cerebellar diseases, such as an abscess, a tumor, or an injury, that the difficulty in locomotion exhibited by them is due to vertigo. At any rate, in post-mortem examinations of persons who have suffered from locomotor ataxia the cerebellum is usually found in a state of health, while the column of Burdach in the spinal cord is found diseased.

The antero-lateral column has nothing whatever to do with sensibility, and of course a patient suffering from disease of this portion of the cord would exhibit no aberration whatever of sensibility, neither in paræsthesia nor anæsthesia, so long as the disease remained in that portion of the cord. Sometimes, however, the membranes of the cord become involved, and then there are some painful sensations. Again, the disease may spread to the posterior column, as it sometimes does, and then there is derangement of sensibility. But so long as the disease is confined to the antero-lateral column there is nothing whatever but derangement of motility, because there is nothing in that portion of the cord but motor fibres. That being the case, when you have patients affected with disease of the antero-lateral column you expect to find derangement of motion and nothing else. We will now see how these statements accord with the histories in these two cases. First, however, let us turn to one other point.

In sclerosis of the antero-lateral column and of the posterior column the disease is always symmetrical. There are some diseases of the spinal cord which are not symmetrical. But in these two both sides are affected, and usually to the same extent. Not always exactly to the same extent, because it does not always affect both sides at the same time, and the disease gets ahead on one side faster than on the other. But after a year or two you will find the manifestations of the disease are about the same on one side as on the other.

With that basis for an examination of these patients we will take up their cases one at a time and see how they correspond with the facts just gone over.

The first patient tells us he is eighteen years of age; that he has been sick about four years. It dates from a fall on the back of the head. About four months afterward people began to notice that in his walk he kept next to the curbstone, or by the houses. After his attention had been called to this peculiarity in his walk, he found that unless he gave attention to where he stepped he would have a tendency to fall.

He says he did have pains in the legs, but they have disappeared. The pains were short and shooting. When asked whether he felt distinctly with the bottom of his feet he replies no, and when asked whether he seemed to be walking on cushions he replies that the bottom of his feet seemed to come to a point as if he were walking on skates, and they wabbled. He says he passes a great deal of water, but he is able to pass it when he desires. He cannot restrain it long after the desire comes. He has had feelings as if a rope were tied around the waist at about the crest of the ilium. He sometimes sees double, and things look blurred. He sometimes has pains in the head. He has had trouble with speech for sometime. There is no trouble with the arms; no numbness, he tells us; but on inquiry, we find that he is unable to pick up a pin until after several trials. He has trouble with his fingers; they do not feel natural.

The tongue is not tremulous. He says his food seems to stop when being swallowed. He complains of difficulty in the pronunciation of words, and when asked to repeat the words "truly rural" and "national intelligencer," he does it with difficulty. He can repeat the sentence, "Peter Piper picked a peck of pickled peppers," but he does it very slowly and only by placing his whole attention upon it. I have given him these words to test his labials and linguals, —the power to use his lips and his tongue. Ordinarily we do not have to give our attention to speech which has become automatic; this young man, however, cannot pronounce linguals and labials without thought.

This patient has not the Argyle Robertson symptom of locomotor ataxia. In a patient having that symptom the pupil does not contract for light, but it does contract for accommodation. In this patient the pupil contracts for light and also for accommodation. His pupils are larger than usual in patients who have locomotor ataxia; in fact the pupils are usually much contracted in this disease, and are likely to be equal. This man's pupils are about equal; the right may be a little larger than the left, but they are both larger than we commonly observe in this disease.

None of the symptoms, as far as we have gone, are exactly characteristic of locomotor ataxia. A man might have all of his symptoms as far as we have discovered them, and yet have some other disease than locomotor ataxia. Now we will inquire with regard to some pathognomonic symptoms.

When asked to stand with his eyes closed, he separates his feet, widens his base; and when asked to walk with his eyes closed, he goes but a short distance when he begins to stagger and fall. He has lost his ability to bring his muscles into such harmonious action as will result in exact movement. He cannot place his feet where he wants to except by concentrating his whole attention upon his movements. If he were to try to mount a horse, he could not place his foot in the stirrup. If he were to try to get into a carriage, he would probably miss the step. He has lost to some extent the power of co-ordination, which is one of the symptoms of locomotor ataxia, and without which locomotor ataxia never exists.

But there are other diseases in which there is want of power of co-ordination besides locomotor ataxia. We see the same thing in multiple neuritis. That symptom is not, then, sufficient in itself to establish the existence of locomotor ataxia, although there cannot be locomotor ataxia without it.

We now proceed to test his patellar tendon reflex, and we find that it is present in nearly a normal degree. That settles the point beyond a doubt that this is not a case of locomotor ataxia pure and simple, for when that disease exists alone, there is always abolition of the knee-jerk ; that is, when the legs are crossed and you strike just below the patellar, there is no rebound of the foot. I have never seen a case of pure locomotor ataxia in which there was exaggeration of the patellar tendon reflex. Not that the abolition of the tendon reflex is itself pathognomonic of the disease, for it is not. There are some people who never have had the tendon reflex, who have been born without it, who are not suffering with locomotor ataxia. But if you put those two symptoms together, the diminution or abolition of the patellar tendon reflex and co-ordination, such as is seen in this patient, they are sure indications that the person is suffering from locomotor ataxia, or sclerosis of the column of Burdock—the external part of the posterior portion of the spinal cord.

When I began the examination of this patient, I was as much surprised as any of you to find that the patellar tendon reflex had not been abolished, for I had been prepared to look for the further symptoms of locomotor ataxia which up to that point in the examination were present. But the case is proving all the more interesting, as it will probably enable me to demonstrate a combination of the two affections, the general anatomical features of which I have just described.

This patient speaks of a sensation of constriction around the waist. That line is supposed to mark the upper limit of the morbid process in the cord. But in many cases which have come under my observation I am perfectly satisfied that there has been disease above that line. Therefore I am unable to explain satisfactorily what that sense of constriction is due to. I do not even know what the condition in the muscles or nerves is which gives rise to it. It may be a sort of contraction of the muscles, or it may be some trouble with the nerves themselves.

This patient states that his legs have not felt stiff, they do not shake, but they sometimes suddenly spring forward after a sleepy attack. He has not ankle clonus. The only regular symptom of locomotor ataxia which is absent in his case is the patella tendon reflex. This is not, I think, exaggerated, but it is not abolished. The only way in which we can account for that is the existence of a condition called spastic ataxia, which consists of a combination of locomotor ataxia and antero-lateral sclerosis.

It is characterized by the symptoms of both diseases to a certain extent. Suppose this man's symptoms began, as they probably did, in the posterior columns of the spinal cord, and that in the first place there was diminution or abolition of the tendon reflex. Suppose the morbid process extended until it involved the antero-lateral columns. The tendency of disease of the antero-lateral columns is to produce exaggerated tendon reflex. Consequently, as the disease advanced in this case, the loss of the tendon reflex would be replaced by its return or by an exaggeration of it. We cannot determine whether the disease began as an antero-lateral sclerosis or as a postero-lateral sclerosis until we shall have examined further. As long as the disease is confined to the posterior columns of the cord, there is no loss of power of motion. Let the patient straighten the leg, and you will find yourself unable to bend it at the knee. I have seen patients who could not stand with their eyes closed, nor walk with the eyes shut, nor feel the ground with the soles of their feet, yet they were as strong as you or I, and it was impossible to flex their legs against their will.

If this man has a combination of the two diseases under consideration, he has lost power in the legs. If he is as strong in the legs as he ever was, he has a disease which I have never seen. If he has the combination of locomotor ataxia and antero-lateral sclerosis, he must necessarily have lost some strength in the legs, for in antero-lateral sclerosis there is loss of power.

For the purpose of comparison, we will now obtain the history of the second patient, who is supposed to have antero-lateral sclerosis. His age is forty-six. He says he has always been quite weak. By weakness he means his toes have always dragged on the ground; that is to say, he could not raise his foot. This is the manner in which antero-lateral sclerosis begins. He had a tendency to fall down. His legs got a little stiffer every year; they would jump at night, and they twitch occasionally yet. As you see, in his walk the toes drag on the ground. When asked to cross the legs while sitting, he is compelled to lift one leg over the other with the hands. When asked to lift the heel while the toes rest on the floor, he does so, and says that sometimes the foot begins to shake, but it rests quietly now because it is too stiff. When it starts going, he has no power whatever to stop it except by putting the heel down. He has, then, three symptoms which are characteristic of antero-lateral sclerosis, viz., rigidity, paralysis, and exaggerated tendon reflex.

If this patient should come back again in three or four years, we should find that he had no exaggerated tendon reflex as he now has, he would have lost that ankle clonus, but he would preserve his other symptoms and, in addition, he would have loss of co-ordinating power. The rigidity of the muscles is sometimes so great that when such patients in walking have contraction of the muscles on the anterior face of the thigh and legs, the one leg becomes locked over the other, and they are unable to proceed. This patient says he has not been troubled in that way.

When asked whether he has any pains, he replies that he has not, that he only feels stiff. Whatever pains he may have are due to stiffness in the muscles and to outside disturbances, not to disturbance in the cord.

When asked to stand with his feet close together and the eyes shut, he is able to do it without any unsteadiness whatever. The other patient, you will see, is quite unsteady.

The second patient is also able to walk as well with the eyes closed as with them open. The first patient says that in the beginning of his symptoms his legs felt too heavy, and that when sitting in a chair or lying on the sofa he imagined he was going to fall. That is not a characteristic symptom of locomotor ataxia. His legs, he says, never felt stiff, nor does he think they are weak. He imagines his legs would be all right if he could place his feet where he liked. On testing the strength of his legs, we find they can be bent at the knee very easily, while on one of the healthy men present we are unable to overcome the power of the extensors of the leg. That shows this patient must have some other affection than merely locomotor ataxia, for the strength of the muscles is not affected in the last-named disease. The explanation of this symptom would also account for the absence of the Argyle Robertson pupil, which is almost always seen.

There ought also to be something else, to which I have not yet alluded, which is a complicating feature. It is trouble with speech and swallowing. The patient says he sometimes chokes, that he has trouble in moving his tongue and in bringing his lips together. When saying "Peter Piper," etc., he did not bring his lips closely together. That is evidence that he has some bulbar disease in addition to antero-lateral and postero-lateral sclerosis; that is to say, he has indications of beginning glosso-labio-laryngeal paralysis. If he comes here within a year or two you will find that he cannot swallow, that the saliva dribbles from his mouth, that he has lost ability to articulate, and the lower part of his face is almost, if not quite, paralyzed. All this, he says, came from the blow on the back of his head. That would account for the bulbar disease, but it would not in itself account for the other symptoms—those depending on disease of the spinal cord. But it might all be accounted for by the commencement of the disease lower down in the cord, and ascending, involving the other parts. He now says that his eyesight is blurred. All the symptoms go to show advancing disease, having already reached the ganglia at the base of the brain, affecting the third pair of nerves and the optic nerves.

When we began to examine this patient we expected to find locomotor ataxia. While I am disappointed, I am glad that it turned out not to be a simple case of that disease. Besides the interest in the case itself, it illustrates very well the importance of not stopping and making a diagnosis after elucidating what appear to be one or two characteristic symptoms of any affection which you think the patient may have. I was astonished to see his leg fly out when testing the patellar tendon reflex. It was something which I had not at all expected, for the other symptoms pointed to uncomplicated locomotor ataxia. But that occurrence disabused me of my first impression of the nature of his disease. And so you will often find that as you proceed with the examination of a case you may change your mind several times as to its real nature. All that is instructive.

The third patient before you is a woman, past her thirtieth year, who, when questioned regarding the symptoms we have discovered in these other patients, seems to think that she too has some of them. She makes believe that there is ankle clonis at both ankles, and so with some other symptoms. Instead of having antero-lateral sclerosis, she is simply hysterical, and has no affection of the cord at all. There is no real disease about her, at least none such as we are looking for. All that is simply simulation of disease. Her walk shows that she has no paralysis, no locomotor ataxia; the manner in which she stands shows that she co-ordinates well. The symptoms which she has are only those which she has seen us experimenting with and which she has imitated, although but poorly. She is hysterical; she has a disease, a disorder, but it is not such as these other patients have. I had her come in simply to show one of the manifestations of hysteria—how it may simulate anything at all. I had a woman patient four or five years ago who was strongly hysterical, and who of all persons whom I have seen was most under the influence of the principle of suggestion. I could make her believe anything at all. I suppose we can generally do that with women, but at the same time we cannot always impose on them in the way I could impose on that one. I would take up a book, for instance, ask "What is that?" She would reply, "It is a book." I would say, "It is not a book, it is a watch." She would say, "No, it does not look like a watch." I would say, "Look at it again, and see if it does not look like a watch." She would then say, "It does look like a watch." "Have you the hardihood to tell me it is not a watch?" "I beg your pardon, doctor; it is a watch." "What sort of a watch?" "I hardly know." "Is it a silver watch?" "Yes." "Now look at it again, and you will see it is not silver; it is gold." "Oh, yes, doctor; it is gold."

With such persons you could do anything, make them believe anything, for they lose the power of voluntarily directing their thought. The patient before us is one of that kind, except that she has more volition remaining than had the one to whom I have referred.

Let us return for a few minutes to the first two cases.

In the one, we have found that he has an affection of two portions of the cord; that he has locomotor ataxia and also antero-lateral sclerosis. Consequently his gait is not purely that of locomotor ataxia, it is a mixture of the gait peculiar to these two affections. The gait of locomotor ataxia is perfectly characteristic, and I am enabled by it to tell while patients are walking through a passage way of some thirty feet to enter my office that they have locomotor ataxia, although I may never have seen them. Such patients make two distinct movements with the foot and two distinct sounds. This patient does sometimes, but not always. If he had pure locomotor ataxia he would do it always. The heel strikes first, making a distinct sound, and then the sole strikes, making a second sound.

This patient has not very well defined symptoms in the sole of his feet, such as patients suffering from locomotor ataxia usually have. They usually consist of sensations as if the patient were walking on a cushion, on sand, or velvet, or as if the feet were too large for the boots, or as if something were crowding the toes. At the same time this patient has some change in sensation in the sole of the feet. He describes it as if walking on skates, and as if his feet were inclined to roll. He probably exhibits in a very mild degree, if at all, the retardation of sensibility which is one of the characteristic symptoms of locomotor ataxia. By that I mean that the impression made on the cutaneous surface of the feet, for instance, is not appreciated by the brain for a space of time considerably longer than would be necessary in a person normally constituted. Sometimes that interval is so long that it is noticed by every person who may be in the room. I had a patient once at my clinic at the Bellevue Hospital Medical College suffering from locomotor ataxia in whom, as he walked about, I stuck two pins, one into the calf of each leg, up to the head. Some persons present took out their watches, and it was found that two minutes and a half elapsed before he felt the pins, and he then jerked up the feet as if something hurt him. As he said, you might pour boiling water on his legs and he would not know it until the flesh had fallen off.

I do not believe this patient is affected in that way. I do not suppose it will take more than a fraction of a second in his case for the sensation produced by the stick of a pin into the feet to travel to the brain. You see he complains apparently immediately after he is pricked, and there seems to be no diminution of sensibility. It is about the same on both sides.

Sensibility travels along the nerves at about the rate of eighty-one feet a second. Call it over four feet to his brain, he ought to feel the sensation in about one-twentieth of a second, and that probably is about the time in which he does feel it. You can appreciate, then, how great the retardation must have been in the patient at Bellevue College.

In the second patient, who has antero-lateral sclerosis, we do not expect any retardation of sensation, for he has no trouble with the posterior columns of the cord; and he has no retardation of sensation as is shown by experiment.

Another word with regard to the reason why the first patient is unable to stand with the eyes closed. He can stand much better with the eyes open, and he can walk pretty well when he has use of his sight. We know that when walking in the street we do not have to look where we are placing our feet, or just the place where we are going. We look and get a general idea of the direction, but we do not watch our feet, nor do we have any guidepost before us. But these patients cannot walk in that way. They must have a guiding spot somewhere. As long as a patient suffering from locomotor ataxia keeps his eyes on the guiding spot he walks pretty well. As the disease advances he shortens that spot, brings it nearer, until finally he has to keep his eyes directed to the floor and see where he is next to place his feet. The moment he finds his eyes off of his feet he begins to fall. That shows that there is something in us when well, aside from the eyesight, which enables us to walk. That is called by some persons the muscular sense. We know, in a way which we do not understand, the exact state of contraction that our muscles are undergoing. If you take off all your clothing and bend your arm, you will know just the degree to which you are bending it without looking at it. How do you know that your arm is bending, or how much it is bent? I do not know. But it is attributed to the muscular sense. These patients do not know what use they are making of their muscles by that sense. They have lost the muscular sense, and cannot tell what their muscles have done unless assisted by sight. They have to see what they are doing. Whether that muscular sense can be called a sense or not, there is no doubt that it is a perception of some kind which conveys the impression to the brain and gives cognizance of what is going on. This patient has lost that perception not only in the legs, but also to some extent in the arms. When he is asked to close his eyes and bring the index finger of the outstretched hand to the tip of the nose or to the middle of the eyebrow, he misses the mark by half an inch, and he does not fully correct the mistake after several trials. Those of us who are in normal health make very slight, if any, error, even at the first trial, and when we miss the mark at the first trial, we correct it at the second.

One reason why I brought these two cases before you to-day was to sharply define the difference between the symptoms of locomotor ataxia and antero-lateral sclerosis of the cord. The case illustrating the former disease turned out to be not a simple, but a complicated case, and therefore somewhat confusing. Almost every case of antero-lateral sclerosis which is sent to me by physicians who have not made more or less a specialty of diseases of the nervous system is sent as one of locomotor ataxia. You now see how essentially distinct are the two affections. The distinction is not only a matter of scientific import, it is also one which directly concerns the patient, as the treatment is different in the two classes of cases. Antero-lateral sclerosis is much more amenable to treatment than is locomotor ataxia. Of all the organic affections of the spinal cord, locomotor ataxia is the most common, while it is the least frequently cured. We shall have to postpone the consideration of the treatment until a future date.

Society Reports.

NEW YORK ACADEMY OF MEDICINE.

Meeting of April 5th, 1888.

The President, Dr. A. JACOBI, in the Chair.

A CONTRIBUTION TO THE DIAGNOSIS AND SURGERY OF CEREBRAL TUMORS

was presented by Dr. E. C. Seguin and Dr. R. F. Weir, in the first part of which they related a case. The patient was a German, a resident of Bridgeport, under the care of his family physician, Dr. Godfrey, who first brought him to Dr. Seguin's on August 12th, 1887. In Dr. Seguin's absence an examination was made by Dr. Booth. The patient was a strong, healthy-looking man, with no history of syphilis, and no history of epilepsy in childhood. The family history was supposed to be good, but it had afterward been learned that the mother had died of cancer, probably scirrhus, of the liver. The patient had been healthy until 1882, when he had malarial fever. During this illness he had a good deal of pain in the head, and one day, when feeling strangely, he got up to go to the window, when his wife observed spasm of the right cheek and neck. Consciousness was not lost. One or two similar attacks occurred before 1885. These spasms in the face and neck on the right side were the only symptoms of cerebral disease for three years. In 1885 the symptoms became more marked. One day during that year he fell unconscious and bit his tongue. He had similar attacks at long intervals afterward. These epileptic attacks were preceded by an aura, which was followed by twitching and jerking in the right hand and arm and the right side of the face, and loss of consciousness. No exciting cause had been observed. The memory was not so good as formerly; the speech had become thick. Dr. Seguin saw the patient for the first time on August 26th; he had had no purely motor attack in the hand alone; it had always been affected after the right cheek. There was no history of injury to the head. The patient was awkward with the right hand; the arm and hand were the seat of a numb, heavy sensation. The left side and lower extremities were not affected. At this time the tongue did not deviate, but at a later examination it deviated a trifle to the right. The patieut was seen and examined on September 21st and October 19th, when his symytoms were found to be rapidly progressing, and an operation was advised. The diagnosis was that of a tumor in the left motor zone, in the facial centre. There was slight diminution of the tactile sense in the right cheek and arm. There was slight impairment of the muscular sense in the right hand. There was constant loss of saliva from the right buccal angle. The strength of the right hand and arm was about two-thirds that of the left. Subsequently tenderness developed over the motor zone of the left hemisphere. The temperature test over the scalp was negative. Whether the tumor was cortical or subcortical was not determined. Treatment by iodide of potassium had no effect. Dr. R. F. Weir performed the operation in the New York Hospital, November 17, 1887. The head was shaved the previous day, and great pains were taken to make the operation perfectly antiseptic. The spray was not used. A minute perforation was made through the scalp and the point marked with a lead-pencil which was to be the centre for the trephine. Two pieces were then removed with the trephine, and the openings being joined, left one large opening two by three inches. The dura mater bulged only a little, and appeared normal. When it was cut the brain bulged somewhat into the opening, but its surface was normal. The finger recognized no tumor until firm pressure was made, when deep resistance was felt in a mass of small size under the suspected convolution. It was of the shape and of about the size of the end of the forefinger, or of an almond. It was readily lifted out by a Volkmann's spoon, blunted for this purpose. A little portion of brain, of about the size of a pea, was removed with it. The finger passed in an inch and a half to the bottom of the wound. There was no hæmorrhage from the brain itself. A rubber drainage tube was carried to the bottom of the cavity and out through the posterior margin of the wound; the dura mater was stitched together except where the tube emerged. After the final dressing the wound was washed with corrosive-sublimate solution (1 to 5,000). The discs of bone were replaced. At the close of the operation the pulse was 125, and the general condition was good. Dr. Peabody pronounced the tumor an infiltrating sarcoma.

Commenting on the operation, Dr. WEIR said he thought there had been more hæmorrhage from the vessels of the scalp than he would allow at a future operation. The patient had gone to his home a month after the operation.

Dr. SEGUIN gave the history of the case from the time of the operation until about the 1st of April. There was almost complete hemiplegia with aphasia just after the operation, but these subsided, leaving the man in about his previous condition. He had no convulsion until the 18th of December. Driveling had ceased. After his return to Bridgeport he again contracted a form of intermittent fever, and the symptoms due to the cerebral lesion were most pronounced when he had the fever. There was jaundice with the malarial symptoms. The wife had observed no twitching in the muscles of the face since his return home after the operation. He stated that he knew the word he wished to use, but could not utter it. The right hand no longer felt numb. The muscular sense was practically perfect, and sensation seemed normal in the right hand. Dr. Seguin thought life had been prolonged by the operation. The paresis seemed to be somewhat greater, but he attributed this to the patient's general health. The sensibility of the face, hands, and fingers was improved. The aphasia was about the same as before the operation. There was no indication of increased intracranial pressure and no evidence of a relapse of the disease. The speaker was not positive that the tumor had been entirely subcortical. It might have involved to some extent a deep gyrus located in its neighborhood. The case was particularly interesting when considered with reference to recent advances in the physiology of the brain and the application of such knowledge to surgical interference in a general way as well as with regard to tumors. The diagnosis of tumor of the brain for the guidance of the surgeon was, as a rule, reached gradually. Then the locality of the tumor would be determined by empirically acquired knowledge due to the studies of Broca and others, and the laws of cerebral action as elicited by Hitzig and others. The speaker here considered the signs of a tumor in the motor and sensory zones, the symptoms due to irritation or excitation of the part as distinguished from those due to its destruction, etc. Speaking of the significance of limited spasm or paresis, he had long looked upon the early spasm as a guide to correct localization of the disease. It was of so great importance that it should always be traced if possible. He would call it the signal symptom of cerebral tumor. The diagnosis of a tumor situated in the sensory zone for sight, he thought, could be made just as positively as if it were located in the motor centre for the hand, face or leg. It was of importance to determine not alone the region in which the tumor was located, but also, if possible, whether it was cortical or subcortical. He thought that at present we were unable to distinguish a cortical from a subcortical tumor by the symptoms. Regarding the significance of headache, his conclusion had been negative. Indeed, headache was an unreliable symptom of tumor of the brain independent of location. His conclusion with regard to the significance of scalp temperature had also been negative. The surgeon would be influenced as to whether he should or should not operate by the question of the probability of the tumor being multiple. The presence of tuberculosis or of cancer in other parts of the body would contra-indicate an operation. Combined symptoms of tumor in the motor and sensory zones, whether sensory or motor, would point to multiple tumor. Where more than one growth existed in a limited area of the brain, it could not be determined during life; the surgeon might remove one and overlook the other.

Dr. WEIR then considered the surgery of cerebral tumors, and read a portion of his paper, giving a synopsis of the operations performed by Bennet, Godlee, Victor Horsley, and others. From the facts presented it would seem that an exploratory operation would be justified when symptoms pointed to progressive brain pressure, whether from tumor, abscess, or an intracerebral blood-clot, or to continuous irritation.

Dr. KEEN, of Philadelphia, related briefly the history of a case which he would at a future time publish in detail—that of a patient, aged twenty years, who had sustained a fall when three years of age, which had left a scar over the left motor region. It was not until some months before the speaker saw him that he had begun to have epilepsy and paralysis of the right leg, the right arm, and the face, and had aphasia. He removed a large tumor from the left motor zone, symptoms of pressure developed afterward, and he had to change the dressing and remove a large blood-clot. Hernia-cerebri then developed and gave considerable trouble, but the patient recovered. In this case the blood-vessels were very brittle, and he had had much difficulty in controlling hæmorrhage. He employed for this purpose hot water, ligatures, and pressure. He thought the entire scalp should be shaved, to reveal any possible scars to which attention had not been drawn.

PERISCOPE.

By DRS. G. W. JACOBY, N. E. BRILL, LOUISE FISKE-BRYSON AND GRACE PECKHAM.

ANATOMY OF THE NERVOUS SYSTEM.

On the Minute Structure of the Corpora Striata and the Optic Thalami. Dr. Vittorio Marchi, (Rivista Sperimentale di Frenatra e di Medicina Legale.

In an elaborate and exceedingly interesting paper on this subject, illustrated with some very fine plates, the writer reaches the following conclusions.

I. Two types of cells are found in the corpora striata, large and medium, which are furnished with numerous processes, one alone of which is distinguished by special characteristics and is the nervous process, the others correspond to protoplasmic processes.

II. The nervous processes comport themselves in two ways, one after a short distance from their origin, divides completely into a fine *rete nervosa* the other constitutes the axis cylinder of a fibre, not however, without first giving off a few delicate branches.

III. In the nucleus of the corpus striatum cells of both types are found ; this predominance, however, is less pronounced in the lenticular nucleus.

IV. In the optic thalamus, isolated groups of cells are not found, but they are scattered irregularly through the whole mass of the gray substance. The larger size prevail and are very similar to those of the anterior horns of the spinal cord. These, as in the corpora striata, show the single nervous process and the numerous protoplasmic processes.

V. Differing from the corpora striata, the first type of cell prevails in the optic thalami.

VI. Corresponding to the double manner of the disposition of the nervous processes, the fibres join themselves with the cells of the corpora striata and the optic thalami, either directly uniting with the nerve process of the cells of the first type, or indirectly losing themselves in the fine network formed from the nerve processes of the cells of the second type, together with the lateral branches emanating from the same prolongations from cells of the first type.

VII. The internal capsule contains fibres which directly unite the peduncles of the brain to the corona radiata. Others which leave the peduncles stop at the basal ganglia; others go from there to the corona radiata, others arising from the cells contained in the substance of the same capsule take ascending or descending direction.

VIII. With reference to the protoplasmic processes, setting aside those which form the rete nervosa, observation leads him to think that the finer ramifications stand in relation to the cells of the neuroglia and to the vessels.

IX. The neuroglia is essentially represented in cells having numerous long, fine processes which ramify many times, and by means of various expansions insert themselves in the walls of the vessels.

X. Finally, the ependima which covers the corpora striata and the internal surface of the optic thalami is composed of cylindrical conical cells, the thinner portion of which continues with a process which, after considerable ramification in most instances, is inserted into the vessels.

The writer deduces the following conclusions in regard to what he styles the much debated question of the functions of the basal ganglia. From the fact that the two types of cells prevail in both the corpora striata and the optic thalami, he argues that they have mixed functions of motion and of sensation; but as in the corpora striata the cells with numerous processes corresponding to the multipolar cells of the posterior horns of the spinal cord, therefore the corpora striata pertains mostly to sensation; while in the optic thalami the cells corresponding to those found in the anterior horn prevail. He finds in these experiments a corroboration of the law formulated by Golgi, that in all parts of the central nervous system, the specific function, whatever may be its nature, ought to be effected not by the isolated or individual action of its single ganglionic elements, but by the conjoined action of extensive groups of cells.

It may be well to add the method of preparation which was employed to obtain the fine results in staining shown in the plates. The specimen is best taken from young animals, must be carefully and well-hardened. Before taking out the brain, he makes repeated injections of a two per cent. solution of bichromate of potassium into the carotid artery. This makes the hardening more even. After this he cuts the brain into pieces and leaves them in Müller's fluid for twenty-four hours, after which he makes sections with a razor into smaller pieces of a cubic centimeter. These he puts again into Müller's fluid, leaving them at an ordinary temperature for eight or ten days ; if it is cold it is necessary to leave them for a longer time. He then places them in a solution of about eight parts of Müller's fluid and two parts of a solution of one per cent. of osmic acid. This mixture accelerates the hardening. The fluid should be abundant and clear, and contain only a few pieces. After twenty-four hours they should be placed directly into a solution of sixty per cent. nitrate of silver, which should be changed after about half-an-hour, because of the precipitate which it forms. In this last solution it is necessary that the pieces remain at least twenty-four or forty-eight hours, longer will not cause any alteration. The sections should then be made, after which they are washed in common alcohol, then passed directly into creosote and left until they have acquired an evident transparency. Then wash them a number of times in turpentine, leaving them for twenty minutes, after which mount in Canada balsam without covering.

PHYSIOLOGY OF THE NERVOUS SYSTEM.

TRAUMATIC CORTICAL HEMIEPILEPSY. Dr. E. Houzé (Bulletin de la Société de Médicine Mentale, 1884, p. 48).

Patient, male, æt. 22, entered hospital February 5th,

1886. Three years previously had received a severe blow with an iron plate on the top of the head. Deep wound. No loss of consciousness. After a few days, convulsive movements in the right leg, and on the fifteenth day a convulsive attack of the entire right side with loss of consciousness. Frequent repetition of the attacks, after which walking became difficult. Severe headaches. After several months, convulsive seizures in right hand and arm; since six months, of face also. At this time signs of amnesic aphasia.

Examination shows occasional contractions in territory of right facial. Speech hesitating, tongue affected with spasmodic movements. Speech scanning; amnesia; lingual ataxia. Agraphia due to convulsive movements of arm. From these symptoms a lesion at the foot of the lower frontal convolution was assumed. The fingers of the right hand are alternately flexed and extended. When the foot is placed upon the ground, rapid movements of the flexion and extension also occur. Walk, dragging, hemiparetic. The convulsive attacks occur ten to fifteen times daily. These are usually confined to the right side, but are sometimes generalized.

In addition to these symptoms of motor irritation, hyperæsthesia of the right foot and hand is observed. Muscular sense is also reduced; no ataxia. Tendon reflexes normal. Cephalometric measurements were employed for the determination of the precise location of the bone cicatrix. These showed the scar to be situated near the central fissure and bordered by the central convolutions.

After a long period of medicinal treatment the patient improved until only convulsive movements in the right leg and occasional epileptic attacks upon that side were present. After five months of slow improvement, trepanation was performed. A small spicula of bone was found at the superior extremity of the central convolution (the symptoms of irritation of the arm and face centres are considered secondary, due to indirect pressure), and removed.

When discharged from the hospital he had no attack for two months, and the twitchings in the foot were rare and not severe. G. W. J.

NERVOUS DISORDERS AND SECONDARY SYPHILIS; HYSTERIA; EPILEPSY; NEUROSES OF THE SYMPATHETIC.

Prof. Fournier (Journal de Méd., p. 202, 1888).

Secondary syphilis incontestibly has a modifying or productive influence upon various neuroses.

Hysteria which for a long time has been dormant is is often reproduced by the syphilitic virus.

Epileptic attacks also become more frequent under its influence.

Can secondary syphilis primarily produce these disorders? As far as hysteria is concerned, this is certain. Hysteria is often seen to occur for the first time simultaneously with syphilis, occasionally in persons hitherto normal, generally in those of a neuropathic constitution. These disorders are usually hysteroid, and not true hysterics, and disappear with the cure of the syphilis. There are exceptional cases, in which the secondary syphilitic stage is characterized by the occurrence of epileptic phenomena.

Cases are also mentioned which show the occurrence of true epileptic attacks coincident with the occurrence of the secondary symptoms, and which disappeared with the cure of the syphilis. Under these conditions only "grand-mal" occurs, never "petit-mal," and mental symptoms are never observed. The prognosis is good, and the treatment that of the primary disorder.

The sympathetic system may be influenced in various ways by secondary syphilis. Caloric disorders occur either as localized or general coldness. Coldness of the extremities, of one arm or leg, may prove sufficiently annoying to prevent the patient from working. Continuous chilly feeling without increase of temperature is frequent. These disorders are generally tenacious, and last from five to six weeks or even several months.

The viscera may also be affected, and visceral neuroses produced. Dyspnœa, occurring in attacks and lasting several minutes, is rare. Palpitation of the heart with precordial oppression occurs more frequently. Irregularity of the heart's action is also encountered. Disorders of the digestive system are much more frequent, particularly in women.

The gastralgic and dyspeptic occurrences show nothing of particular interest. In certain cases the syphilis produces vomiting analagous to the vomiting of pregnancy. This may be coincident with the gastralgia, or there is gastric intolerance without pain in which everything is rejected by the stomach. Comple anorexia, similar to hysterical anorexia, also occurs. In such cases inunctions must be employed. In contradistinction to anorexia, bulimia occurs in the early part of the secondary stage. The duration of the latter disorder varies from a few weeks to several months, and is generally the sign of a severe form of secondary syphilis. The specific treatment must be energetically pushed. Hydrotherapy is also of value. G. W. J.

ETIOLOGY OF BASEDOW'S DISEASE. Société Médicale des Hôpitaux, meeting of May 14th, 1888 (Gazette des Hôpitaux, p. 519, 1888).

Rendu relates the history of a patient who has been under observation for ten years. A young woman, æt. 28, always well until her eighteenth year, when she became chlorotic, with severe cardiac palpitations, vertigo, and symptoms of cerebral anæmia. Under treatment she improved, then married and had two children. Severe domestic troubles. In the spring of 1877, shortness of breath in ascending stairs, pain in walking, and severe pains in the shoulder; angina pectoris; frequent attacks during five to six days. Rendu prescribed iodide of potassium, two grammes daily. She improved greatly, but presented symptoms of iodism; became loquacious; complained continually of feeling too warm, and showed generalized symptoms of arterial excitement. Soon all the symptoms of Basedow's disease appeared, minus the goitre. Rendu believes that in this case there is a relation between the administration of the iodide and the appearance of Basedow's disease. G. W. J.

HYPNOTISM AT THE NANCY SCHOOL. Dr. Bernheim (Gazette des Hôpitaux, p. 337, 1888).

The Nancy doctrine of the phenomena of hypnotism has so frequently been misrepresented that B. here gives the features observed at Nancy in so far as they differ from those seen at the Salpêtriére.

1. The three phases—lethargy, catalepsy, somnambulism—are never observed. In all subjects cataleptic and somnambulistic phenomena may be produced by simple suggestion. Neither opening of the eyes nor friction of the vertex in any manner modifies the phenomena, if suggestion be excluded.

Transferred through magnets,muscular hyperexcitability symptoms of cerebral localization (by touching certain parts of the cranium), are never observed unless suggestion is employed. All these phenomena can be produced, if the patient believes that they are to occur. The three so-called phases of the hypnotic state are due entirely to suggestion.

2. In hysterical patients the hypnosis does not differ from that in other subjects. It is always due to suggestion.

3. Hysteria is not a good soil for the study of hypnotism. Auto-suggestion serves to obscure the clear picture and to confuse the inexperienced operator.

4. The hypnotic state is not a neurosis; the phenomena are natural and psychological, and may be obtained in many subjects during natural sleep.

5. The hypnotic state is not peculiar to or more easily obtained in neurotic individuals than in others.

6. It is not asserted that *all* somnambulists are pure automatons, but that among this class there are some in whom the power of resistance is so greatly reduced that they are at the mercy of the operator.

7. All procedures for hypnotizing may be summed up under the word "suggestion..' No procedure will succeed if the subject does not know that he is expected to sleep.

8. Suggestion is the key to all hypnotic phenomena. Every physician who, in his hospital ward, does not succeed in hypnotizing eighty per cent. of his patients must acknowledge to lack of experience and refrain from expressing an opinion upon the subject. G. W. J.

A CASE OF PSEUDO-TABES. A. Pitres (Archiv de Neurologie, p. 337, 1888).

Certain cases are known which during life presented all the symptoms of tabes dorsalis, but at the autopsy the central nervous system was found to be almost normal. Such cases led Trousseau to look upon the disease as a neurosis. Others reserved their interpretation of them until science should have advanced our knowledge. It soon became possible to explain those cases which upon autopsy showed degeneration of the posterior columns, but intra-vitam had not shown any of the usual symptoms of tabes (sclerosis of the columns of Goll). Then also it became possible to explain a certain class presenting tabetic symptoms intra-vitam, but showing no lesion of the cord on autopsy (multiple neuritis). Thus the term pseudo-tabes must be restricted to cases presenting the symptoms of tabes, but upon autopsy showing the intactness of the central nervous system, the spinal roots, and the peripheral nerves. Such a case is here described.

CASE.—Male, æt. 45, no heredity, no syphilis or alcoholism ; venereal excesses. In 1877, at age of 36, attacks of sharp, lancinating pains, beginning in the right hip and extending later to the left. Polyuria. In 1880, belt pain ; unsteady gait ; sensation of swelling of feet ; Romberg's symptom. From 1881–'86, disorders of micturition, rectal tenesmus, gastric crisis.

Typical fulgurating pains. Continuation of motor incoordination. No disorders of sight, or trophic disturbances. Patellrar tendon-reflexes preserved. Death due to tuberculous pleurisy.

Autopsy : No sclerosis of the cord. No atrophy of posterior roots. Integrity of peripheral nerves. G. W. J.

ON THE DISEASE PHENOMENA AFTER CONCUSSION OF THE SPINAL CORD IN RAILWAY ACCIDENTS (Transactions of the Berlin Society of Internal Medicine. Meeting of January, 1888).

Dr. Oppenheim in discussing this subject stated that the symptomatology of this class of affections had not been materially changed since first described by Erichsen.. Only certain symptoms which had been neglected as subordinate by him in his description have an increased importance, owing to newer and more accurate methods of investigation. It was then presumed that the injury was confined to the spinal cord, and all symptoms were referred to that organ, hence the term railway-spine. Certain known cerebral symptoms were disregarded, whereas now more and more attention is paid to the latter, so that the terms "railway-spine" has become converted into "railway-brain," and traumatic meningo-myelitis into traumatic hysteria.

The symptomatology is varied and motley, so that one cannot speak in this relation of a definite set of symptoms excepting of certain nuclear symptoms which appear in every case. The most prominent change occurs in the *psyche*, especially in the affective sphere. Emotional depression and abnormal irritability represent the psychical disturbance. The patients are moody, busy themselves with painful delusions, love to be alone. Two conditions serve to differentiate this psychical change from that of melancholia :

1. Abnormal irritability.
2. Hypochondrical nature of the psychical change.

Sometimes it becomes necessary to treat those cases, which show the condition of fear, in asylums.

Disturbances of sensation and of the functions of the organs of special sense occur frequently. Seldom is the typical girdle sensation experienced.

Motor disturbances show themselves by the energetic character of muscular movements, by the position of the body in walking, etc. Some patients can walk backwards better than forwards. Tremor is most frequent.

Speech disturbances are also present and are anomalous in character. Certain phenomena in the circulatory apparatus, which have heretofore been undiscovered, are quite characteristic. Very frequently there are attacks of cardiac palpitation with increased pulse rate and accompanied by a feeling of fear and unequally dilated pupils; during these attacks the pulse may record 160 beats per minute. The least emotional disturbance or mental impression increases an already increased pulse beat, the entire body becomes flushed and perspiration freely flows from the surface of the body. The area of cardiac pulsation is increased. In one case hypertrophy and dilatation of both ventricles developed.

It is thus seen that this disease is essentially a combined psychosis and neurosis. At any rate almost all the disease manifestations point to a cerebral basis, and hence the term "railway-spine" should be abolished. Nor should traumatic hysteria, or Page's traumatic neuræsthenia be substituted. Under the conception of traumatic neurosis and traumatic psychosis the greatest majority of the cases may be included. One limitation should be made for those cases which, few in number, show bladder disturbance, iridoplegia, and optic nerve atrophy; cases which show material changes in the central nervous axis concerning which we have not yet been enlightened. N. E. B.

PRIMARY ACTINOMYCOSIS OF THE HUMAN BRAIN (Munchner Med. Wochenschrift, 1887, No. 41).

The patient, a female of sixteen years, complained one year before death of severe headache, to which there was added a paresis of the left abducens. Six months after this she was delivered of a healthy boy. The headache progressively increased in severity, and attacks of unconsciousness, short in duration, supervened. Later there were vomiting, coma, death. The diagnosis was a cerebral tumor.

The autopsy showed a tumor the size of a hazel nut in the third ventricle, chronic internal hydrocephalus, with marked distension of all the cerebral ventricles. The tumor contained an albuminous mass, containing mucin and large granulation cells and innumerable characteristic colonies of actinomyces. N. E .B.

THE DIAGNOSIS OF PREMATURE CRANIAL SYNOSTOSES.

At a meeting of the Imperial Society of Physicians in Vienna, Meynert presented two cases of premature closure of the cranial sutures. The one a boy of 9½ years, who was also hydrocephalic, and whose cranium, as a result of synostosis of the entire sagittal sutures, was remarkably long, very much arched from behind forwards, and presented the appearance of a boat whose keel is directed upwards (scapho-cephalic). In addition the eyes were very deeply set, which was explained by the fact that the antero-posterior diameter being so remarkably long the orbits developed in a similar manner and thus became very deep, and since the bulbi were not long enough to fill the orbits, they appear to lie deeply therein.

The second case was one of a man 35 year old, who had had epilepsy since his 12th year, and whose head was dome shaped. This was explained by Meynert to be due to a premature synostosis of the lateral parts of the coronal sutures, whereby a shortening of the anterior part of the cranial cavity occurred and similarly involving the orbit so that the individual appeared to be flat-eyed (oxy-cephalic). Meynert attempts to elucidate with these two cases a diagnosis *in vivo* of premature cranial synostosis. Synostosis of the middle portion of the coronal suture produces a bulging in the occipital region, and a diminution in size of the frontal and parietal regions, the brain being crowded backwards. Synostosis of the lateral parts of the coronal suture the growth of the frontal and parietal regions is hindered and a shortening of the orbit (flat-eye) results. Ossification of the posterior part of the sagittal suture, the breadth of the cranium, is diminished, and the cranium becomes posteriorly very small.

The following points are characteristic in defining a synostotic (oxy-cephalic) cranium :

1. Subnormal horizontal circumference (Nanna-cephalus).
2. Vertical diameter larger than the horizontal.
3. The vertical diameter, however, must possess this character : the vertical index must be brachycephalic.
4. The flat eye.

N. E. B.

Typical Diphtheritic Paralysis.

This affection comes on some little time after the acute illness has subsided. The paralysis (which involves sensory and motor nerves alike) tends to be of symmetrical distribution, to creep from part to part, and to subside in one region as it invades another. Sensory disturbances are not limited to the extremities, but are apt to involve districts situated in the middle line of the body, including the tongue and mouth and the senses of taste and smell. Paralysis of diphtheritic origin is liable not only to affect the arms, legs, respiratory muscles, muscles of the trunk and accommodation, and of swallowing, but also the vocal cords and even single nerves. Knee-jerks are first exaggerated, then abolished, and the affected muscles acquire the characteristic reactions of degeneration. Many of the phenomena are such as would seem to be best explained on the assumption that they are due to spreading neuritis. Others (such as sensory affections along the middle line, the early undue briskness of knee-jerk, and nystagmus) seem rather to be due to some spreading central lesion. I am inclined to believe that, in diphtheritic paralysis, a wave, so to speak, of slight inflammatory mischief spreads not only through the medulla oblongata and cord, but along the nerve trunk also.—J. S. Bristowe, in British Medical Journal. Feb. 14, 1888. L. F. B.

DENTITION AMONG IDIOTIC AND BACKWARD CHILDREN.

Dr. Alice Sollier (Ancienne Externe des Hôpitaux de Paris et de l'Hôpital des Enfants-Malades) finds that idiocy, with or without epilepsy, predisposes ninety-one per cent. of the victims to dental deformity and disease. The record includes one hundred clinical cases. Congenital idiocy has no more special influence in this respect than idiocy developed during the first dentition. Deformities and lesions are almost exclusively associated with the second dentition. The appearance and shedding of the milk teeth are usually delayed. In fourteen per cent. there were dwarfed teeth ; in eleven per cent., giant teeth. Other abnormalities were found in fifty-three per cent, and absence of teeth in eleven per cent. Giant teeth were often found where others had not developed, or else they represented anchylosis of two adjacent teeth. In only two per cent. were supernumerary teeth discovered. Anomalies of implantation were common (thirty-four per cent.), but anomalies of position in relation to other teeth rare. Crookedness, obliqueness, etc., were the most frequent of all malformations, and chiefly affected the canines and incisors. Erosion (loss of substance of the enamel over a greater or less area of the surface of a tooth) was often associated with convulsions, but was most frequent in cases where there were no convulsions; thus Dr. Sollier concludes that idiocy, with or without epilepsy, can by itself cause erosion. In forty-one per cent. of the whole series there was longitudinal grooving of the enamel; in fifty-eight per cent. the edges of some of the teeth were notched. Both these peculiarities, especially the notches, more often coincided with convulsions than did erosions. The articulation of the upper and lower dental arches was defective in forty-three per cent. In thirty-eight per cent. each arch showed deformity. Madame Sollier calls attention to an undescribed anomaly of this class, where the level of the two halves of the arch is unsymmetrical. In forty-five per cent. of the cases the palatine vault was deformed. In nine cases of idiocy, and imbecility with epilepsy, none of the above characteristic lesions could be discovered.—British Medical Journal, Feb. 28, 1888.

L. F. B.

SLEEPING WITH THE HEAD NORTH.

The superstition that human beings should sleep with their heads to the north is believed by the French to have for its foundation a scientific fact. They affirm that each human system is in itself an electric battery, the head being one of the electrodes, the feet the other. Their proof was discovered from experiments which the Academy of Sciences was allowed to make on the body of a man who was guillotined. This was taken the instant it fell and placed upon a pivot free to move as it might. The head part, after a little vacillation, turned to the north, and the body then remained stationary. It was turned half-way around by one of the professors, and again the head end of the trunk moved slowly to the cardinal point due north, the same results being repeated until the final cessation of organic movement.—Pacific Record.

L. F. B

INEBRIETY A MENTAL DISEASE.

The following questions proposed by the English examiners in psychological medicine to the candidates for certificates, show that inebriety is recognized among the mental diseases of the old world.

"What forms of mental disorder may be classed under alcoholic insanity?

"Trace the relationship of alcohol and syphilis to general paralysis.

"Describe the condition commonly known as chronic alcoholism; give the prognosis and treatment."—Quarterly Journal of Inebriety. L. F. B.

MELANCHOLIA AND OTHER DEPRESSIVE MENTAL AFFECTIONS IN OTOPIESIC DISORDERS OF THE EAR. Boucheron, *Gazette des Hôpitaux*, p. 1,184, 1887.

That affections of the ear may produce various nervous symptoms, such as vertigo, convulsions, mental depression, and even mania, is acknowledged. These symptoms have however, in cases thus far published, always been the result of gross lesions of the ear, and the relation between cause and effect has been easily recognizable.

B. here refers to a class of irradiated nervous affections caused particularly by obstruction of the Eustachian tube, which the atmospheric pressure not being counterbalanced (on account of the vacuum in the tympanic cavity) produces an excitation of the labyrinth and of the acoustic nerve, by compression—otopiesis.

In such a condition the excitation may be transmitted to the nervous centres, and various symptoms, according to the predisposition of the patient, may be produced. If transmitted to the medulla oblongata or to the cord, this excitation causes epilepsy, pseudo-meningitis, or various forms of convulsions; if to the cerebellum, disorders of equilibration, vertigo, rotary movements, etc.; if to the cerebrum and cortex, light or severe mental symptoms, with the predominant character of depression. The less severe symptoms are: loss or diminution of memory, of power of reflexion, and of quickness of conception, diminution of affection for relatives, ideas of suspicion, defiance, persecution, and hypochondriasis. The severe symptoms may be those of acute melancholia, insanity with delirium, hallucinations, and illusions of hearing, loss of consciousness, e c.

The treatment of these symptoms is self-evident. Insufflations of air into the Eustachian tube. G. W. J.

THERAPEUTICS OF THE NERVOUS SYSTEM.

Treatment of Disease by Nerve-Pressure.

Dr. Anders Wide (Nordiskt Medicinskt Arkiv., Nov. 10, 1887) reports three cases treated by nerve-pressure, a method originated by Ling. The first, a woman twenty years old, suffered from tremblings of the promotor and supinator muscles of the arm, causing oscillations in the arm and hand at the rate of two hundred a minute. Pressure was made upon the radial and median nerves about the middle of the humerus, first with the fingers, then with a tourniquet, in the beginning from two to four hours, then eight to ten later. After eleven days of treatment, during which the tourniquet was applied seven times, the tremblings ceased. This was a case in which electricity and other remedial measures had proved of no avail. A spasm of the spinal accessory of the right side, drawing the head backward and to the left with such force that the patient was often obliged to hold it with both hands to prevent suffocation, was greatly relieved by nerve-pressure. Strong and continued pressure with the fingers was made on the nerve at its entry into the trapezius muscle. Afterwards a strap in the form of a figure of eight bandage round the shoulders to exercise a sustained pressure upon the nerve. This insured freedom of breathing. The third case—paresis and atrophy of the right forearm in a girl of eleven—was rapidly benefited by pressure upon the radial nerve. Electricity, massage, and medical gymnastics had been tried in vain. The power of contraction returned to the affected muscles, and the nutrition of the arm gradually improved. The author claims to have brought about some beneficial change in locomotor ataxia by this method. Pressure may be applied to most of the nerves of the head, trunk, and extremities, and also to the sympathetic. Affections of the stomach have been relieved by pressure applied to the cœliac plexus. —London Medical Recorder, Feb. 20, 1888. L. F. B.

ONE VIEW OF NEURALGIA.

Many conditions are classed under the head of neuralgia. There is the pure form without apparent and definite cause during life and with no discoverable lesion after death. The reflex form may be, and frequently is, due to dental caries and other remote causes, the removal of which gives speedy relief to the symptoms. Another form is due to actual inflammation of the nerve—a true neuritis and not neuralgia at all; others due to poisons circulating in the blood, as lead, syphilis, gout, and malaria; and some to pressure upon the trunk of the affected nerve or to some irritation still higher in its course. The treatment of neuralgia may be considered as: 1, external; 2, external remote; 3, internal.

1. Of the local methods of treatment, such operations as incision and division of the nerve explain themselves. Stretching the nerve and acupuncture seem to aid in the same way, probably by causing a solution of continuity in the sheath, and thus relieving tension and diminishing the swelling of the nerve. Callender considers it probable that "stretching is of use by numbing the nerve for a short time." If this be really true, the natural explanation would be that the sensory irritation resulting from the hyperæmia would for a time cease, and on its cessation the reflex dilatation of the blood vessels would also be brought to an end. Electricity, again, in common with belladonna and heat or locally applied, would contract blood vessels. Belladonna, volatile oils, and their stearoptenes (solid crystalline compounds separable from volatile oils by cold), such as thymol and menthol, also chloroform, not only contract the blood vessels, but also diminish the sensibility of the part, while aconite locally applied acts purely as an anodyne.

2. Counter-irritation over the upper part of the dorsal spine, which apparently does good in some cases in a way not easy of explanation, is one method of external and remote treatment. Ligature of the carotids is eminently successful. This heroic measure has been tried with gratifying results by Dr. Patruban in many cases of severe neuralgia. The idea of the operation originated from a knowledge of the relief obtained in some cases by simple pressure on the carotid.

3. For the internal treatment of neuralgia, the substances naturally fall into four groups:

(1.) Those which contract blood vessels, as strychnia, atropia and its allies, bromide of camphor, digitalis, ergot, volatile oils such as turpentine, etc., chloride of ammonium, and quinine. Several of these drugs have also a specific action on sensory nerves.

(2.) This group consists of bodies which act as general tonics, such as quinine, iron, strychnia, phosphorus, and arsenic. Arsenic and phosphorus seem to have some more direct action than that of simple tonics.

(3.) A third division consists of simple anodynes or sedatives, such as bromide of potassium. This, however, like chloral, diminishes the pulse tension and, in addition, diminishes reflex excitability by depression of the peripheral sensory filaments. Other drugs, such as cannabis indica and morphia, seem to act as similar anodynes.

(4.) The fourth group consists of the usual tell-tale drugs, which are always appearing, and of the action of which no very apparent explanation can be given. Among these gelsemium is a prominent member, and probably also arsenic and phosphorus.

There are symptomatic, pathological, etiological, and therapeutic reasons for believing that the blood vessels are at fault in cases of neuralgia, and therefore it seems necessary to consider true neuralgia as a sympathetic neurosis, affecting certain tracts of cerebro-spinal nerves, resulting in simple dilatation of the vessels of these nerves, brought about by unknown causes, or by reflex irritation, or possibly by the specific action of certain poisons in the blood.—James R. Whitehall, Edinburgh Medical Journal, Dec., 1887.

L. F. B.

NEURALGIA, CHLOROFORM, AND THE CONSTANT CURRENT.

Marvellous results are claimed by Prof. Adam Kiewicz (Progrès Medical) from the combined action of chloroform and the constant current in facial and other forms of neuralgia. The electrode is made of hollow charcoal into which the chloroform is introduced, and from which the current sends it into the tissues. That this power of penetration may be thus obtained is thought to be shown in the fact that when chloroform is colored with gentian violet and applied in the manner described to the ear of a rabbit, the tissue becomes dyed. In experiments with the human subject, the writer notes at the commencement the triple action of the constant current, the chloroform, and a condition of cataphoresis followed by a burning sensation and finally anæsthesia. Several remarkable cases of cure are cited. Anæsthesia is not obtained when the nerves are deep-seated, nor in sciatica.—Medical and Surgical Reporter, Feb. 18, 1888.

L. F. B.

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Original Articles.

THE RELATION BETWEEN TROPHIC LESIONS AND DISEASES OF THE NERVOUS SYSTEM.[1]

By E. C. SEGUIN, M.D.,

PRESIDENT OF THE AMERICAN NEUROLOGICAL ASSOCIATION, ETC.

THE question chosen by the council of the Association for discussion to-day, viz.: "*The relation between trophic lesions and diseases of the nervous system, excluding changes within the central nervous system itself*," is one which brings the critic face to face with an enormous accumulation of more or less well-observed, widely diverse, and not necessarily correlated clinical and experimental facts or data.

The very terms of the question involve to my mind a petitio principii: for the essential query in any review of the data is whether the lesions referred to are really trophic, in nervous causal relation.

Your referee has been informed that "permanent vasomotor changes, in so far as they can be shown to influence nutrition," may or should be included in the discussion. This I consider a wholly different field of physio-pathology and one whose introduction into the discussion would only tend to obscure, complicate and indefinitely extend the search after the true relation between trophic lesions and nervous diseases; and I shall consequently omit it from my remarks.

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[1] Referee's paper read before a joint meeting of the Association of American Physicians and the American Physiological Association during the Congress of American Physicians and Surgeons at Washington, D. C., Sept. 20, 1888.

We must also insist upon a strict definition of the word lesion, as meaning a positive histological alteration in tissue, thus excluding retarded or imperfect development, simple quantitative reduction of tissues, and alterations in local circulation and calorification (as observed in cerebral and spinal paralyses occurring before the full growth of the body, and occasionally after it).

Thus simplified the question may be re-stated in the following terms:

What are the lesions which may be supposed to be directly produced by disease of the nervous system (brain, spinal cord, and nerves); and what is the essential causal relation between the two factors?

To attempt to enumerate separately in a systematic manner all the lesions of the non-nervous tissues which have been observed clinically and experimentally, and ascribed to a direct morbid nervous influence (positive or negative), would be an extensive work, far beyond the time limits of this discussion.

Such "trophic lesions" have been described as occurring in almost all the non-nervous organs and tissues, internal and external. We find medical literature filled with examples of such lesions in the cutaneous tissue and appendages, in the muscular tissue, in bones and articulations, in vascular walls, in parenchymatous organs, and in internal epithelial structures. In other words, wherever nerve fibres terminate, and even where none can be demonstrated (as in cartilage) such lesions have been described.

Some simple mode of classification of these data must be chosen to facilitate discussion, and looking at the subject from the standpoint of the pathologist and practical physician I would suggest the following:

FIRST CLASS.—"Trophic lesions" occurring in parts whose sensibility is more or less reduced by the nervous disease, and which are exposed to the action of traumatic and infectious influences.

This class includes by far the largest number of the data, such as cutaneous ulcerations, fall of nails and hair, altered appearance of skin and nails, articular changes (arthropathies), fractures of bones, deep eschars, necrosis of digits, and most of the lesions observed in the hollow viscera lined by epithelia. To put it in another way, this class includes the various lesions observed in the course of posterior spinal sclerosis, injuries of the spinal cord, forms of "myelitis" so-called, and injuries to nerve-trunks. Speaking before this audience it is useless to further specify the "trophic lesions" referred to.

SECOND CLASS.—Trophic lesions occurring in deeper parts, not exposed to bacterial infection, and upon which traumatic influences cannot be demonstrated to act: in other words, the apparently spontaneous trophic lesions. This class is made up of the muscular atrophies occurring in the course of nervous diseases, and of a limited number of cutaneous lesions. Possibly some alterations in glandular function, due to nerve lesion, may belong to this class. Clinically these lesions are met with after nerve section, in the course of neuritis, and of disease affecting primarily or at least chiefly the ventral cornua of the spinal cord and their homologues in the cephalic prolongation of the spinal axis.

With respect to the first class of so-called trophic lesions, a careful study of the conditions under which they arise, and a comparison of them with the similar peripheral lesions which present as complications at the close of some non-nervous diseases with tendencies to asthenia and inanition, make it somewhat doubtful whether they can rightly be considered as direct results of suppressed or perverted nervous action. Even the strongest partisan of the truly dystrophic nature of these lesions admits that extraneous influences (as trauma and bacterial infection) play a certain though wholly secondary part in their genesis. But other observers hold an opposite extreme view, and claim that the real or efficient causes of the lesions are trauma and infection acting upon parts which have lost their automatic defence through anæsthesia, and whose circulation and general nutrition are lowered, but not specifically altered, by disease and inertia.

This second view is, I must say, supported by negative evidence, experimental and clinical, of such importance that it needs to be stated.

(*a.*) Ulceration of the cornea and even panophthalmitis are results of experimental and pathological injury to the trigeminus nerve, more especially of its ophthalmic branch and of the Gasserian ganglion. These ocular lesions are fully described in text-books, and are generally looked upon as typical trophic changes due to the nerve disease. Yet thirty years ago H. Snellen[1] and M. Schiff[2] separately demonstrated by the simple experiment of sealing the eye by sewing the edges of the lids or by fastening one of the animal's ears over it, that ulceration of the cornea could be prevented after section of the trigeminus. At some time prior to 1872 von Gudden[3] proved the same thing by a beautiful experiment. He took newly-born rabbits and produced perfect closure of the eyelids by an operation (artificial ankyloblepharon). When the wounds were healed, and the eyes absolutely sealed, he cut the trigeminus nerve by the usual intra-cranial method. Upon opening the eyelids from eight to fifteen days after the nerve-section he invariably found the cornea normal. These experiments make it clear that the nerve lesion is not the real or efficient cause of the corneal changes in the usual experiments, and probably not in human cases of disease of the fifth nerve.

(*b.*) Section and other injuries of nerve-trunks have long been known to be followed by so-called trophic lesions in the distal parts supplied by the injured nerve. Changes in the skin and hairs, falling of the nails, ulceration, and even extensive necrosis or gangrene have been elaborately described in animals and in man. Yet it is nearly forty years since Brown-Séquard[4] showed that (in animals) if the parts supplied by the injured nerve be kept perfectly clean and protected from traumatic influences, ulcerations, etc., did not ensue. He also demonstrated that wounds made in parts supplied by an injured nerve-trunk healed as well as wounds made elsewhere. These experiments (confirmed by numerous observers) show that the nerve injury in such experiments or cases is not the true efficient cause of the ulcerations, etc., and also (what is fully as important) that the nutritive functions which go to repair wounds are fully active in anæsthetic and paralyzed parts. In the practice of medicine we have frequent occasion to apply these data, in the prevention and treatment of ulceration, bed-sores, etc., by mechanical and antiseptic measures, after injuries to nerves or to the spinal cord, as well as in cases of paraplegia. In human cases of section of sensory nerves, while certain quantitative changes in the anæsthetic area are apparently inevitable, actual histological lesions can, I believe, be indefinitely prevented by guarding against traumatic influences.[1] As regards bed-sores in paraplegia you have probaby all seen them show healthy reparative action while the spinal disease was growing worse.

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[1] H. Snellen, De invloed der zennwen, op. de onsteking. Dissert. Utrecht, 1857. Also in Archiv. f. d. Hollandische Beitrage zur Natur. Heilkunde, Bd. I. 3, p 206 (1857)

[2] M. Schiff, in Canstatt's Jahresbericht, I., p, 121, 1857.

[3] Von Gudden, cited by Kondracki in his thesis, Ueber die Durchschneidung des Nervus Trigeminus, Zurich, 1872.

[4] Brown-Sequard, Gazette Medicale, 1849, p. 880.

(*c.*) It is a remarkable fact that such lesions as perforating ulcer, arthropathies, fractures, etc., which occur in the course of posterior spinal sclerosis and other nervous affections, are extremely rare in patients whose circumstances enable them to avoid over-exertion in the later stages of the disease, and to receive every needed care. This certainly would point to traumatism as a potent factor in the production of the so-called trophic lesions of tabes.

(*d.*) Cystitis was until a comparatively recent time considered one of the symptoms of myelitis and of injury to the spinal cord; though I suppose most of us to-day would speak of it as a complication preventable by the use of aseptic catheters introduced with the greatest care.

The negative demonstrations and arguments to the effect that the greatest number of the most formidable of the so-called trophic lesions of the first class are preventable and curable, appear to my mind almost overwhelming proof that the efficient cause of these lesions is not a suppression or perversion of nervous action or influence.

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[1] Except some lesions of the second class which are unpreventable, though it would seem not invariable results of nerve injuries.

Consequently I would refuse the name of trophic lesions to the phenomena embraced in the first class of data.

We are now brought to the study of the second class of "trophic lesions," those in which extraneous or traumatic causes cannot be shown to act. It is perhaps in the study of these that the problem of the relation between the nervous disease and the lesion can be best approached.

The most typical lesions of the second class are muscular atrophy with degeneration, and the cutaneous affection known as herpes or zona. Probably other so-called skin diseases belong to this group, but full demonstration of their nervous origin is wanting.

(*a.*) The natural history of neuro-muscular atrophic degeneration is well known to all of you. Within a few days after section of a nerve-trunk, or after destruction of the ventral ganglion cells with which a nerve-trunk is associated in the spinal axis (clinically, in cases of nerve injury, neuritis, poliomyelitis, chronic degeneration of ventral ganglion cells, etc.), the nerve fibres distal of the point of injury or disease, and all the muscles innervated by the fibres lose certain properties known as conductibility and irritability, react abnormally to electrical stimulation, and if examined with the microscope show distinct and invariable alterations. Later, the affected muscles undergo a marked reduction of volume.

In adult animals, after certain lesions, an extreme degree of atrophy is established and persists. In young animals, after certain lesions (simple nerve injuries and neuritis more especially), a process of regeneration sets in which in a few months leads to return of a normal anatomical state of the nerves and muscles, and to renewed functional activity. In some cases there are also more diffused changes produced, arrest of development of parts, quantitative modifications which should not be confounded with actual lesions. What

I wish to emphasize is that we have here to deal with *qualitative or histological changes in nerves and muscles, which occur with fatal necessity when the cause has acted,* and which are demonstrable by microscopic examination and by electrical tests (reaction of degeneration). Further, that these lesions are unpreventable, and in one sense incurable; no traumatic or infectious influence can be traced in their genesis, and no amount of care or any form of treatment will prevent the appearance or thwart the evolution of the changes.

(*b.*) The so-called herpetic lesions of the skin. The vesiculo-pustular affection appears all at once or in successive crops upon areas of skin supplied by one or more of the cerebro-spinal nerves; the distribution of the primary eruption and of the subsequent scars corresponding exactly with nerve territories. Hence the names for varieties of herpes, such as H. frontalis, H. corneæ, H. progenitalis, H. intercostalis, etc., etc. Along with the eruption there are not rarely subjective symptoms of nerve irritation, such as burning, prickling, pain, or numbness. In some cases pain (neuralgia) persists long after the eruption has subsided. Usually, scars remain, and they may be extremely deep. Autopsies have shown, beyond room for doubt, that in such cases the nerve trunk supplying the affected cutaneous area, and especially the ganglion upon its dorsal root, are the seat of inflammatory and degenerative processes. Unfortunately, with the means at our command the lesion has not yet been traced into the terminal filaments and end-organs of the affected nerve in the cutis and epidermis. The demonstration is, however, almost complete that we are here in presence of a neuro-cutaneous and continuous lesion, corresponding to the continuous lesion observed in (*a.*) the neuro–muscular apparatus. Here again we have to deal with an evidently non-traumatic and non-infectious lesion (the skin lesion), revealed by regular and constant symptoms, unpreventable by mechanical means and incurable in a strict sense of the word.

Similar herpetic cutaneous lesions are observed after injuries, more especially such as give rise to irritative and inflammatory conditions of the nerves. Simple section is more apt to be followed only by cutaneous alterations of the first class. It is, furthermore, possible that traumatic neuritis may produce other true lesions of the skin besides herpes, but this is not yet proven.

To these two varieties of lesions, embraced in the second of the classes which I propose, I am ready and willing to apply the term *trophic lesions* in the true sense of the word; *i. e.*, they are *histological alterations set up directly and fatally by the nerve disease, without the intervention of accidental or extraneous causes.* The relation of cause and effect seems indisputable, and we may therefore say that the efficient cause of the trophic lesion is disease of a part of the nervous system.

And, now, as to the mechanism or physiology of these trophic lesions. We cannot go far in this direction without entering the domain of pure speculation. It is only a few weeks since I heard my illustrious master and friend, Professor Charcot, state in a clinical lecture that we know absolutely nothing of trophic nerves and their mode of action. In this negation I most fully concur, especially if it be applied to the confused or unclassified mass of so-called trophic lesions about which so much has been written. The existence of trophic nerves as such is unanimously denied by physiologists, and much of the speculations of physicians have been made without scientific basis in anatomy and physiology.

The attempt to simplify the problem which I herewith submit to the Association may not advance our actual knowledge, but it may possibly serve as a step toward a more exact study of the subject, and may give rise to a beneficial discussion. Allow me in closing to attempt to show in what way this analysis may be a slight step in advance.

I have, in the first place, rejected from the category of trophic lesions all vaso-motor, calorific, and metabolic phenomena, as well as all mere quantitative reductions in tissues and organs; reserving the name for such alterations as are characterized by demonstrable histological changes.

This will doubtless be objected to, as in a certain sense the excluded phenomena have much to do with nutrition, and the word "trophic" leads the mind inevitably to think of *changes in nutrition*, which for me is far too vague and general a conception to prove of help in the study of our subject. Besides, since Claude Bernard's, Brown-Séquard's and Ludgwig's discoveries a sort of antagonism has been revealed between mere vaso-motor variations and the activity of the cellular life (salivary secretions, etc.).

In the second place, I have attempted to show that histological lesions apparently due to nervous disease may be divided into two classes; one in which the morbid nervous influence is of doubtful or at least of secondary causal value, while the active or efficient causes of the lesions are extraneous and accidental (traumatism and infection); whereas in the second class (by far the smaller at present) extraneous causes are unimportant or even wholly wanting, while, as far as our present means of observation go, the efficient cause of the lesions is a morbid state of the nervous system.

The phenomena which make up the first class I hold to be mere complications having a complex etiology, while those of the second class are really trophic lesions due to disease of the nervous system.

I would not be understood as claiming that the classification here proposed is final or absolutely exact in all its details. For example, lesions of the second class may co-exist with others of the first class in paralyzed parts: *e. g.*, atrophic or herpetiform lesions in paraplegia of the traumatic form especially. Again, a reasonable doubt may be entertained as to whether arthropathies always belong to the first class of lesions. It would be necessary to make a new analytical study of all varieties of so-called trophic lesions, and classify them according to their histology, etiology, in the light of the subdivision here proposed.

Third.—While not pretending to be able to throw any new light on the intimate nature of real trophic lesions, I desire to point out that possibly (and I say this with all due reserve) the mechanism of these alterations, or as the question puts it, the relation between the trophic lesions and disease of the nervous system, lies or is embraced in a law of *inter-dependent life in continuous tissues.* As regards the neuro-muscular changes of our second class, the operation of such a law seems highly probable. Anatomically and physiologically the neuro-muscular apparatus from the ganglion cells of the ventral cornua of the spinal cord to the striated muscular substance is a unity or becomes one before the completion of fœtal life. Whether the ultimate nerve fibrillæ and other prolongations of neural substance which lie under the sarcolemma actually blend with the sarcous substance, is, I know, an unsettled point; but their coaptation and physiological continuity are established. Besides, in lower animal forms true neuro-muscular structures do exist.

With reference to the neuro-cutaneous apparatus, we need still more delicate and reliable histological researches to show what is the true relation between ultimate nerve fibrillæ and the peripheral neural substance with the cells of the cutis and epidermis. While some observers claim actual blending of the two substances, by penetration of nerve fibrillæ into epithelia and by the interposition of cell-like nervous expansions in among the cells of the skin, others are in doubt as to the arrangement. Still, at the present time, the weight of evidence is in favor of the existence of continuity between sensory nerves and some of the elements of the skin. The same statement may be made with reference to the relation existing between the termination of glandular nerves and the epithelia of gland.

I therefore venture to suggest that *disease of the nervous system produces true trophic lesions when it interferes with the associated or inter-dependent life of continuous tissues.*

TRANSACTIONS OF THE AMERICAN NEUROLOGICAL ASSOCIATION.

FOURTEENTH ANNUAL REPORT.

Tuesday (First Day), Morning Session.

The American Neurological Association convened at Willard's Hotel, Washington, D. C., September 18th, 1888, and was called to order by the President, Dr. James J. Putnam, of Boston. The President then delivered the annual address.

ADDRESS OF THE PRESIDENT, DR. JAMES J. PUTNAM.

Gentlemen of the Neurological Association :

It is my pleasant duty to bid you all welcome to Washington, in the name of yourselves ; and to declare our meeting to be formally opened.

The Council has done, and will do, everything in its power to reduce to a minimum the routine business, and to leave you free to attend to the scientific communications which we have come to hear.

The programme is a full one, and I shall detain you only a few moments from attacking it.

I wish to ask your attention, during the space that your courtesies allow to the presiding officer, to the consideration of the question, whether the time has not come when this Association might increase its efficiency by adopting some plan of co-operative work, to supplement and assist the individual work of its members.

I purposely avoid for the moment the term "collective investigation," because I do not mean to propose that we should necessarily adopt the exact method indicated by that name, but only to express my belief that, in a general way, we have reached such a point in our development, that we can count on each other enough, personally and scientifically, to be able to look to each other for support during the progress of our undertakings, instead of only for criticism at their close.

The possible advantages to be expected from co-operation in some portions of our work are, I think, incontestible; the difficulty lies in realizing them.

That which usually gives to a scientific enterprise its pith and point, is the genius, faith, energy, or personal ambition of a single man, and I do not propose that we should lose sight of this fact. Nevertheless, I think we may make a step forward by attempting systematically what we now do in a desultory manner, in the way of mutual assistance.

No one of us, not even the most talented and industrious, is able to utilize for the purposes of original investigation more than a small proportion of the vast array of facts that crowd in upon him on his daily rounds; but we should be ready to devote ourselves with increased zeal to studying and classifying those that remained, if we were sure that our observations were being made in such a way that each one would lend its testimony to the proving or disproving of some particular hypothesis of pathology or physiology or therapeutics. For this purpose, however, uniform methods of examination and tabulation would be necessary, and the knowledge that some one stands ready to collaborate the material that has been accumulated, so that our time and labor should not have been thrown away.

Again, there are many subjects of the very highest practical importance, those namely, where the problems at stake are of a statistical character, which can only be dealt with satisfactorily through some species of co-operative inquiry. And co-operation, directed to this end, means simply an attempt to avoid the annoying differences and errors as regards method of examination and point of view, that come in to vitiate the statistics of observers working at different times and places and without mutual understanding.

I am speaking as if co-operative investigation were a new thing, whereas, as we all know, it has been tried, in various forms, a hundred times, sometimes successfully, sometimes unsuccessfully, more often, no doubt, with partial and temporary success, then to be given up or changed for something better. Such temporary success even is often justification enough for the experiment. One does not look for institutions of millennial permanence, but for ever new steps in advance.

Let us glance at the history of one or two of the prominent co-operative efforts of recent times.

The first Collective Investigating Committee of the British Medical Association was appointed in 1881, with Dr. Mahomed as paid Secretary. Two volumes of records have been publishd, besides reports in the *British Medical Journal*, on various subjects.

At the Industrial Congress at Copenhagen in 1884, an address was made by Sir William Gull, favoring the appointment of an International Committee for a similar purpose, and the motion was warmly seconded and the committee appointed. The Verein für innere Medicin, in Berlin, had already given its sanction to this method of research, and had formed a committee with Leyden as chairman.

It could not be claimed, perhaps, that the work of these committees has always been of the first quality. The information was collected through circulars sent about rather broadcast, and the answers must have been of varying degrees of merit. There can be no doubt, however, that something substantial has been gained, not only of the nature of harvest, but also of seed-grain, since those who took part in the inquiry must have been trained and stimulated, so that another time they would both observe and report to better advantage.

An Association like ours would have a better chance of success in carrying out a plan of investigation of this kind than where the profession at large was called upon to give its co-operation; since most of our members are familiar with the methods of accurate research, and would understand the bearings of the investigation that we should make.

To speak of institutions nearer home, some of the New York physicians formed, a number of years ago, a society for collective investigation in therapeutics. It lasted, I believe, but for a few years, but during that time published papers of real value.

If care were taken to select questions for inquiry which were of fundamental and practical importance to every observer, such a scheme would have a greater prospect of permanence.

It is not, however, at the history of committees for collective investigation that I look with so much interest, as affording examples for our instruction, as to the work of individuals whose influence and energy have created schools and inspired followers; men like Prof. Charcot, to name but a single instance.

I have also in mind the results that have been gained, especially in the department of physiology, by dividing up an important inquiry into a number of related parts, which are assigned to different persons acting temporarily as assistants to one person, by whom the investigation is mainly undertaken.

Charcots are not to be had for the asking; but we have our share of workers with ability, energy, and zeal enough to be good workers in given directions, and worthy of support. My own feeling is that there is more vitality in a scheme for which one or two persons make themselves responsible, especially if they are already identified with it, than in the work of a committee, where the interest is less personal and the responsibility more divided; and that the efforts of our Association should be directed to furthering the plans of such persons, by moral and scientific support and by furnishing funds for printing circulars if necessary, and so forth.

I can see no better way of accomplishing this than that the proposer of any investigation should appeal, either personally or through the Secretary, and, if necessary, at the expense of the Association, to the members, stating his plan and asking for support. If his scheme recommended itself to others so far that he should be able to form a sufficiently large working committee, he should confer again with them and submit details. The rest of the committee should have the right of criticism, and of withdrawal if not satisfied, and the publication of the results should be made in the name of the committee with the originator as chairman; or, if it was agreed that the originator should do the greater part of the work, and make himself responsible for the conclusions, he might publish the results in his own name with those of the rest as collaborators; and the question as to whether the Association should vote an appropriation for his assistance, if any was required, and the amount of the appropriation, should be decided in any way that the members of the council might determine.

It is not my purpose to discuss in detail what subjects would be best suited for investigation in this manner, and I will refer only to one or two by way of illustration.

In the first place, there are those such as were proposed for the British Medical Society, including the vast subject of heredity; the laws of degeneration and (as being of quite equal importance) of regeneration in families and races; and the strange transmutation or evolution of diseases from one form to another, which seems to be entirely distinct and different.

To come nearer home, there are various questions in therapeutics, such as the action of iodide of potash on non-syphilitic brain tumors, as one member suggested to me.

Again, we all know that Dr. Dana has been studying the racial relations of the neuroses, a subject which Clifford Allbutt also has recently touched upon in his suggestive address, and for the investigation of which no country could be better suited than ours. It would be an easy matter for us to collect large numbers of facts under his guidance upon these points, and the comparison of the experience of the different cities would be particularly instructive.

The subject of cranial measurements, for which large masses of facts are also needed, is another fit matter for such methods of research, and I am glad to be able to say that a valuable introduction to investigation of this kind is among the candidates' papers for this year.

I have myself been interested for a number of years in an inquiry which I think could soon be brought to a fairly definite conclusion in this manner, namely, the effect of very small doses of lead as predisposing to disease. I published, last year, the report of the analysis for lead of the urine of eighty-six persons with various symptoms of nervous disease, functional and organic, but not presenting unequivocal symptoms of lead poisoning. In forty-eight cases, or more than fifty per cent. of the whole number, lead was found, and for certain groups of cases the proportion ran up to a much higher figure. Out of a group of eleven cases, on the other hand, made up of persons who were practically in good health, lead was found in only two cases.

These facts seemed to me of importance; but it was also evident that their clinical significance would be diminished if it should appear, on further investigation, that the urine of most or of many healthy persons also contained lead, showing that its presence need not be injurious. I therefore made arrangements to have the urine of a large number of medical students analyzed, and up to the present time I have reports on twenty-three cases. In only three of these, or thirteen per cent., lead was found, and then in very small amounts.

Even these few affirmative cases, together with others of a similar kind which I have collected but which do not belong to this particular set, show that the mere finding of lead in the urine is not an indication of active poisoning; but, on the other hand, it remains to be proved that the majority of persons can carry lead about with them all their lives and still keep their tissues as healthy and as resistant as if it were absent; and it is still an important and open question, and one that only the testimony of large numbers of facts can answer, whether the first signs of poisoning are always the classical symptoms with which we have long been familiar, or whether lead, like syphilis, may predispose the central nervous system to degenerative changes of an entirely different order from those seen in typical cases of poisoning.

Such an investigation is too expensive, and requires too much material, for a single person to deal with satisfactorily; but if a large number were to take part, reasonable conclusions could soon be reached; and it is certainly important for us to understand accurately the effect of influences to which the whole community is more or less exposed.

But I do not think it is by collective investigation alone that the members of the Association can be useful to each other, and to the profession, through co-operative work.

In the first place, I hope that another year we may do still more than we have done this year in the way of notifying members of the contents of papers to be read. The Surgical Association has, I think, set us a good example in printing quite a summary of the points to be raised in their discussions, and I feel sure that others besides the principal speakers will be stimulated thereby to take an active part.

Another piece of joint work which has occurred to me is the formation, either actually or on paper, of a collection of microscopic or other specimens, to be kept by the secretary or by an officer appointed for that purpose, or deposited in the Army Nat. Museum. If the collection should consist of actual specimens, they would naturally be duplicates; if it should consist of a list only, this might contain a statement of the preparations which members were willing to lend, under suitable conditions, and of those which they would only allow to be consulted in their own laboratories.

Perhaps this scheme is chimerical and would lead to nothing. If the specimens were likely to be largely consulted, I admit that their owners might be put to inconvenience. As a matter of fact, however, I presume they would be consulted but little, but I think that occasionally this would be done, and with the greatest possible benefit.

If this plan should lead to the formation of a central museum of such specimens as crania and brains which had been described, and would otherwise lie idle on a top shelf, or of photographs, drawings and casts of such specimens, the plan would, I think, be justified. Possibly we might some day publish sets of illustrations on the plan of the new Iconographie of the Salpétriere.

[The National Army Museum, as Dr. Billings assured us in his address, is ready to take charge of microscopic and other collections, and what could be a more pleasant and fitting memorial of our Association than to have its name linked with such a collection, and to have been the first to set an example which would be sure to find followers.]

I have intended, in these remarks, only to ask you to reflect whether some way cannot be found through which, by combining, we can reach certain ends either of the nature of actual discovery, or of critical suggestion, which could not be reached by one person alone; and I have tried to indicate certain methods by which we should be the most likely to arrive at the best, though perhaps not the most conspicuous results, with the least danger of failing through overreaching ourselves.

If the general suggestion pleases you, I dare say better means than mine can be suggested for carrying it out. The time and place seem to me appropriate for initiating such an enterprise. This is the first meeting of the Congress of all the principal Associations of the country for their mutual stimulation and support, and our first meeting in the capital of the country.

What is, however, of more importance, we have, I think, reached a period in our development, when such a step is justifiable.

We have learned to know each other better personally, and we have all of us learned to do at least fairly good work, and some of us work of the highest quality, and all of us have learned to appreciate in what really good work consists.

A remark made to me ten years ago by one of the most prominent neurologists of Germany, that he could not make much use of American cases, they were so inadequately reported, would now not be in place.

We need now to bring our best workers more and more prominently to the front, and give them a chance to exert all the influence of which they are capable; to show that we appreciate that our specialty is bound to be of more vital importance than almost any other, and that it is, above all, the specialty of research.

And it should be remembered, also, that we are not a local, but a national Association, with natural functions of the nature of encouraging broad and liberal enterprises of education and investigation, and not merely a collection of workers, bent only on prosecuting our individual aims, and with no bond of union but our yearly meetings.

REPORT OF THE TREASURER.

Dr. Græme M. Hammond, of New York, Secretary and Treasurer, presented the Annual Report, which, on motion, was accepted. There was a balance in the treasury of $217.60.

SCIENTIFIC COMMUNICATIONS.

Dr. Philip C. Knapp, of Boston, read a paper on Nervous Affections following Brain Injury ("Concussion of the Spine," "Railway Spine," and "Railway Brain").

(For paper see October number.

Dr. ISAAC OTT read a paper entitled

HEAT CENTRES IN MAN.

In this abstract I will give only the principal points of my paper on this subject.

There have been localized in the lower animals six centres whose injury causes increased temperature. The cruciate about the Rolandic fissure, the sylvian at the junction of the supra, and post-sylvian fissure; the caudate nucleus, the tissues about the corpus striatum, a point between the optic thalamus near the median line ; and the antero inner end of the optic thalamus. These centres have their own laws, which serve to distinguish one from the other. Dr. W. Hale White[1] has published a paper containing a number of collected cases bearing on this subject, and I have mainly used them to support the theory of localized heat-centres in man. He has ruled out all cases where the cause is not purely nervous.

In support of a heat-centre about the Rolandic fissure, there are two cases of injury in this region, which were followed by a temperature of 104° F. There is also a case of softening of the brain in the neighborhood of the Rolandic fissure, with a temperature of 105° F. reported.

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[1] Guy's Hospital Reports, 1884.

Corpus Striatum.—There are recorded two cases, one of degeneration of the corpus striatum, another of echinococcus—cysts in it which caused an elevation of temperature. These cases are very clear, and undoubtedly seem to show the corpus striatum to be a thermic centre.

Thalamus Opticus.—The cases bearing on this point are not very definite.

Subnormal Temperatures.—There has been recorded, one by Dr. Farnham of Cambridge, a case of typhoid whose preceding high temperature was succeeded by a fall to 95.4° F. In my experiments upon the heat centres it was found that the elevated temperature was succeeded by a fall below normal, and heat production also corresponded to it. Dr. Williams also found the temperature in phthisis characterized by an afternoon pyrexia and a subnormal temperature (94° F.) in the morning.

In the convulsive affections I have shown that tetanus mainly causes heat through the stimulation of thermic centres, and not by muscular movement. In chorea Prof. Osler, from an analysis of 410 cases, arrived at the conclusion that fever takes place when rheumatism sets in, when endocarditis becomes severe, and in some cases of chorea insaniens. There may be the most intense and violent movements without any rise of temperature, and in a great majority of cases remain afebrile throughout.

In epilepsy, according to Lemoine, in nearly two hundred observations of isolated attacks, the average rise was 1.2° F.

Tumors and lesions of the spinal cord, oblong medulla, and pons varolii are usually followed by a high temperature due to a removal of the influence of the thermotaxic centre, permitting the spinal thermogenesis to become exaggerated.

Low temperatures are due to the excitation of the thermotaxic centres, or their fibres, or to affection of the thermolytic apparatus.

High temperatures have been reported ranging as high as 125° F. to 128° F. These cases, if true, and I am inclined to believe them possible, are paralelled by Allenberg and Landois' experiments upon the Rolandic centres in dogs, where they found a difference of 23° F. between the extremities. The rapid and high elevation of temperature often seen in malarial paroxysms indicates, to my mind, an action on the thalmic heat-centre. This is the only centre whose rise and fall of temperature corresponds in time and degree to that seen in these conditions.

Cases of temperature of 110° in children have been reported which lasted for about an hour and then fell to normal, the patients recovering. In the several hundred of temperature experiments upon animals, in only one case have I seen the preceeding cases paralelled. Thus, after a grain and a half of atropin per jugular,the temperature sank to 97° F., but on applying sciatic irritation the temperature rose rapidly to 102° F., a gain of five degrees in sixteen minutes. It is well known that a dose of atropin of this size would prevent the sensory irritation from having much effect upon the vaso-motor apparatus in producing contraction of the vessels. Atropin in large doses paralyzes the main vaso-motor centre.

Diagnostic Value.—If the symptoms point to a lesion of the pons certainly high temperature will be an important aid. If the symptoms indicate a lesion about the corpus striatum then high temperature with general muscular wasting will be strong corroboration of the diagnosis.

These points will be either confirmed or refuted by lesions in man, and I hope this paper will stimulate neurologists to make more frequent observations upon temperature.

DISCUSSION ON DR. OTT'S PAPER.

Dr. DERCUM introduced Dr. Hare, who referred to the experiments of Dr. Ott. The fact that destruction of a centre caused elevation of temperature could not be said to prove that the centre in question was a heat centre, but rather that it was an inhibitory heat centre; its function was to inhibit rather than to stimulate heat production. There was an apparent fallacy, too, in the statement in regard to atropin. Atropin does not paralyze the vaso-motor centres. The chief action of atropine is upon the motor rather than upon the sensory system.

Dr. GRAY referred to a case of leptomeningitis above the fissure of Sylvius, in which the temperature was at no time above the normal.

Dr. LLOYD referred to a case of subnormal temperature after injury of the cortical motor region.

Dr. OTT explained that he had stated simply that destructive excision of the centres caused a rise of temperature; he had not stated whether those centres were inhibitory or not. In regard to atropin, he had stated that atropin inhibited the vaso-motor reflexes. The subnormal temperature in Dr. Lloyd's case would be explained by irritation of that region of the cortex, by the removal of which temperature was increased. It was not usual to observe the temperature of patients suffering with nervous disease, and variations might be overlooked. The fact that lesions about the fissure of Rolando were not always followed by an elevation of temperature proved nothing one way or the other. Paralysis also did not always result.

Dr. E. D. FISHER, of New York, then read a paper entitled:

CLINICAL REPORT OF CASES OF EPILEPSY FOLLOWING CEREBRAL HEMIPLEGIA.

Of late much has been written in regard to cerebral hemiplegia in children, or cerebrale kinderlähmung, both in this country, in England, and on the continent. In recording the following cases I would, however, lay particular stress on the epileptic seizures associated with the disease. The picture of these cases has been so often drawn that it has become a familiar one. The hemiplegia unilateral and bilateral, as it is sometimes termed, coming on at birth, or in the first few years of life, is similar in most respects to that of adult hemiplegia, with the additional symptoms of more or less interference with the growth of the parts and a more marked condition of contracture. These cases are to be found in every almshouse or home for the feeble-minded. There is usually marked contracture of the upper extremity, the fingers closed in the palm and resisting in many cases all efforts to draw them out. The lower extremity is also contractured, the gastrocnemius being involved, causing the patient to walk on the ball of the foot with the heel drawn up ; the face is but slightly affected, at least in the latest stages of the disease. There is some interference with the growth of the parts, and while this was present in all of the following cases, in only one was it marked, affording at once a differential diagnosis from infantile spinal paralysis. In reference to this point I would refer to three cases reported by H. Quinke in the Deutches Archiv. f. Klin. Med., bd xlii., h. 5, of Glioma involving the central convolutions, in which, along with the paralysis, marked and rapid wasting was present, leading the author to believe that a trophic centre must exist in close proximity to the motor centres. The author does not consider it possible that the wasting could be due to inactivity, as is the case usually, or as the result of descending degeneration of the lateral columns, as it occurred so soon after the paralysis, and microscopical examination revealed no changes in the anterior cornua.

We have also present in these cases, imbecility or idiocy, and epilepsy, conditions not existing in the spinal lesion. All these symptoms, with the exception of the last two, may exist wherever the motor tract is interrupted in its course, whether in the cortex, sub-cortical region, basal ganglia, capsule, crura, pons, medulla, or cord. It rarely occurs in other than the cortical and sub-cortical regions, and if accompanied by imbecility and epilepsy, can only be referred to the cortex.

Wallenberg, in Westphal's Archiv. f. Psychiatrie, bd. xix., h. 2, reports a typical case of spastic cerebral hemiplegia of childhood, in which the lesion involved the cornua with descending degeneration and as was to be expected there were no mental symptoms or epileptic seizures. In regard to the epileptic seizures, there is no rule as to their commencement on the paralyzed or on the non-affected side, indeed they more often resembled in their onset idiopathic epilepsy, and if a warning were present, consisted in an epigastric aura, vertigo, &c. As Jacksonian epilepsy may resemble idiopathic epilepsy in its onset and course, so may the idiopathic form simulate in every particular the Jacksonian.

When the hemiplegia is congenital, the cause is probably in the majority of cases, meningeal hemorrhage, induced either through injury in utero or at time of birth. Porencephalus is also a factor in these cases. We find evidence of the hemorrhage in the form of cysts or adhesions, between the dura and the pia mater, with sclerosis and atrophy of the convolution, accompanied by secondary degeneration extending into the cord. Both hemispheres may be involved, and very commonly over the convex surface. This would sustain Gowers' statement that in congenital cases the hemiplegia is more often of the double form than in the acquired cases.

In these latter cases, occurring usually at the age of two or three years, Gowers believes that thrombosis of the longitudinal sinus is a very frequent cause, with thrombosis of the veins entering the sinus resulting in capillary hemorrhages, atrophy and sclerosis of the convolution with secondary degeneration. Embolism is more likely to occur in cases of more advanced years, or to follow the exanthemata rheumatism, &c.

Poli-encephalitis and intra-cerebral hemorrhage are probably not as frequent causes as those previously mentioned.

In an interesting paper read before this Association in July of last year, Dr. P. C. Knapp enters very fully into its etiology, and also gives a complete bibliography of the subject. I would also refer to Dr. Osler's lectures on the subject in the Phila. Medical News. I would differ with Dr. Knapp in reference to the interference with the growth of the members affected, agreeing with Prof. Henoch that as compared with infantile spinal paralysis it is comparatively small, the wasting of the muscles being almost exclusively one of disuse, as seen in those cases with permanent contraction; whereas, when athetoid or choreiform movements were present, the muscles were often well developed.

In three cases affected with congenital choreiform movements either constantly present or increased by voluntary action, I found the muscles well developed, and although muscular power seemed somewhat decreased, it appeared to be more awkwardness in movement than actual loss of power. These cases represent, to my mind, congenital injury to the brain substance with a multiple sclerosis, not entirely destroying the cortex or involving all the motor fibres and leading thus to irregular motor impulses. These cases are similar to those of the congenital type of spastic hemiplegia, the lesion, however, being more diffuse in distribution and not so destructive in character. I would refer here to an article on intention tremor by Dr. Stephan in the Archiv. für Psychiatrie und Nerven Krankheiten bd. xviii., h. 2.

My cases are mostly congenital, or occurring between the ages of one and three. In one case, however, resembling in every particular the spastic hemiplegia of childhood, with marked contractions, equino varus, exaggerated reflexes and epileptic seizures, the paralysis occurred at 23 years of age,followed one month later by epileptic attacks, always commencing on the paralyzed side.

In regard to longevity, in most instances the average of 20 years, as given by Henoch, was far exceeded. The presence in greater or less degree of mental weakness is always marked ; this is accounted for by the occurrence of the disease in the early stage of cerebral development ; epilepsy did not occur in many of these cases for some time following the paralysis.

The contractures decreased in some cases in the course of years, so that in one instance the hand became a fairly useful member. Relaxation during sleep of the spastic condition was not observed. The extreme spasticity caused in voluntary acts was illustrated in cases 1 and 18. In the latter the whole of the affected side, including the muscles of the face were thrown in violent spasm on the slightest attempt to rise or even extend a limb ; in the former, flexion of the arm caused flexion at the wrist, while extension produced excessively violent extension of the wrist, this was beyond the control of the patient and was only overcome by great force.

As remarked in the beginning of this paper my special interest in these cases was the associated epilepsy, and it has been my study to inquire into the nature of the attacks, their onset and course, in order if possible to see whether any difference existed between them and those of idiopathic epilepsy, inferring if none can be found, or at least if no well-founded and persistently acting difference exist, that in all probability, the seat of the disease in idiopathic epilepsy must be in the same region and probably of like nature as in spastic hemiplegia, although not so marked in its changes. Perhaps the most distinctive characteristic of epilepsy is the loss of consciousness, coming on suddenly. In Jacksonian epilepsy the consciousness is often involved, but here we have a case of convulsions due to mechanical irritation corresponding to the physiological experiments of the laboratory. The intelligence of the patient is also much less apt to be affected than in petit mal, even when the latter is not associated with grand mal.

In cases of epileptic seizures from tumors of the brain, the convulsions may be due to a general pressure or disturbance of cerebal circulation and its attacks are not localized but commence bilaterally, and are preceded by loss of consciousness.

A statement of Luciani, sustained by many autopsies, may be of interest here, that while a paralysis may be strictly localized as to the cerebral lesion, it is not possible to make more than an approximate localization in epileptic seizures. This would the more readily occur where the tumor was of large size or of such nature, or so situated as to produce a general disturbance of the circulation. Such cases are found in the epilepsy of spastic hemiplegia, and although the lesion is unilateral I find that the attacks are more often of this character than that of the Jacksonian type.

This then, approaches the form of idiopathic epilepsy where the marked feature is the disturbance of consciousness which precedes the convulsion, and is indeed the most serious phase of the case, as here the higher centres, the latest evolved, and rerepresenting all the lower centres, according to Hughling Jackson, are affected, and we have the resulting dementia, or at least feeble mindedness so often present. When, however, the lesion can be localized as due to a circumscribed pachymeningitis following insolation for instance, the attack commencing with a localized tremor or tingling preceding the loss of consciousness, although the convulsion may become universal and of great severity, still such patients may live on for years with but slight if any mental impairment.

My cases as I have said, resemble the idiopathic epilepsy rather than the Jacksonian form,contrary to the expectation from the localization of the lesion ; the explanation probably lies however in the consideration that the disease in question is one of early life, during the most active period of cerebral development, and is always accompanied by interference with this development and the growth of the cells of the cortex.

We are justified by analogy to infer that in idiopathic epilepsy the seat of the disease lies in the cortex cells, and consists in a primary change in their nutrition. That it is primary and not induced by any disturbance of circulation following the convulsions would seem to be clinically proven by the fact that in many cases of petit mal, when convulsion can be excluded the mental disturbances are the most marked.

In looking over my cases of idiopathic epilepsy I find mental weakness almost invariably present. As Luciani has well stated, "the central organ for epilepsy upon which its pathology substantially rests is always the complex of the motor centres of the cortex, whether the irritation arises directly or reflexly." Thus, any internal organ or any portion of the periphery may become a epileptogenous zone for the origin of an epileptic seizure, but this does not result unless the cortex is in a condition to respond to the irritation, in other words, unless we have a morbid condition, in which case the equilibrium or stability of the cells is easily disturbed.

Thus it is that heredity becomes as important a factor in prognosis as in other mental disorders. In conclusion, it is noticeable in these cases that the great majority are of the hemiplegic type, only one side being involved, and in several instances the paralysis was but slightly marked, so that only a careful examination revealed the fact, that there was some slight interference with the growth of one side, with exaggerated patellar reflex of that side.

This would lead us to a more careful examination of all cases of epilepsy occuring in early life, and probably many now referred to hereditary or unknown causes can be more properly classed as due to cerebral injury in utero, at birth, or during the first few years of life. These facts ascertained however,do not improve the prognosis, as we may see from the review of the cases detailed. In regard to treatment I have but little to say. Trephining after changes have occurred in the cerebral and spinal structure would seem useless, The removal of some portion of the cortex where the epileptic seizures are excessive and unilateral in character, at least in the beginning, has proved in some cases beneficial. The bromides appear to act as beneficially as in ordinary epilepsy.

CASE I.—Geo. T., aet. 25, had severe fall of two stories when a child. Paralyzed in right arm; lower extremity unaffected; arm and hand held in position of flexion, but can be voluntarily extended. Seizures occur about once a month. Patient very unintelligent ; reflexes somewhat increased, especially of right side. Warning of present tremor commencing in right side.

CASE II.—Henry H., aet. 17, paralyzed at age of two years on right side ; patellar reflex exaggerated in affected side ; marked contractures ; gait spastic ; right limbs smaller than left ; speech slow, patient unintelligent ; left internal rectus weak. Attacks occur every three or four days, commencing on paralyzed side.

CASE III.—James J., aet. 56, paralyzed on right side at six years of age ; one year after had epileptic seizures. Attacks occur every four months ; increased by drinking ; no warning. Patient presents usual spastic condition of these cases.

CASE IV.—Henry D., 43; hemiplegia at 13 years of age; no history of rheumatism, no cardiac lesion. Four years later first attack. Patient has been under observation for the past ten years at the almshouse. Attacks come on without warning ; generally falls suddenly backwards ; attacks vary from eight or ten a month to five in one night ; any excitement, joy or sorrow, will bring one on ; speech unintelligible and general intelligence growing less.

CASE V.—Geo. Y., aet. 25, spastic hemiplegia from 3 years of age. Attacks about once a week ; no warning ; not limited to or beginning on paralyzed side , dementia.

CASE VI.—James L., 55, right leg amputated six years ago, following injury. Three months later epileptic seizures; now about three in a month. Memory good.

CASE VII.—Arthur C., 50, paralyzed on right side when seven years old. Attacks very frequently ; not beginning or limited to one side ; no warning ; speech unintelligible ; dementia.

CASE VIII.—John K., 19, right hemiplegia following fall at two years of age. Attacks nine to fourteen a month; generally no warning ; dementia. Reflexes exaggerated on both sides, most marked on the right.

CASE IX.—Wm. L., 26, hemiplegia resulting from a fall at age of three. Attacks five to nine a month ; generally warning of dizziness ; reflexes but slightly exaggerated ; dementia.

CASE X.—John H., 24, congenital paralysis of right side. First seizure at age of thirteen; no warning; generally about one a month ; feeble-minded.

CASE XI.—Martin K., 28, congenital left hemiplegia with epilepsy. Attacks six to nine a month ; warning of dizziness. Attacks not limited to or commencing unilateally ; weak-minded.

Case XII.—Wm. H., aet. 26, hemiplegia of right side following blow on left side of head. Arm and leg much contracted. Equino varus ; seizures two or three a month; warning dizziness ; attacks general ; speech slow, scarcely intelligible ; partial dementia.

Case XIII.—Patrick C., aet. 49, family history negative. Patient gives history of syphilis and alcoholism, no cardiac lesion. Ten years ago had attack of vertigo and nausea; unconscious for two months, and gradual loss of power on left side. One year later had first epileptic seizure, which now occur about every three months ; no warning, never bites the tongue. Attacks general, heretofore commencing on the paralyzed side.

Case XIV.—Frank R., aet. 22, perfectly well up to nine years of age, when he fell, breaking bridge of nose. Left leg dragged slightly in walking ; both patellar reflexes somewhat exaggerated. Patient says that the attacks begin by twitching in the left leg which passes to the right leg, and then he loses consciousness. At outset cries out, "No ! no !" as if in fear. This can be checked. About one year ago, while attacks were very frequent for ten weeks had spasmodic attacks in one arm (?) in which hammer-like strokes were kept up by the hour. The patient semiconscious and moaning as from fear. Attacks of late under large doses of bromide much reduced ; patient is unable to read well, is slow of speech, feeble minded, but possesses considerable talent in drawing.

Case XV.—John H., aet. 32. Hemiplegia following scarlet fever at age of three. No history of otitis ; hearing good ; spastic condition excessive ; marked tremor on voluntary action ; athetoid movements of fingers ; equino varus. First fit at fourteen years of age; generally preceded by dizziness. Patient fairly intelligent.

Case XVI.—Henry Z., aet. 30. Congenital right hemiplegia ; marked flexion elbow and wrist ; gait markedly spastic; reflexes exaggerated. Attacks begin on right side and always more severe on that side ; no warning ; imbecility ; athetoid movements of fingers of right hand.

CASE XVII.—John H., aet. 19. Right spastic hemiplegia from a fall at two years of age. Spastic condition marked ; constant tremor of right eyelid ; patellar reflex not exaggerated. Attacks usually at 5 A.M.; no warning and generally occurring about once a week. Patient unable to read or write.

CASE XVIII.—James B., aet. 48. Left hemiplegia at one year of age, with convulsions. Left leg smaller and shorter than right ; left hand tightly flexed until age of fifteen years. Can now open it ; arm well developed, showing but slight shortening. Excessive tremor or spasm of left side of face on talking. Patellar reflexes greatly exaggerated on both sides ; gait that of spastic paraplegia ; no epileptic seizures since childhood ; intelligence fair ; can read and write.

CASE XIX.—John V., aet. 35. Patient has had epileptic seizures since two years of age. Attack commences with feeling of numbness on left side. On examination slight paresis, with exaggerated patellar reflex on that side. Patient able to read and write a little.

CASE XX.—Mary B., aet. 47. Specific history, left hemiplegia three years ago ; reflexes exaggerated ; has had two seizures since in hospital ; denies any previous attacks; no warning, feels suddenly both sides equally affected. Paralysis more marked for several days following attack. Intelligence impaired.

CASE XXI.—Mary T., aet. 29. Right hemiplegia with convulsions following small pox ; intention tremor ; reflexes exaggerated ; seizures very frequent, beginning with choking sensation ; uncertain whether they begin on paralyzed side ; patient imbecile.

CASE XXII.—Maria M., aet. 32. Right hemiplegia with convulsions, between two and three years of age. Walk markedly spastic; attacks about once a month, generally no warning. Imbecile.

CASE XXIII.—Ida C., aet. 32. Left spastic hemiplegia with convulsions at six years of age. Attacks about once a month; petit mal daily; feeling of dizzines precedes attack. Patient unable to read intelligently.

CASE XXIV.—Ada C., aet. 34, left spastic hemiplegia, with epilepsy since childhood; attacks about once a month at time of menstruation, warning in nature of peculiar feeling; does not commence unilaterally; feeble minded.

CASE XXV.—Mary C., aet. 19, congenital right hemiplegia; arm flexed at elbow and fingers in hand; impossible to overcome contracture by force; leg flexed at knee and foot in position of equino varus; patellar reflex exaggerated on both sides, especially on right side; right side much shorter than left and muscles atrophied; seizures mild but very frequent and always commencing on paralyzed side; speech scarcely intelligible; dementia.

CASE XXVI.—Louise W., 32, congenital left hemiplegia; contractures very marked; muscles atrophic and considerable shortening of upper and lower extremities; head unsymmetrical. Attacks first came on at two years of age; warning, as of sensation of choking; commences usually on left side. No attacks for past three years; speech intelligible, but patient very stupid, unable to read or count.

CASE XXVII.—Michael S., aet. 31, attacks followed measles at age of two; did not return until twenty-seven years of age; patient says, was never paralyzed, but on examination find paresis of right side, flatness of right side of face; reflexes exaggerated; patient says that he has always used left hand more than right. Although he went to school until fourteen years of age, can scarcely read; patient ascribes this to defective eyesight. Attacks of late becoming more frequent, about one a month; no warning and commence bilaterally.

CASE XXVIII.—A.C., aet. 2½, (the mother, a primipara at 42 years of age, pregnancy not being suspected, had by advice of physicians, bandaged herself rather tightly and taken rather violent exercise to remove the supposed flatulence;

delivery however, natural and easy. The child made no attempt to walk or talk till two years of age). About one year ago had epileptic seizure, commencing on right side and becoming general ; remained more marked on that side. Has had two similar attacks since. On examination, child large and well-developed ; gait spastic, pushing one foot before the other and walking on the ball of the foot ; reflexes exaggerated, especially on the right side ; child able to say a few words and beginning to show desire for certain things ; expression of face, idiotic.

DISCUSSION ON DR. FISHER'S PAPER.

Dr. KNAPP. suggested the existence of trophic cell centres in the cortex. He had seen cases of infantile hemiplegia in which the atrophy was as great as that in many cases of spinal paralysis.

Dr. G. E. WALTON described a local epileptic attack. The patient had been healthy to within a few weeks when the attacks had commenced. The convulsion involved the left side of the face ; there was slight dilatation of the pupils, and the head was turned over the left shoulder. The hands were not affected, and there was no loss of consciousness, but the patient talked thick after the attack. This seemed to be due to difficult pronation rather than to aphasia. The speaker referred to the question of operation. In monkeys the centre for turning the head was located in the first and second frontal convolution near the ascending frontal. The centre for the face and mouth would be lower down.

Dr. FISHER did not find the wasting infantile hemiplegia so marked as that in Dr. Knapp's cases. There was always some wasting, but he thought not more than would be explained by disuse. Another proof that the epilepsy of this condition was not of the Jacksonian type and dependent upon local lesion was the fact that there was no aura or only an epigastric aura in these cases. According to Luciani epileptic seizures had but little value for localization compared with paralysis.

Afternoon Session.

Dr. Robert T. Edes, of Washington, D. C., read the following paper, entitled

THE RELATION OF RENAL DISEASES TO DISEASES OF THE NERVOUS SYSTEM.

The connection between renal and nervous diseases is twofold. First, the influence of renal disease in the production of diseases on the part of the nervous system ; and, second, the influence of nervous diseases in producing renal symptoms. It is only the first of these to which I shall do more than allude.

A great deal of observation and experiment, to say nothing of much theorizing, has been lavished on the first part of the question, to determine the precise nature of the relationship; the fact of such a close relationship being one of the earliest observed and most important facts in the pathology of Bright's disease.

This connection is usually expressed by the word "uremia," and as it is my belief that a considerable confusion not only of words, but of ideas, has arisen from the general and undefined use of this term, and that it is easier to get rid of the errors connected with it by simply dropping the whole thing together, than by endeavoring to establish a correct definition, I shall use "uremic" only in the old sense to denote phenomena connected with the nervous system occurring in the course of renal disease, and not as carrying with it any theory whatsoever ; and adopt for purposes of classification certain others which do express, as I consider, more accurately the true pathology.

The word "uro-toxic" speaks for itself, and means—pertaining to poisoning by the retention of substances contained in the urine. Another class of symptoms might be called "uro-septic," but with these we have less to do than the surgeons, to whom it is of great interest in connection with lesions and operations in the lower urinary passages.

For the symptoms dependent upon affections of the vessels, which are recognized as playing so important a

SYMPTOMS.	*Neurotic.*	*Angio-neurotic.*	*Angio-notheutic.*	*Anæmic and Hydræmic.*	*Urotoxic.*	*Uroseptic.*
Neuralgia, "rheumatism," itching	◇					
"Dead fingers."		◇				
Symmetrical gangrene.		<	>			
Skin diseases		<		?		
High tension, hypertrophied heart		◇ <	>			
Headache, alone		◇				
Headache, with eclampsia		<			>	
Headache, preceding paralysis			◇			
Vomiting	◇				◇	
Dropsy		<		>		
Dyspnœa		<		>		
Cheyne-Stokes respiration		<			>	
Delirium			?		?	
Insanity			<	>		
Muscular twitchings					◇	
Convulsions		<			>	
Insomnia					◇	
Coma		<	◇ Cerebral hemorr.	>	◇	
Hemiplegia (usually)			◇	>		
Paralysis (localized)		<			◇	
Hiccough						
Permanent dimness of vision			◇		>	
Temporary loss of vision		<				
Polyuria		◇				
Pollakiuria	◇					
Fever						◇

The closed parallelogram opposite any symptom and under any heading shows that the symptom may be wholly due to the cause signified. Half of the parallelogram shows that the cause noted at the head of the column is one factor.

part in the pathology of renal disease, the word "angiopathic" may be used, with the subdivisions "angio-neurotic" already in use, and "angio-notheutic," a word which I owe to the kindness and learning of my friend Dr. Fletcher, and which refers to symptoms or lesions dependent on organic disease or degeneration of the vessels (*αγγέιοϰ*, a vessel, *υορευδις*, degeneration).

We find as symptoms accompanying acute and chronic diffuse nephritis in their various forms often enough to be legitimately recognized as having an intimate relationship therewith, and not being present merely as coincidences; convulsions, coma, delirium sometimes becoming insanity, headache, dyspnœa, failure of vision and hearing, paralyses, neuralgia, itching, sometimes cutaneous eruptions, "dead fingers," rarely symmetrical gangrene. These with vomiting, which is sometimes at least a nervous symptom, make the group known as uremic.

It is the object of this paper to insist upon the fact that they are not all urotoxic and to suggest the nature of the pathogenic connection.

The most obvious assumption, that which holds its own to the present day in some form and which does actually cover a part of the ground is, as the name suggests, that uremic symptoms are due to a retention in the blood of some excrementitious substance, whether water, urea, or some product of its decomposition, extractive, potash salts, or the totality of the urinary solids. Perhaps we may add to this list an alternative which seems to me to have a considerable degree of probability in its favor, but as yet little evidence, that new toxic agents perhaps in the nature of a ptomaines may be present in some cases. This might account for the difference between the great toleration of anuria from the arrest of the healthy secretion by mechanical means, and the rapid supervention of symptoms from a much less serious retention in chronic cases. This, however, is for the chemistry of the future to decide.

Such an accumulation of urinary solids is assumed to take place when the secretion becomes deficient in quantity, and it has been demonstrated by actual chemical analysis in a certain number of cases, though as a matter of fact in much fewer than might be supposed from the frequency of the allusions, the water and the urea as being the larger part of the constituents are naturally those which have been most frequently examined. Data in regard to the others are too scanty to have great value

It is not at all certain that urinary constituents are accumulating in all cases in which renal disease, even advanced, exists, and there can hardly be a better instance of the inconsistencies of medical reasoning than the indifference with which the removal of one kidney is spoken of in trust that the other will carry on the work of excretion thoroughly, and the promptness with which any symptoms that may arise in any stage of Bright's disease are attributed to a so-called uremia. A sort of reasoning in a circle takes place by which the pathological theory of uremia depending on an accumulation of urea is proved by the coincidence of these symptoms with known renal disease, and on the other hand, the diagnosis of renal disease is confirmed by the occurrence of symptoms supposed to depend upon the presence of urea in the blood.

A deficient secretion of urea or a secretion considerably less than that stated as a physiological average may undoubtedly take place in nephritis, even in the earlier stages; but it must be remembered that the thirty grammes per diem, which are often taken as the standard and which, as I have elsewhere[1] tried to show, are much too large an estimate for a considerable number of healthy persons, are not applicable to all persons, and certainly not to those whose digestion is imperfect, whose blood is deficient in red corpuscles, and who are undoubtedly making a much less than the proper amount of nitrogenous excreta. The quantity of urea varies from day to day both in health and disease, and a single observation cannot be properly regarded as conclusive of a diminished secretion. Unfortunately quantitative observations extending over several days are not so numerous as is desirable. Twenty grammes is probably a better average for many persons, and low feeding and old age as well as many diseases which have nothing to do with the kidneys, may bring it much below.

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[1] Read before Association of Physicians, 1888.

The most convenient question with which to approach this inquiry is not: What are the nervous symptoms of Bright's disease? for this may refer either to the early stages, when the exact amount of secreting tissue thrown out of action is not known, but which may, in a great many cases, be fairly assumed to be much less than that removed by the ablation of one healthy kidney out of the pair, a loss which we know is perfectly well borne; or it may, and more frequently does, refer to the culmination of a long series of changes involving not only the kidneys, but heart, arteries, stomach, and blood, and perhaps organic changes in the nervous system itself.

A much simpler one, and one there is an abundance of clinical material to answer, is this, What are the symptoms of a total loss of function of both kidneys?

This is easy to answer. Cases of suppression of urine occurring in persons free from chronic diffuse nephritis, or where such a nephritis as bearing on only one kidney, has been unconnected with constitutional disease, have been recorded in considerable number, and in fact they present to us a series of pictures of as much uniformity as can be expected in clinical observation. It is interesting to notice, by the way, how strong is the influence of preconceived ideas, in the frequency with which authors introduce into their comments and into their titles remarks on the absence of the "usual" symptoms of uremia.

Dr. Roberts says: "When even the suppression is absolute, seven or eight days elapse before the special symptoms of uremic poisoning make their appearance; but when these do appear the end approaches rapidly, and death is not delayed beyond two or three days. Up to the rise of the proper uremic symptoms the condition of the patient is as a rule wonderfully calm and free from distress; the functions generally proceed tranquilly and the intelligence is undisturbed. The most distinctive and invariable of the special uremic signs are muscular twitchings. I believe that these are never wanting. Contraction of the pupils is also a constant sign, but later in development than the muscular twitchings."

An examination of the cases collected by Dr. Fowler[1] and a number of later ones, although confirming the general accuracy of this description would lead to somewhat different conclusions in details.

The muscular twitchings, although occasionally mentioned, are not invariably so. They might, however, more easily escape observation than some other symptoms. The pupils are in some cases distinctly mentioned as dilated. Somnolence with restlessness, and, on the other hand, insomnia are very common. Convulsions are occasionally noted but they are by no means so common as one might expect from a comparison of these cases to Bright's disease, or in fact as the authors themselves seem to have expected. In the fatal cases death has come in two quite different ways. In one set the patient dies very quietly in the full possession of his faculties, and often a few hours or minutes after having been up, or engaged in conversation, *i. e.*, without any of the "usual" uremic symptoms. In the other, the end comes more in the usual way after an interval of the more classical sopor and coma.

Among the symptoms which have a special interest for our present inquiry, headache is sometimes, but not invariably, noted, not often spoken of as severe, and sometimes a sense of pressure in the head. Dyspnœa is noted in a certain but not large number.

We may describe these cases in general by saying that the poison of the urine is a slow one which produces but little disturbance for a number of days, *i.e.*, in small dose; and that a patient may recover after days of anuria, having hardly had an inconvenient symptom, or at a later period after those which are distinctly urotoxic.

When the blood has been thoroughly impregnated with the poison an effect upon the nervous centres is perceptible, which may go on to what is generally considered uremia, but which may produce death by an action on the heart (and muscles of respiration ?) before this condition has become at all prominent.

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[1] Suppression of Urine, New York, 1881.

We may reckon as purely urotoxic symptoms: insomnia, restlessness, somnolence, mild delirium, sopor, and coma; muscular twitching, muscular weakness, rarely convulsions, sudden paralysis (of heart and respiratory muscles ?); sometimes headache, sometimes dyspnœa, sometimes hiccough, frequently vomiting.

It is not necessary to our present inquiry to determine what is the special agent in the urine the accumulation which gives rise to these symptoms. Many theories, which I will not take up your time in detailing, have been framed and put to the test of experiment and clinical observation. The objections to assigning this place to urea were seen at an early day, and led to the making of the well-known ammonia theory of Frerichs with its supplements and modifications. Recent experiments have shown that large amounts of urea injected into the blood of animals deprived of the power of re-eliminating, produces symptoms comparable to uremia, and a proportion of urea somewhat approaching that which remains in the blood of these animals has been found in a few cases of uremia in man. It is possible that urea is the chief poison in these cases of suppression, but it is probable that it is at least assisted by the other normal constituents.

More recent observations attach importance to other constituents like extractive, and potash salts, but the most hopeful line of investigation at present lies in the investigation of self-formed poisons of greater subtlety and power.[1]

How far are these facts applicable to the symptomatology of chronic Bright's disease in any of its forms?

We meet with these phenomena, complicated with extreme anemia and debility, with œdema, not only of the subcutaneous tissue but of internal organs and with cardiac weakness, in the later course and at the end of chronic nephritis, and then there is no reason to find fault with their usual explanation as depending on a failure of the kidneys to do their work. They are then not only uremic in the ordinary sense, but truly urotoxic, and we see saturation of an enfeebled organism by a slowly administered poison, of which the last few, perhaps larger, doses bring the symptoms rapidly on.

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[1] At the time this paper was read the author had not seen the suggestive but not quite conclusive work of Ch. Bouchard, "Sur les Auto-intoxications," Paris, 1887. This author assigns to urea little or no part in the total toxicity of the urine, much more to the potash salts, but concludes that there are several poisons in the urine not yet to be chemically defined. They are probably not alkaloids.

There are cases, however, where these and other nervous symptoms are met with in the earlier periods of nephritis of either form before the renal disorganization has reached an extreme grade. In some the progress is undoubtedly toward atrophy with resulting uremia; in others the renal symptoms, though undoubtedly present and perhaps giving the name to the disease, are subordinated to the vascular and nervous ones.

So great may be the disproportion that Mahomed, whose early death was so great a loss to this department of pathology, used to speak of cases of " Bright's disease without nephritis," an error in nomenclature as it seems to me, since Bright's disease ought to be what Bright wrote about, which was undoubtedly the disease of the kidney, but yet indicating a view to which sufficient attention is not always paid.

In these the word "uremic" loses its usual or etymological signification, and is no longer synonymous with urotoxic. It is more by analogy than by actual demonstration that these symptoms are supposed to depend upon the same accumulation as takes place in the suppression we have been considering.

It is found in some of these that there is at the time of the outbreak a diminution in the amount of urine and of urea, but in many others more or less decided symptoms occur while the usual flow is going on or, at most, only some hours after it has become diminished.

This outbreak of symptoms very soon after the flow of urine has become diminished as regards the water, but where an increased specific gravity indicates that a considerable quantity of solid matter is still being carried off, is called uremic; and yet if the kidneys, which are still doing some work, were removed entirely, as happened in the case of Dr. Polk (N. Y. Med. Journal, Feb. 17, 1883), or were entirely thrown out of action by any of the numerous accidents which may happen to the lower urinary passages, it would require eight or ten days for sufficient of the poison to accumulate to produce the symptoms.

It may be said that in such cases there has been, notwithstanding an abundant secretion of water, a retention of the solid elements of the secretion until they have accumulated to just on the brink of a poisonous dose which the temporary anuria causes them to exceed. Such a kind of retention seems possible when we consider the separate functions of the Malpighian bodies and the epithelium of the tubes, and, as regards any one constituent, it is difficult to disprove; but if we consider the water, the urea, and the total solids, we find that they may be excreted up to or even during the day of such an attack in a quantity which is not, to be sure, equal to the physiological average, but is not below what may be a fair quantity for persons with a much diminished tissue metabolism.

It may be remarked, as bearing on the probability of this form of retention usually taking place, that most of the substances found in the urine of which we have definite knowledge are diffusible, some of them highly so, and that in many cases of destruction of the kidneys by any of the various lesions of the lower urinary passages it is often remarked how small a quantity of renal tissue suffices to produce a large amount of water, and that in these cases uremic symptoms are deferred long after this destruction has been going on.

It is to be noticed also that the form of Bright's disease in which we hear most about its latency, where an outbreak of uremia occurs in the midst of apparent health, is not that in which the epithelium is the first to undergo degeneration, but the interstitial, where the vascular element is the predominant one.

In looking over a somewhat voluminous literature to find how close a correspondence exists between a diminished diuresis and an attack of well-marked uremia, it is quite clear that in a large majority of cases either a gradual diminution in the amount of concentrated urine passed, or a somewhat slighter diminution with a low specific gravity, takes place when uremic symptoms are about to occur, and that the amount of water and solids not infrequently increases coincidently with the remission of the symptoms; but there are cases enough of an exceptional character to show that while we must regard the ordinary theory of the pathogénesis of the severer and more general symptoms of uremia, and, in particular, convulsions and coma without hemiplegia, as the correct one, yet there are cases to which it does not apply, and where we must seek, if not for an entirely different explanation, at least for another factor of great or controlling influence. In these cases are found chiefly headache, dyspnœa with Cheyne-Stokes respiration, coma with hemiplegia and unilateral convulsions, but also coma without hemiplegia, and general convulsions.

Bouvat (These de Lyon, 1883,) reports the case of a man of twenty-two with an acute nephritis, probably of scarlatinal origin, who had headache and eclampsia. The urine contained no albumen, but numerous granulo-fatty casts and much urate of sodium. During the time at which the attacks of eclampsia were taking place, there was no mention of the quantity of urine, but it was examined on every day and was not said to be scanty; two days after the last eclamptic attack, and while the patient was still somnolent, there were seventeen grammes of urea in two litres of water passed in the twenty-four hours. While the patient was still eclamptic there were found in the *blood* seventeen and nineteen centigrammes of urea per litre, an amount which the author thinks sufficient to prove the uremic character of the attacks, but which is so little in excess of the normal amount (ten centigrammes to the litre) and so insignificant in comparison with the amount which it is necessary to inject in order to produce decided symptoms in animals,[1] and which was found in some patients by Grehant and Quniquaud,[2] that the case seems to prove the contrary theory. It is interesting to note that delirium lasted for a week after the free discharge recorded had been going on.

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[1] Six per cent. found in blood.

[2] Two to four per cent. found in blood. Comptes Rendus de l'Academie des Sciences, 1884, p. 383.

Eiselt (Aertz. Ber. der K. K. Krankenhaus. Prag., 1884,) reports the case of a man who had well-marked nephritis and at one time maniacal attacks, supposed to be uremic, with other more usual symptoms. At a later period, after passing 1,200, 1,500 grammes of urine of sp. gr. 1,008 on two successive days, he had an epileptiform attack on the day on which the quantity rose to 2,840. On the next day it was 2,640, and he felt very well. On three days, at a subsequent period when passing 1,300 to 2,600, with sp. gr. of 1,006 to 1,016, he had vomiting and headache. Later the urine diminished in quantity, and he had eclampsia and somnolence under the usual circumstances, and the autopsy showed granular kidneys. Thomayer in commenting on this case speaks of other cases reported by B. Stricker and by Budde, in which uremic symptoms occurred with a daily amount of urine from one to two litres. The original reports of these I could not find.

The frequent occurrence of headaches, which are often so characteristic of interstitial nephritis for years before renal atrophy has advanced far enough to interfere with the secretion of a sufficient amount of urea, is well known.

The following cases show some of the relations of headache to urinary excretion.

C. H. W., æt. 40, commercial traveller. Headaches for a long time, growing worse and more frequent lately. Urine copious, with albumen and casts. Albuminuric retinitis, high arterial tension, severe headache and vomiting, relieved by morphia. Then left hemiplegia with relief of headache. From time to time until death, delirium, constant polyuria.

Solids in the urine as calculated from the total quantity and the specific gr., (which of course only gives a rough approximation),Sept. 21—87 grammes; Oct. 2—77; Oct. 13—51; Oct. 17—18; Nov. 1—100. At the time of the marked diminution of the middle of October it was noted that on October 10th he had morphia with relief of headache and was more intelligent. The scantiness of urine lasted for about a week, during which he had no more severe headaches, but was slightly delirious, and the next mention of headache and restlessness occurs on October 30th, several days after the urine had again become abundant, and the day before one on which the calculation gives 100 grammes of solids. Does not this sequence seem much more like a fall and rise of tension than an accumulation of urinary solids?

The tracing of October 19th, during oliguria, but after the headache was relieved, shows a pulse of less than his usual tension, though no tracing was taken during the subsequent period of polyuria.

The patient died on December 13th, and the autopsy showed hypertrophy of the left ventricle with tracts of thickening here and there in the aorta. The kidneys were firm, granular, cystic and atrophied. The brain was unusually firm on section, with several small cyst-like cavities in the white and gray substance and spots of reddish-brown in various parts of the cortex and corpora striata. The arteries at the base had yellowish patches.

The diagnosis between Bright's with headache on the one hand, and cerebral disease with polyuria, no uncommon combination, on the other, is not always easy. Albumen and casts may be present in either. Localization of the headache, localization of any paralytic or spastic symptoms that may be present, study of the eye ground and of the heart and more than a single careful urinary examination may for a considerable time be all that can be depended on to make the distinction.

Headaches and polyuria are among the angio-neurotic phenomena which may belong to more than one disease, and it may be that they are not only not uremic, but, even when albumen and casts are present, not intimately connected with the local renal disease.

While recognizing the presence and importance of the uro-toxic origin of headache, I cannot believe this to be its usual one, considering the absence or rarity of headache in complete suppression of the urine, where it ought to be present and to increase in intensity pari passu with the duration; and also considering its coming and going in ordinary cases in a way and under therapeutic influences more consistent with a neurosis than with any form of poisoning. If it be said that the relief which is afforded in some cases to the headaches of renal disease by the hot air bath is due to the elimination of the accumulated urinary products it may be answered that the bath relaxes the arterioles as well as carries off urea, and that the relief is often of too long duration to be explained simply by the removal of a certain excess of poison which must constantly be in process of renewal.

P. W., æt. 43, syphilitic ? headaches for two years. Queer feeling in head, left arm and leg. Convulsion and delirium. Urine; low sp. gr., albumen and casts, no retinitis. Fits repeated several times. Urine improved in character. Headache. Physical signs of consolidation and catarrh at apex of right lung. Gain in weight. Deep sleep, delirium and death.

Scalp thick, calvaria dense, thickening of dura, thickening of arteries at base, two tumors beneath the posterior part of the first temporal convolution on the right side. Left kidney small, but normal in structure to the naked eye. Right normal in size with slight depressions and adhesion of the capsule (chronic interstitial nephritis in incipient stages?) cavity in apex of right lung with cheesy contents (quiescent).

Miss J. P., very severe sick headaches with vomiting. Intervals of freedom. Albumen and casts in abundant urine, during intervals as well as during attacks. Mind clear till last day of life.

D. A., æt. 34. Headache, albuminuric retinitis, renal hemorrhage, albumen; granular, fatty, and blood casts; abundant urine, slight hypertrophy of heart; pain in back, copious renal hemorrhage soon followed by very severe headache, coma, and death. Probable cerebral hemorrhage.

Epilepsy may exist side by side with chronic nephritis, and a short observation would lead to either one of two opinions, either of them erroneous, that the epilepsy produced sufficient congestion of the kidneys to cause casts and albumen, or that the fit was of a uremic character.

Thos. Fl., hospital repeater, æt. 40, epileptic as a child but not of late years until the present attack began, when he used to have one or two fits after drinking. He generally remained in the hospital a few days and was discharged "relieved" or "well." The urine always contained albumen casts, which became worse in character, and he died with chronic meningitis, hypertrophied heart, and interstitial nephritis.

The mental disturbances occurring in connection with renal disease have been discussed at some length, and spoken of as if some special relation existed between the two conditions. An attempt has even been made to give such a connection a medico-legal importance.

If we go back to our typical cases of retention, where we have the purest type of urinary poisoning, we find that many of them are distinguished by almost absolute mental clearness up to a very late period, and that in others a very mild delirium toward the last, and especially at night, is the utmost that can be observed in this direction.

It may be said with much confidence that insanity and delirium are not early, or distinctive, or common urotoxic symptoms.

At the termination of chronic Bright's disease, delirium is more common and more marked; but here we are dealing not only with retained secretion but with anemia, malnutrition, cardiac weakness, and other conditions which produce delirium in many other diseases.

An association with the peculiar nervous restlessness of interstitial nephritis is quite common.

In examining those cases, of which there is no inconsiderable number, where insanity in various forms, mania with excitement, lypemania with fixed delusions are present together with renal disease, it may be remarked, in the first place, that some of them do not bear a rigid scrutiny as cases of nephritis. In those where both affections are undoubtedly present, it is true that in the later stages of the renal disease the urinary poisoning is one factor, but in others the insanity is too early a symptom to be attributed to urine poisoning. In one case given by Dieulafoy (Gaz. Hebd., 1845), where a man had attacks of hysterical violence, terrific hallucinations, lypemania, delirium of persecution, the urine was scanty but contained 39.9 per litre of urea. The patient recovered so far as his mental symptoms were concerned, and died two years later in coma without delirium.

A case reported by Raymond (Arch. Gen. de Med., 1882,) is of much interest as showing the relations of delirium to the excretion of urea.

A woman, æt. 66, had severe dyspnœa, with Cheyne-Stokes respiration, and entered the hospital on July 20th. On this day she passed 1,450 gr. of urine with 18.7 of urea in the twenty-four hours.

She continued to pass from 12 to 18 grammes every day until the 27th, when she began to be delirious.

There was then a period of three days in which no urine was passed (or none reported), and after this a much diminished secretion. It was not until nearly a month after the first diminution of urine that the patient became comatose.

As the patient was on an exclusive milk diet it is hardly to be supposed that with this amount of excretion there could have been an accumulation of urea at the time when the delirium began, though afterwards the increasing somnolence was probably due to gradual accumulation of the urinary poison.

The autopsy confirmed the diagnosis of chronic nephritis.

The connection is that of insanity with any severe depressing disease with a brain which may be disturbed by any one of several causes.

It may be perfectly proper to speak of the insanity of Bright's disease, or of "Folie Brightique," as a concise method of indicating the exciting cause, but not as denoting a special form of insanity.

Much interest has been recently awakened in a class, or two classes, of cases highly important in themselves and calculated to throw light on the pathology of so-called uremia. I refer to those where symptoms that would naturally lead to a diagnosis of a localized cerebral lesion, such as hemorrhage or embolism, are shown, either by the rapidity with which they disappear or by an autopsy, not to be so caused; or where the suddenness and severity of an apoplectiform attack, especially but not exclusively in old people, lead to a similar diagnosis with a similar result. These are the "serous apoplexies" of a former generation and the uremia of latent Bright's disease of the present.

They are not, however, necessarily uremic even in the widest sense of the word, although it is true that a great many of them are. Still less are they urotoxic. From the character of the lesion they may occur in any disease with a feeble circulation and that condition of the vessels and blood which permits an easy and rapid escape of serum into the surrounding tissues; and as these conditions are frequently met with in advanced Bright's disease, it is not strange that the combination should be a common one.

In many of the cases reported the urine at the time of the accident has been found to be scanty, but it is by no means invariably the case that there is any evidence of a long accumulation, nor is the urine always *extremely* scanty, and as most of these cases occur in old persons where the normal amount of urea is considerably reduced (say to eight or ten grammes per diem), it is not at all certain that the full amount formed may not be secreted.

The great frequency of cerebral hemorrhage in interstitial nephritis, referrible to disease of the arterioles and to high vascular tension, is well known. A certain proportion of unilateral symptoms, and some general ones like coma, and perhaps rarely convulsions, are thus caused; and the headaches which precede them, as in a case already reported, are probably connected with the organic changes in the vessels, or with their condition of fullness rather than with any assumable excess of urea. To these cases, however, having an actual and well-defined lesion, it is very properly not usual to apply the word uremic even if they do accompany renal disease. It is understood that it is the vessel and not the blood which is at fault.

It is certainly repugnant to our notions that a poison gradually diffusing itself throughout the system should be supposed to affect only one side of the brain, and where we fail to find hemorrhage or closure of the vessels we naturally look for some other factor to determine the localization. In most of these cases a state of things is found to exist in the brain which has before been invoked to explain the general symptoms of uremia when the chemical theories have been deemed insufficient ; but having been found not to exist in all cases, and thus being inadequate for a general theory, has been overlooked or regarded as of little importance. It is not so obvious as hemorrhage or softening, and may easily escape attention, especially when the tendency is to regard it as a common condition and not distinctive of anything in particular. This is œdema of the cerebral tissue with or without an increase of fluid in the ventricles. Such a condition may give rise either to no symptom at all or to the most serious ones, according to its degree, and the symptoms, when present, may be either generalized or local according as the lesion involves a large part of the cerebral centres or a limited area in the motor region.

One obvious objection to accepting this lesion as an efficient one, beside the fact that it is often present without symptoms, is that in the great majority of cases where hemiplegia has been present there has been no discernible difference in the two sides of the brain.

A slight difference in the degree of œdema between the two sides, or even more narrowly limited, would be very difficult to demonstrate, but might yet be sufficient to make the distinction between the paralysis of one side and the freedom of the other. It is certainly much easier to imagine this than a restriction of the action of a poison circulating in the blood to one side of the brain. Œdemas limited to a small area have been noted in other parts of the body. In a case of Dewevre (Lyon Med., 1886, p. 133), a patient who had "uremic" hemiplegia, had a few days previous a transitory, circumscribed œdema on the back of one hand.

In a few cases, however, a difference in the degree of œdema on the two sides has been noted, or a limited area of well-marked anemia, or a difference of dilatation of the lateral ventricles. What should give rise to a greater degree of œdema upon one side than the other, it is impossible to say with certainty; but one who has watched the rapidity with which, especially in debilitated, emaciated, and flabby patients, the fluids of anasarca will seek the dependent parts, will have no difficulty in admitting the possibility that even so slight a circumstance as a person sleeping upon one side rather than the other might be enough to make the difference.

To illustrate the difference in symptoms which may come from a moderate inequality of pressure on one side when both sides are affected by the same lesion, I may mention the case of a man picked up unconscious, where coma and a well-marked hemiplegia led to a diagnosis of hemorrhage or softening, but where an autopsy showed that an acute meningitis of the convexity was the real lesion, the prominence of the paralytic symptoms on one side being accounted for by the greater thickness of the purulent layer upon the opposite surface of the cerebrum.

So it is not taking a long step in the dark to infer that the symptoms which, according to all physiological laws, ought to, and in the great majority of cases do, point to a focal lesion of some sort, do so in these cases as well, and that, as no other lesion is found, œdema is sufficient.

If we wish to ascertain, by an examination of the excretion, whether there is a more or less remote connection between urinary retention and the appearance of the symptoms, that is, whether, even if we cannot look upon them as directly urotoxic, they may not be indirectly so, we have by no means so many facts to guide us as might appear from the literature of the subject, already becoming voluminous in comparison with the length of time which has elapsed since it was considered that the presence of a distinct paralysis settled the diagnosis as against uremia. Most of the reports of these cases content themselves with a statement as to the presence or absence of albumen or sugar, which of course only implies that there was not absolute anuria. Even when a little more than this is stated, it is curious to see how few physicians remember that to get a product you must have a multiplicand and a multiplier both. "Urea diminished" usually means percentage diminished, a statement of very little value alone.

Neither is the fact that at the moment of a paralytic or convulsive seizure the urine is scanty, any proof of an accumulation. As we have repeatedly had occasion to recall, it requires a week or more to fully develop purely urotoxic symptoms. Any severe shock or nervous affection may check for a time the flow of urine, and a few hours' suppression is much more likely to be the result than the cause of an apoplectic attack. In fact we know that alone it cannot be the cause in so short a time.

In the cases given by Raymond (Revue de Med., 1885), in not one have we the means of judging even approximately of the amount of urinary solids discharged. We can only say that there was not actual suppression, or near enough to it to excite remark. The cases of Chantemesse and Tenneson give no available data except that the urine is not expressly stated to be scanty, and in one case it had the specific gravity of 1017.

Florand and Canniot (Gaz. Med. de Paris, 1886, p. 532) report two cases, in the first of which the urine was dark and scanty; and in the other, where a hemiplegia lasted from the 4th to the 12th of October, with severe headache but no loss of consciousness, the urine was clear, not albuminous, and passed frequently in small quantities. In their remarks they use the expression "normal condition of urine." In this case there was no obvious focal lesion, but œdema of the brain and granular kidneys.

Buckling (Brit. Med. Jour., 1886, II. 1076) reports the case of a woman of 62 with hemiplegia of this kind, which recovered, and who passed an average of forty ounces a day with one per cent. of urea. This would give about twelve grammes of urea, a quantity below the assumed physiological average, but perhaps not below what we have a right to expect from a patient of that sex, age, and condition; nor approaching the condition of anuria, which, we must constantly repeat, takes a week at least to produce decisive results.

It is certainly a most unfounded assumption, in the face of the statements of the diminution of urea in old age, to suppose that all nervous symptoms in old persons not accounted for by some other obvious lesion are due to the senile kidney and consequent uremia. As is elsewhere remarked, there is no reason to suppose that the atrophy of the kidney is out of proportion to the diminished vital metamorphosis.

Hemiplegias without hemorrhage or softening are not confined to renal cases. An elderly colored woman, who had had for months swelling of the face, legs, and abdomen, with headache, dyspnœa, and palpitation, entered the Boston City Hospital with general anasarca of a passive kind affecting the hands and the dependent portions of the body. There was nothing decisive about the heart. The urine contained considerable albumen and casts, not of blood or waxy. The urea was noted as forty-one grammes per litre. A few days after she had a sudden hemiplegia without loss of consciousness.

The autopsy, made by an exceedingly careful pathologist, disclosed no plugging of the arteries or hemorrhage in the brain.

There was some dilatation of the heart, though the muscular substance was firm. The kidneys, though slightly reduced in size, showed no signs of nephritis, the diagnosis of the pathologist being "chronic cyanotic induration."

In some other cases reported it is very possible that the renal element may not be the controlling one, but rather a condition of cardiac failure and more or less localized vasomotor paralysis.

Level (These de Paris, 1888), who rejects the œdema theory, reports cases of this kind where nothing was found in the brain—one of these which seems to be of the kind where the exception proves the rule. A woman, æt 70, was brought into the hospital with complete resolution of the four limbs, conjugate deviation of the eyes to the right, and deviation of the labial commissure in the same direction. There was absolute insensibility of the left side.

Two days afterwards the paralysis had disappeared except the deviation of the eyes, but the coma continued, and she soon died. There was atrophy of the kidneys depending on cancer of the uterus, and the consequent stoppage of the ureters. The heart was hypertrophied, but the brain was absolutely healthy, with very little atheroma of the arteries at the base. Such a case as this is open to any theory, but œdema, which may be local and transitory, can certainly be made applicable quite as easily as uremia which certainly is never local.

Certain experiments of Raymond, which show that a unilateral lesion, the obvious effects of which have disappeared, is sufficient to give a unilateral form to accidents due to urinary poisoning at a subsequent period are very interesting.

This hypothesis of a lesion which does not destroy the nervous elements, but is in fact compatible with a complete and rapid restoration of their function, or which may on the other hand so completely throw a large portion of the brain out of action as to cause rapid death, is not a new one in pathology. Besides its having always held a sort of reserve position in the present connection we are familiar enough with something like it in the so-called "congestive attacks" of coma or hemiplegia or excitement in the course of general paresis, or in the partial and often more or less transitory paralyses of cerebral syphilis attributable to a spasmodic action of arteries already narrowed by a specific endarteritis.

Œdema is well known to depend, it not always, at least often, upon a certain vaso-motor condition, a condition which may be present in the brain as well as in the subcutaneous cellular tissue, and it seems probable that we have in many of these nervous symptoms connected with renal disease the results of various vascular changes, ranging from spasm to paralysis acting as exciting causes upon a substratum of anemia, hydræmia, and cardiac debility, and perhaps often of contamination of the blood by retained urinary elements.

There are many other nervous symptoms which there is not time to treat in detail, especially as my object has been not so much to describe them all as to comment on the erroneous pathology which groups them altogether as uremic. The "dead fingers" are so obviously a vaso-motor phenomenon that they speak of themselves against so comprehensive a classification. Amaurosis and some at least of the troubles of audition are probably referrible to the same causes as eclampsia. A very important nervous symptom, and one which is certainly at times a urotoxic phenomenon, is dyspnœa. This may arise in the course of Bright's disease from several causes, as œdema of the glottis or of the lungs. The one which concerns us is where none of these conditions is present. It is then very likely to be associated with Cheyne-Stokes respiration. Cuffer (These de Paris, 1878,) refers its causation to anemia and the diminished capacity of the tissues for oxygen.

In many cases of dyspnœa we find that the urine is diminished, but often it occurs when the urine is copious, very early in the disease, or at least early in the known course of the disease.

Uribe (These de Paris,1886), gives a case from Rosenstein, where attacks of dyspnœa are noted at intervals, while the patient is passing an abundance of urine, as 5,000 of 1,008 sp. gr. and afterwards from 2,300 to 2,600 of sp. gr. 1,009 to 1,012. When intense dyspnœa was noted the amount was 500 grammes of sp. gr. 1,018. Still later when there was "extreme and continuous oppression" there was 1,500 of sp. gr. 1,015.

There was a certain amount of actual pulmonary lesion in this case, but the dyspnœa is evidently not attributed entirely to this by the reporter. There was hypertrophy of the heart and granular kidneys.

In a case of Hervier (These de Paris) the urine had always been normal in quantity, the mind was clear, and there was no œdema.

Fifteen days after beginning of the dyspnœa there was a sudden loss of consciousness with complete left hemiplegia. In some other of his cases the urine was diminished in quantity and deficient in urea.

Waldenburg reports the case of a man with uremic asthma, headache and advanced renal disease. He improved under treatment, and did very well until he had a fit of passion, when the headache reappeared with vomiting and diminution of urine and tenderness on pressure in the region of the kidneys. There was relief from dry cups on the loins, but he began again to have dyspnœa, the urine being diminished one half. Digitalis gave relief, but did not much increase the diuresis, but the reporter states that, although the urine was much less than it had been, it was *about equal to that of a man in health.*

In a case of my own the patient's wife informed me that her husband, at a time when he was still going into town to attend to his business, used to hold his breath so long when asleep at night that she was frightened. I supposed this to be Cheyne-Stokes, which he afterwards manifested most distinctly. When he was dying in coma, and his face was encrusted with crystals of urea, the Cheyne-Stokes disappeared and the breathing was of the usual stertorous character. A brother of this patient, with interstitial nephritis, had the same symptom in the most typical form, but in his case it was among the terminal symptoms.

The case of Raymond, already quoted in connection with delirium, shows the coincidence of extreme dyspnœa with an amount of urea excretion fully equal to the formation.

The causation of the various nervous symptoms of renal disease (and the same may be said of many others which are beyond the limits of this discussion), is a complicated interaction of several causes, in one instance one condition and in another, another assuming the predominace.

For the sake of simplicity, we may speak of four principal factors:

1. Degeneration of the blood (anemia, hydremia, and perhaps other less understood conditions).

2. Poisoning of the blood (urea, potash salts, extractives, ptomaine ?).

3. Disordered vaso-motor action (spasm, paralysis).

4. Vascular degeneration (endarteritis, endophlebitis, fatty degeneration, miliary aneurisms).

Some of the earlier symptoms may be of a purely neurotic character.

In some cases mechanical obstruction or sudden paralytic or congestive anuria may bring the blood-poisoning to the front at once and without complication, but, with this exception, we are seldom in presence of so simple a state of things.

In chronic Bright's disease we are likely to find operative in the earlier stages those causes which have the least to do with marked organic changes. Angio-neurotic phenomena, and perhaps anemia, are in the foreground—the dead fingers, the headaches, the polyuria, and the asthma. As degeneration of the vessels progresses, we have hypertrophy of the heart, albuminuric retinitis, cerebral hemorrhage, and cerebral œdema; and when the kidneys fail to provide for the necessary excretion, the vomiting, convulsions, and coma, with a continuance of many of the former symptoms.

The action of certain drugs and therapeutic measures is very suggestive as to some of these points of pathology, as well as practically.

Ball and Jennings (L'Encephale, 1887, p. 295), in examining the state of the arterial tension in a case of chronic morphinism, find that during the period at which the effect of the dose has passed off and the demand for a new one is imperative, ["etat de besoin"] the sphygmograh gives the tracing of high tension which resembles, as is evident to any one familiar with this feature, the condition found in many cases of Bright's disease, and in fact considered by some persons as characteristic as the state of the urine. The comparison is expressly made by these authors. This indescribable condition of distress of the morphinist, which probably brings as much suffering bodily and mentally as anything short of severe and constant pain, is by no means dissimilar to the nervous restlessness often noticed in the patient with chronic interstitial nephritis, which probably has relations on the one hand with convulsions, and on the other with insanity.

Ball and Jennings find, as might be expected, that the dose of morphine which brings the well-known comfort, reduces also the arterial tension to the normal point, but not only this, that some other drugs which stimulate the heart without raising or while reducing the tension also bring relief. These are sparteine and nitroglycerine. Is it not more probable that the great relief from many uremic symptoms by both well-known agents, morphine and nitroglycerine, should be much more closely connected with their action on the vascular tension than on any power which they might be supposed to have of assisting elimination, a power which there is not the slightest independent reason to attribute to them? So far as opium is concerned the effect is undoubtedly in the opposite direction.

These facts suggest a query which I propose to this society with a due sense of responsibility involved in an affirmative reply, and which I should hesitate much in putting before a less judicious body, namely, whether a physician would be justified not only in using morphine, as we all do, for the relief of nervous symptoms in the advanced stage of Bright's disease, but in instituting a treatment by small doses as soon as a diagnosis of chronic interstitial nephritis with high tension is established, using, of course, all measures to diminish as much as possible the injurious effects, in the way of diet, exercise and cathartics; or in other words making his patient a careful morphinist? We might go further and suggest the contrast between the calm of the satisfied opium eater and the restless worry and drive which I believe to be more than a coincidence in the middle aged business man who so frequently finds himself unexpectedly with Bright's disease in full force.

Copious diaphoresis by the hot air bath, or more recently under the influence of pilocartine is well known to be among the most efficient means of relief in uremic attacks, and the explanation usually given is that the urea or other urinary poison is carried off by the skin. It is true that a good deal of water is thus got rid of and a certain amount of urea and other solids accompany it; but no analysis has yet shown that all such supplementary outlets together cover the normal amount for the day, to say nothing of an accumulation for a long time, such as is demanded by the retention theory. The same is true, so far as the solids are concerned, of the vomiting which carries off some solids ; and probably of the fecal discharges also. All these emunctories undoubtedly afford some relief, but the mere eliminative action cannot account for the more continued good effects which sometimes follow.

Such measures have, however, this in common with the drugs we have considered, that they relax a more or less extensive territory of arterioles, and diminish the general tension. Dry cups on the loins which carry off nothing are often useful in re-establishing diuresis. Bleeding, which can remove but a small fraction of the total urea in the body and which certainly cannot diminish the percentage in the blood, is of undoubted value in some cases of convulsions and has also been followed by prompt recovery in cases of cerebral œdema.

DISCUSSION ON DR. EDES' PAPER.

Dr. F. X. DERCUM agreed in a general way with the reader of the paper, but thought that one should be careful about making deductions which assume that urea is not injurious to the organism, even in the highest degree. It does not produce nerve symptoms under the conditions quoted only because the other emunctories act vicariously. The animal ultimately dies when these become insufficient. We know too little about the subject to be able to state whether the phenomena of uremia are due to the retention or the perversion of this product.

Dr. SEGUIN added that nerve symptoms which might be attributed to uremia were the nerve symptoms of the prealbuminuric stage of Bright's disease. These were of frequent occurrence, consisting of a succession of headaches and paresthesiæ about the head coexisting with high arterial tension and a negative condition as regards symptoms of cerebral disease. A previously typical migrane would be changed in character and become more frequent. A characteristic headache of the prealbuminuric stage of

Bright's disease was, however, of the occipital form, bilateral pain extending often into the cervical region. Occasionally there was increased action of the heart, and usually hyaline casts were found in the urine, but albumen rarely at this stage. Symptoms of indigestion, too, were found in the formative stage of interstitial nephritis. The paresthesiæ about the head are probably due to increased arterial tension. The pressure, confusion, and often vertigo are generally wrongly referred to hyperæmia of the brain. The speaker was not opposed to the trial of the morphia treatment if the nerve symptoms were prominent and persistent, and if proper precautions could be insured to prevent abuse of the drug. Still, advising such a treatment must always entail a heavy responsibility, partly justified by the fatal nature of the disease.

The speaker suggested that dementia paralytica might give rise to a confusion in diagnosis. There was a high arterial tension in this condition ; we might find other signs of a coexisting interstitial nephritis, so that in some cases the apoplectiform and epileptiform attacks may in reality be due to "uremia."

Still, referring to the difficulty of diagnosis, the speaker related the history of the case of a little girl suffering from the ordinary parenchymatous form of Bright's disease, having also hemiplegic symptoms with localized spasms in one hand. There were atrophic changes in the optic nerve and retina. The question was whether we had here a condition of uremia affecting one hemisphere with albuminuric retinitis, or whether we had a coincidence of diseases, a tumor of the motor region of one side explaining the convulsions in the hand and the hemiparesis. The change in the fundus oculi consisted in hemorrhages and whitish patches of degeneration of evidently nephritic origin.

Dr. L. C. **Gray** referred to cases in which the differential diagnosis was observed. A young gentleman of 28 years, of immaculate habits, came home one night with a slight headache. At an early hour the sister heard a noise in his room, and going in found him dancing a jig. Afterwards he lay down and went to sleep. When the speaker saw him he was apparently oblivious to his surroundings, yet would respond to requests, but with the jerky action of a jumping-jack. This patient finally had convulsions and died. There was found chronic interstitial nephritis, and, curiously enough, there was in each middle cerebral artery a little slit through which the blood exuded, lifting the pia. The pia was not opaque. The hemorrhage had not affected the integrity of either the pia or the cortex. The arteries were not examined through accident.

Another case was that of a gentleman for whom he had been consulted in regard to commitment to a lunatic asylum. The speaker had made it a rule in all doubtful cases to examine the kidneys. There was found well-marked pyeluria, which was treated with dietetics, laxatives, quinine, and rest in bed. With the disappearance of the pyeluria, the mental symptoms improved. After some indiscretion, the pyeluria returned and with it the mental symptoms.

In another case of Bright's disease with convulsions and death, there was found a plug of the basilar artery, and, in another, extensor paralysis, symmetrical and resembling that of lead poisoning, supervened a few days before death. The occipital headaches of nephritis often had a quasi periodicty, with marked relief for many days from a good dose of quinine.

The speaker also referred to a curious case of hematurea with granular casts, during which condition the patient passed into coma vigil, when pulsation of the superficial veins of the thorax appeared.

On several occasions the speaker had verified the test proposed by Dr. McBride for differentiating coma of Bright's disease from an attack of hemiplegia. A man woke up to find himself on the floor, and it became uncertain whether he had fallen under the influence of apoplexy or the coma of Bright's disease. The cutaneous and tendon reflexes presented the differential test.

In treating cases of nephritis the speaker thought it most necessary to avoid strain upon the cutaneous capillaries. He carefully confined such patients to the room or bed, thus shielding them from the variations of temperature. Neglect of this precaution has been followed by dangerous attacks; while with it cases will get along without any active medication.

Dr. DERCUM stated that it was his custom to treat all doubtful cases as though cases of Bright's disease. A man 68 years, without any previous seizure, had twenty-six convulsions within a short time, limited to the right side of the body and face; he had also one general convulsion. This man was sweated, and recovered consciousness, and was now apparently well. There had been evidently no hemorrhage. In a suitable case the speaker would try the morphine treatment recommended by Dr. Edes.

Dr. ZENNER suggested localized œdemas as the origin of the symptoms in such a case or possibly a plug which had been subsequently washed away.

Dr. EDES stated that it had been pretty well settled that urea was not a highly dangerous product. The relief afforded by diaphoretics was very slight compared to the kidney elimination. Morphine if used at all in these cases should be used with the greatest caution in the smallest doses which would produce the stimulant effect. The speaker hoped that no one present would regard him as having recommended the procedure promiscuously, and that if any one did use it, he would do so upon his own responsibility, and with regard to the special case in hand.

The speaker thought it questionable whether Dr. Seguin's instances of prealbuminuric headache had preceded the kidney lesion. The outbreak of these symptoms did not mark the beginning of the lesion, which has usually existed for a long time.

In regard to Dr. Gray's case of coincident renal and mental disease he could not admit its oppositeness. The case was described as one of "pyeluria;" the secreting structure of the kidneys might not have been invaded at all.

Reviews.

APPLIED ANATOMY OF THE NERVOUS SYSTEM, by A. L. Ranney, A.M., M.D. 2d Ed. D. Appleton & Co. 1888.

Dr. Ranney's book on the Anatomy of the Nervous System is well known to most American neurologists, and does not call for extensive notice in this journal. The criticisms passed upon the first edition were so decided in their tone that we naturally looked for many changes in this second edition, which the author has "rewritten, enlarged and profusely illustrated." That it is rewritten and enlarged is very evident; that it is profusely illustrated falls a little short of the truth. The illustrations are so numerous that the printed matter appears to be a mere accompaniment to the text, and we are bound to add that the illustrations are not always happily chosen. They are culled from all possible sources, and many of them from works that are decidedly antiquated. The author claims "originality of treatment * * *, because diagrammatic illustration forms an important feature in the author's system of teaching." Ingenious diagrams are, to be sure, a great aid in teaching, but it is not sufficient merely to alter diagrams of other authors, it is of greater importance still to present the subject in a novel, or at least an interesting fashion. This, Dr. Ranney has not done; he presents an immense number of theories and controversial facts on every point that is at all in doubt, and never ventures to express a decisive opinion. The book shows too plainly that the author's own researches and studies do not entitle him to speak with any sort of authority on any subject treated in this book. The author's reading too, seems to be of a peculiar sort; "home products" seem to make a deep impression upon his mind, altogether out of proportion to their true worth, entire pages are quoted from "home" articles, the conclusions are taken up in the main body of the work, and then a little foot-note is added to say that there may still be some doubt on this or that point. This is supposed to be a text-book from which the student is to gather accurate information, and Dr. Ranney should have resisted the temptation of placing all this recondite lore before the student who would rise from a perusal of these pages with the idea that almost anything and everything can be maintained with regard to cerebral anatomy, provided you can produce a few neat looking diagrams to prove the case.

We have spoken in general terms, but it would be impossible to criticise in detail. The author's judgment is sufficiently characterized by the fact that he reproduces Luys' absurd theories regarding the thalamus which every one else has abandoned ; his account of the lemniscus is a mixture of the views of Flechsig, Meynert, Spitzka and others, and we challenge any student to form any sort of idea of what the lemniscus is and what it is not. In reporting Goltz's views the author refers to a method of experimentation which Goltz abandoned fully six years ago. Ecker's well-known diagram of the convolutions is said to be after Ferrier ; Flechsig's diagrams are taken at second and third hand, and after they have passed through this process are slightly altered by the author.

Such criticisms would be unjust, if the author did not point to the illustrations as *the* important feature of his book.

If the author wishes to see in what way diagrams can be made to subserve a useful purpose, let him consult Edinger's little book—a book, by the way, which he might have consulted with great advantage to himself. If the public calls for a third edition of the "Applied Anatomy," we hope the author will start afresh, that he will avoid all controversial facts, and will give the student a more concise account of the anatomy of the central nervous system, and fewer "pictures."

Letters to the Editor.

To the Editor of THE JOUR. OF NERVOUS AND MENTAL DISEASE.

Sir:—The admirable abstract of Marchi's paper in your journal for August, 1888, pp. 515-517 contains the terms *optic thalami* six times, and the term *corpora striata* nine times in addition to the title. If I am right in holding that no misconception could possibly arise from the employment of the mononyms *striata* and *thalami*, then one-fiftieth of the entire abstract, representing two whole lines of your valuable page, has been needlessly occupied by really irksome repetitions, involving an appreciable loss of time and energy in writing, printing and reading.

BURT G. WILDER.

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THE Journal OF Nervous and Mental Disease.

Original Articles.

THE ANTIPYRETICS—ACETPHENETIDIN AND ANTITHERMIN.

By ISAAC OTT, M.D.

ONE of my pupils, Dr. Peter J. Martin, in his prize essay[1] has shown that kairin, hydrochinon, antipyrin and thallin reduce the heat production and heat dissipation of animals thrown into an experimental fever by puncture of the four basal heat centres. Drs. Wood, Reichert and Hare previously had utilized the discovery of Drs. E. V. Bergmann and O. Angerer,[2] that pepsin would produce fever. They studied the effect of antipyrin on this fever and found H. P. reduced more than H. D. Dr. Girard[3] has recently studied the effect of antipyrin in the same manner as Dr. Martin, by puncture of the corpus striatum. He concluded that antipyrin in physiological conditions lowers the temperature of the rabbit, that this agent efficaciously combats the hyperthermia caused by irritation of a thermic centre, that in antipyrinized animals the puncture of the caudate nucleus produced its usual result. Sawadowski[4] has also shown that antipyrin acts upon heat phenomena through the caudate nucleus, for when the copora striata were removed this antithermic had no effect.

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[1] University of Penna., 1887. Published in Therapeutic Gazette, 1887.

[2] Festschrift zur dritten Saecularfeier der Alma Maximiliana, gewidmet von der Medicinischen. Facultat Wurzburg, Band 1, 1882.

[3] Revue medicale de la Suisse Romande, No. 11, 1887.

[4] Centralblatt f. med. Wissenschaft, 1888.

In my laboratory Dr. E. W. Evans[1] has exhaustively studied the effects of antifebrin upon the heat production and heat dissipation of the normal and fevered animal. The fever was produced by deutero-albumose.

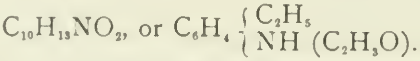
Antifebrin was found to reduce the heat production of animals normally and in a state of experimental fever. Dr Hobart A. Hare has confirmed these researches of Dr Evans.

These researches inclined me to study the latest antipyretics. In my experiments rabbits and cats were used, the drug being given by the stomach either by catheter or capsule. The insolubility of these drugs caused me to select the stomach as the medium for exhibition of the drug. Heat production and heat dissipation were measured by means of d'Arsonval's calorimeter. To produce an experimental fever, cats were used. In them the cruciate heat centres of Eulenberg and Landois were removed, which permitted the temperature to rise and to remain so till the death of the animal, The operation was performed a day or two before the exhibition of the antipyretics. No food was given to them for three days previous to the experiment.

The cruciate centre was preferred to the Sylvian for injury, as Sylvian lesions on both sides of the brain would soon cause death. How these centres act in the production of fever I have fully detailed in a previous number of this Journal (Feb., 1888). To remove the cruciate centres the skull was perforated near the median line on both sides with Pasteur's trephine. The dura mater was divided, and the cortex broken up with a blunt probe, care being taken to avoid injuring the longitudinal sinus. The effects of the drugs upon heat production and heat dissipation was not tested till an hour after their administration.

ACETPHENETIDIN.

This body is a grayish powder whose formula is



$$C_{10}H_{13}NO_2, \text{ or } C_6H_4 \begin{cases} C_2H_5 \\ NH\,(C_2H_3O). \end{cases}$$

being in fact benzol C_6H_4 in which two atoms of hydrogen are replaced by compound radicals. It has neither taste nor smell, almost insoluble in water. It dissolves in alcohol in the proportion of one in twenty. It arrests the alcoholic fermentation of grape sugar. In order to test the action of the drug on the healthy body, Dr. Georgieoski took thirty grains a day in three doses of ten grains each, separated by intervals of an hour. The only sensation produced was a slight feeling comparable to the beginning of alcoholic intoxication. No reduction of temperature was produced though the drug was taken for some days. The color of the urine was unchanged, but when a few drops of solution of chloride of iron were added to it, it gave a reddish-brown or black color; sulphate of copper gave a similar reaction. The urine was affected in half an hour after taking the dose. Observations were also made by Dr. Georgieoski upon thirty febrile patients, including cases of phthisis, typhoid, pneumonia, erysipelas of the face, acute rheumatism, quinsy, diptheria and pleurisy. A single dose of three to five grains was sufficient to lower the temperature, usually in half an hour it had gone down nearly 1° F. This reduction continued, the lowest point being reached in three to four hours after the dose had been taken. The subsequent rise which then began was very gradual, the original height not being reached for five to six hours more. As a rule, a three-grain dose reduced the temperature 1.8°, and a five-grain 3.6°. The nature of the disease seemed to have a decided influence upon the effect of the medicine; thus in typhoid, where the temperature curve generally shows great variations the drug produces a greater and more prolonged reduction than in the diseases like pneumonia, where it is less variable. This drug is also an analgesic. Acetphenetidin has a composition analogous to antifebrin.

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[1] Therapeutic Gazette, 1887.

In the following table the results upon acetphenetidin are arranged. H. P. is heat production; H. D., heat dissipation.

	Before drug.			*After drug.*		
EXP.	H. D.	H. P.	R. T.	H. D.	H. P.	R. T.
1	29.20	26.51	103	20.86	23.21	102.5
2	36.71	31.10	102.6	20.86	20.30	101.9
3	20.86	20.86	102.2	20.86	18.95	102.4
4	33.76	34.76	104.8	37.54	33.25	101.5
5	43.80	43.47	102.1	36.29	34.63	101.9
6	10.43	12.15	104.8	14.60	11.15	102.2

Increrse or decrease of H. D. after drug.	*Increase or decrease of H. P. after drug.*
— 8.34	— 3.30
— 5.85	— 10.80
.0	— 1.91
+ 3.78	— 1.51
— 7.51	— 8.84
+ 4.17	— 1.0

By an examination of the above tables and Fig. 1 it is seen that with one exception the temperature is reduced by acetphenetidin. Heat dissipation is reduced in all experiments except two, whilst heat production is diminished in all except one.

This is due to an action on the thermal centres, for when blood pressure experiments were made there was but little change in cardiac frequency. The slight fall of pressure in the arteries would not suffice to diminish heat production as much as has been shown. There was no important change in the movements of the respiratory apparatus.

ANTITHERMIN.

Its formula is

$$CH_2C(C_6H_5N\text{–}NH)\ CH_2\text{–}CH_2COOH.$$

This body has a chemical relationship to antipyrin and is composed of phenyl-hydrasin with levulinic acid. It is one of the intermediate products in the chemical production of antipyrin. It is composed of colorless crystals, of a slightly bitter taste, which causes an unpleasant grating

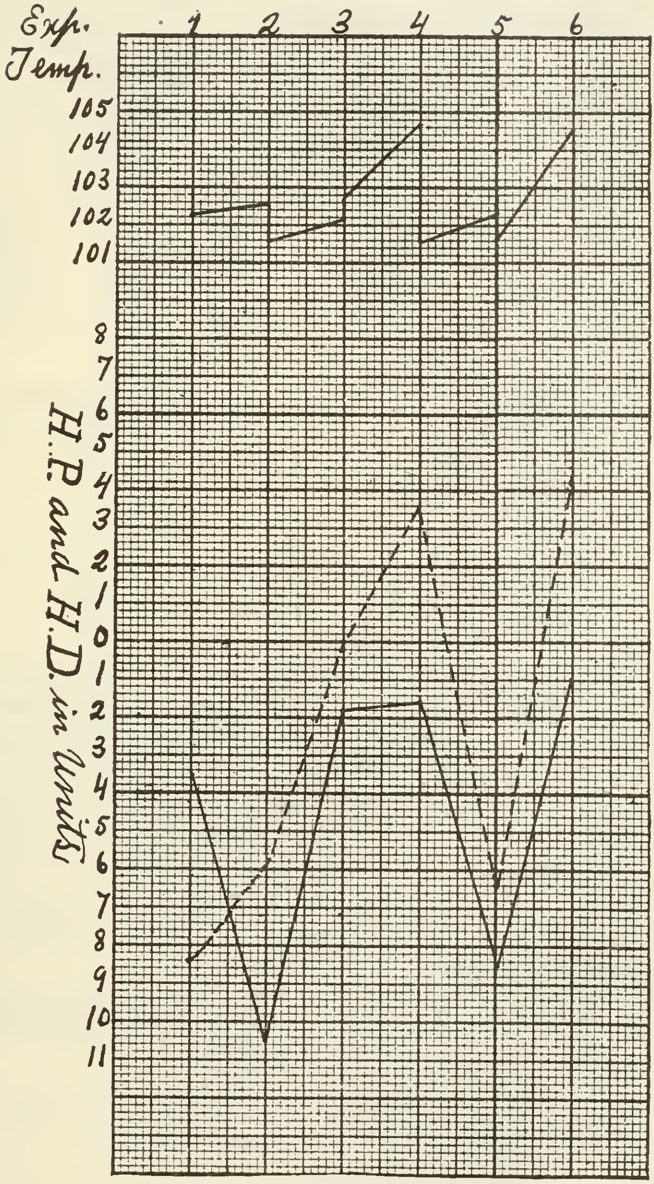
Exp.
1
2
3
4
5
6
Temp.
105
104
103
102
101
8
7
6
5
4
3
2
1
0
1
2
3
4
5
6
7
8
9
10
11
H.P. and H.D. in Units

Fig. 1.

when ground between the teeth. It is insoluble in water and but sparingly soluble in alcohol.

	Before drug.			*After drug.*		
EXP.	H. D.	H. P.	R. T.	H. D.	H. P.	R. T.
1	35.46	34.27	101.6	33.37	33.37	101.6
2	33.37	32.04	104.4	27.11	24.25	101.2
3	31.29	29.90	102.5	29.20	29.20	102.6
4	47.92	43.74	103.1	45.90	35.96	99.8
5	46.72	43.44	102.8	35.46	29.99	100.1
6	16.68	16.12	102.7	22.94	21.25	101.5

Increase or decrease of H. D. after drug	*Increase or decrease of H. P. after drug.*
— 2.09	— .90
— 6.26	— 7.79
— 2.09	— 0.50
— 2.02	— 7.78
— 11.36	— 13.45
+ 6.26	+ 5.13

In the experiments upon antithermin, the temperature fell in all except one, and heat production and heat dissipation also were diminished in all cases save one, see Fig. 2. The cardiac frequency was not perceptibly altered by antithermin. The arterial tension was not greatly diminished. Its diminution would not account for the lessening of the heat production. No marked change was noticed in the respiration. These experiments lead to the conclusion that acetphenetidin and antithermin diminish temperature, heat production and heat dissipation as a rule.

As antipyrin acts upon the thermal centre of the corpus striatum to reduce temperature, it is highly probable that these antipyretics act in a similar way.

Appended are some of the experiments upon which the preceding statements are based.

A. T. is air temperature.

E. T., temperature of exit-tube.

R. T., rectal temperature.

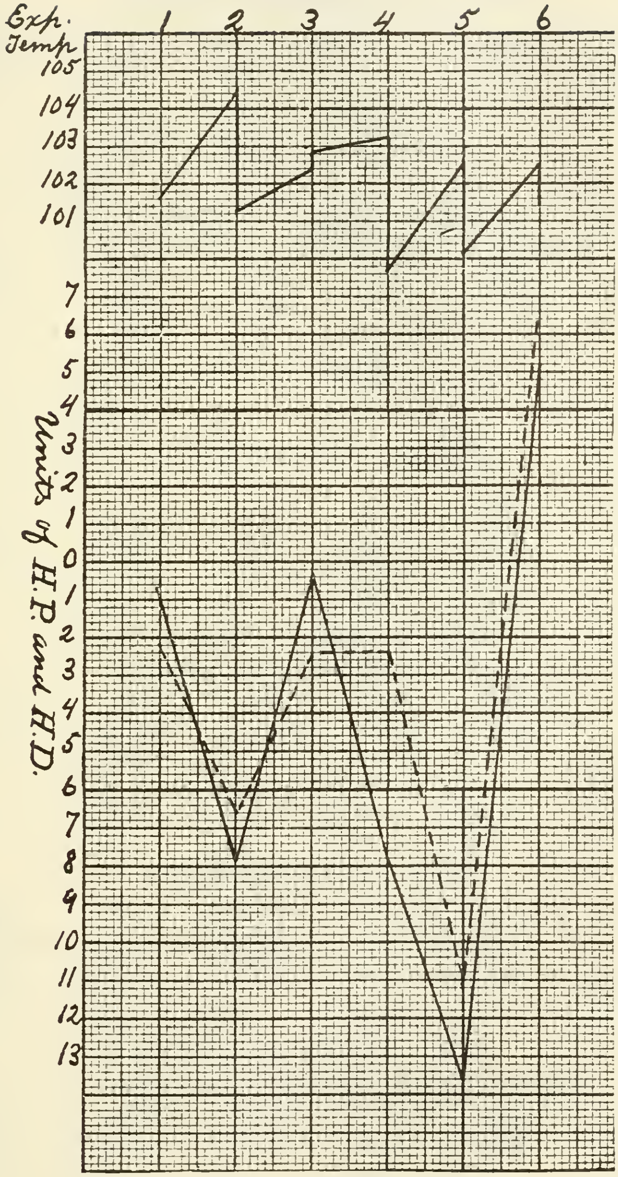
Exp.
1
2
3
4
5
6
Temp
105
104
103
102
101
7
6
5
4
3
2
1
0
1
2
3
4
5
6
7
8
9
10
11
12
13
Units of H.P. and H.D.

Fig. 2.

EXP. I.—White rabbit; wt. 4.06 lbs.

P. M.	A. T.	C. T.	E. T.	R. T.	METER.
1.30	70	73	22.1	10.30	22,424
2.30	78	73.7	24.0	102.2	22,674
		+ .7		— .8	

H. D. = 29.20. H. P. = 26.51.

2.40 Five grains of acetphenetidin by the stomach.

P. M.	A. T.	C. T.	E. T.	R. T.	METER.
3.40	75.3	73.65	24.2	101.8	22,674
4.40	75.0	74.15	23.8	102.5	22,904
		+ .50		+ .7	

H. D. = 20.86. H. P. = 23.21.

EXP. II.—Rabbit, wt. 3.40.

P. M.	A. T.	C. T.	E. T.	R. T.	M.
12.10	67.8	66.8	19.5	102.65	77,980
1.10	69.0	67.68	20.4	101.00	78,336
		+ 88		— 1.65	

H. D. = 36.71. H. P. = 31.10.

1.28 Ten grains of acetphenetidin by stomach.

P. M.	A. T.	C. T.	E. T.	R. T.	M.
2.28	69.5	67.6	20.5	102.1	78,336
	69.8	68.1	20.4	101.9	78,652
		+ .5		—.2	

H. D. = 20.86. H. P. = 20.30.

EXP. III.—Rabbit, wt. 3.84.

P. M.	A. T.	C. T.	E. T.	R. T.	METER.
12.7	66.0	64.6	19.1	102.2	78,652
1.7	65.2	65.1	18.4	102.2	79,034
		+ .5		.0	

H. D. = 20.86. H. P. = 20.86.

1.25 Ten grains of acetphenetidin by the stomach.

P. M.	A. T.	C. T.	E. T.	R. T.	METER.
2.52	68.2	65.1	19.1	103.0	79,034
3.52	67.0	65.6	19.2	102.4	29,284
		+ .5		— .6	

H. D. = 20.86. H. P. = 18.95.

EXP. IV.—Rabbit, wt. 3.36.

P. M.	A. T.	C. T.	E. T.	R. T.	METER.
12.32	62	61.35	16.8	104.8	79,284
1.32	64	62.15	18.3	105.3	80,975

H. D. = 33.76. H. P. = 34.76.

1.34 Ten grains of acetphenetidin by stomach.

2.40	68.8	62.4	19.0	103.1	80,975
3.40	68.8	63.3	18.5	101.5	81,126
		+ .9		— 1.6	

H. D. = 37.54. H. P. = 33.25.

EXP. V.—Rabbit.

A. M.	A. T.	C. T.	E. T.	R. T.	METER.
9.58	66.8	64.3	19.1	102.1	81,126
10.58	66.5	65.35	18.8	102.0	82,290
		+ 1.05		— .1	

H. D. = 43.80. H. P. = 43.47.

11.00 Ten grains of acetphenetidin by the stomach.

12.8	66.5	65.28	18.8	102.4	82,290
1.8	69.0	66.15	19.6	101.9	83,028

H. D. = 36.29. H. P. = 34.63.

EXP. VI.—Cat (cruciate fever), wt. 2.6.

P. M.	A. T.	C. T.	E. T.	R. T.	METER.
4.20	83.2	82.45	28.5	104.8	45,144
5.20	83.4	82.7	28.4	105.6	45,474
		+ .25		+ .8	

H. D. = 10.43. H. P. = 12.15.

5.30 Five grains of acetphenetidin by the stomach.

6.05	83.8	82.7	29.2	103.8	45,474
7.05	83.4	83.05	28.6	102.2	45,960

H. D. = 14.60. H. P. = 11.15.

EXP. VII.—Rabbit, wt. 4.78.

P. M.	A. T.	C. T.	E. T.	R. T.	METER.
2.10	82.6	76.3	27.2	101.6	20,789
3.10	81.7	77.15	26.0	101.3	21,080

H. D. = 35.47. H. P. = 34.27.

3.20 Five grains of antithermin by stomach.

4.13	81.8	77.3	29.1	101.6	21,080
5.13	81.2	78.1	26.2	101.6	21,336
		.8		.0	

H. D. = 33.37. H. P. = 33.37.

EXP. VIII.—Cat (cruciate fever), wt. 5.36.

P. M.	A. T.	C. T.	E. T.	R. T.	METER.
3.10	91.1	85.6	31.7	104.4	49,969
4.10	87.4	86.4	30.4	103.1	20,272

H. D. = 33.37. H. P. = 32.04.

4.30 Fifteen grains of antithermin by the stomach.

5.20	87.3	86.45	31.3	101.8	50,272
6.20	87.7	87.1	30.9	101.2	50,535

H. D. = 27.11. H. P. = 24.25.

EXP. IX.—Rabbit, wt. 3.84.

P. M.	A. T.	C. T.	E. T.	R. T.	METER.
1.30	81.9	78.45	27.7	102.5	21,843
2.30	83.6	79.2	26.8	102.0	22,111
		+ .75		— .5	

H. D. = 31.29. H. P. = 29.70.

2.40 Five grains of antithermin by stomach.

3.35	83.9	79.35	28.4	102.6	22,111
4.35	82.5	80.05	27.0	102.6	22,422
		+ .70		.0	

H. D. = 29.20. H. P. = 29.20.

EXP. X.—Rabbit, wt. 5.10.

P. M.	A. T.	C. T.	E. T.	R. T.	METER.
2.29	69.2	66.1	20.0	103.1	83,028
3.29	69.5	67.25	20.0	102.1	83,832
		+ 1.15		— 1.0	

H. D. = 47.92. H. P. = 43.74.

3.30 Ten grains of antithermin by capsule.

4.30	70.6	67.2	20.9	101.3	83,832
5.30	71.2	68.1	20.9	99.8	84,710
		+ .9		— 1.5	

H. D. = 45.90. H. P. = 35.96.

EXP. XI.—Rabbit, wt. 4.40.

A. M.	A. T.	C. T.	E. T.	R. T.	METER.
8.32	68.5	68.28	20.4	102.8	84,710
9.32	72.5	69.4	21.8	101.9	24,889
		+ 1.12		— .9	
	H. D. = 46.72.		H. P. = 43.44.		
9.40	Ten grains of antithermin by capsule.				
10.33	70.8	69.4	21.5	101.6	84,889
11.33	71.7	70.25	21.8	100.1	85,756
	H. D. = 35 46.		H. P. = 29.99.		

EXP. XII.—Rabbit, wt. 3.41.

A. M.	A. T.	C. T.	E. T.	R. T.	METER.
11.40	71.7	70.3	21.8	102.7	85,756
12.40	72.7	70.7	22.1	102.5	87,348
	H. D. = 16.68.		H. P. = 16.12.		
1.42	73.8	70.7	23.1	102.1	87,348
2.42	73.0	71.25	22.5	101.5	87,979
		+ .55		— .6	
	H. D. = 22.94.		H. P. = 22.25.		

EXP. XIII.—Rabbit.

P. M.	PULSE.	ARTERIAL PRESSURE.
3.15	63	120
3.25	Five grains acetphenetidin by the stomach.	
4.46	64	110
4.48	60	126
5.10	58	112

EXP. XIV.—Rabbit.

A. M.	PULSE.	ARTERIAL PRESSURE.
11.15	60	108
11.25	Five grains of antithermin by stomach.	
P. M.		
12.25	62	108
12.42	63	90

PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, October 22d, 1888.

THE VICE-PRESIDENT, CHARLES K. MILLS, M.D., IN THE CHAIR.

Dr. WILLIAM OSLER presented a

NOTE ON PACHYMENINGITIS HEMORRHAGICA.

There are several points of interest in this condition:

First. Is it in any sense of the term an inflammatory process? Practically, we see one of three conditions in these cases:

a. Subdural vascular membranes, often of extreme delicacy.

b. Simple subdural hemorrhage.

c. Combination of the two—vascular membrane and blood-clot.

In two specimens which I showed last year at the Pathological Society, I remarked on the absence of any of the features to which we could apply the term inflammatory.

Dr. Joseph Wiglesworth, in the January number of the *Journal of Mental Science* for this year, takes the same ground. Apart from injuries, dura-arachnitis is rare. I have seen two instances in the specific fevers. In both there was delicate fibrinous exudate between the dura and the arachnoid.

Second: Which is antecedent, vascular membrane or hemorrhage?

It is usually stated that the hemorrhage occurs first, and from the blood-clot, when organized, the vascular sheet arises. This may be the case, and the fact that subdural hemorrhage is often found alone—in fifteen out of forty-two cases in Wiglesworth's series—lends weight to this view. On the other hand, the vascular membrane may exist without a trace of past hemorrhage; neither staining nor melanin grains, simply a fibrous sheet, varying in thickness and permeated with large vessels. The specimen here shown illustrates this condition: It was removed from a man, aged fifty-six, who died of aneurism. The skull cap was unusually thick and dense and extremely vascular. The meningeal arteries ran in deep grooves through the bone. There were many points of vascular union between the inner table of the skull and the dura. In spite of this, the calvaria was removed without much difficulty. The inner surface of the dura was everywhere covered with a fibrous, highly vascular membrane. Toward the vault it was from half a line to a line and one-half in thickness. Toward the base it was extremely thin. It was chiefly made up of large veins forming in places beautiful arborescent tufts. On section, where the membrane was thickest, the veins bled freely, but there was no trace of clot. Microscopically, the membrane was composed of bundles of connective tissue forming a supporting framework for the numerous vessels. At one small area alone, in the middle fossa on the left side, there was a brownish-red staining on the surface of the vascular membrane.

In the two specimens which I studied last winter the identical condition existed, though in neither was the blood supply abundant, nor the vascular membrane thickened.

Third. Whence the hemorrhage, dural or pial?

I have always thought the former, but I see that Wiglesworth, in referring to the atrophy of the convolutions as a possible cause for hemorrhage, speaks thus: "The pia mater over a variable area may be so intensely congested as actually to resemble an ecchymosis. . . . It is manifest that the conditions may be highly favorable to actual rupture, and I doubt not that this frequently occurs."

Certainly in the instances I have seen the subdural membranes were intimately associated with the dural vessels, and it seems more probable (and is, I believe, generally acknowledged) that from these the hemorrhage always proceeds.

Fourth. The explanation of the occurrence of this singular structure is by no means clear. The cases are commonly met with in asylums. In general hospitals years may pass without seeing an instance. During eight years in the post-mortem room of the General Hospital of Montreal, no instance occurred. The first specimen which I saw, after having been for fourteen years interested in morbid anatomy, was demonstrated by Virchow, in 1884, at the Pathological Institute. At the Philadelphia Hospital cases are by no means uncommon. Within the past two or three months there have been four specimens found, usually in the bodies of persons from the insane department, but in the three instances to which I have referred, the patients came from the medical wards, and had not shown any mental symptoms.

One reason urged why the subdural hemorrhage is much more common in the insane, is the atrophy of the convolutions so constantly associated with this condition. But there must be something more than this, for if atrophy alone is the chief factor, we certainly would meet with it in phthisis and in other cachectic conditions in which the cerebral wasting is quite as common as in general paresis.

The frequency of this condition in asylum work may be gathered from the fact that Wiglesworth's paper is based upon forty-two specimens which occurred in a series of four hundred unselected post-mortem examinations in which the persons died of various forms of insanity. Wiglesworth's contribution is one of the most interestingand valuable that has been made upon the subject.

Dr. F. X. DERCUM asked if Dr. Osler accepted the view of Wiglesworth that this condition of the dura is really due to hemorrhage from the pia. He could not understand how a hemorrhage from the pia could become so intimately connected with the dura. He had always regarded the formation as dural.

He asked Dr. Osler if he had any explanation to offer for the formation of the curious cysts in old cases of pachymeningitis.

Dr. JAMES HENDRIE LLOYD said that Dr. Osler had stated that in the case reported there was aneurism of the aorta. He would ask if there was any connection between the two conditions. Were there any brain symptoms?

The idea had prevailed heretofore that we mostly find this condition in connection with brain troubles such as general paresis, trauma, and chronic alcoholism.

Dr. CHARLES K. MILLS said that the paper was interesting in connection with a case of a child nearly three years of age, with spasms of a peculiar type, for which he advised trephining and the operation was done by Dr. John B. Roberts at the Philadelphia Polyclinic. After the removal of the skull and dura mater, a membrane was found which was easily separated from the pia arachnoid. It was a vascular membrane, between the dura mater and the pia arachnoid. Œdema and great vascularity of the pia mater were also present. After the escape of some serous fluid, the brain apparently receded. The history was that a clock had fallen upon the child's head, and the question was whether or not a slight hemorrhage beneath the dura had not taken place, or whether there might not have been the formation of a membrane without the presence of a clot. It may be that this is the explanation of some of the cases of infantile spasm of peculiar type. This child often had from eight to ten attacks a day. The day after the operation it had two slight attacks, but since then none. He had seen a considerable number of cases of pachymeningitis hemorrhagica at the Philadelphia Hospital, some of which he had reported.

Dr. OSLER did not see how the hemorrhage could be connected with the pia as Dr. Wiglesworth thought. The cases he had seen had been in medical wards, and had presented no indications of cerebral trouble. Atrophy of the convolutions does not always coexist. If atrophy were one of the prime factors in the causation of this condition, we would expect to see it more frequently in phthisis and the chronic cachexias, in which the cerebral wasting is often quite as marked as in general paresis. There is often as much thickening of the pia arachnoid and gelatinous œdema between the convolutions in phthisis as in general paresis, yet, so far as he knows, subdural hemorrhage has not been described in connection with phthisis except in the inmates of lunatic asylums. The condition is rarely met with in general practice. He had himself twelve years' experience in general autopsical work before he saw a case of pachymeningitis hemorrhagica. The first case that he saw was demonstrated by Virchow in 1884, in the Pathological Institute in Berlin. At the Philadelphia Hospital the condition is met with two or three times a month, usually in patients from the Department for the Insane.

Dr. OSLER then presented a communication entitled

INJURY TO THE CAUDA EQUINA AND SYPHILOMA OF THE CORD AND CAUDA EQUINA.

Dr. F. X. DERCUM reported

THREE CASES OF SPINAL ACCESSORY SPASM UNSUCCESSFULLY TREATED BY EXCISION OF THE NERVE.

The following cases were placed on record, not only to confirm the inutility of the treatment by excision of the nerve, but also to provoke discussion on this most distressing affection.

Case I.—J. F., aged forty years, married, and a gas-fitter by trade, presented himself at the Nervous Dispensary of the University Hospital, with the following history: He had had rheumatism at various times, one attack of which had kept him in bed nine weeks. Eight months after this last attack he had rheumatism of the muscles of the shoulders, and then began to have turning of the head to one side, which, up to the time of his first examination, had lasted a year. The movements were at first slight and painless, as they still are, but about one month after their first appearance the movements became excessively violent, coming on in paroxysms, and interfering with sleep. His wife described these attacks as irritable convulsions. However, they were limited to the neck, and were unattended by loss of consciousness. These excessively severe paroxysms were, later on, merely occasional. Short, sharp, jerky movements were, however, never absent. The sterno-cleido-mastoid of the right side felt hard and contracted, and was evidently the principal muscle at fault. Indeed, in the movement observed by us it appeared to be the only one involved, though in the severe paroxysms described by the patient the other neck muscles, doubtless, played a part.

Some years ago, Dr. H. C. Wood successfully treated one of these cases by severely cauterizing the back of the neck with the hot iron; and acting upon this hint, the Paquelin cautery and antimonial ointment were freely used in the present case, but without any relief of the spasm.

Finally, the patient was admitted to the hospital under the care of Dr. Agnew, who excised a portion of the right spinal accessory nerve. The immediate effect of the operation was a disappearance of the spasm, but as this man had left the hospital, in which he remained twenty-five days, the small, jerking, rotary movement of the head had again appeared, and he could discover little, if any, difference in the character of the movement. Whether any of the more severe paroxysms recurred he was unable to say, as the patient has not since been under observation.

Case II.—E. L., aged fifty-eight years, married, a watch-case-maker, likewise presented himself at the University clinic two years ago. When apparently in good health, he commenced to have slight twitching in the neck. This twitching had gradually become marked, and, at the time of making the note, was present in the form of a severe clonic spasm. There was no history of rheumatism or syphilis.

Examination showed that the left sterno-cleido-mastoid and trapezius muscles were affected. The spasm was slow and irregularly recurring, and was accompanied by pain in the mastoid and occipital regions. It interfered greatly with sleep, the patient being kept awake two or three hours after lying down. Phonation also seemed to be occasionally interfered with.

The patient refusing to be cauterized, various internal remedies were prescribed, all without avail. Finally, he was admitted to the surgical wards, and likewise operated on by Dr. Agnew, who again removed a portion of the nerve. However, within a few hours after recovering from the effects of the ether, we might say almost immediately, the spasm recurred, and it persisted during the entire stay of the patient in the hospital, some three weeks. I should remark, however, that the spasm appeared to be less severe. Certainly, the movements were of less extent.

It is exceedingly probable that in this and the preceding case, other muscles than those supplied by the spinal accessory nerve were involved, and to this, in part at least, the failure is to be ascribed. However, I determined to give the method another trial in the following case, which seemed more favorable.

Case III.—M. K., female, aged fifty-five, a housekeeper, presented herself at the clinic with constantly recurring slow spasm of the right sterno-cleido-mastoid. This, she said, had existed for two years. At its commencement she had been in her usual good health. Examination showed the right sterno-mastoid to be markedly hypertrophied, further, that the right trapezius also participated, but to a slight extent. It could not be determined that any other muscle was, or had at any time been involved. The patient complained also of occipital pain, and pain along the dorsal spine.

After a rather prolonged trial of the cautery, galvanism, and various drugs, the knife was again appealed to, Dr. Ashhurst this time being the operator. Owing to the apparently limited character of the spasm, he was hopeful of a good result. Dr. Ashhurst performed an extremely thorough excision, removing over four inches of the nerve-trunk, including not only the supply to the sterno-mastoid, but also that to the trapezius. Previous to making the excision, the nerve was thoroughly stretched, very marked force being used. After the operation, and during the entire stay of the patient in the hospital, some twenty-one days, no spasm whatever was observed. However, that an inference as regards cure would have been altogether premature, was proved subsequently. The operation was performed on the 2d of June, and in the early part of the following August he received a letter from the patient, bitterly complaining that the spasm had returned, saying that it was " very bad again." Of the extent of the recurrence he could not speak with certainty.

Whether in these three cases the recurrence of the spasm was due to the spreading of the affection to other muscles, or to the vicarious nerve-supply of the sterno-mastoid and trapezius, is, he thinks, a matter of minor importance. The belief that these spasms are centric in origin, certainly gathers additional force, and the problem that presents itself is, whether the centres affected are spinal or cerebral, and how this differentiation shall be made. Secondly, whether a more radical surgical treatment should be invoked. These were the points to which he invited discussion.

He purposely refrained from speaking of the morphia treatment of these cases, inasmuch as its application to dispensary patients, who can be so loosely controlled, would be of more than doubtful propriety.

DR. C. K. MILLS added another to the list of unsuccessful cases treated by this operation. On the 6th of this month Dr. Roberts operated on a case which had been sent to Dr. Mills by Dr. Tomlinson, of Wilmington. Dr. Roberts cut down on the spinal accessory nerve of the affected side, and, after identifying the nerve by the faradic current, excised one and a-half to two inches. Two weeks after the operation the patient left the hospital, the spasm somewhat improved, but certainly not cured. Dr. Mills had had a number of these cases, and some years ago wrote a paper on the subject. Two of the cases reported recovered largely under the use of the actual cautery.

One of the questions which arises in a consideration of these cases is whether some of them are cortical in origin, due to lesion of the centres for conjugate deviation of the head. He believed that such a view should be entertained, but probably not for such a case as he had referred to, or for such as Dr. Dercum had reported. The persistence of the spasm and its tonic character in most cases are against the supposition of cortical lesion.

DR. DERCUM said that the point is, How are we to differentiate between the spinal and cerebral cases? If conjugate deviation of the eyes occurred with the movements, there would be reason for supposing that there was a cortical lesion. It is, however, rare to have this association. It was not present in his case.

As an indication of the utter hopelessness of operations on the muscles and nerves, it would be well to bear in mind that the movement is one in which a large number of muscles are involved. There are nine muscles, in addition to the sterno-mastoid and trapezius, which are called into play in the act of turning the head and neck, besides two others which act as rotators at times.

DR. J. H. LLOYD reported the following

CASE OF ALCOHOLIC MULTIPLE NEURITIS,

which presented such a characteristic clinical picture of a disease which is not common, that he was led to report it in spite of the fact that the pathological study was incomplete.

Margaret M., aged about thirty-five years, was admitted into the nervous ward of the Philadelphia Hospital in May, 1888. She had a family history of phthisis on the father's side. The patient had never been very strong, but had had no sickness except the ordinary diseases of childhood. She was married at seventeen and had had four children, the last labor occurring five years before admission. She denied syphilis, and presented no lesions of it. She also denied excessive drinking, but an inquiry of her friends revealed the habitual use of alcoholics, sometimes in excess, from her childhood. Four weeks before admission she awoke one morning to find both feet paretic. For one week preceding this she had had severe pains in both legs.

On examination the following facts were noticed: The patient was anæmic, very despondent in expression, and rather emaciated. She presented double wrist-drop, with wasting of the arm muscles, also more marked on the left side. There was paralysis, quite complete, of the anterior leg muscles on both sides, with consequent foot-drop and wasting. The extensor muscles of the left forearm contracted sluggishly to the faradic and galvanic currents, and showed the serial reactions of degeneration: A.C.C. >C.C.C. The extensor of the feet (muscles especially supplied by the anterior tibial nerves) also reacted sluggishly and showed reactions of degeneration to galvanism. The patellar reflexes were abolished. There were small areas of anæsthesia over the legs and feet. She would believe that a pin was touching the foot when it was touching the leg, and confused the legs. The sensory symptoms were very significant. She suffered from severe burning pain on the soles of the feet, and later in the palms of the hands, causing her to complain. The nerve trunks were acutely sensitive. Pressure over the popliteal and peroneal nerves of the main trunks of the arm was productive of most severe pain. The peroneal nerves especially were sore. The muscles of the leg were extremely sensitive to touch, and those of the calves especially were very tender.

The lungs were normal. The heart presented a faint systolic murmur at the apex ; not always heard, and possibly anæmic(?). The pulse was small in volume, very rapid, constantly ranging from 122 to 140, and easily compressible. Her temperature was constantly afebrile, except on two days, when it rose to $101\frac{4}{5}^{\circ}$. For more than half the time she was under observation (about four weeks) the temperature was rather subnormal, falling as low once as $96\frac{3}{5}^{\circ}$. It was taken in the axilla. The loss of appetite was complete, and the tongue was furred. She slept poorly and her mind wandered. At times she was in a well-marked delusional condition, and was restless always and complaining.

The patient, as she lay in bed, with the wrist-drop and foot-drop, bore a striking resemblance to the picture which Gowers, has in his recent work, of a patient with this disease.

The indications for treatment were especially to relieve pain and sustain the power of a failing heart. It was soon evident that the patient would not respond to treatment. The heart, in particular, never improved in its action. Digitalis was given in the form of tincture, infusion, powder, and poultices of the leaves, but the pulse remained rapid and feeble. A full liquid diet was administerred in oft-repeated quantities, but alcohol was not given until toward the last, and then in small doses. It is not necessary to detail all the treatment. Partial relief was obtained from the burning pains in the soles of the feet by lotions of carbolized water; and small cantharidal blisters were used with some benefit over some of the most painful nerves, as the peroneal. The patient gradually sank from a weak heart and died rather suddenly about the end of the fourth week of her sojourn in the hospital.

He regretted to say that the specimens from this case became decomposed, in some unaccountable way, and were unfit for microscopic study. The brain, cord, and affected nerves, clear to their ramifications in the muscles, were removed, and did not exhibit any marked change on gross examination. It was thought that some parts of the posterior columns were sclerosed; an observation which, he believed, has been made before in some of these cases. The history and observation of the case do not point to an original posterior sclerosis, and it may be a question whether such a change in the cord in a case like the one described could not be secondary to the original nerve lesions. It is also a question whether the obstinately feeble action of the heart in these cases may not be due to changes in the pneumogastric similar to those which occur in the other nerves. The heart was small and the valves normal. The liver was fatty and the kidneys slightly congested and enlarged.

DR. W. T. SHARPLESS reported a case of

EMBOLISM OF THE MIDDLE CEREBRAL ARTERY.—LEFT HEMIPLEGIA, ULCERATIVE ENDOCARDITIS, AND LATE GENERAL CONVULSIONS, AND SKIN INFARCTS.

M. D., aged thirty-one years, married (service of Dr. C. K. Mills, at the Philadelphia Hospital), had nothing special in family history. He had been very intemperate for several years, and there was a probable history of syphilis although he denied it. He had acute articular rheumatism last winter which was his only attack. In March last he was suddenly paralyzed on the left side and entered the hospital on April 18th. His condition Aug. 1st, when he came under Dr. Mills's care, was as follows: Left hemiplegia; paralysis marked on the arm, less so in the leg, and not at all in the face; œdema of feet and legs, extending to the knees. He had a very loud double aortic murmur that could be heard as far down the back as the sacrum. Area of cardiac dullness was very much enlarged, as was also that of the liver and spleen. The urine contained albumen in considerable quantity, and later hyaline, granular, and a few epithelial casts were found. In testing his sensations it was found that he could not perceive a light touch as well on the paralyzed side as on the other; but the sense of pain was preserved. Paralyzed muscles responded to the faradic current and showed no reactions of degeneration. His temperature rose to 101° to 102° every evening and regularly fell to normal in the morning.

On Sept. 6th he had two general epileptiform convulsions, and later he had another of a similar character.

On Sept. 15th petechial spots were noticed on his legs and ankles, which increased in size and depth until his death, which occurred Oct. 15th. These spots varied in size from that of a pin's head to a dime and showed no disposition to heal. They were probably skin infarcts.

Autopsy, October 15th: Right middle cerebral artery close to, but just beyond its orgin in the internal carotid, was closed by an evidently old embolus. All branches of the middle cerebral beyond this were thready and cord-like, and could be distinctly seen lying in a bed of yellowish softening, which extended over the island of Reil and the internal surface of all convolutions which cover in the Sylvian fissure. The cortex of the motor zone, except as just stated, was not softened. A cut through about the middle of the lenticular body revealed softening of the entire breadth of the internal capsule and adjoining lenticular and caudate bodies. This softening was in the middle part of the capsule and did not extend into the anterior and posterior third. Evidently some collateral circulation had been established between cortical branches of the middle and the anterior and posterior cerebral arteries. This was plain in several places where the narrowed and atrophic vessels beyond the embolus again took up their calibre.

The heart was enormously hypertrophied. On the tricuspid and aortic valves were the vegetations of recent ulcerative endocarditis, with a perforation on the septum leading from the left to the right ventricle, just below the aortic ring.

Probably the sequence of events in this case was as follows: First, the abuse of alcohol, and the syhilitic infection; then disease of the aortic valves resulting in their thickening and contraction; next, in March last, the embolus lodging in the middle cerebral artery and producing the hemiplegia, and recently the ulcerative process engrafted on the sclerotic valves.

DR. WILLIAM OSLER said that we are apt to think that the cortical cerebral arteries have not special anastomoses, but an examination of many of these cases of total occlusion, particularly of the middle cerebral artery, shows that a collateral circulation can be established, usually, as in this case, by the enlargement of the anterior cerebral, so that the nutrition is maintained in the central gyri.

TRANSACTIONS AMERICAN NEUROLOGICAL ASSOCIATION.

FOURTEENTH ANNUAL REPORT.

Continued from September Number.

The following paper was read :

NERVOUS AFFECTIONS FOLLOWING INJURY, "CONCUSSION OF THE SPINE," "RAILWAY SPINE" AND "RAILWAY BRAIN."

By PHILIP COOMBS KNAPP, M. D.

It is rather singular that the two most elaborate works on those affections of the nervous system which are supposed to follow injury should have been written by surgeons, and should have been based on the evidence of railway cases. One of these works, the influence of which is not yet dead, is based upon the ideas in regard to the pathology of the nervous system which obtained twenty years ago, and treats of all forms of injury of the central nervous system under the most misleading heading of "concussion of the spine;" the other, which reads like the work of a special pleader for the railway companies, discusses case after case of obscure nervous disease without mention of the condition of the reflexes. Fortunately, however, the attention of neurologists has of late been directed to the subject, and since the appearance of Page's first treatise in 1881, many valuable contributions to our knowledge have been made in this country and in Europe, and the work done in Germany, especially, has brought the matter more fully to our attention.

Erichsen's composite of "concussion of the spine" has been found to be compounded of too many distinct conditions to be trustworthy, and, from the vagueness of his classification, his ideas on prognosis proved misleading ; yet his work has had such an influence that the English railway companies are said to have paid eleven million dollars in damages in five years, and I have no question that it has also had an influence upon the great sums that have been paid in this country. The reaction, of course, followed, and it was aided by the cynicism that naturally arises when we see a man, who has claimed to be permanently injured, walk off as well as ever when the "damages" have been paid. It has seemed to me, however, that this reaction has gone too far, and therefore I have thought it worth while to go over the subject once more, and to review some of the recent work that has been done upon it. Before discussing controverted points, however, I will mention briefly certain definite lesions of the nervous system which may unquestionably follow injury.

Among the commoner results of injury are the affections of the peripheral nerves. The obstinate pain and persistent weakness of the shoulder which so often follow an injury are probably due to an implication of certain nerve fibres in the periarthritic process. Beside that, we often see various local paralyses, due to all sorts of lesions of the nerves, from simple pressure to severe crushing,—paralyses of all forms of intensity, from the transitory forms with normal electrical reactions to the severe atrophic forms with reaction of degeneration. The prognosis, of course, varies with the degree of injury, and is governed by the ordinary rules.

Trauma may produce certain definite lesions of the spinal cord and its coverings, beside the vague and questionable results of pure "concussion." It may, in the first place, cause fracture or dislocation of the vertebræ, and, secondarily, affect the cord itself. In these cases it not infrequently happens that the patient exhibits the symptoms of injury to the cord, while the injury to the vertebræ is noted only at the autopsy. With or without injury of the vertebræ, however, we may find serious injury to the cord, —hæmorrhage into the meninges or into the cord itself, rupture of the pia with hernia of the cord, or acute myelitis.[1]

In addition to the cases of what may be called "acute injury to the cord," where the symptoms develop immediately after the accident, it is a well-attested fact that chronic degenerative processes may be due to injury; and here, of course, the symptoms are very insidious in their onset. Spitzka[2] and Gowers[3] cite cases of tabes dorsalis due to injury, and Hoffmann[4] has just reported a very interesting case of tabes from Erb's clinique at Heidelberg due to a prolonged daily concussion of the whole body, especially the abdomen. Dana,[5] too, has cited a case where tabetic symptoms followed a railway injury to a syphilitic subject, where he thinks the accident determined the localization of the morbid process. Besides tabes, injury may produce lateral sclerosis, progressive muscular atrophy,[6] diffuse sclerosis,[7] and disseminated sclerosis,[8]—the last two affections especially being extremely difficult to diagnosticate in their early stages.

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[1] E. Leyden. Klinik der Rückenmarkskrankheiten, i. 371, ii. 61, 92, 139.

There is, or rather was, another lesion of the cord which was once deemed of great importance and was regarded by Erichsen[9] as the chief source of the symptoms of his "concussion of the spine,"—namely, spinal lepto-meningitis. We used to hear of it, but lately the cases have become rare, and, in fact, few now disagree with Strümpell's dictum,[10]—"A case of primary chronic lepto-meningitis, which can be surely and convincingly proven clinically and anatomically, does not exist."

Finally, in the brain and its coverings injury may produce various lesions,—fracture of the skull, meningeal and intra-cerebral hæmorrhage, pachymeningitis interna, hæmorrhagica, meningitis, softening, abscess, tumor, and various functional disorders, such as epilepsy, paralysis agitans, and chorea.[11] Furthermore, injury may give rise to various psychoses[12] and chronic degenerative processes, especially paretic dementia,[13] and of some of these, and of certain functional nervous affections, I will speak later.

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[2] E. C. Spitzka. The Chronic Inflammatory and Degenerative Affections of the Spinal Cord. Pepper's System of Medicine, v. 855.

[3] W. R. Gowers. Diseases of the Nervous System, i. 289.

[4] J. Hoffmann. Beitrag zur Ætiologie, Symptomatologie und Therapie der Tabes dorsalis. Archiv f. Psychiatrie u. Nervenkrankheiten, xix. 438, 1888.

[5] L. Dana. Nervous Syphilis following a Railroad Injury. The Post-Graduate, April, 1888.

[6] W. R. Gowers. *Op. cit.*, i. 450.

[7] W. R. Gowers. *Op. cit.*, i. 238.

[8] E. C. Spitzka. *Op. cit.*, p. 884.

[9] J. E. Erichsen. On Concussion of the Spine, p. 85.

[10] A. Strümpell. Lehrbuch der speciellen Pathologie und Therapie der inneren Krankheiten, ii. 1, 450, 4te Aufl.

Thus far all is clear and well defined. It would require an exhaustive treatise to speak fully of all these conditions and to dwell on their diagnosis and prognosis. They are met with more or less often, and usually they can be readily recognized. Besides these affections, however, there are other cases of a more obscure character, where our diagnosis is often doubtful and our prognosis sadly at fault.

Whether there is a true "concussion of the spinal cord" is still a matter of doubt. By this term I mean a paraplegia following injury, where the cord has sustained no coarse mechanical lesion, where "molecular changes in the finer nerve-elements have occurred, giving rise to an immediate and complete functional paralysis,"[14]—a condition analogous to the commoner concussion of the brain. Page[15] questions the possibility of such an affection, but cases have been reported which clinically answer the requirements.[16] The anatomical relations of the cord naturally render it difficult for true concussion to occur; and, moreover, in simple concussion there is apt to be recovery, so that post-mortem evidence is lacking. Cases have been reported,[17] however, where paraplegia came on suddenly after injury and terminated fatally, although no lesion could be found after death. Some of these cases are, of course, untrustworthy, as they were observed at a time when the methods of examining the cord were less exact, so that it is hard to exclude the existence of contusion or punctate hæmorrhages into the cord. Duménil and Petel,[18] however, still hold to a belief in commotion of the cord, which may be the origin of consecutive inflammatory lesions or sclerosis, and Dana[19] admits the existence, rarely, of true concussion. Some writers, Obersteiner[20] among them, hint at the existence of chronic concussion in men who are constantly exposed to jarring, as railway employes, but such cases are more likely to be classed among the degenerations of the cord, as in Hoffmann's case already cited.

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[11] Ch. Bataille. Traumatisme et Névropathie.

[12] Hartmann. Ueber Geistesstörungen nach Kopfverletzungen. Archiv f. Psychiatrie u. Nervenkrankheiten, xv. 98, 1884.

[13] R. v. Krafft-Ebing. Lehrbuch der Psychiatrie. i. 166.

[14] W. H. Erb. Diseases of the Spinal Cord. Ziemssen's Cyclopædia, xiii. 347.

[15] H. W. Page. Injuries of the Spine and Spinal Cord, p. 33.

[16] Wm. Hunt. Concussion of the Brain and Spinal Cord. Pepper's System of Medicine, v. 913.

[17] E. Leyden. *Op. cit.*, ii. 93.

Beside the true concussion there are a host of obscure affections which have been classed by Erichsen under the general head of "spinal concussion," and about which there has been much controversy. Dr. R. M. Hodges,[21] in a paper read before the Boston Society for Medical Improvement eight years ago, was one of the early dissenters from the views of Erichsen. He showed that a strain of the muscles or ligaments of the spine was capable of explaining many of the symptoms, and he believed that many of the cases were cases of functional nervous disease. Soon after Page[22] advocated the same views with somewhat more detail, ascribing the symptoms in cases of "railway spine" to a traumatic lumbago (that is, a strain) and a traumatic neurasthenia caused by the shock and terror of the accident.

Two years later Walton[23] found that in a number of cases of injury there was anæsthesia or hemianæsthesia, often involving the special senses, and, calling our attention[24] anew to the fact that many of the symptoms were cerebral, he suggested the term "railway brain," as more suitable than "railway spine." About the same time Putnam[25] reported similar cases, and they both suggested the relation between hemianæsthesia and hysteria. This theory has been further elaborated by Charcot,[26] who states that "these grave and obstinate nervous states, which are presented as the result of railway collisions, rendering their victims unable to return to their work or resume their ordinary occupations for periods of several months or even years, are often only hysteria, nothing but hysteria." Much of Charcot's last volume is devoted to the description of cases of traumatic hysteria, and a number of his pupils have published further studies upon the subject.

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[18] Duménil and Petel. Commotion de la moelle épinière. Archives de Neurologie, Jan., Mar., May, 1885.

[19] C. L. Dana. Concussion of the Spine and its Relation to Neurasthenia and Hysteria. Medical Record, 6th Dec., 1884.

[20] H. Obersteiner Ueber Erschütterung des Rückenmarks. Med Jahrbücher, p. 531, 1879.

[21] R. M. Hodges. So-called Concussion of the Spinal Cord. Boston Medical and Surgical Journal, 21st, 28th April, 1881.

[22] H. W. Page. *Op. cit.*, p. 116 *et. seq.* Also Boylston Prize Disertation for 1881.

[23] G. L. Walton. Two Cases of Hysteria. Archives of Medicine, Aug., 1883.

[24] G. L. Walton. Possible Cerebral Origin of the Symptoms usually classed under "Railway Spine." Boston Medical and Surgical Journal, 11th Oct., 1883.

Before all this, in 1880, Westphal[27] had reported three cases of "railway spine," and had advanced the theory that the symptoms were due to small foci of myelitis or encephalitis caused by trauma, and that they were analagous in their symptoms to multiple sclerosis. Since then Westphal's assistants, Thomsen and Oppenheim, have made[28] an elaborate study of sensory disturbances in all forms of nervous disease, including railway spine, and have shown that hemianæsthesia is not pathognomic of hysteria; and Oppenheim,[29] in a later paper, has carefully studied a second series of cases of "railway spine," with the result of substantiating Westphal's views.

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[25] J. J. Putnam. Recent Investigations into the Pathology of so-called Concussion of the Spine, etc. Boston Medical and Surgical Journal, 6th Sept, 1883. The Medico-legal Significance of Hemianæsthesia after Concussion Accidents. American Journal of Neurology and Psychiatry, Nov., 1884.

[26] J. M. Charcot. Leçons sur les maladies du système nerveux, iii. 251.

[27] C. Westphal. Einige Fälle von Erkrankung des Nervensystems nach Verletzung auf Eisenbahnen. Charité-Annalen, v. 379, 1878.

[28] R. Thomsen und H. Oppenheim. Ueber das Vorkommen und die Bedeutung der sensorischen Anästhesie bei Erkrankungen des centralen Nervensystems. Archiv f. Psychiatrie u. Nervenkrankheiten, xv. 559, 633. 1884.

[29] H. Oppenheim. Weitere Mittheilungen über die sich am Kopfverletzungen und Erschütterungen (in specie: Eisenbahnunfälle) anschliessenden Erkrankungen des Nervensystems. Archiv f. Psychiatrie u. Nervenkrankheiten, xvi. 743. 1885.

Before discussing these various theories I will cite, as briefly as possible, some cases of nervous affections following injury that I have seen in the last three years. I have not selected these cases in support of any theory, but I have picked out cases of different types, representing as fairly as possible the whole number of cases that I have seen. In only three cases was there any question of damages. One of these was a child, and another was seen after the award had been made, although a question of appeal was pending. Thus we can eliminate at the start two factors which have tended to obscure the subject and to bias opinion—the idea of simulation and the excitement that naturally attends litigation and is often a hindrance to recovery. This gives a more satisfactory basis to reason from, for, as the late Dr. Curtis said[30] in the discussion of Dr. Hodges' paper, "treatises based, like Erichsen's work" —and the same may be said of Page and Rigler—"upon the evidence of railway cases are certainly the last sources of information from which one may learn to make a correct diagnosis and prognosis, and to escape being deceived by the voluntary or involuntary exaggeration and simulation so commonly observed in plaintiffs seeking damages." The sub-heading of "railway" spine and brain is hardly appropriate, for railway accidents were not the cause of the trouble in the majority of cases, and none of them were victims of a great railway accident, like that at Roslindale. Of course a railway accident has no specific effect, except that in it are brought to play the most tremendous forces that we employ in our daily lives, and the terror and horror of any great railway catastrophe has a vastly greater psychical effect.

I give the cases as briefly as possible, omitting unessential symptoms. I regret that in some of them my investigations in the domain of the special senses have not been as thorough as I could have wished.

I. Jeremiah C., 37, m., railway employee, consulted me in March, 1887. A year and a half before he was knocked off a cable car, striking his back, and losing consciousness. On return of consciousness he went back to work, and kept at it for an hour or two, but afterwards he was laid up for seven weeks. Now he has pain in the back, especially on motion, with rigidity of the spine, and lumbar tenderness. His arms feel helpless; he has numbness and tingling in the hands and at times in the legs; the legs are not as strong, and he has had cramps in them. Occasional vertigo, and rush of blood to the head. Nervous, fretful, low-spirited, and poor memory. Some vesical tenesmus, and loss of sexual power, Some exaggeration of knee-jerks. Some improvement under faradism and the actual cautery.

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[30] Boston Medical and Surgical Journal, 5th Feb., 1880.

II. John D., 30, m., organ finisher, consulted me in February, 1888. Fell down stairs a month ago, striking small of back and buttocks. Great pain in the back. Diminished power in left leg. He cannot bend his spine, and has great tenderness in the lumbar region. He has a desire to empty his bladder most of the time, and, when he passes water, he thinks he is through before he really is, occasionally wetting himself. No sexual power since the accident. Quite nervous and rather alarmed as to his condition. Knee-jerks rather quickened, a tap setting up a general shrinking, as if from pain.

III. Martin H., 46, m., draw-tender. Referred to me at the Boston Dispensary, in August, 1886. Two years ago fell from a mast, thirty-six feet, striking back. Since then has had sharp pains in the back and abdomen, shooting down the legs. The legs are easily fatigued, feel numb and prickly, and as if a pad were between them and the floor. "Drawing" girdle sensation. Twisting or bending the spine, or riding in the cars, is painful. Faint spells and vertigo; severe headache at times. Nervous, low-spirited, and a poor sleeper. Short breath, palpitation, and a "drawing" feeling in the stomach. Poor appetite and digestion. Arms feel numb and fingers feel as if asleep. Diminished sensation in arms and legs, and some tenderness of nerve-trunks in legs. Lumbar spine flat, slight lateral curvature to the right, tender below tenth dorsal vertebra, the tenderness being greater by the sides of the spinous processes. Reflexes and electrical reactions normal. No ataxia.

IV. Bateman C., 59, m., electrician. January, 1886. Some months before he fell from a ladder, striking on his buttocks. No loss of consciousness. Nausea and vomiting till two months ago. Costive. Since accident loss of power and prickly throbbing in legs, worse in the right leg, which has wasted. Legs at times feel hot or cold, both subjectively and objectively. No distinct pain in legs. Water had to be drawn for a week after his fall. A month ago fell, rupturing a vessel in his knee, and the knee had to be aspirated. Right leg two inches smaller, marked diminution of sensibility. Muscles of thigh do not react to either current on right, and very feebly to strong galvanic current on left. Much fibrillary contraction of right quadriceps. Distinct gain under galvanism. With improvement in strength and sensation in legs has decided pain in them.

V. Jeremiah O'D., 50, m., carpenter. Consulted me in August, 1885, being anxious to get a pension on account of his disability. Was stabbed in the abdomen in 1865, and had peritonitis after it. In 1870 the pension board rejected his application, thinking his hemiparesis was the result of apoplexy, but he denies any history of apoplexy. On recovery from the peritonitis, the left leg began to be weak, and he had pain and stiffness in the left hip. He could not walk without staggering and getting exhausted. Hard to lift left leg up stairs. Severe pain in left side and abdomen, and left side of head. Depressed, poor memory, slight mental impairment, vertigo, and diplopia; some tinnitus. Numbness gradually developed over his entire left side, less marked in the hands and feet, but amounting in some places to absolute anæsthesia and analgesia. Tingling and prickling on left side. Smell impaired; poor vision in left eye from cataract, field not contracted; taste poor on left; hearing worse on left. Diminished tactile sensibility over entire left side; left arm a little smaller and weaker. Cannot put left leg into a chair without great effort. Sways with eyes shut. Knee-jerk and cremaster reflex most marked on right; knee-jerk weak. Speech rather indistinct. Slight tenderness over left posterior tibial. March, 1888: Question of pension still pending. Has not improved since 1885. Symptoms much the same. Still has anæsthesia, which is most marked on the left, although tactile sensibility is blunted on the right. Considerable difficulty in walking, drags left foot. Trouble in locomotion increased on trying to make any quick movement.

VI. Susan W., 46, m. December, 1886. Neurotic taint. Fell on ice last winter, striking left hip and elbow, and causing hernia. Now the slightest effort causes pain across the chest and in the back. Lifting causes a "hot water" feeling in the hernia. Very severe headache, impaired vision, and increase of deafness in left ear. Tinnitus. Short breath and pleuritic stitch. Weak stomach; very costive; frequent micturition. Considerable pain in the arms, numb feeling on left side; the left hand and foot get cold readily. Staggers on walking, and the left leg gives out. Cramps in the legs; numbness, prickling, and pain in the left leg. Great spinal tenderness; tender over stomach and lower ribs on each side. Tender over left ulnar, sciatic, and posterior tibial nerves. Diminished sensibility in left ulnar region, over left chest, and on outer side of left leg. Electrical reactions normal. Knee-jerks exaggerated, front-tap contraction. Eyes and ears not examined. March, 1888: Worse since last seen. Pain in left side and back; prickly feeling all over body. Much vertigo. Pain in right foot. Poor vision. Very nervous. At times has much trouble in passing water. Troubled greatly with leucorrhœa and piles. Field of vision good, *vou* 20–50. Fundus normal Cannot hear watch with either ear, or through bone. Marked opacity of membrana tympani. Cannot stand with eyes shut. Slight tremor of hands. Extreme spinal tenderness.

VII. Annie S., 45, m. Seen in consultation February, 1886, with Dr. E. S. Boland, who has reported the case in full.[31] Not neurotic. Two years before she was thrown down an embankment by the sudden starting of a train, and had recovered damages, although the case was still in dispute. Much pain in head and lumbar region, and considerable vertigo. Sleeps poorly and has a poor memory. Poor appetite; very costive; has had jaundice since the accident. At one time had xanthopsia, and another melanopsia. Menses irregular and painful. Urine scanty. Wets and soils herself at times. Very marked anæsthesia over whole body, with analgesia. Some sensation in tip of nose, left ulnar region, and right cheek. All muscular efforts slow and weak. Cannot stand without support. Muscles do not react well to faradism; knee-jerks weak. Field of vision contracted, especially in right eye; *vod*, can count fingers; *vos*, 2–20. Monocular diplopia, *od.* Pupils react sluggishly to light. Loss of smell and taste. Hearing to watch, contact *ad*, four inches, *as*. Gained under treatment for three months. Later right ankle became weak. When last heard from, December, 1887, she was still far from well, being quite lame, and having much pain in her back.

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[31] E. S. Boland. Symptoms following Injury to the Head and Back. Boston Med and Surg. Journal, 10th Nov., 1887.

VIII. Chas. L., 14, s., school-boy. Referred to me by Dr. Cutter, of Leominster, in May, 1886. Nervous heredity. Posthumous child, always nervous and irritable, had convulsions in infancy. Six years ago fell from a bridge, striking forehead. Signs of shock after it. "Shoulders drew up and spine got crooked." Delirious after fall, and very nervous since. Said to have lateral curvature, but it was not detected. Three years ago eyes began to trouble him, with dim vision and pain. Much headache, irritable, surly, and heedless. Poor appetite, chronic diarrhœa. Palpitation. Passes much urine. Rheumatic fever a year ago. Muscles weak, pain in legs with numbness and prickling in hands and arms. Fell again last fall, striking head. Worse since then, and has had two attacks: in one unconscious, rigid, trembled and screamed; sleepy after it. Two attacks of aphonia. Left leg said to be drawn up at times. Very fat. Field of vision normal, *vod* 20–20, *vos* 20–100, astigmatism of 4 D, *os*. Quite tender over spine, and more or less tender all over. Smell, taste, and hearing normal. Slight diminution of electrical sensibility on left. Knee-jerks only on re-enforcement. In June, 1888, reported to have had chorea, and after that to have had an increase of all his symptoms, with one or two more attacks.

IX. Chas. D., 12, s., school-boy. Mother consulted me in September, 1886, for an opinion in relation to a suit for damages. Not neurotic. Two years ago he fell down a coal-hole, striking head. Unconscious for a time, and delirious for several days. Scalp-wound, and question of depressed fracture. Headache and vertigo since; could not go up or down stairs safely, owing to vertigo. Violent headaches induced by any effort or excitement. Very forgetful, peevish, fretful, and sleeps poorly. Has had attacks of severe headache, with nausea, and "raving attacks," when he would call out various phrases referring to his accident, and twisted about, his limbs working. Exhausted after these. Sudden attacks of pallor and vertigo. Some diplopia and tinnitus. Capricious appetite; has not grown much or gained in flesh since accident. Slight furrow over right parietal, and cicatrix over occiput. Some spinal tenderness in upper dorsal region; jarring causes vertigo. Field of vision and tactile sensibility normal. Special senses unimpaired. No knee-jerks. Gradual improvement, but in December, 1887, was still subject to severe headache and vertigo on any exertion or excitement. No attacks since May, 1886.

X. Charles C., 61, w., machinist. June, 1887. No special taint. Eighteen months ago he was thrown from a wagon, and was unconscious for six' and a-half hours after it. No external injury or fracture. Hot water bottles were put to his feet, burning them so badly that he was kept in bed for four months. On getting about, his shoulders and right leg began to feel heavy and his arms ached. His head felt sore, and he has had sharp pains in it. Discouraged, low-spirited, and irritable, but mental power is not impaired. Short breath, poor appetite, constipation. Prickly aching and burning in the arms, which feel weak and heavy. Right leg feels numb and prickly, and both legs ache. He has pain and a hollow feeling in the back, which hinder his walking. Says he is growing worse. Marked myopic astigmatism, field of vision normal. Fibrillary tremor of tongue. It hurts him in the lower dorsal region to bend his spine, but it is not tender. Some inco-ordination of the left arm, and a little tremor of the hands. Speech is a little thick. Epigastric reflex present only on left. Triceps, radial, ulnar, and patellar reflexes exaggerated. Slight patellar clonus; front-tap contraction. March, 1888: Condition not improved. Complains greatly of his back, and of inability to use his arms well. Numbness of both legs. No inco-ordination of hand. Reflexes exaggerated. No consciousness of events immediately preceding accident. Was thrown from a carriage fifteen years ago, and after that had some stiffness of left arm, which recovered. A year before his last accident, however, this arm had been rather weak.

XI. Dennis B., 37, m., printer. Referred to me by Dr. Post, in August, 1885. In January, 1885, was struck by shafting, the right side being most injured. Right instep and right little finger broken; right thigh and leg much bruised. Laid up until May. Two weeks ago tried to go to work on a hot day, worked an hour and a-half, and had to go to bed. Memory began to fail after injury, and he has had constant severe headache ever since. Has vertigo so badly that his wife is afraid to let him go out alone. Forgetful since his injury. Very restless at night. Much more irritable and excitable. Considerable diplopia. Slight palpitation and shortness of breath. Poor appetite, some vomiting. At times has to wait before he can pass water, and at times the stream stops. Cannot close right hand as well. Frequent and severe pain in the right leg after using it, and constant numbness and prickling. Right leg weaker, somewhat wasted, and is easily fatigued. After his attempt to go to work, was in bed for a week; headache and vertigo much worse, felt dazed, and has been more or less confused; had constant nausea and vomiting for a week. Field of vision good, *vod* 20-20, *vos* 20-30, left disk paler. Some weakness of external recti, with nystagmus on excursion outwards. Other senses and tactile sensibility unimpaired. Arms strong, no inco-ordination, some tremor of hands. Right leg smaller than left, vastus internus does not react to faradism, lower leg muscles require a stronger current than on left. Nerve-trunks in right leg rather tender.

Reflexes normal. His symptoms gradually increased ; he grew steadily weaker, the mental deterioration became more marked, and about a year later he died. No autopsy.

XII. Wm. M., 49, m., engineer. Consulted me in March, 1887. Gonorrhœa. Considerable tobacco, little alcohol. Struck by a stone from a blast twenty years ago, breaking right forearm and thigh, and cutting radial (?) nerve in upper arm. Anchylosis of right elbow joint. Arm has been partly paralyzed since. Ever since accident has been nervous, "shattered." Sleep is very restless ; feels unstrung. Any excitement or any considerable exertion uses him up, and makes him put out of breath. Some "rheumatic" pain in legs. Cannot move right arm at shoulder much, or at elbow at all ; can flex and spread wrists and fingers, but cannot extend them; supinates a little, pronates well. Arm two or three inches smaller round; muscles wasted, do not react to faradism. Diminished sensibility in distribution of radial nerve. Sensation, motion, and reflexes elsewhere normal. March, 1888: Condition much the same. The pain in the legs is of rather recent date, and is of a rheumatic character. He is able to work, but since his injury he has been of a nervous, unstable disposition. Before it he could stand anything ; since, everything excites him very much, and makes him very nervous.

These last two cases are of further interest from the fact that Erichsen[32] claims that when injury produces fracduces fracture of any bone, the nervous system is apt to escape from the effects of concussion, the violence of the shock expending itself in producing the fracture. These cases, as well as a good many others that have been reported, show at least that Erichsen's rule is not without exceptions.

These cases certainly present divers groups of symptoms which demand a little consideration. The commonest among them point to some cerebral disturbance. Eight had headache or pain in the head, and eight had vertigo ; ten had some psychical disturbance, nervousness, restleness, irritability, inability to make prolonged effort, depression, anxiety, loss of memory, and, in at least one case (XI.), distinct mental impairment; two (VIII. and IX.) had some sort of convulsive seizure; one only (IX.) seemed to have been affected by any terror, and in him the effect was slight.

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[32] J. E. Erichsen. *Op. cit.*, p. 73.

Motor disturbances were not uncommon. Seven patients had muscular weakness, which sometimes amounted to actual paralysis, although chiefly when there was neuritis. Several had tremor; two (V. and VI.) Romberg's symptom; and one (X.) inco-ordination. Several had muscular wasting, and four diminished electrical excitability, chiefly from neuritis. The knee-jerks were increased in three cases, diminished in three, and absent in one.

Sensory disturbances were less common. In only four cases was there poor vision, due generally to definite causes independent of the injury. In three cases the other special senses were impaired, generally on one side. Three patients had diplopia, and one (XI,) nystagmus. One (VII.) had monocular diplopia, sluggish pupils, and xanthopsia; the last symptom was noted in one of Oppenheim's cases. Contracture of the field of vision was found but once, but in a few instances the fields were not examined. Anæsthesia in varying degrees was noted in seven cases, due, in two at least, to neuritis. In four of these seven cases, and in two others, there was paræsthesia. In no case, unless possibly in Case VIII. was the anæsthesia strictly unilateral—hemianæsthesia.

Digestive disturbances were occasionally seen, and in five cases there were vesical symptoms—signs of paresis of the bladder. Two men reported impotence.

Pain in the back was found in seven cases, and several others had pain in the side, limbs, or abdomen. The pain in the back was usually associated with tenderness over the spinal muscles and was increased on motion. In a few instances it was associated with tenderness over the spinous processes.

What is the cause of such an array of symptoms? Is there "only hysteria, nothing but hysteria?" Is there merely a strain of the muscles of the back, with neurasthenia added to it? Is there merely a functional derangement, or is there some structural change in the nervous system? Of course it is not possible to find any one diagnosis to fit so many different cases, but these cases and their attendant symptoms may furnish us with some data to aid in considering the whole subject of so-called "concussion of the spine."

Before discussing the question further it is essential to give some sort of a definition of what is meant by the two vague terms hysteria and neurasthenia. Neither of them can rightly be regarded as a disease. They are both conditions of the individual, the latter being well defined as "a bodily condition which is frequently associated with various chronic disorders, and not rarely coexisting with perverted functional activity of the nervous centres."[33] These states are often erroneously spoken of as if they were distinct diseases, and the names are often used as convenient "dumps" for cases where we can make no diagnosis. Hysteria is the state in which ideas can control the body and produce morbid changes in its functions,[34] while neurasthenia is a state of exhaustion from over-strain. The two states may be combined, or may complicate other affections. There is a hysterical symptom-complex that is so definite that it may fairly well be spoken of as a disease, and that is the group of symptoms that make up the *grande hystérie* of Charcot. To that alone I will apply the term hysteria; other conditions I will speak of as the hysterical or neurasthenic states.

That psychical disturbance can produce functional parralyses has been known for many years, and these paralyses have been discussed under many different names, such as "emotional paralysis,"[35] "paralysis dependent upon idea,"[36] or the "Schrecklähmung" of the Germans.[37] It was reserved for Charcot,[38] however, to give us the explanation of their origin. He has found that in certain hysterical patients at La Salpêtrière, who were easily hypnotizable, he could produce, by suggestion when hypnotized, paralyses precisely similar to those seen in other patients as the result of an injury. He therefore suggests that the mental state occasioned by the nervous shock of the accident is similar to the sommambulistic stage of hypnotism—there is an "obnubilation of the *ego*." The idea of injury occurring in this state of nervous shock or obnubilation, acts as a traumatic suggestion, producing the same results as ordinary suggestion in a hypnotized patient. The patient develops his own idea and suggests it.

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[33] H. C. Wood. Nervous Diseases and their Diagnosis, p. 18.

[34] P. J. Möbius. Unter den Begriff der Hysterie. Centralblatt f. Nervenheilkunde, 1st. Feb., 1888.

[35] R. B. Todd. Clinical Lectures on Paralysis.

[36] J. Russell Reynolds. Remarks on Paralysis and other Disorders of Motion and Sensation dependent on Idea. British Medical Journal, 6th Nov., 1869.

Charcot's brilliant reasoning proves beyond question the existence of a traumatic hysteria in his cases, but, after a careful study of these cases, and of others collected by Rendu,[39] Poupon,[40], Lyon,[41] and Berbez,[42] I am unable to trace any resemblance between them and the cases cited above, or the cases reported by German observers. Charcot's patients present either typical grand hysteria, with hemianæsthesia of the skin and organs of special sense, and with hystero-epileptic attacks with grand movements, passionate attitudes, and the *arc de cercle* ; hysterical monoplegia ; or hysterical articular neurosis. The characteristics of hysterical monoplegia are paralysis, with or without contracture ; anæsthesia of the paralyzed limb, not following any nerve tracts but having a sharp line of demarcation and encircling the limb like a bracelet ; little or no atrophy, and normal electrical reactions. The joint affection is that first described by Brodie, simulating severe organic disease, and attended with great pain, cutaneous hyperæsthesia, and tenderness and stiffness of the joint, the stiffness disappearing under ether. The diagnosis of these conditions is not difficult, and in the cases reported above they were not present. I have seen all three of the conditions,—grand hysteria, hysterical monoplegia, and articular neurosis ; but I have not yet seen these conditions appearing as the result of injury.

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[37] E. Leyden. *Op. cit.*, i. 173.

[38] J. M. Charcot. *Op. cit.*, iii. 355, 392 *et seq.*

[39] H. Rendu. Contribution a l'histoire des monoplégies partielles du membre supérieur, d'origine hystero-traumatique. Archives de Neurologie, Sept., 1887.

[40] H. Poupon. Paralysies hystero-traumatiques. L'Encéphale, Jan., 1886.

[41] G. Lyon. Note sur l'hysterie consécutive aux traumatismes graves. L'Encéphale, Jan., 1888.

[42] P. Berbez. Hystérie et Traumatisme. Paris, 1887.

The researches of Thomsen and Oppenheim have shown conclusively that hemianæsthesia is not pathognomonic of hysteria. Charcot formerly held [43] that general anæsthesia was rare in hysteria, that the anæsthesia was usually unilateral, the median line forming the limit. This claim has been abandoned, for many of his cases show anæsthesia of only one limb, and Berbez has found hemianæsthesia in only thirty-eight out of ninety-three cases. The commonest sympton in sensory anæsthesia—a point in which both French and Germans observers agree—is the peripheral limitation of the field of vision. Thomsen and Oppenheim have found sensory anæsthesia, which in many cases was unilateral, in epilepsy, hysteria, neurasthenia, nervousness, chorea, "railway-spine," multiple sclerosis, Westphal's pseudo-sclerosis, organic cerebral disease, certain psychoses, and other conditions ; peripheral limitation of the visual field being the most constant symptom. Furthermore, the investigation of the committee of the Société de Biologie, consisting of Charcot, Luys, and Dumontpallier, have shown [44] that in hemianæsthesia due to undoubted organic disease of the brain the phenomena of transfer can sometimes be excited by the application of metals. Thus it seems that not only is hemianæsthesia not pathognomonic of hysteria, but also that transfer phenomena in hemianæsthesia are not pathognomonic of hysteria.

Thomsen and Oppenheim oppose the hypothesis of hysteria as an explanation of their cases on other grounds. To their argument that the anæsthesia is fixed and unvarying, and that the disposition is not that of the hysterical, Charcot has replied by citing cases where the anæsthesia in hysterical patients has not varied for years. Oppenheim's further objections, however, seems to be more conclusive. He finds that the patients are anxious, hypochondriacal, and depressed ; complaining of headache, vertigo, faintness, and occasionally of epileptiform attacks. They have pain in the back, anæsthesia, and peripheral limitation of the field of vision ; their movements are slow and feeble ; and they try to guard their spines from jarring; there is often tremor ; the vesical functions may be disturbed ; the pulse is often rapid ; in one case Oppenheim found unequal pupils which did not react to light, in another optic atrophy. This latter symptom is not very rare; it has been noted, for example, in cases reported by Rigler,[45] Walton,[46] and Wharton Jones.[47] Not only are these symptom not hysterical, but they are not even functional ; and Oppenheim justly argues that if they be ranked as hysterical the firmest pillars of our knowledge are overturned. Westphal,[48] too, asserts that these cases cannot be brought under the rubic of hysteria,

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[43] J. M. Charcot. Leçons sur les localisations, etc., p. 115.

[44] Premier Rapport fait à la Société de Biologie sur la métallo-scopie et la métallothérapié du Dr. Burq. Gazette Médicale, 28th April, 1877.

Within a few months Oppenheim has reviewed the subject again, [49] and concludes that the cases with signs of undoubted organic disease, immobile pupils, optic atrophy, and vesical symptoms are in the minority, and that most cases represent, not a typical neurosis like hysteria or neurasthenia, but a more general and complex psychoneurosis, from which the patient never recovers.

In the cases reported above the leading symptoms were certainly not those of hysteria. The psychical conditions noted—depression, anxiety, loss of memory, mental impairment ; the tremor ; the exaggerated reflexes, and the swaying with closed eyes ; the pronounced paræsthesia ; the vertigo and headache (persistent headache being confessedly not a symptom of hysteria);[50] nystagmus ; vesical paresis—all these point to something beside hysteria or the hysterical state. Moreover, incontinence of urine, nystagmus, and exaggerated reflexes are symptoms which we should expect to find in organic rather than in functional disease. Case XI., especially, is strongly suggestive of disseminated sclerosis, and the fatal termination renders the diagnosis of organic disease assured.

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[45] J. Rigler. Ueber die Folgen der Verletzungen auf Eisenbahnen, insbesondere des Verletzungen des Rückenmarks. Berlin, 1879.

[46] G. L. Walton. Art. cit.. Boston Medical and Surgical Journal, 11th Oct., 1883.

[47] Quoted by Eichhorst. Handbook of Practical Medicine. Am. Trans., iii. 144.

[48] Archiv f. Psychiatrie u. Nervenkrankheiten, xvii. 282. 1886.

[49] H. Oppenheim. Wie sind die Erkrankungen des Nervensystems aufzufassen, welche sich nach Erschütterung des Rückenmarkes, insbesondere Eisenbahnunfällen, entwickeln? Berliner klinische Wochenschrift, No. 9, 27th Feb., 1888.

It seems to me that the theory of an organic lesion, possibly the lesions suggested by Westphal, is the one which is the most satisfactory in many of these cases. Bramwell, [51] who is certainly sceptical as to the frequency of organic change, suggests that there may be multiple capillary hæmorrhages in the brain and cord, which give rise to inflammatory processes, and finally to sclerosis. These hæmorrhages are so small as easily to escape notice, and later, if there be sclerosis, the recent methods of staining would be necessary to detect it. An interesting corroboration of this theory is afforded by the general lesions found in the bodies of some of those killed at the great railway accident at Charenton in 1881. In several cases Vibert[52] states that there were found very abundant punctuate hæmorrhages in the upper part of the body, and suggests that they arose from lesion of the nervous centres. Willigk[53] found in one case dilatation of the finest vessels, with infiltration into the perivascular spaces, and degeneration of the coats of the vessels. Page,[54] however, asserts that in these railway cases " Mors silet ;" which is as correct as many of his statements. Autopsies and experimental pathology furnish us with various facts which prove an organic change

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[50] J. M. Charcot. Leçons sur les maladies du système nerveux, iii. 268.

[51] B. Bramwell. Diseases of the Spinal Cord, 2d ed., p. 312.

[52] Ch. Vibert. Etude médico légale sur les blessures produites par les accidents de chemin de fer. Paris, 1888.

[53] A. Willigk. Anatomischer Befund nach Hirnerschütterung. Vierteljahrschrift f. die prakt. Heilkunde, cxxviii. 19, 1875.

[54] H. W. Page. *Op. cit.*, p. 82.

In regard to experiment, Mendel,[55] believing that hyperæmia was an important feature of the early changes in general paralysis, sought to excite an intense chronic hyperæmia in dogs. For this purpose he fastened the animals on a revolving table, with their heads toward the periphery. Rapid revolution, 125 to 130 a minute, continued for half an hour, produced punctate hæmorrhages. Slower revolutions (110) for six minutes a day, produced, after some weeks, symptoms of general paralysis, and, on killing the animals, he found adhesions between the skull, the meninges, and the brain, an increase in the nuclei and cells of the glia, an increase in the number of vessels, and changes in the ganglion cells. This condition finds a clinical representation in a case recently reported by Bernhardt,[56] where symptoms of general paralysis developed gradually after a railway injury. Fürstner[57] has repeated Mendel's experiments, with fewer revolutions (60 to 80) for a shorter time (1 to 2 minutes) and continued for months. He found double primary degeneration of the lateral columns and of a particular part of the posterior columns, changes in the optic nerves and changes in the brain similar to those found by Mendel. Similar changes in the lateral columns have been found after death, in patients who had suffered from "concussion," by Dumenil and Petel, and also by Edes,[58] who has called attention to the occurrence of symptoms of spastic paralysis in certain cases.

In undoubted organic disease, however, no matter what the case may be, there are often symptoms which are due to a superinduced hysterical or neurasthenic state, and these symptoms may so overlay the symptoms due to the structural changes as to render the diagnosis extremely difficult. I did not rehearse these symptoms; they were present in several cases where I believe there was also organic disease, and I have seen organic disease not due to injury so masked by hysterical or neurasthenic symptoms as to make the case a puzzle.[59] One symptom of the neurasthenic state is not very rare, and that is the hyperæsthesia over the spinous processes of the vertebræ.

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[55] E. Mendel. Ueber paralytischen Blodsinn bei Hunden. Ref. in Neurolog. Centralblatt, 15th May, 1884.

[56] M. Bernhardt. Beitrag zur Frage von der Beurtheilung der nach heftigen Körpererschütterungen, in specie Eisenbahnunfällen, auftretenden nervösen Störungen. Deutsche medicinische Wochenschrift, 29th March, 1888.

[57] Quoted by Hoffman, art. cit.

[58] R. T. Eddes. The somewhat frequent occurrence of Degeneration of the Posterior-lateral Columns of the Spinal Cord in so-called Spinal Concussion. Boston Medical and Surgical Journal, 21st Sept., 1882.

Apart from this mixture of functional and organic symptoms we must, however, recognize the fact that there are two great classes of cases which are the result of railway accidents and other injuries,[60] one where the symptoms are due, as I have endeavored to prove, to organic changes in the central nervous system; the other where the symptoms are due to functional disturbance.

As a subdivision of this latter class may be mentioned the more purely psychical disturbances, which are not rare. Naturally these are seen more markedly after railway accidents than after the accidents of ordinary life, such as falls. The horror of a scene like that at Roslindale is not soon forgotten by the ordinary spectator, and to one who has been an active participant, with the terror of sudden death or some fearful injury imminent, the effect must be still greater. Immediately after the accident at Charenton Vibert[61] noted a pronouced psychical change in almost all of the survivors, characterized by nervous excitability, insomnia, frightful dreams when sleep did come, tremor, headache, and a sort of semi-consciousness or cerebral automatism. This condition, he states, disappeared after a short time,—a few days or weeks,—but I believe that such disturbances are often of much longer duration; at any rate I have known the psychical shock of a railway accident to be apparent for a good many months afterwards. Moeli[62] has noted some of the more permanent psychical conditions. The patient becomes sensitive to sensory impressions, and they are irritable, anxious, and depressed. They are easily fatigued, weak, nervous, tearful and tremulous ; they are unstable and incapable of mental application, and all thought becomes difficult. They generally have headache, which is increased by mental effort or by slight amounts of alcohol. Moreover they are dominated by the thought of the accident, which is a constant source of oppression to them and haunts them night and day. Thomsen[63] has lately described a case under the name of "acute railway brain," where there was a slightly different symptom-complex. Immediately after the accident there were maniaca symptoms, with absolute and complete anæsthesia, confusion, and delusions of persecution. Latter the maniacal symptoms disappeared, the anæsthesia was less complete, but the man became lachrymose, hypochondriacal, depressed, irritable and unable to work on account of headache and weakness.

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[59] For a fuller discussion of this point see E. C. Seguin's article on Hysterical symptoms in Organic Nervous Affections, in his Opera Minora, p. 180.

[60] F. Kalliefe. Ueber Rückenmarkserschütterung nach Eisenbahnunfällen (Railway-spine), p. 27.

[61] C. Vibert. *Op. cit.*, pp. 11, 35.

[62] C. Moeli. Ueber psychische Störungen nach Eisenbahnunfällen. Berliner klinische Wochenschrift, 7th Feb., 1881.

Beside these psychical disorders there are other manifestions of functional nervous disease. I believe that hysteria (the "grand hysteria" of Charcot) is only rarely the result of injury, but there are a large class of cases which, after injury, suffer from symptoms which are a part of the neurasthenic state. After severe concussion, or the psychical trauma of injury, the victim is thrown into a pronounced neurasthenic state, which may last for years. His nervous system is utterly shattered, or, to use the phrase of the day, he is "all broken up." He is nervous, emotional, irritable, perhaps hysterical ; he is overcome by trifles ; he is exhausted by the slightest effort. He may present no objective symptoms, but he remains an utter wreck. There is a general weakening, and a decline from the normal standard in the functions of the central nervous system, especially in the domain of the thought, the will, and the emotions.[64] The symptoms may be milder, as in Case XII. Here, the patient finds himself changed : instead of being capable of continued exertion or strain, he is easily upset, trifles annoy him, he is irritable and quick tempered, he has lost the power of rebounding after pressure, of maintaing the calm, good tempered spirit which perhaps he had before ; his sleep is not sound, he starts when a door slams, his children annoy him, he is fretful and fault-finding. It may be that such a man is able to work as well as before, and to earn as much money, but, if he be in some responsible position, perhaps his judgment is less sure, or his bearing toward his associates less agreeable ; he is no longer a "good fellow," but nervous and disagreeable. These things, of course, are trifles, for which no jury would award damages ; but they are trifles which mark an enfeebled nervous system,and it is these very trifles which are like sand in the bearings of the carriage : they decide whether life is agreeable or disagreeable, and they are trifles which may continue for years ; in fact, the man may never recover his old buoyancy, his consideration for others, and his good nature.

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[63] R. Thomsen. Vorstellung eines Falles von acutem schweren Railway Brain. Verhandl. der Gesellschaft der Charité-Aerzte in Berlin. Berliner klinische Wochenschritt, 18th Aug., 1887.

[64] M. Bernhardt. *Art. cit.*

I have not spoken as yet of the subject of strain of the muscles of the back, upon which so much stress has been laid by Page. It is, I believe, the chief source of the marked rigidity of the spine so often seen, and by it or by "spinal irritation" is to be explained the spinal tenderness so often met with, for spinal tenderness has as little to do with disease of the cord as pain in the back has to do with renal disease. Although, however, it is often present, I believe that it usually exists as a complication and not as the sole cause of the symptoms. The first two cases reported are good examples of muscular strain, but in neither of them was that the only trouble. The first man had paræthesia in the arms, vertigo, psychical disturbances, and vesical symptoms, and the second had vesical disturbance and impotence. Page[65] seeks to ascribe incontinence and impotence to the strain alone, but I am unwilling to accept his conclusions. With strain of the muscles of the back there may be a little difficulty in emptying the bladder, but, when there is pain only on motion, I cannot see how the strain should paralyze the sphincters. In severe twists of the spine the nerves may be implicated, and it is possible that some of the nerves of the vesical plexus may be among them, yet that is by no means clear. Impotence is so common in all conditions of pain or weakness as to be of no significance. Strain of the muscles may often present such prominent symptoms as to mask the symptoms of nervous disturbance that lie beneath, yet I believe that careful inquiry will, in most cases of "concussion," reveal symptoms referable to the nervous system. As to Page's claim[66] that these cases do not recover owing to excessive doses of bromide of potassium, it is too much the argument of the railway attorney to merit consideration.

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[65] H. W. Page. *Op. cit.*, p. 182.

Of differential diagnosis I will say but little, because there is so much to say that it might easily fill a book; nor will I dwell upon the possibility of simulation, which has been so prominent in the minds of some writers as almost to conceal the fact that there was a real affection to be simulated. It is perhaps needless to add that all objective symptoms which indicate structural disease should be noted with care: the pupils and the optic nerve, the electrical reactions, the condition of the reflexes, the presence of tremor, especially fibrillary tremor of the face and tongue, ataxia, and Romberg's symptom. It should also be borne in mind that peripheral limitation of the field of vision, which is regarded by Thomsen and Oppenheim as one of the most constant attendants upon anæsthesia, and is considered by Charcot one of the chief stigmata of hysteria, may be the earliest recognizable manifestation of atrophy of the optic nerve.

In spite of the most rigid examination, however, there are many cases which furnish us no objective signs, notably the cases of purely psychical disturbance, and the conditions of neurasthenia, the purely "functional" affections. Here we can say only this, that if the patient's statements be true, he has suffered severe and perhaps incurable injury. If, in such cases, we had the opportunity of long-continued and constant observation, we would be in a better position to judge of the patient's veracity; but it may be as difficult to decide, and may require as long-continued study as it does in certain cases of insanity.

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[66] H. W. Page. On the Abuse of Bromide of Potassium in the Treatment of Traumatic Neurasthenia. Medical Times and Gazette, 4th April, 1885.

The prognosis in these cases varies, of course, with the character of the affection. Although meningeal hæmorrhage may be absorbed, other distinct structural affections of the brain or cord, of course, seldom recover. A noteworthy instance of recovery after paralysis of four years' duration is the case reported a number of years ago by Dr. Webber.[67] The prognosis of neuritis, which was present as a complication in several of the cases I have reported, depends, of course, upon the amount of injury to the nerve as shown by the electrical reactions. Strain of the muscles of the back, by itself, is an affair of long duration, and, as a complication, I have found it very persistent, lasting several years. Never having seen grand hysteria as a result of trauma, I am unable, from my own experience, to judge of the prognosis; but I see no reason why, when a person is once thrown into that state, from injury, he should recover, in the sense of getting out of the state, any quicker than one who gets into it from other causes, except that the absence of a hereditary nervous taint is in his favor. I suspect, however, that comparatively few cases of hysteria are induced from injury in persons who have not the taint. Charcot's cases are chiefly "hereditaires," but Berbez and Lyon are in doubt as to the preponderance of hereditary taint in the cases reported. Their conclusions are less decisive, however, as many persons, without heredity, may be of a neuropathic type. I find my own opinion supported by the recent testimony of Bataille: "It is apparent that predisposition is alone capable of making us comprehend the development of these attacks of hysteria." Of course, in hysterical cases the individual symptoms—anæsthesia, paralysis, contracture—may disappear, but the underlying state remains, capable of reproducing all the symptoms at short notice.

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[67] S. G. Webber. Recovery after four years' paralysis following railroad injury. Boston Medical and Surgical Journal, 18th July, 1872.

What has been said of the hysterical state holds true in a measure of the neurasthenic state. Where the symptoms are mild, and there is no neurotic taint, the patient's chances of recovery are of course better. Even in severer forms of neurasthenia the prognosis is not utterly bad, although, unfortunately, there are many cases that never recover, whether the neurasthenia be of traumatic origin or not. Nevertheless, in many cases, even though there is so great improvement that the patient can return to work and do as much as before, there are still the subtler changes of which I have spoken which show that the recovery is not absolute.

The prognosis of purely psychical disturbances is also grave. Hartmann has also called attention to the fact that psychoses may develop a long time after an injury, especially if it has caused headache, vertigo, irritability, and a loss of power of intellectual application, as was seen in Case IX. Krafft-Ebing[68] also shows that injury may make the brain the place of least resistance ; there is increased irritability, intolerance of heat or alcohol, and disturbance of vaso-motor innervation, which favors the devolopment of psychoses. Thomsen also regards the prognosis of his "acute railway brain" as grave.

In the majority of cases the symptom-complex is something like this : the patient has headache and vertigo ; he is depressed, irritable, and hypochondriacal, with a diminished power of application ; he may have some visual disturbance, he often has a contracted field of vision and occasionally optic atrophy ; there is some tremor and perhaps inco-ordination ; he has some anæsthesia, usually not limited to one half of the body, and with it numbness and pricking ; his movements are slow and weak ; his tendon reflexes are exaggerated ; there is often some lack of control over his bladder ; and he may have pain and stiffness in the back from muscular strain. Here I believe the condition is due to a disseminated miliary sclerosis, or, in the early stages, to a hæmorrhage or inflammatory process. The prognosis is like that of multiple sclerosis. With rest, freedom from excitement,—such as comes when litigation is over and the anxiety about money matters is settled,—and judicious treatment, the patient may improve. The same holds true of tabes dorsalis. The ultimate prognosis is bad. Oppenheim has never met with a recovery; Gowers[69] has found a good many cases where "damages" have not brought about a cure, and considers that where there has been a late or gradual onset of symptoms there is far less tendency to arrest or subsidence than with earlier lesions; and Strümpell,[70] in his latest edition, warns us against regarding these conditions as mild or insignificent. "The patients actually suffer greatly from them, and the suspicion of exaggeration and simulation is certainly much more rarely justified than it is pronounced."

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[68] R. v. Krafft-Ebing. *Op. cit.*, i. 166.

The following conclusions seem justifiable:

(1) Concussion of the spine, in the strict sense of the term, although probable, is still a matter of doubt.

(2) Muscular strain, spinal irritation, and peripheral neuritis are not uncommon complications.

(3) Injury may give rise, not only to gross mechanical lesions of the central nervous system, with symptoms coming on soon after the accident, but also to typical chronic degenerative processes of insidious onset.

(4) Injury may also give rise to various functional affections of the nervous system, including psychoses, hysteria, and neurasthenia.

(5) Hemianæsthesia is not pathognomic of hysteria, but is found in other conditions.

(6) Psychical disturbances—anxiety, hypochondriasis, depression, emotional disturbance, and lack of power of application—may exist alone or in conjunction with other affections.

(7) The neurasthenic state is often produced by injury, but true hysteria is rare.

(8) Both the hysterical and the neurasthenic states may be superimposed upon organic disease, obscuring the diagnosis.

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[69] W. R. Gowers. *Op. cit.*, i. 453.

[70] A. Strümpell. *Op. cit.*, ii. 1, 164.

(9) There is a fairly typical symptom-complex, with psychical disturbances, paræsthesia, anæsthesia, slow and feeble movements, exaggerated reflexes, etc., which is not uncommon, and is probably due to organic disease.

(10) The prognosis of these conditions is grave. Improvement is not uncommon, but complete recovery is rare.

DISCUSSION ON DR. KNAPP'S PAPER.

Dr. SEGUIN referred to the difficulty of diagnosis. As elements of error he mentioned uncertainty or unconscious deception by the patient who ascribed to his injury symptoms which had commenced before the accident. To these were to be added intentional deception and auto-suggestion. The more enlightened school of hypnotism has demonstrated the production of various symptoms of nerve disease in the healthy person. Bernheim and his pupils have shown that these symptoms might be produced not only by the suggestion of the operator, but by the suggestion of the patient himself, in whom a firm belief and a constant thinking may result as in the hypnotic state in an apparent loss of power, anæsthesia, spasm, etc.

In regard to the hysterical element, he referred to a paper written in 1875, in which he had shown the frequent coexistance of hysterical symptoms with organic disease of the brain and cord. This combination was not peculiar to traumatism, but was found also in morphinism and after moral shock.

Dr. L. C. GRAY also alluded to the influence of suggestion. The psychical lesion was so large as to make the majority of cases undiagnosible. So soon as a person was injured he was besieged by runners for legal firms. By means of these runners and the lawyers he was impressed with the danger to which he had been subjected. The suit then followed, running on for two or three years, during which time the patient could not afford to get well, as he would thus become liable to a suit for conspiracy. Finally, after having kept up the disease for the two or three or four years during the long trial, habit would prolong it for at least three or four years after.

In several cases he had advised the counsel to settle the case, and recovery had occurred in a few months.

It was a curious fact that patients suffering from organic injury usually did not have neurasthenia. The exaggerated manner of the neurasthenic contrasted curiously with the calmness of the man with serious organic injury.

The psychical results of injury would entitle the sufferer to some recompense, but of course not to so much as miliary hemorrhages or other wide-spread disease.

Dr. ZENNER referred to the fact of hysterical symptoms conjoined with organic disease, referring to a case of lead poisoning associated with tremor, which disappeared under hypnotism and after two or three applications was cured. Deception on the part of the patient should not, on the other hand, lead the exclusion of other serious disease. Pure malignering was a very rare occurrence. Deception n itself presupposed disease.

Dr. KNAPP recognized the tendency to auto-suggestion and the exaggeration of symptoms produced by these medico-legal trials, but neither sources of error were present in his cases, as suits for damages were not in progress, and all had been anxious to get well.

The following paper was read :

THE CORTICAL LOCALIZATION OF THE CUTANEOUS SENSATIONS.

By CHARLES L. DANA, A.M., M.D., New York.

I desire to present the evidence, so far as it is now attainable, regarding the question of the cortical localization of the cutaneous senses, viz., tactile, thermic and pathic.

It is well known there are now two very antagonistic views with regard to this subject. Ferrier contends that these centres are in the hippocampal gyrus and gyrus fornicatus. He bases this view upon experiments upon eleven monkeys, certain anatomical considerations, and the clinical fact that very often lesions of the convex motor cortex of the brain do not cause any sensory symptoms. His experiments are confirmed by those of Horsley and Schäfer. No positive pathological evidence is brought forward to support this view.

The other view is that the cutaneous sensory centres are more or less identified with the motor centres, being situated perhaps a little more posteriorly. Munk, Tripier, and Luciani and Sepilli, in particular, contradict the statements and experimental evidence of Ferrier. Luciani and Sepilli record experimental observations made upon ten dogs and four monkeys, besides pathological observations in forty-seven cases.

There is still a third view, viz., that both the motor cortex and the limbic lobe are concerned in the representation of cutaneous sensations.

Thus, in brief, the matter stands at present.

In pursuing an investigation of this kind, we make use of several kinds of evidence:

1. That obtained by clinical and pathological observation on man.

2. That obtained by experiment upon the lower animals.

3. That obtained by studying the development of special tracts in the embryo.

4. That obtained by the study of brains genetically imperfect.

5. That obtained by studies in comparative anatomy and physiology.

The atrophy method of Gudden cannot be directly applied to this question. Of the foregoing, it is the first class of evidence which is by far the most important and decisive, outweighing all the others. Perhaps the next most important is the experimental evidence obtained by observations on monkeys. At least it seems to be shown that the representations of motor areas in the brain of the *macacus* is closely identical with that in the brain of man.

My paper deals chiefly with pathological and clinical data, and these I shall present first.

I. A few cases of lesions of the brain cortex with sensibility disturbances were collected by Nothnagel in his work published in 1879. They were insufficient for him to form a conclusion as to localization. Exner in 1880 collected twenty-two cases, Starr in 1884 collected forty-one cases, Luciani and Sepilli in 1885 forty-seven cases, illustrating sensory localization, these latter including some of Starr's and Exner's. I have consulted the originals in Starr's list, and eliminated some in which the reports did not seem satisfactorily complete. Adding the remainder to Luciani and Sepilli, I found that I had eighty cases. Searching through the literature of the last four years, I have been able to find fifty-eight cases. To these I add four cases of my own, making a total of one hundred and forth-two.

These are all cases in which symptoms were observed during life,and autopsies were made, except in nine instances. In these latter there was injury of the skull and rupture of the middle meningeal artery. Trephining proved the nature of the lesion. Beside this, in the way of negative evidence, I have collected twenty cases of lesions of the gyrus fornicatus or hippocampus.

The cases have been tabulated in such a way as to show the location and nature of the lesion, the leading symptoms aside from the anæsthesia, and the kind and degree of the disturbance.

The numbers of the cases have then been marked upon charts of the brain cortex somewhat after the manner of Exner and Naunym; only, instead of dividing the brain surface into small squares, as was done by the observers mentioned, I have divided the chief convolutions into thirds and halves. In reading descriptions of post-mortem appearances, it seems to have become a custom to describe lesions as occupying certain thirds, as, for example, of the frontal, central, and temporal convolutions. Descriptions are rarely more minute than this. In reading over some of Exner's cases, one is a little astonished at the confidence with which he marks down a lesion in a minute square from the vaguest possible description. A good deal of scientific imagination must be used in dealing with the squares. The divisions I have made therefore are, I venture to think, more natural, less arbitrary, and better fit into ordinary descriptions of post-mortem findings. The results of my tabulation upon the brain charts are shown here. *Figs. 1 and 2.*

These charts, however, do not give a perfectly just idea of the sensory areas, for I have also marked down the areas involved in extensive or multiple lesions, for example, lesions involving both central and occipital convolutions. In these cases the lesion in the extra motor zones caused other symptoms, or, as shown by other cases, had nothing to do with the cutaneous sensory disturbances. It will be found that no lesion in the occipital, temporal, or anterior part of the frontal lobes caused cutaneous sensory troubles unless there was also some involvement of the motor areas. Even in the inferior parietal lobule there are but four lesions causing cutaneous sensory disturbances, and not extending into the motor regions.

The clinical and pathological evidence thus collected shows that the motor areas of the cortex contain also the representation of cutaneous sensations.

A study of the cases shows that the sensory centres for special parts of the body, i. e., face, arm, or leg, are in general identical with the motor centres for those parts, but are larger and more diffuse. It takes an extensive and, as it seems to me, a deep lesion to cause marked anæsthesia. For example, in the cases in which there was distinct and very marked anæsthesia, the pathological condition was:

Abscess, - - - - - - - -	2
Softening (from thrombosis or encephalitis),	27
Tumors, - - - - - - - -	7
Meningitis, - - - - - - -	3

In very few cases does one find a record of hæmorrhage; hence, the sudden appearance of anæsthesia with hemiplegia is rather a sign of necrotic or inflammatory softening than of rupture of vessels. Surface clots, however, do sometimes cause incomplete hemianæsthesia along with the paralysis.[1]

---

[1] Thus Wiesmann, in a collection of over two hundred cases of fracture of the skull with rupture of the middle meningeal, found incomplete hemianæsthesia in seven cases. I have noted two other cases, one of my own and one of Wilkes's.

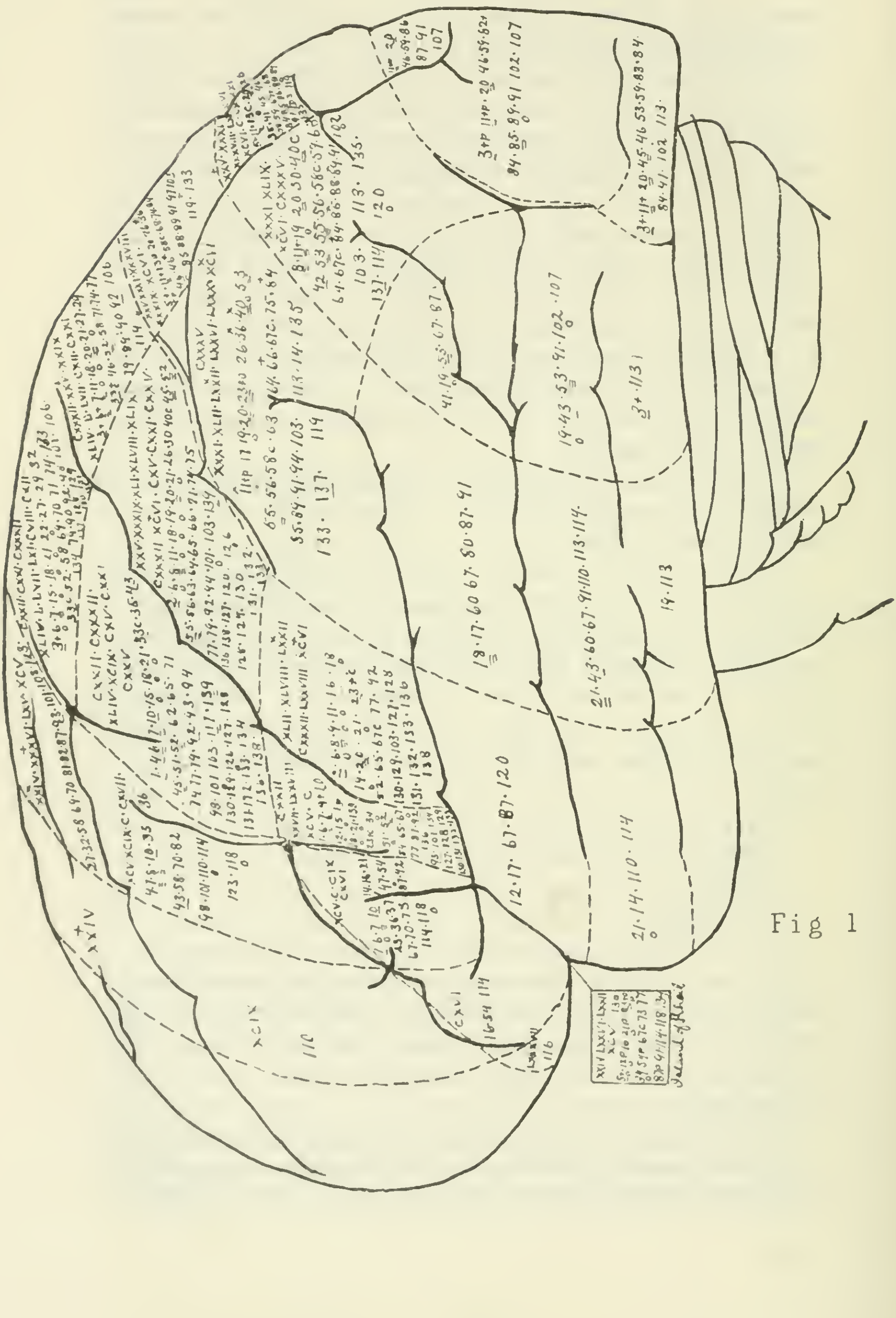

19·17·60·67·80·87·91
12·17·67·87·120
21·43·60·67·91·110·113·114
21·14·110·114
19·113
19·43·53·91·102·107
3+113
41·19·53·67·87
XCIX
110
116
CXVI
16·54 114
Island of Reil

Fig 1

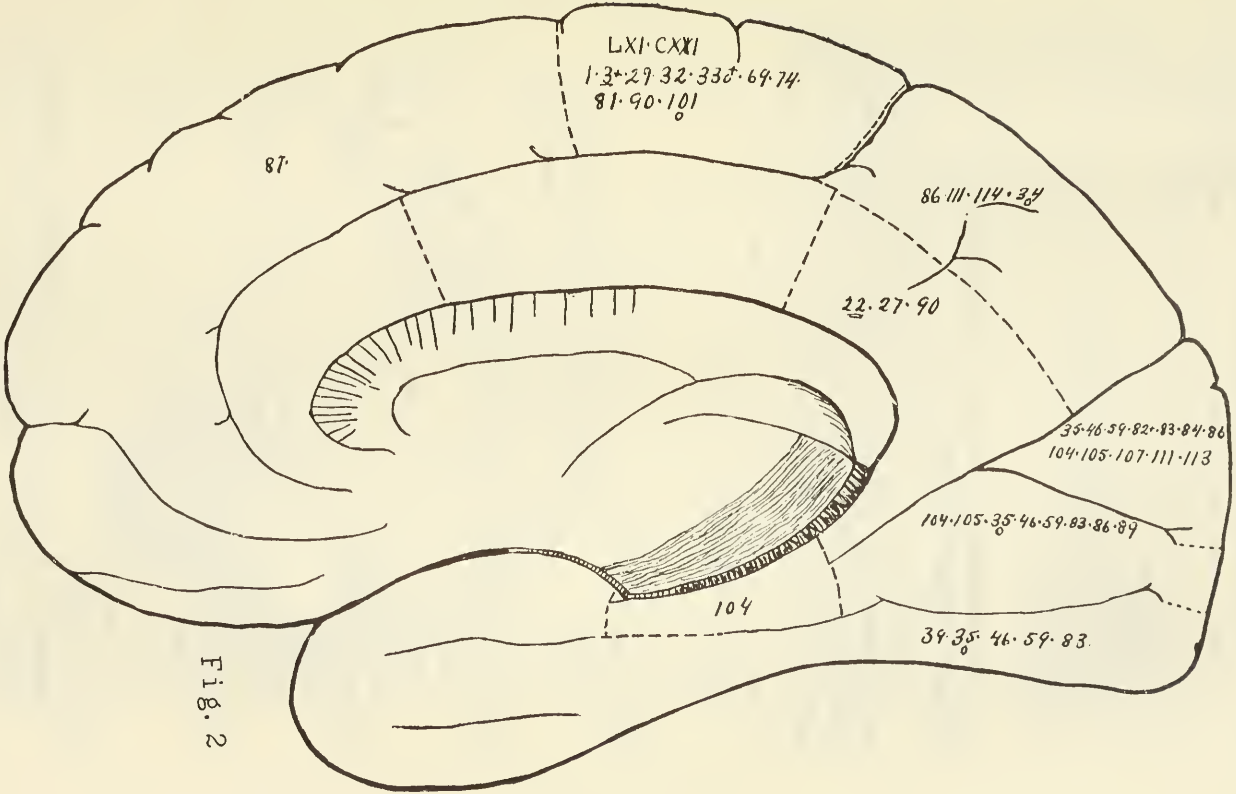

LXI·CXXI
1·3+·29·32·33♂·64·74·
81·90·101
87·
86·111·114·34
22·27·90
35·46·59·82+·83·84·86
104·105·107·111·113
104·105·35·46·59·83·86·89
104
34·35·46·59·83.

Fig. 2

In the cases of paræsthesia alone, the lesions were:

Tumors	16
Softening,	7
Meningo-encephalitis,	5
Meningitis,	1
Clot,	3

Thus it seems that the slowly developing lesions oftener produce only paræsthesia.

An analysis of the cases with reference to localization in the right or left hemisphere shows a preponderance of nearly two to one in favor of the left hemisphere, especially in the central convolutions, e. g.:

Frontal lobes, except paracentral lobule—	
Right hemisphere,	14
Left "	27
Not stated,	1
Ascending central—	
Right,	45
Left,	85
Not stated,	4
Superior parietal—	
Right,	17
Left,	12
Not stated,	4
Inferior parietal—	
Right,	17
Left,	25
Not stated,	3
Paracentral—	
Right,	4
Left,	6
Not stated,	2
Island of Reil—	
Right,	12
Left,	5
Not stated	2

In the island of Reil the lesions are much oftener on the right side.

The preponderance indicated may be due to a greater frequency of recorded lesions in the left hemisphere. The paræsthesiæ alone were caused by lesions about equally distributed in each hemisphere—r. 13, l. 12. The severer cases of anæsthesia were also due to lesions about equally distributed—r. 17, l. 14.

It may be stated here that the cases collected, so far as they go, confirm the view that the seat of muscular memories is in the inferior parietal lobule.

An analysis of the different cases of paræsthesia alone, of analgesia, and of tactile, thermic, and pathic anæsthesia, two or all forms being present in the cases, is shown on the accompanying charts.

FIG. A.

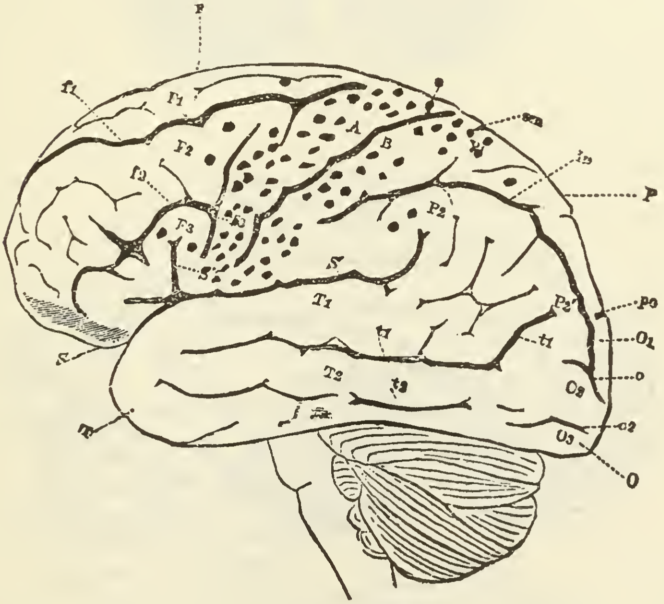
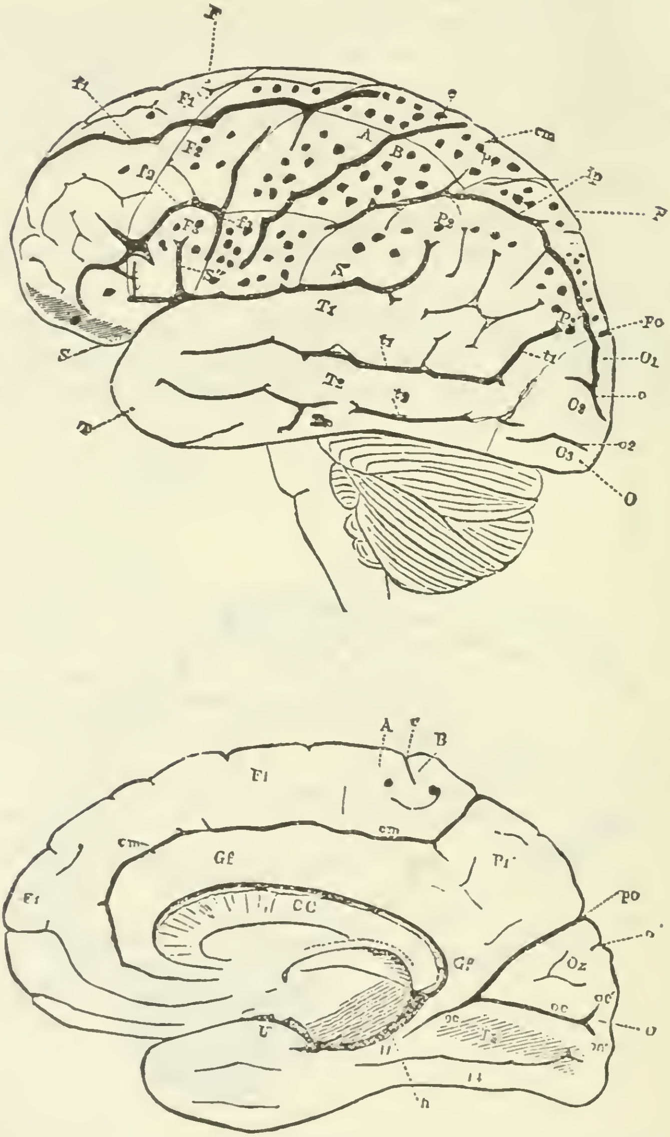


Diagram showing localization and number of lesions in cases in which there was analgesia.

FIGS. B, B1.


F
F1
F1
F2
f2
F3
f3
S''
S
A
B
c
cm
P1
ip
P
P2
T1
t1
T2
t2
T3
po
O1
o
O2
o2
O3
O
S
T
A
c
B
F1
cm
cm
Gf
F1
CC
P1
po
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Oz
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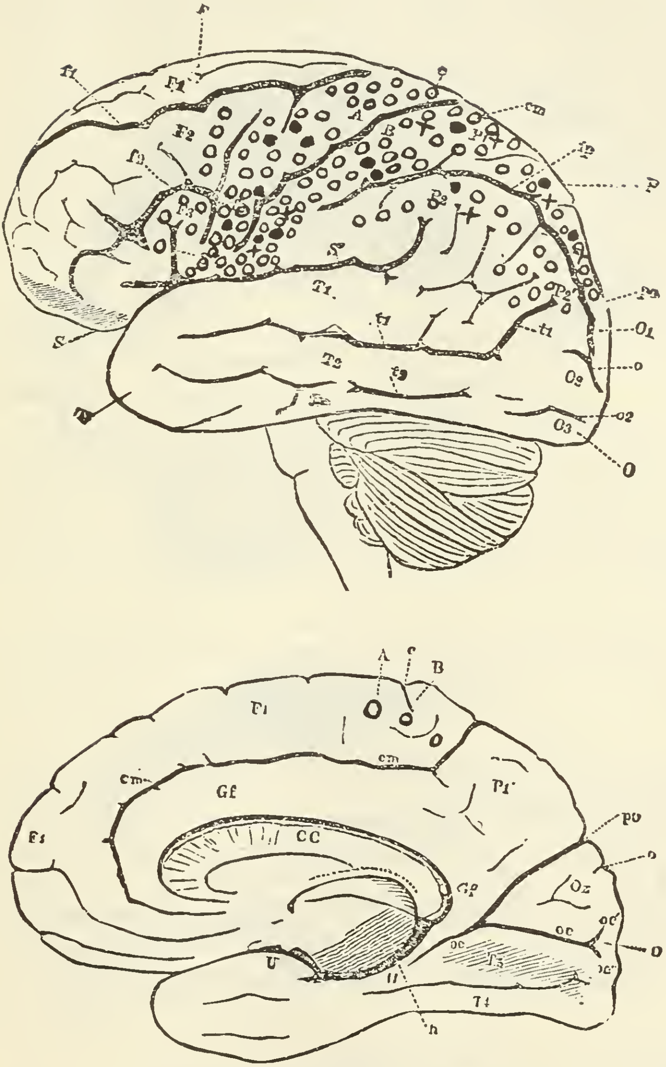
oc
o
U
h

Diagrams showing localization and number of lesions in which there was paræsthesia.

FIGS. C, C¹.


F
f1
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F2
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B
ip
P
F3
P2
S
T1
P2
t1
t1
O1
T2
t2
o
O2
o2
O3
O
A
c
B
F1
cm
cm
Gf
P1
F1
CC
po
o
Gp
Oz
oc
oc
O
U
oc
T4
h

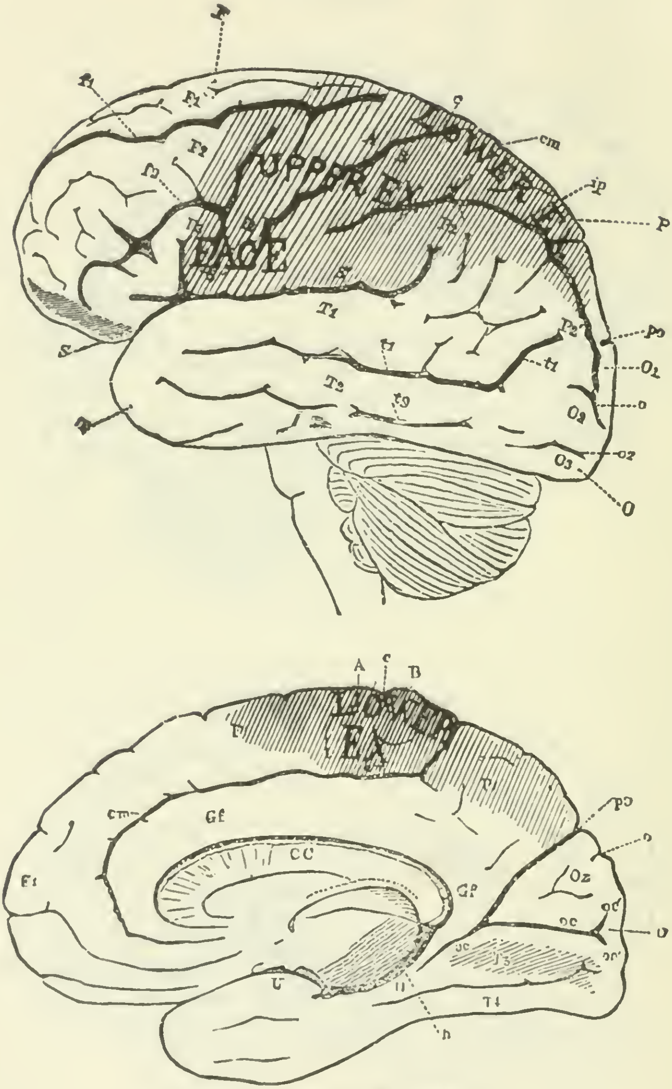
Diagrams showing number and localization of lesions :

● = tactile anæsthesia.

+ = tactile and thermic anæsthesia.

○ = combined tactile, thermic, and pathic anæsthesia, or tactile and pathic anæsthesia alone.

FIGS. D, D¹.


F
F1
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F3
f3
c
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UPPER EX
LOWER EX
FACE
S
T1
T2
t1
t2
P2
po
O1
o
O2
o2
O3
O
A
c
B
cm
Gf
CC
F1
Gp
Oz
oc
oc'
oc
T3
U
H
T4
h

Cortical areas for cutaneous sensations.

The tables and cuts do not show any especial difference in the localization of the three different sensations. If anything, tactile sensation seems to be more strictly confined to the motor areas. On the whole, the pathological histories show that tumors and slowly growing lesions in the motor areas, either in or just below the gray matter, produce paræsthesia and slighter degrees of anæsthesia. Softening, thrombotic or inflammatory, of the same areas coming on suddenly produces localized anæsthesia if it is extensive, and completely destroys the parts. Pressure clots from meningeal hemorrhage, if extensive and severe, will cause partial anæsthesia of the opposite side, as well as profound hemiplegia.

There is no known region of the cortex, lesion of which will cause anæsthesia without accompanying hemiplegia. The nearest approach to this is in lesions of the supramarginal lobule, where a slight degree of tactile anæsthesia without paralysis has been occasionally noted.

It is not possible for a cortical lesion to cause total unilateral anæsthesia without the lesion being so extensive as to be rapidly fatal. That there is a total crossing of the cutaneous sensory nerves, however, seems probable from the fact that the crossing is practically total in the cord, medulla, pons, and capsule.

Besides, lesions of the lower part of the central gyri have produced almost absolute anæsthesia as in Case I. of my own and Case III. of Petrina. It seems like an "absolute" cortical area for sensation.

From the foregoing we deduce further general laws, that *cutaneous anæsthesia of organic cortical origin is always limited to or more pronounced in certain parts, e. g., the face, or arm, or lower limb of the body, and it is generally incomplete.*

Total hemianæsthesia is either of functional or subcortical origin.

Cortical anæsthesia is always accompanied with some degree of paralysis.

II. *The experimental argument.*—It must be admitted that at present many facts obtained by experiments upon monkeys favor the idea that the limbic lobe is the seat of cutaneous sensations. We must remember, however, that Ferrier is alone in denying that anæsthesia is caused by removal of parts of the motor areas. Schiff, Luciani,[1] Tripier, Munk, all have noted sensibility disturbances after these lesions. The positive experiments made upon the limbic lobe are as yet few in number, and the operation is confessedly a very difficult one.

III. *The embryological evidence.*—The study of the development of sensory tracts in the embryo has so far failed to show that any but a very small part of the sensory tract turns in toward the limbic lobe. Most of the fibres pass up in the direction of the parietal lobe (including the postcentral convolution). Von Monakow has shown, by the atrophy method, a connection between the parietal cortex and a part of the sensory tract.

IV. *The evidence from teratology.*—Zuckerkandl[2] has collected two cases in which infants were born with absence of the olfactory lobe on one or both sides. In these cases there was decided lack of development of the cornu Ammonis; the middle and posterior horn of the lateral ventricle in one case was absent, and the hippocampus and gyrus fornicatus in both cases were small.

He refers to two other cases shown him by Kundrat, but I cannot find any details.[3]

V. *The anatomical and comparative anatomical evidence.*—Mr. Victor Horsley finds in the anatomy of the cortex an evidence of sensory function. At the discussion on cerebral localization before the Congress of American Physicians and Surgeons, September 19th, 1888, he said that, as the large pyramidal cells were undoubtedly motor, the smaller cells were probably sensory, and he showed the following diagram:

---

[1] Luciani and Sepilli. in ten dogs and four monkeys, found that extirpation of motor region caused an. and atax. Tactile sense much affected, muscular sense even more, pain and thermic sense less.

[2] Ueber das Riechencentre.

[3] Tiedman reported the case of a child born without olfactory nerves, Ammon's horn and fornix not developed perfectly; cited by Zuckerkandl, p. 107, op. cit.

Rudolphi, 1814, cited by Zuckerkandl, p. 107, op. cit., described the brain of child with absence on right side of I., II., III., IV., VI., cranial nerves. The corpus callosum was shorter on right side, the right lateral ventricle smaller, anterior horn short, descending and posterior absent, Ammon's horn very small, ditto fornix and corpora albicantia.

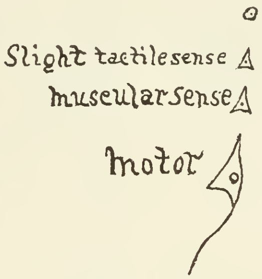


Diagram A.

Muscular sense, as I understand him, he takes to be compounded of (*a*) a sense of present movement or innervation, (*b*) a memory of past movement. It can only be the first, as it seems to me, which is in the motor areas.

The muscular memories, by which co-ordinated purposive movements result, seems to be, so far as is shown by the evidence of human pathology, in the inferior parietal lobule.

Finally, the evidence of comparative anatomy is quite against the view that the limbic lobe is the centre for tactile or pathic sensations.

The elaborate comparative anatomical studies of Broca some years ago and of Zuckerkandl (Ueber das Riechencentrum, F.Enke,Stuttgart, 1887) show that in anosmic animals like the dolphin this lobe is rudimentary. In animals with a highly developed olfactory nerve like the dog, this lobe is very large. Zuckerkandl gives the following table showing the relative development of the gyrus fornicatus:

GYRUS FORNICATUS.

ABSENT.	RUDIMENTARY.	PRESENT.
Monotemes.	Cetacea.	Ungulates.
Marsupials.	Primalis.	Carnivora.
Some families of chiroptera.	Some prosimians.	Insectivora.
		Man.

And Zuckerkandl concludes that the limbic lobe is the seat of olfactory sensation.

Objections.—In answer to the facts which I have brought forward, two objections will be raised: first, the large number of negative cases in which no sensory disturbance occurred despite extensive destruction of the motor tract; second, the positive experiments of Ferrier and of Horsley and Schäfer upon the limbic lobe in monkeys. To the first of these objections one cannot yet offer a perfectly satisfactory answer. To be sure, it is not true, as Ferrier states, that one negative case upsets the whole argument. This is not logic, or, if it is, it is a logic which works both ways. We would have to give up our cortical motor centres; for there still occur every now and then cases in which there is profound and extensive destruction of the motor areas without paralysis, as witness the cases recently reported by Byrom, Bramwell (*Intracranial Tumors*), and Gaskill (*Brit. Med. Jour.*, vol. I., 1888). On the other hand, in late years, with more careful observation, the cases of serious lesions of the motor areas without sensory disturbance are becoming more and more rare. Incomplete observation, late observation when compensation has occurred, small lesions, slowly developing lesions, are the factors which may explain the frequent absence of sensory disturbances.

As to the second point, a word may be said first regarding Ferrier's anatomical argument, viz. (loc. cit., p. 326), that there is a bundle of fibres in the outer third of the foot of the crus which does not degenerate downwards, which is connected probably with the sensory tract, and which bends round towards the hippocampus.

This statement has been disproved by the observations of Bechterew, who shows that this bundle does degenerate downward, and that it connects the frontal lobes with the pons nuclei fibres, passing thence to the contralateral cerebellum (*Archiv f. Psych.*, 1888). The experimental facts adduced by Ferrier are, however, most weighty. I am not prepared to deny that it is beyond possibility for the limbic lobe to be shown to be a centre for cutaneous sensations in man; but the facts of human pathology so far do not give it the slightest support. The few cases of lesion of the gyrus fornicatus collected by Exner showed it to be a latent region. I have been able to find other cases making in all twenty, in which the gyrus fornicatus or hippocampus was more or less involved. In none of these was any anæsthesia that could be fairly attributed to the lesion in the gyr. fornicatus observed.

LESIONS OF GYRUS FORNICATUS AND HIPPOCAMPUS.

1. Von Monakow, Arch. f. Psych., xi. Sarcoma of falx pressing on gyr. forn. No symptoms.
2. Von Monakow, Arch. f. Psysh., xiv., Hft. 3, 1883. Quoted by Luciani and Sepilli. Softening of lingual lobe and posterior part of hippocampus, 1st and 2d temp. cuneus. Blindness, hemiplegia, dementia.
3. A. McL. Hamilton, N. Y. Med. Jour., 1882, p. 575. Uncinate slightly, also hippocampus; sensory epilepsy.
4. Wildebrandt, Arch. f. Ophthal., 31, p. 119. Cuneus involving hippocampus and lingual (5th T.) slightly. Hemianopsia, slight hemianæsthesia.
5. J. A. Vorthnis, Brain, July, 1886. Upper 3d, central convol. Paracentral lobule extending into g. fornicatus. Hemiplegia and, later, hemianæsthesia in foot.
6. Mills, Phila. Med. Times, Jan. 18th, 1879. Tumor of right posterior ½, 1st of and 2d frontal, small segment of anterior gyrus forn. and corp. callosum. Spasm and mental disturbance.
7. Seguin. Tumor and cyst of paracentral lobule, part of first frontal, and involving part of gyrus fornicatus. No marked anæsthesia at any time. Jacksonian epilepsy.
8. Bristow, Brain, 1884, Oct., Case 2. Tumor corp. callosum involving 1st and 2 frontal postcentral and anterior ½ corp. callosum. No anæsthesia.
9. Reinhard, Arch. f. Psych., xviii., Case 13. Softening, fusiform, cuneus, convex occip. con., sup. parietal convolutions, ang. gyr., 2d temp. con. Blindness, motor aphasia, dementia.
10. Seguin, Arch. de Neurolog., 1886, No. 32, Case 45. Softening of right cuneus, 5th temporal, part of 4th, extending forward into part of hippocampus. Hemianopsia, slight ataxia.

11. Haab, 1882, quoted by Seguin, loc. cit., Case 28. Softening lower ½ cuneus, part of hippocampus and 5th temp. conv., extending into ventricle. Hemianopsia, temporary hemiplegia, no hemianæsthesia.
12. Huguenin, quoted by Seguin, Case 29, loc. cit. Tumor of right cuneus, extending forward and involving slightly the hippocampus. Hemianopsia.
13. Cassy, quoted by Luciani and Sepilli, op. cit., p. 324. Complete atrophy of cornu Ammonis and hippocampal gyrus. No anæsthesia. See also cases of atrophy of this region in epilepsy cited by Meynert, Snell, Hencks, Pfleger, Tamburini, etc.
14. Only general convulsive attacks. Involvement of first frontal præcent., and anterior ½ of gyr. forn. Charcot and Pitres (Rev. Mensuelle, 1878, p. 810, obs. xix).
15. No motor or sensory disturbance. Involvement of occipital convexity, cuneus, præcuneus. and posterior of gyr. forn. Boyer (Les. Cortic, p. 58, obs. 31).
16. General paralysis ; no anæsthesia or paraly sis. Lesion of frontal lobes and anterior 3d of gyr. forn. Baraduc (Bull. de la Soc. Anat., Marz, 1876, p. 277).
17. Tumor involving inferior and anterior surface of temp.-sphen. lobe and extending into hippocampus. Only slight visual disturbance ; right hemiplegia. Boyer (Les. Cortic., p. 48, obs. ix).
18. Claus, Der Irrenfreund, 1883, No. 6. L.—Softening (1) posterior ½ fusiform lobe or T. ; (2) spot of softening 3¼ ctm. long in frontal lobe, 5 ctm. behind anterior end gyrus fornicatus, in its white substance close under the cortex. Mental disturbances, aphasia, no paralysis or anæsthesia.
19. Bannister. This was a case reported to me verbally by Dr. Bannister. The patient was a lunatic who died with general cerebral symptoms, but without any general anasthesia. A very finely limited, cyst-like cavity was found occupying the lower part of the paracentral lobule and the adjacent part of the gyrus fornicatus. It was about 1½ inches in diameter.
20. M. Jastrowitz. Localization un Gehirn, Leipsig and Berlin, 1888, p. 49, Case VI. Tumor of right upper central convolutions, and lower anterior part of præcuneus and adjacent gyrus fornicatus. Hemiplegia of leg and arm with some loss of cutaneous sensation which, however, followed the gradual deveelopment of the paralysis.

Again, anæsthesia is not a symptom of tumors of the corpus callosum.

As to the theory that cutaneous sensations may be represented in both the motor areas and the limbic lobe, the case cited above by Jastrowitz (Case XX.) is instructive. Here there was a tumor with softening in the central convolutions and another tumor destroying part of the gyrus fornicatus. Yet the anæsthesia was not marked and it progressed with the paralysis. There was. however, decided muscular anæsthesia.

It is hard to conceive of a sensory mechanism so complicated and clumsy as that required by a hypothesis of compound sensory centres, one in the limbic lobe and one in the motor cortex; and no one has, I think, ventured to explain how much an anatomical arrangement could be put in harmony with the psychology of sensation. The mean reaction-time for tactile sensation is a little longer than that for sound, a little shorter than that for sight, (Wundt), e. g.

Sound,	0.167 sec.	or about	1-6 sec.
Touch,	0.213 “	“	1-5 “
Sighi,	0.222 “	“	1-5 “

The mean figures given by Hirsch, Hankel, Exner, Auerbach and Kries give the same relations. If the tactile senses were a compound mechanism involving the interaction of two centres, a longer reaction-time might be expected. As it is, since tactile sensations have a longer distance to travel on the peripheral nerves it may be fairly inferred that the actual cerebral portion of the tactile sensation reaction is shorter than that of sound or sight-sensation.

The objection which may be raised against a number of the cases quoted, that the lesion was not a purely cortical one, and that by pressure or extension, or involvement of the posterior capsular fibres anæsthesia was produced will not hold for a very large number of cases.

Owing to the immense typographical difficulties of printing an elaborate tabulated analysis of 134 cases I have omitted publishing it. I am certain, however, that *no amount of scrutiny can explain away the numerous cases in* *which superficial cortical lesions have caused monoplegias and monoanæsthesias.* I would refer as good examples of cases that illustrate anæsthesia from purely cortical lesions to Cases III. and VI. of Petrina, *Ztschr. f. Heilk, 1881*, Case I. of Luciani and Sepilli, Loc. cit. p. 302; case of Leyden, Beitrag. zur Lehre von der Loc. im Gehirn, 1888, p. 13. Some of Reinhardt's cases (loc. cit.) also furnish examples of superficial lesions causing anæsthesia and paralysis.

Finally, it has been plentifully shown that the anterior part of the frontal lobes, the occipital and the tempero-sphenoidal lobes, are latent regions so far as cutaneous sensory disturbances are concerned.

If now we can exclude the gyrus fornicatus and hippocampus, as I believe we can, nothing is left as centres for these sensations but the central and the parietal lobes. My cases certainly appear to show that it is not in the parietal, lobes, if we except the upper lobule and post-central gyrus which are motor areas. By exclusion, then, as well as by positive evidence, we must conclude that the sensory and the motor areas are identical.

Conlcusions.—The cortical areas for touch, pain, and probably temperature, are identical with the motor areas.

The representations for the different parts of the cutaneous surface are, in the main, identical with the corresponding motor segments.

The areas for the different segments overlap and diffuse into each other.

The brain cortex of the left hemisphere, except the island of Reil, shows lesions causing sensibility disturbances twice as often as the right.

The anæsthesia produced by lesions in these areas is partial, as a rule; it may be total for a limited area, but never total for the entire half of the body. The anæsthesia from cortical lesion is limited to or more pronounced in certain parts of the opposite side, such as the face, arm, or leg.

Slowly developing lesions like tumors are especially apt to cause paræsthesia or sensory auræ if there is epilepsy. Sudden or extensive softening causes the most marked and profound forms of anæsthesia. This softening need not necessarily be deep but is often so.

Pressure lesions like superficial clots can cause some anæsthesia, but always with pronounced hemiplegia. Superficial syphilitic and tubercular lesions may cause partial anæsthesia.

No case of pure lesion of the limbic lobe has yet been observed (and reported) in which anæsthesia was produced. This lobe, or a part of it, is probably the seat of olfactory sensation.

The comparatively slight involvement of sensibility in lesions of the motor cortex has yet to be satisfactorily explained. It may be that each body segment has a larger area of representation on the cortex for sensation than for motion ; or it may be that each segment is represented to some extent in both hemispheres, and hence compensation easily occurs. It is just possible that there is some sensory function in the limbic lobe.

The occurrence of slight sensibility disturbances with spasms and paralysis, points to lesions high up near or in the cortex.

Profound hemianæsthesia, with or without motor symptoms, points to a lesion lower down in the internal capsule or still nearer the cord.

Hemianæsthesia is always accompanied with some spasmodic or paralytic symptoms.

APPENDIX.

The following is a record of my own cases :

CASE *I.—Sudden right hemiplegia, motor aphasia, dysphagia, right hemianæsthesia very marked in face, intelligence dulled but present, early rigidity, hyperæsthesia on right side. Progressive weakness, bed-sores. Death in two months. Autopsy. Yellow softening; deep involvement of 2d and 3d left frontal and part of lower half of precentral convolutions, including cortex.*

A. G., female, age 70, native of Ireland, widow. No family or personal history obtained, except that the patient was quite well up to April 21st. On the morning of that day she suddenly became unconscious, and fell to the floor.

She was found to be paralyzed on the right side. She could not swallow. Her condition remained unchanged until admission next day.

On admission she was still but partially conscious. The right side of the face and right arm were partially paralyzed. There was some paralysis also in the leg. There was marked rigidity in the arm and leg. Sensation, motion, and reflexes were normal on the left side. *Sensation, tactile and pathic, was nearly or quite abolished on the right side of the face.* The mouth was drawn to the left side, and the right corner of the mouth was lower than the left. The right cheek was more flaccid than the left, and the right eye was not so closely shut as the other; it was suffused and congested.

Patient does not speak, but comprehends some remarks addressed to her. Her intellectual power is, however, notably impaired. She grasps anything with her left hand when told to do so, and, when asked to put out her tongue, attempts to pull it out with that hand. She continually puts her hand to the left side of her head, as if in pain. She cannot swallow, and grows very red in the face on making the attempt. Her eyes are turned to the left.

Patient frequently yawned. Left axilla more moist than the right.

Pulse slow, resp. normal, temp. 100.

Urine clear, normal in appearance, s. g. 1030, and contained no albumen.

Patient is a large and muscular woman. Her nutrition is good; her liver and spleen are normal. The signs of general bronchitis are present.

Ordered ol. tiglei gtt. ij. in the afternoon. During evening she had two passages.

Of milk and whiskey ℥ij. were introduced into patient's stomach by means of the stomach pump. Three nutritive enemata were given at intervals of four hours. The first one was not retained. Half hour before the second enema a suppository of tannic acid was given, and the remaining two were retained.

April 22d, 7.30 P.M.—Temp., right axilla, 100. Temp., left axilla, 97¾.

9.30 P.M.—Temp., right axilla, 100. Temp., left axilla, 95.

April 23d.—Patient about the same; recognized friends who called. Bowels constipated; retention of urine. Pulse 80, resp. 22.

Fed by enemata and stomach pump.

April 24th.—No change. Patient conscious.

" 25th.—Patient perspires on right side of face alone, and right arm. Conjugate deviation of eyes to left. Rigidity still present in right leg, but not in arm.

April 26th.—Pulse 75, resp. 24.

" 27th.—Patient smiles at remarks made, and sometimes cries. Still has aphemia; vomits occasionally.

April 28th.—No change.

" 29th.—Can move right leg a little.

" 30th.—Patient shows more intelligence, tries to speak. Swallows a little for first time.

May 1st.—Continues to swallow very well. Bowels opened with ox-gall enemas. Is more intelligent.

	RIGHT AXILLA.	LEFT AXILLA.
May 3d, 8 A.M.—	Temp. 99	Temp. 99
7.30 P.M.	" 98½	" 98
" 4th, A.M.	" 99	" 99
P.M.	" 99½	" 98
" 5th, A.M.	" 99½	" 96
P.M.	" 99	" 98½
" 6th, A.M.	" 99¼	" 95½
P.M.	" 99½	" 97
" 7th, A.M.	" 99	" 98
P.M.	" 100	" 99

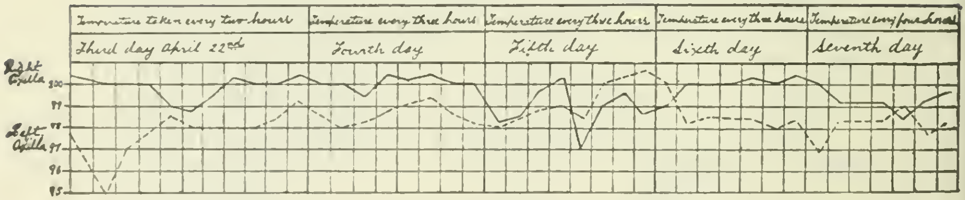
Pulse 80, resp. 18. Patient has pain in the right arm and leg, which she tries to move with her left hand.

	RIGHT AXILLA.	LEFT AXILLA.
May 7th, 12 P.M.—	Temp. 100	Temp. 98½
" 8th, A.M.	" 99	" 96
P.M.	" 99	" 96¼
12 P.M.	" 99½	" 96
" 9th, A.M.	" 99	" 98
P.M.	" 99	" 98¼

Patient still complains, by means of signs, of pain in her right arm and leg. She usually retains her food well, but vomited once.

May 10th.—Patient shows signs of more intelligence. She moves her right leg rather freely and her right arm a little. She has not yet spoken.

		RIGHT AXILLA.	LEFT AXILLA.
May 10th,	A.M.—	Temp. 98	Temp. 98½
	P.M.	" 99½	" 98
" 11th,	A.M.	" 98	" 98½
	P.M.	"	"
" 12th,	A.M.	" 98	" 98½
	P.M.	" 99½	" 99
" 13th,	A.M.	" 97	
	P.M.	" 99½	
" 14th,	A.M.	" 98	
	P.M.	" 99	
" 15th,		" 100	
" 16th,		" 99	



Patient keeps her right leg flexed all the time, unless it be extended by force. She moves it much more freely than her right arm. The leg is carefully but fully extended morning and evening. Patient eats with evident relish, and retains eggs, corn-starch, milk, beef-tea, etc. Bowels regular. Urine passed naturally.

May 17th.—Reflex less. The contracted muscles do not relax in sleep.

May 22d—Extension apparatus with 4½ lb. weight attached to contracted right leg.

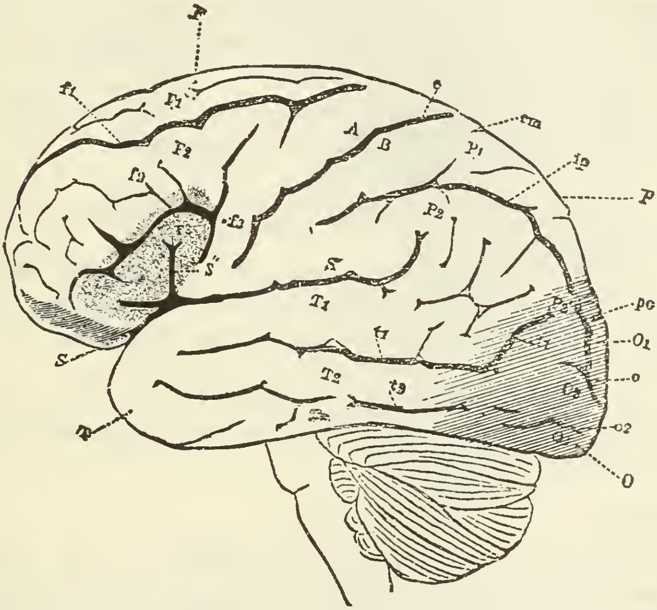
May 24th.—Vomited three times.

May 31st.—Temperature no longer taken. Extension still used. No vomiting. Bowels regular.

During June patient gradually became weaker. *Sensation continued absolutely abolished in the right half of the face* *and imperfect in the arm and leg. The left lower extremity continues to show rigidity, Vomiting occurs daily. Bed-sores developed on nates and sacrum.*

On June 16th she died.

Autopsy held twelve hours after death. Body somewhat emaciated.



Case I.—Thrombosis of branch of left middle cerebral.

Brain very œdematous, its tissue not firm. Over the 2d and 3d left frontal convolutions was seen a light brownish discoloration, somewhat depressed and soft. The left occipital convolution seemed softer to the touch than the middle. The arteries at the base were very atheromatous and patent. The cerebral arteries were followed up and no occlusion found. In the 3d left frontal convolution, extending into 2d and slightly into precentral, and into the island of Reil, was a yellow softening. The tissue was yellowish, of nearly the consistence of pus, which it resembled in gross appearance, but under the microscope showed only granular and fatty matter with many compound granular corpuscles. The softened space was of the size of a pigeon's egg, and was just in front and outside of the caudate nucleus, the outer portion of which was slightly involved. The cortex over the affected part was profoundly affected. Nearly the whole of left occipital lobe and part of the parietal were white and soft, but not broken at all, evidently a recent change. There was no evidence of a hæmorrhage there. Most of the parietal lobe, the basal ganglia, and the right side of the brain were normal ; also the cerebellum, pons, and medulla. Ventricles normal.

Heart.—Mitral valves very slightly thickened, and two to three patches of atheroma on endocardium.

Other organs normal.

Probable blocking of the 1st (and 2d) branches of the middle cerebral.

The points of interest here are the unilateral fever and hyperidrosis, dysphagia, motor aphasia, early rigidity without involvement of the ventricles, total anæsthesia of the face.

The occipital lesion was evidently a late one, and could not have had any relation with the anæsthesia which was present from the onset.

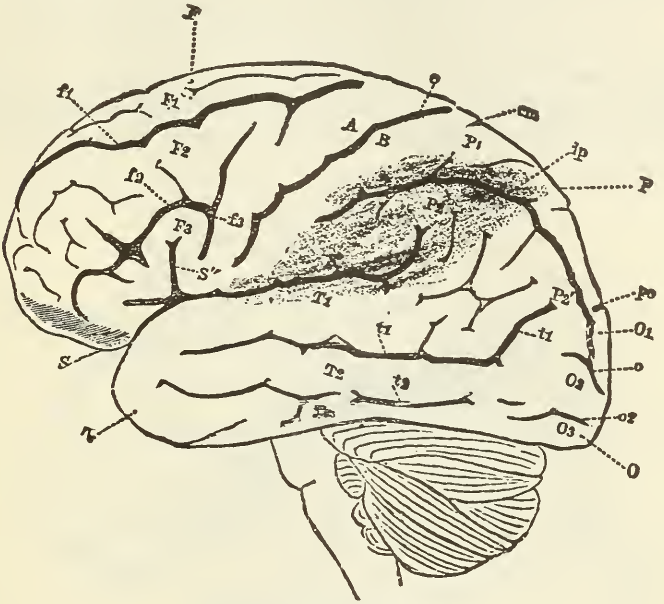
CASE II.—A. D., female, age 60.

The history of this case is very imperfect, as the patient when seen was entirely aphasic. I depend on the statement of friends, who assured me that five years before she had had a total left-sided paralysis, and that for some time she had no feeling in the right leg. From the positive statements made, and the fact that the symptom was remembered so long, it must be concluded that the sensory disturbance was very great.

She was brought to the hospital, February 22d, with right-sided hemiplegia and complete motor aphasia; no anæsthesia noted, intelligence being preserved. She died next day.

Autopsy : The brain showed a recent hæmorrhage in the left hemisphere and an extensive convolutional atrophy in the right hemisphere due to a lesion which must have involved the supramarginal gyrus, part of superior parietal, and part of middle third of posterior central, and part of first temporal.

There was a blocking up probably of the third branch of the Sylvian artery.



Case II.—Right hemisphere.

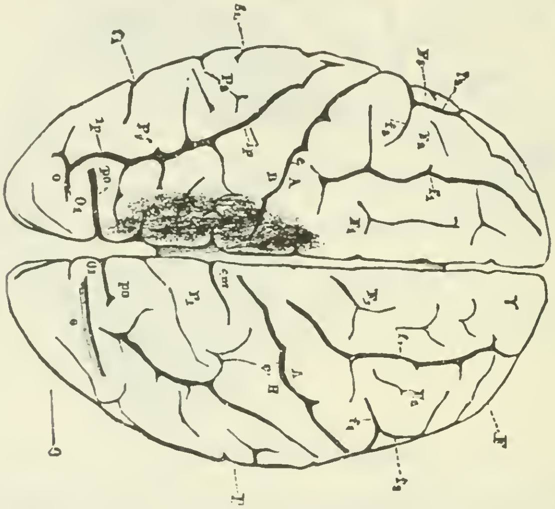
Case III.—The specimens in this case were shown, and the case reported briefly at a meeting of the N. Y. Neurological Society (*Medical Record*, March 15th, 1887, p. 280).

Pachymeningitis hæmorrhagica, with large meningeal hæmorrhage pressing chiefly on leg-centre—right hemiplegia, total paralysis in leg, aphasia, hemianæsthesia, convulsions limited to arm and face.

The patient was a woman about 68 years old, and came into the hospital with complete motor aphasia, and unable to give any previous history. She had no paralysis at first, but three days after admission she had a general convulsion, followed by right hemiplegia—total in the leg, and some right-sided analgesia. On the second and third days she had a series of brief localized convulsions, involving the face bilaterally and the right arm. The movements were clonic, beginning in the muscles of the lower jaw. The other peculiarities were these:

1. The pupils remained small during the convulsions.

2. The conjugate deviation of the eyes was at first, and very temporarily, toward the side of the lesion, and away from the paralyzed side. The head also was turned toward this side. When this occurs it is ordinarily spoken of as a paralytic deviation. This does not explain it here, since almost immediately the head and eyes were turned strongly to the opposite and paralyzed side.



CASE III.—The darkened area indicates where the severe compression was found.

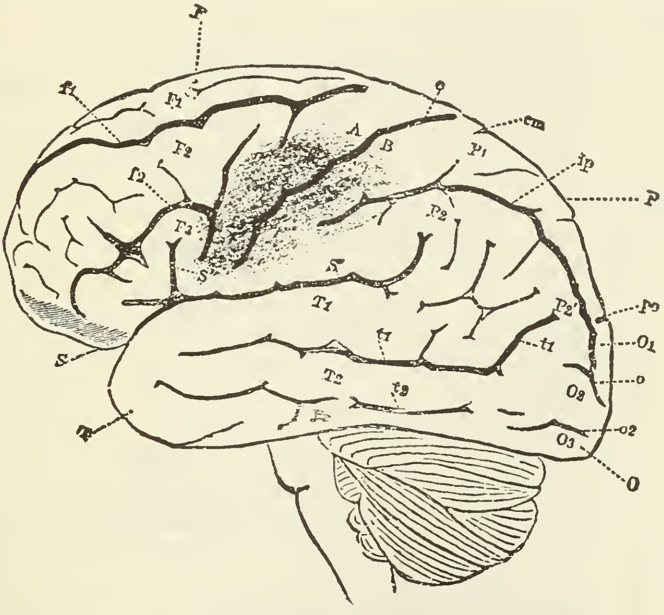
3. The temperature on the paralyzed side was one degree higher than normal, and higher by a degree than that of the other side. In meningeal hæmorrhages the temperature is often below normal, according to Minot.

4. The presence of hemianæsthesia.

The patient died on the third day. Post-mortem showed chronic pachymeningitis over both convexities, but more on the left side. On the left convexity there was a very extensive, fresh meningeal clot, pressing upon and flattening especially the upper half of the central convolutions and the superior parietal lobule. Brain substance normal.

CASE IV.—This case, taken from the Bellevue Hospital records, though imperfectly recorded, seems to be of some value.

A woman, aged 58, was admitted to the hospital with a history of having had a sudden attack of right hemiplegia. On admission she was entirely aphasic, but conscious. The face was but little paralyzed, but there was a total loss of motion and sensation in the right arm and leg.



CASE IV.

Autopsy showed a thrombosis of a branch of the left middle cerebral, involving the middle and part of the lower third of both central convolutions, and the white substance beneath, and the anterior part of the corpus striatum (caudate nucleus). The internal capsule was normal.

There was a probable blocking of the second branch of the Sylvian artery and perhaps of anterior central artery.

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The cases are arranged as numbered in the table. The first forty-seven cases are taken from Luciani and Sepelli's work, and are numbered in the order as given by those authors. These cases are given in abstract, and I have not gone over the original reports myself. They are analyzed in my tables, however.

Thirty-three cases are taken from the table prepared by Starr (*Jour. Nerv. and Men. Disease*, July, 1884, p. 401). All of these, as well as the other cases reported by that writer, I have verified. The remaining sixty-four cases are those collected by myself, including the three original cases reported by myself.

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DISCUSSION ON DR. DANA'S PAPER.

DR. C. K. MILLS considered the paper interesting and valuable, yet its inferences were not, he thought, sustained. He had himself recently gone over a large number of cases with the same object as Dr. Dana. It was probable that the cases gone over by both were at least in part the same. He had concluded that in the limbic lobe, the precuneus, and the postero-parietal region were probably situated the cortical areas for cutaneous sensation. In more than thirty out of the forty-one cases collected by Dr. Starr, the lesions involved the superior or inferior parietal lobule in addition to the central convolutions. Dr. Starr had quoted four of his own cases. In all of these he thought the lesion sufficiently deep to have involved the tracts going to or coming from the gyrus fornicatus, the procuneus, or the postero-parietal region. Lesions of association fibres between related sensory and motor areas would, he thought, account for some of the sensori-motor phenomena. The speaker referred to a case in which the fibroma, starting from the first and second frontal convolution, invaded the gyrus fornicatus and the corpus callosum. It did not involve the optic or trigeminal nerves, yet conjuntival anæsthesia and ulcerative keratitis were present. In a case of Loffler, referred to by Hitzig, where the skull was driven in about median line in the leg region, both legs were hyperæsthetic, probably from actual or inflammatory invasion of the gyrus fornicatus; Dr. Mills spoke of several cases of his own. To say the least, the question was still an open one ; for himself he believed that cortical sensory centres were distinct from cortical motor centres.

DR. M. A. STARR considered Dr. Dana's paper one of great value. All admitted that the location of the cortical sensory centers was an open question, hence very addition to to the facts was of exceeding value. Dr. Mills, in his remark that it was impossible to tell whether fibres were sensory or motor, did not recognize the fact that the most of the work done in tracing fibres was now done upon the brains of fœtuses where the distinction between the sensory and motor tracts could be readily made out by the time and direction of their developement. In the posterior part of the internal capsule, behind the pyramidal tract there was a tract known to be sensory, going to the parietal and the post central convolutions. The direction of this bundle of fibres was outward, not into the gyrus fornicatus or to the limbic lobe. These facts aided the view advanced by Dr. Dana, and not the view of Ferrier advanced by Dr. Mills.

In comparative anatomy, Zuckerkandl had on the contary connected the sense of smell with the gyrus fornicatus, which was small in animal, having but a slight sense of smell, though they might have a large general sensory surface ; and conversely large, where the sense of smell was pronounced. While many of the cases in his own collection had been unsatisfactory, there had been none among them which would support the theory of Ferrier, that the centre or general sensation is in the gyrus fornicatus. As to the case which Dr. Mills described as a lesion invading the hippocampus, the crus cerebri was so near that the invasion of the hippocampus without the involvement of the crus would be most difficult of determination. We have undoubtedly a sensory tract in the crus, presumably in the lemniscus. Ferrier in his last edition could mention only one instance in which in experiment upon the hippocampus the crus was not injured. All of these first experiments were performed too, before the adoption of antisepsis and for that reason are unreliable. As to the other instance mentioned by Dr. Mills, it was difficult to think that the trigeminal nerve was not involved, and the probability seemed in favor of a basilar affection involving it.

The signal symptoms of cortical epilepsy, numbness and tingling, showed that there must be a connection between the cortical motor regions and those of sensation, and the fact that the sensory disturbance usually preceded the motor, contraindicated the view that the sensory areas were distant from the motor.

Another fact had to be taken into consideration in interpreting lesions of the cortex. Each half of the body was represented in both sides of the brain so that lesion of one side of the brain would cause only partial anæsthesia with compensation by the other half for the action lost.

Dr. SEGUIN stated that the results of extirpation of the motor zone would support Schiff's theory of a sensori motor function. The peculiar attitude of the fore paw in Schiff's dogs which was dragged without actual paralysis was explainable only by a degree of anesthesia of the foot. The aura of localized epilepsy (which is often a sensory signal symptom) in brain tumor was another evidence in its favor. The speaker referred to a case of non-syphilitic, brain tumor cured by large doses of the potassium iodide. In this case the convulsion commenced in the hand of the left side and extended to the forearm, arm, face and leg; then, extended to the other side when consciousness was lost. These attacks always commenced with the sensation of wires "working" in forearm and hand. The attacks came at longer and longer intervals and were more restricted until only the arm was affected, and finally the attacks ceased. Long after the patient denied any *visible* attack, however, the sensation of wires boring into the hand would recur at long intervals.

Another argument was furnished by the results o operation. The speaker's case upon which Dr. Weir had operated supported the conclusions of Dr. Dana. Before the operation the man had had some anesthesia of the right hand and cheek. Since the operation this had been increased. It was not complete but was still very marked.

It was most marked for tactile sensation ; analgesia was slight, and the muscular sense normal. The anesthesia affected the right hand, cheek, the right half of the tongue and the inside of the mouth. The tumor removed was deeply situated and involving the caudal extremity of the second frontal and the edge of the precentral convolution. A space about the size of a silver dollar was excised. There was no lesion of the fundus, no petit mal. In addition to the anesthesia there was paresis of the arm and cheek.[1]

Dr. Dana acknowledged that with a special sense such as the tactile sense, it seemed more rational that there should be a special centre. He had started in his study of the subject strongly prejudiced in favor of the gyrus fornicatus. The facts of pathology were however opposed to the existence of a sensory centre in the limbic lobe. The speaker referred to the case of Exner in which the anterior half of the limbic lobe was softened without sensory symptoms. On the other hand the lesions of the cortex which gave rise to anæsthesia were not always deep. Luciani and Sepelli had reported several cases of simple superficial softening of the cortex in which anesthesia was present.

In regard to Dr. Starr's suggestion, he thought that it could not be borne out. The sensory fibres crossed in the cord, and almost complete anesthesia was produced by section on the internal capsule, medulla, or cord.

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[1] Cf. American Journal of the Medical Sciences, July, Aug. and Sept. 1888.

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Original Articles.

ON GOLD AS A STAINING AGENT FOR NERVE TISSUES.

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TWO methods of gold staining are in use for hardened tissues. One consists in bringing the section to be stained into a dilute solution of palladium chloride, where it remains for five minutes ; then into an acid solution of chloride of gold, where it remains for twenty-four hours. The myeline sheaths of the coarser fibres are stained violet.

The other method, introduced by Freud of Vienna, is used with tissues hardened in Müller's fluid. It consists in bringing the section to be stained into a one per cent. solution of chloride of gold, where it remains for from four to six hours, then successively into a twenty per cent. solution of caustic soda, and a ten per cent. solution of potassium iodide. In this last solution the section takes, in five or ten minutes, a reddish or violet color. In a successful specimen ganglion cells and axis cylinders are sharply stained, as are often also the myeline sheaths. According to some authorities the result is sometimes an axis cylinder, sometimes a myeline sheath stain. The stain results from a reduction of the gold salt to the form of an oxide, or to metallic gold. The defect of the method is its extreme unreliability, which renders it worthless for staining sections in series.

But reduction of gold chloride may be effected with great certainty by a number of reagents. They must, however, be employed with some care, in order to determine the formation of the purple or red oxide chiefly or entirely in the nervous elements of the tissues.

The following method, if faithfully carried out, will give sufficiently uniform results.

The piece of tissue from which sections are to be cut, is hardened in Müller's fluid for from two to five months ; it is then washed for a few minutes in water, is brought for a day or two into fifty per cent., then into ninety-five per cent. alcohol, where it should remain for two months or longer, until it has taken a greenish tinge. It is then imbedded in celloidin and the sections cut. The sections should remain in eighty per cent. alcohol for a time varying from a few days to several weeks, before staining.

The section to be stained is brought from water into a one per cent. aqueous solution of gold chloride, where it remains for from ten to thirty minutes. It is then washed superficially in water, brought for half a minute into a ten per cent. solution of sodium hydrate, washed again, and brought into the following solution, which is called the reducing fluid, where in a few moments it takes a vivid red color.

Sulphurous acid, - - - -	5 c.c.
Tincture of iodine 5 per cent., - -	gtt. v–x.
Solution of ferric chloride 37 per cent. (officinal),	gtt. i.

The section is then washed in water, and mounted in Canada balsam by the usual manipulation.

Sections, until they are brought into the reducing fluid, should be handled with platinum or some non-metallic substance, as an iron needle streaks the specimen.

The reducing fluid should always be made fresh just before using.

The stain which is taken by a specimen handled as above, varies with the time which has elapsed since the cutting of the section. If the section is stained at once, there is a more or less diffuse stain, which, if the tissues are not too thoroughly impregnated with the bichromate, differentiates in the course of a week or more after the specimen has been mounted on a slide. This probably takes place better in the dark. The axis cylinders, and ganglion cells and their processes, are sharply stained, and the myeline sheaths are somewhat stained. At the same time there is a good deal of color in the other structures, notably the connective tissue nuclei. After a day or two in the dilute alcohol, sections take a stain which is lighter, and almost entirely confined to ganglion cells, axis cylinders and myeline sheaths. Still later ganglion cells stain little or not at all ; after this the larger axis cylinders remain unstained, and a specimen is obtained in which only myeline sheaths, and the fine axis cylinders of the gray matter are stained ; sections of this kind closely resemble those stained by the Weigert method ; at last only myeline sheaths are capable of taking the coloring matter.

The sequence of events in the preceding method is as follows: The potassium bichromate of the Müller's fluid is reduced in the tissue to brown chromium dioxide, or to green chromic oxide, partly by the reducing action of the fresh tissue itself, partly by the alcohol into which it afterwards comes. This process is completed by the action of alcohol and light on the cut section. The oxides of chromium are displaced by the gold salt, which is then converted into the trihydroxide by the sodium hydrate, and into the red oxide by the reducing fluid.

Dilute alcohol acts on chrome salts not only as a reducing agent, but as a solvent ; to this fact is due the comparatively rapid change in the manner of reaction of the sections to the gold salt, when they are kept in eighty per cent. alcohol. To prevent this change, and at the same time obtain a better differentiation of the nerve elements, the following method should be employed:

The sections, hardened as above, are brought immediately after cutting into a mixture of absolute alcohol four parts and glycerine one part. In this solution in the dark the change described above takes place little more slowly.

They may then, if necessary, be washed out for a day or two in water before staining. They are then brought for a day or two into a one per cent. solution of nitric acid in ninety five per cent. alcohol; then from water into a one per cent. chloride of gold solution, to which has been added one per cent. of nitric acid; after remaining in this last for an hour or two, they are brought successively into the soda solution and the reducing fluid as before.

The stain attained is of a bright red color, axis cylinders and ganglion cells and their processes being very distinct, myeline sheaths and connective tissue nuclei lightly tinged. The color may turn to blue or purple on keeping. This is especially apt to be the case if the section is brought into an alkaline solution just before treatment with the gold salt.

The success of the above method depends almost wholly on the hardening of the tissue. Too long a stay in Müller's fluid makes necessary a more thorough removal of the bichromate by a long stay in alcohol; the time allowed varies somewhat with the temperature, which last should be rather under eighty degrees F. than over that point; the tissue should be moderately firm, and of a decided brown color, not blackish, when removed from the Müller's fluid; when this is the case, a clear nerve fibre stain may be obtained by the use of the nitric acid solution, even after a minimum stay in alcohol.

In hardening the tissue, the bichromate may be reduced more rapidly and completely by other means than by alcohol. Harden the tissue in Müller's fluid for two or three months, then bring for a few hours into sulphurous acid. The tissue soon takes a light green color; it should then come into fifty per cent., then into ninety-five per cent. alcohol, and should remain for a few weeks in the latter; it may then be imbedded, and sections cut and stained as above. Although more rapid, this manipulation will probably not be found as advantageous as the other one.

Sections stained by chloride of gold show the great preponderance in the gray matter of the spinal cord, especially in the cervical and lumbar enlargements, of naked over medullated nerve fibres. The larger axis cylinders are surrounded by rings of color, the most distinct one generally corresponding to the outer limit of the myeline sheath. The color in the axis cylinders and ganglion cells has the appearance of being due to a deposition of the metal in the tissue itself, as the cells are readily seen to be lying in their lymph spaces, and the nuclei and nucleoli are differentiated.

The black reaction in the Golgi stain, as shown by Rossbach and Sehrwald (Centralblatt f. die med. Wissenschaft, Juni, 1888), is due to the formation of bichromate of silver in the lymph spaces around the ganglion cells and axis cylinders, and gives the rounded outline of these spaces, not that of the cells.

In sections stained by the Weigert method there are often seen rings of color similar to those of the gold stain, which correspond to the clefts and outer surface of the myeline sheaths. This suggests the probability that the numerous smaller fibres in the gray matter, which are stained by the Weigert method, are naked axis cylinders, the color being due to a deposition of the mordant (chrome salt) around the fibre.

It might be interesting to note in connection with the mechanical theory of mordants, how much of the color in tissue staining is due to mordanting or coloring matter deposited in spaces left by nature, or by unequal contraction in hardening. Undoubted examples of space staining are seen in the demonstration of endothelial cells and of Ranvier's crosses by means of nitrate of silver, and of the corneal cells by chloride of gold. All of the myeline sheath stains, except perhaps Weigert's acid fuchsine stain, are open to this interpretation.

I have seen sections of spinal cord stained by the Weigert method, and imperfectly decolorized, in which the whole thickness of the peripheral horn-spongy tissue was occupied by a black network. This was evidently caused by coloring matter deposited in interstices due to the hardening process. The black network in liver stained by the Weigert method, seen by some observers, may be explained in the same way.

MUSCLE CONTROL.

By SARAH E. POST, M.D.,

NEW YORK.

MRS. WARD'S novel, "Robert Elsmere," touches upon many interesting psychological problems. Among these is that of muscle control by a foreign mind. I refer to the incident in which the heroine, blindfolded, takes her lover's hand and, without previous instruction, advances and kisses a statuette under the influence of his will. The problem here presented is startling from its suggestions of a spiritual entity or an unrecognized medium of force. The first impulse of the reader is to doubt the phenomenon. I would myself doubt it had I not personal knowledge of a similar incident. As a tribute to the veracity of the author of "Robert Elsmere," I venture to detail this incident, which occurred to me in the summer of 1881, while passing a few weeks in a boarding-house devoted to women. I was at the time convalescing from an illness which had been accompanied by great pain, and I was still very sensitive to light, sound, and, indeed, to all forms of irritation. Upon the evening in question there were twenty or thirty of us together in the parlor; and in the dearth of interest common to such occasions, one of the ladies had suggested a game in which, the subject being blindfolded, she was made to do something previously agreed upon by the company, the medium of communication being the operator's hands upon her shoulders. The proposition interested me from its assumption of power to communicate ideas by a method outside of my experience; and others failing to do so, I offered myself as the subject. I expressed myself as willing to do whatever would be required of me, thus narrowing the problem down to the simple communication of the requirement to my mind. I said, "If you can in any way make me know what I am to do, I will do it."

The game is not a new one, and is probably familiar to all.

Under the guidance of the hands on my back, I searched for lost articles, passed from one room to another, and performed a number of other feats.

So far as I had expected anything, I had expected a picture or presentment of what I was to do in such shape as to allow me to exercise my will and marshal my forces for its accomplishment, and I was prepared for considerable mental effort in grasping the idea which was to be projected into my mind. Nothing of the kind, however, happened. I was given no idea. While perfectly conscious, I proceeded blindly. I advanced very slowly, each step being taken in response to a simple loss of equilibrium. Apparently I first inclined in the direction, and then took the step to save myself from falling down. I sat, I knelt, I reached out my hand in response to similar impulses. With my mind fully awake but willingly passive, I was an automaton.

I am aware that similar exhibitions have been explained by conscious or unconscious suggestion conveyed through the hands of the operator, the weight of her body being the propelling power, and I would accept this explanation for my own case were it not for what followed. Finding me so docile, I suppose, it was determined to give me something more difficult to do. With the operator's hands upon my shoulders, I again successively felt the loss of equilibrium, and advanced as before for a number of steps, when I stood upright and then bent my head without any further forward impulse. While wondering a little at this loss of the feeling of instability, I commenced to experience a contraction in the muscle around my mouth. In a few seconds the puckering of the lips became very marked, so much so as to suggest kissing. Upon this I immediately said to myself, "If they require me to kiss any one, it will make a mistake, because that is something which I will not do." Instantly, however, I reproved myself for allowing my thoughts to wander, and said to myself, "I must forget this kissing, or I will not be able to receive the impulse and do what is required of me."

I did then succeed in putting the idea out of my mind, and resumed my previously passive, receptive condition, but no further inclination came to me. Although the operator's hands were still upon my shonlders, I had no further loss of equilibrium nor of impulse in any direction, and the experiment was then declared to have failed. It was then told to me that I had been required to kiss one of the young ladies. I had advanced to her side, but had made no further response.

It will be seen that my experiment resembled essentially that of our author.

In my experiment, as in hers, the mind of the subject had nothing to do with the result. The contraction of the orbicularis oris preceded the idea of kissing, and was the cause of its suggestion. The contraction of the orbicularis oris occurred while my mind was a blank. My experience, however, goes a step further than Mrs. Ward's. I was not led by the hand, and it was not a case of "Two minds with but a single thought, two hearts that beat as one." The operator was not a stranger to me, but neither was she a personal friend. There was also no direct contact between the operator and myself, as would have been the case in a clasping of hands. Quite a number of thicknesses of clothing intervened between us.

Further, Mrs. Ward represents her subject as in an unconscious or somnambulistic state during the experiment. When it is over, she sighs, passes her hand over her eyes, and seems not to know where she is. My experiment was, on the contrary, performed with the subject fully conscious, proving that hypnotism is not essential to the result.

Truth is again stranger than fiction. The control of a circular muscle like the orbicularis is analogous to the control of the heart, said to be possible in some cases. Both would appear to be in the line of proof that, under certain conditions, the muscles of one body may be controlled by the mind belonging to another body, its own mind being passive in the matter. I should not be surprised, too, to learn that such substitution is not rare. It seems to me that the mind of the well substitutes the mind of the sick again and again in the practice of nurses and physicians. I can recall two cases in Bellevue Hospital—cases of otherwise incoercible vomiting which were apparently controlled by such substitution. One of these was a case of pneumonia with a very high temperature during several successive days, and absolute rejection of stimulants, medicine, and food. Her condition was desperate. I remained with her for a night, and the tendency to vomit seemed to be inhibited by my keeping myself very much alert to the necessities of the case. Personal contact seemed to assist in the result. If I left her bedside for five minutes the vomiting recommenced, while holding her hand appeared to strengthen my influence. In this treatment I had at that time no theory as my guide, but simply followed an instinct in the matter. The patient was too much reduced for self-control. I did not address myself to her at all, but simply tried with all my might to do for her that which she was unable to do for herself.

In another case of incoercible vomiting, I recall the same attitude of mind. The forlorn patient was not appealed to, but as I left for the night I said to the nurse, "If that woman vomits, your reputation will be nothing with me." I was the head nurse of the ward at the time and had some dictatorial power. The woman did not vomit and made a good recovery. Both of these cases had been declared hopeless by the physicians—visiting and house; and the nurse had been left entirely without directions.

It will be seen that the experiments with hypnotism which excite so much remark belong to a somewhat different order from these which I have in mind. The hypnotized subject acts under the influence of suggestions. Mr. Croffut, a celebrated operator in hypnotism, in a recent number of the *North American Review*, remarks that he has never obtained compliance without suggestion, that is, without the spoken word, having the subject face to face, or in some other way presenting the matter before him. The subject is instigated to rob a house. The plan is first unfolded to him, and then he proceeds with intention. The operator cuts off certain faculties, but employs others. To cut off the whole brain, or rather the whole intelligence, as in my case, simplifies the experiment. My blundering interference with complete success, is but an additional evidence of the divorcing in the experiment of the mind from the body—of that which controls from that which is controlled. In the preceding experiments my mind had had no knowledge of the meaning of my motions until they were completed. I found things without knowing that they were lost. The motions were, however, simple. The last experiment was more complex, involving in its consummation the bending of the head, the puckering of the lips, and the actual facial contact. Before consummation, it revealed its object. I found myself performing an action which was repugnant to me. The line of reasoning taken by mind is rather interesting. It at first occurred to me that kissing was a part of the experiment ; but not having any idea in my previous performances, the fact of having an idea made the suggestion irrational, and by an effort of the will I banished it. I then reasoned that my mind had wandered, and that I had created the thought which I must get rid of as quickly as possible in order to assure passivity and success. I did get rid of it, but in so doing frustrated the operator's control.

Had I been hypnotized, so as to have eliminated the objecting faculty, the experiment would probably have been carried through ; although Mr. Croffut relates the circumstance of a girl who could not be induced to play cards even when hypnotized on account of the strength of her moral objections.

My own experiment never has been repeated. The circumstances were written out shortly after, and later, until revived by recent discussions, it faded from my mind.

TRANSACTIONS AMERICAN NEUROLOGICAL ASSOCIATION.

FOURTEENTH ANNUAL REPORT.

SECOND DAY.

Wednesday, Sept. 10th—Morning Session.

Continued from October Number.

ON A SUBCUTANEOUS CONNECTIVE TISSUE DYSTROPHY OF THE ARMS AND BACK, ASSOCIATED WITH SYMPTOMS RESEMBLING MYXŒDEMA.

Dr. F. X. Dercum, of Philadelphia, presented the case of a woman, aged 51 years, who suffered from a curious enlargement of the arms and back. This enlargement had existed some two and a half years and had been very gradual. It was stated that the increase in size was due to a dystrophy of the sub-cutaneous connective tissue, and that neither the muscles nor skin contributes to it. Sections of excised pieces of tissue were shown and revealed mainly mucous or embryonal connective tissue. In some fragments fat cells were demonstrated by means of osmic acid, in others no fat whatever could be detected. Some of the preparations exhibited, in addition, vessels with thickened and infiltrated walls besides non-medulated nerves in which inflammatory changes had taken place. Associated with this condition were the following symptoms. Pain increases, confined almost exclusively to the right arm, and while marked along the nerve trunks also diffused throughout the tissue. A peculiar "cakeing" of the swelling was noted at the time of the exises as well as a "bundle-of-worms-like" feel on the inner aspect of the arm. In addition to the pain in the right arm, various patches of anæsthesia were noted—all more marked, if not confined to the right side of trunk and right limbs. The special nerves also showed decided impairment, the visual fields being markedly contracted, and hearing, taste and smell more or less obtruded. Sweating had been scanty, especially in the early history of the case. Occasional bleeding from mucous surfaces was noted. Occasionally it came from the mucous membrane of the mouth or throat, sometimes a bloody bronchial sputum was noted, and occasionally hematomesis. The thyroid gland could not be felt. The urine was normal, no diminution of urea being detected. Counting the blood corpuscles yielded a negative result.

Dr. Dercum, while claiming that the case was one resembling myxœdema, pointed out the difference between it and typical instances of the latter affection. In the first place the skin itself was not involved—secondly, while slight slowing of movement existed it was not marked. Speech disturbance was present for only a few days, and was probably due to the swelling of the tongue and oral tissues accompanying a crisis of pain. The mind was clear but the patient was excessively irritable. No depression of temperature was observed except at one time, when 97° was noted. Subjective chilly feelings, however, were frequently complained of.

DISCUSSION OF DR. DERCUM'S PAPER.

DR. MILLS referred to the case of a woman who suffered from what appeared to be myxœdema of the face.

DR. WM. A. HAMMOND was struck with the analogies between the author's case and myxœdema; yet he was not sure that it should be called a case of that disease. It lacked certain essential features. There was no mental aberration. Of the two cases reported by himself, one was dead, the other was alive. This patient showed the characteristic scanning speech. If asked a simple question a half minute was required in getting a reply. Clubbing of the fingers was marked in both of these cases. Dr. Dercum's case had no involvement of the thyroid which would also seem to be essential to the disease. In his own cases referred to, the thyroid had been enlarged. This was undoubtedly a remarkable case, analogous to those of the enlargement of lateral half of the body. Of this he had seen two cases. If these cases were myœdematous we must revise our notions of that affection.

DR. PUTNAM considered that transition cases were always especially valuable. Pathologically, the affection described would appear to be related to myxœdema, but clinically the relation did not appear distinct.

DR. DERCUM stated that the fluid which exuded in his case resembled lymph rather than the denser fluid of myxœdema. He thought that the case was allied to the type without being a true myxœdema. He referred to the fact, however, that in quite a number of cases of myxœdema, mental symptoms had been marked. In some cases too, the thyroid had not been enlarged but wanting. He thought it difficult to determine minor degrees of enlargement of thyroid through the skin.

The following paper was then read :

SUBACUTE PROGRESSIVE POLYMYOSITIS.

By GEO. W. JACOBY, M.D.
OF NEW YORK.

The increase in our knowledge of the various affections of the muscles has, as is well known, during the last ten years assumed proportions which could hardly have been foreseen. That certain muscular affections are due to disease of the anterior columns of the spinal cord, that others are due to disease of the peripheral nerves, and that still others are entirely independent of such affections, but are primarily localized in the muscles themselves, are facts which have assumed the character of axioms. All the attention necessary for the attainment of these results has, however, been given to the chronic forms of muscular disorders, the forms generally described under the designation of degenerative atrophy, and the acute and subacute disorders, particularly the inflammatory ones, those showing the characteristics of inflammation as seen in other organs, have been markedly neglected. It is therefore our purpose by means of this paper to call to your attention the little that has been done in this direction, and by the report of a remarkable case, one presenting *intra vitam* all the typical signs of inflammation, invading successively nearly all the muscles of the body, ending fatally, and showing microscopically not only the characteristics of acute and subacute inflammation, but also many of those found in the chronic forms of primary dystrophies, to endeavor to trace a relationship, perhaps a connection between the acute and chronic primary muscular affections. The case is as follows:

Patient, F. H., male, æt. 35. Family history is unimportant. Married; three children, one dead. His wife has had no miscarriages. Neither he nor his wife have had syphilis, nor do they show any suspicious marks. Habits as regards alcohol and tobacco good. The patient was always always perfectly healthy until about four years ago. At that time he had an attack of intermittent fever, for which and during subsequent illnesses he was treated by Dr. H. J. Boldt, of this city, by whom the patient was kindly referred to us. Four or five months later he had an attack of pleurisy, and subsequently he was prostrated with a severe attack of typhoid fever which incapacitated him for about three months. The recovery from the typhoid was complete, and he remained perfectly well for more than a year. During this time he attended to his work, that of a machinist, without the loss of a single day, and felt as well as in his healthiest days, weighing more than he did prior to his illness. After the lapse of this year (two and a half years ago), having been in good health the day previously, he complained of a feeling of tension in the right calf. The gastrocnemious muscle "seemed to be severely inflamed." The skin was red and shining and there existed pain to superficial as well as to deep pressure. Slight œdema was also present. The diagnosis of a cellulitis was made and a deep incision effected under antiseptic precautions. No pus was present. The wound healed by first intention, and the patient seemed improved. After a few weeks a similar condition occurred in the left calf, and the right one also returned to the state which existed prior to the incision. The tension increased in both legs and was particularly noticeable in the morning. Finally, on account of this tension and swelling of the muscles, he found difficulty in bending the legs at the knees. The condition varied from day to day, but if one leg seemed somewhat better, the other one was worse. Then the thigh of the right side became similarly affected, and this was followed by the same condition in the left thigh. Then the flexor surfaces of both forearms became involved, and finally the biceps of both arms.

I saw the patient for the first time on May 11, 1888, when he presented the following *status*: He is a tall, thin cachetic-looking individual. The facial muscles are normal. Eyes and pupils, tongue and speech normal. The deltoid and trapezius on the left side were enlarged and painful to pressure. So also the biceps of the left arm and all the muscles on the flexor surface of the forearm. The right arm is similarly affected but to a slight degree. The entire left arm feels harder than the right. The skin over both forearms on the flexor surface shows a slight erythematous blush, which is also more marked upon the left than upon the right side. On the hands the balls of both thumbs seem somewhat tender to pressure, but not swollen.

The lower extremities are both equally affected. The quadriceps cruris and the adductors are very much enlarged and painful to pressure, as are also the gastrocnemii. The muscles of the feet are not affected. The motions of the patient are impeded in accordance with the muscles involved. He can execute all movements, but those requiring action of the affected muscles show a certain effort which at once attracts attention. The affected muscles have a peculiar feeling, recalling very much, as do also all movements of the lower extremities, cases of pseudo-hypertrophic paralysis. They however feel more elastic. The tendons of all the involved muscles can be plainly felt as tense cords. Slight œdema of the skin, which however does not take the impression of the finger is noticeable over all the affected muscles. The pain on pressure is confined entirely to the body of the muscle. Pressure over the nerve trunks does not cause any pain.

Electrical examinations showed the excitability of the affected muscles to be greatly reduced to both currents. A current of 15 m. a. was requisite for the production of cathodic closure contraction. No reaction of degeneration in any of the muscles. Slight patellar tendon reflexes obtainable with great difficulty. Sensation normal. No bladder affection. The mechanical excitability of the affected muscles was not increased, perhaps reduced. Fibrillary twitchings were not present. There was no atrophy discernable in any parts of the body. Heart, lungs and abdominal organs normal. Measurements of the legs and thighs gave the following results:

Thigh, left, upper part, - -	58 cm.
" " middle, - -	53 "
" " lowers, - -	43 "
Leg left, below the knee, -	35 "
" " middle of calf, - -	41 "

The right leg and thigh gave slightly smaller figures.

On the 16th of May, Dr. Kammerer, of this city, kindly excised for me two pieces of muscle, one piece from the gastrocnemius of the right leg and the other piece from the supinator longus of the left forearm. Both wounds healed by first intention.

During the following fortnight there was no particular change in his condition. One day he would be able to walk around, and another day he would be confined to his bed. Then his condition grew decidedly worse, the swelling of the extremities increased, the muscles became exquisitely painful to the touch, but the joints remained freely moveable and free from exudation.

On July 3d, Dr. Boldt, requesting me to see the patient again, writes:

"He has acute cirrhosis of the liver. The stage of enlargement has passed and the organ for the past ten days is getting smaller again. The connective tissue seems to be rapidly shrinking. Examinations of the urine only show decrease of urinary salts."

July 4th.—I found the condition as follows:

Abdomen tense with ascitic fluid. The face and body very much emaciated. Sweats profusely. The affected muscles in consequence of the general emaciation appear much more swollen than actual measurements prove them to be. The figures obtained by measurement are essentially the same as those above given, with the exception of the lower parts of the thigh. There is very great pain upon motion and to pressure. The attachment ends of the muscles are approximated and the tendons stand out hard and unyielding. The skin has a deep erysipelatous appearance over all the implicated muscles. Minute capillary hæmorrhages are also seen along the borders of these discolored territories. In addition to the muscles previously mentioned as being affected, the glutei, the muscles of the back, the intercostals and the external obliques of the abdomen are also more or less involved. That, however, which at once attracts attention on account of the contrast produced is the atrophy which has invaded certain muscles and which is assuredly not due to the general emaciation. Upon both thighs it is seen that the lower part of the quadriceps, particularly the vasti are very much wasted. This is particularly marked upon the left side, which was previously the larger of the two. The contrast caused by the wasting of the vasti and the great hypertrophy of the rectus, abductors and glutei was very marked. A similar contrast was also observable in the arms where the biceps and triceps were very much enlarged while the deltoid and anterior part of the trapezius, which had also been affected were, particularly upon the left side, almost entirely atrophied. The shoulder joints were movable. The dorsal interossei of both hands and the thenar muscles also atrophied, together with the ball of the thumb of the left hand, that of the right hand being swollen and painful.

July 18th.—Condition the same. Consolidation of apex of left lung. No other muscles involved by the atrophy.

July 25th.—General condition much worse. Has difficulty in swallowing. Tongue painful and swollen. Cannot protrude it beyond the edge of the teeth.

Aug. 2d.—Pain in swallowing so great that only by the greatest efforts can he be induced to take a little milk. His condition is a truly deplorable one. Active movements of any kind are impossible. The head cannot be lifted from the pillow and even a slight rotation of the head causes great distress. The forearms are flexed almost at right angles to the arms. Pressure-force of hands *nil.* The legs flexed upon the thighs and cannot be straightened. The feet themselves are freely movable. Extended passive movements cannot be made on account of the resistance of the tense tendons. Face and eyes not affected. Sensation not affected. Pupils, sight, smell and hearing normal. Sensorium perfectly clear.

Aug. 12th.—Death due to general marasmus, and disorders of respiration.

Owing to my absence from town at the time of death, a post-mortem was unfortunately not obtained.

The pieces of muscle exsected from the leg and arm were immediately placed in a half per cent. solution of chromic acid. When sufficiently hardened they were embedded in celloidin and cross and longitudinal sections made. These sections were then stained with an ammoniacal carmine solution and mounted in glycerine. The histological changes found will now be described.

In transverse sections of the piece of muscle exsected from the leg (Fig. I.), the small size of the single muscle fibres, without any noticable decrease in their number in each individual bundle, is marked. I do not, however, attribute this feature to the morbid process itself, since with the exception of a moderate increase of the perimysium internum, the fibres have retained their mutual flattening, and even the sarcolemma sheath appears to be preserved, except in a few muscle fibres which are highly altered. In addition we also, with low powers, at once notice a distinct broadening of the perimysium externum as also of the perimysium internum, the latter not being so conspicuous and not so uniformly observable as the former.

The newly formed connective tissue which goes to make up the augmented perimysium externum, is either myxomatous or fibrous in structure. The latter again is either loose and delicate or dense and fibrous, being then made up of comparatively coarse bundles. In both varieties of the perimysium externum we frequently meet with the following two features.

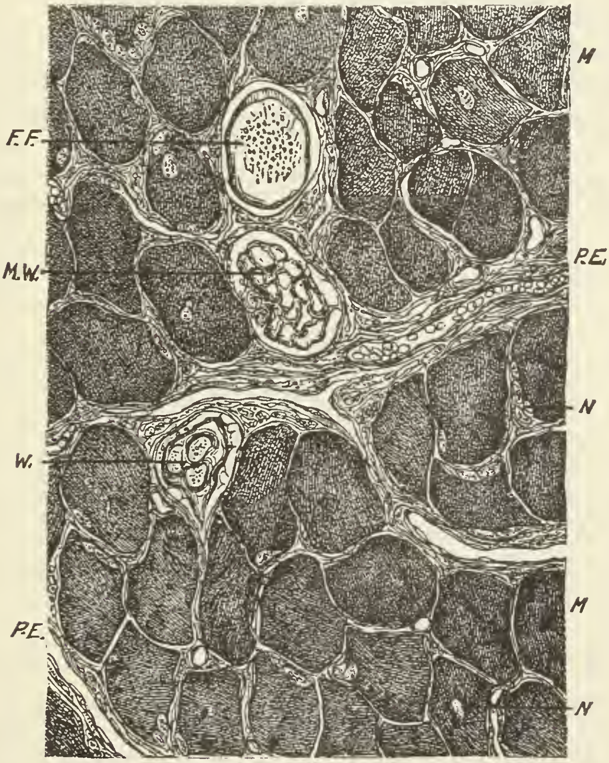

Fig. I.—Transverse Section x 200.

M. M. Muscle fibres of average size in partly transverse, partly slightly oblique sections.
P. E P. E. Perimysium externum with numerous capillaries.
N. N. Nuclei, some in centre, some at periphery of muscle.
W. Muscle fibre in waxy degeneration, holding three protoplasmic bodies.
M. W. Cluster of medullary corpuscles in waxy degeneration.
F. F. Muscle fibre in waxy. possibly combined with fatty degeneration.

Firstly, fat globules in a more or less regular arrangement; and, secondly, waxy degeneration of the bundles of fibrous connective tissue, in some places very pronounced. The perimysium internum, although as already mentioned, augmented, is made up entirely of a delicate fibrous connective tissue, carrying numerous blood-vessels, but nowhere exhibiting fat globules or waxy degeneration.

The muscle fibres themselves apart, from their small size, show peculiar changes, which never invade all the fibres of a single bundle, but only a limited number, which varies from one to eight.

These affected fibres are either transformed into an apparently homogeneous mass, with traces of nuclei in it and still surrounded by an unchanged sarcolemma sheath, or the muscle is transformed into a highly refracting so-called waxy mass which shows distinct nuclei, or is entirely homogeneous, or homogeneous only at the periphery and granular in the centre. It is not uncommon to see muscle fibres which are partly normal in appearance and partly waxy or homogeneous. Many of the fibres contain nuclei in their interior, which, when fallen out, leave open spaces or vacuoles. The number of such nuclei and vacuoles varies from one to half a dozen or more in a single fibre. Other fibres again are in part or entirely broken up into smaller lumps with or without nuclei (Fig. II.). The staining of the specimens with the ammoniacal carmine solution also yielded peculiar results, inasmuch as many of the fibres appear deeply stained in one part and unstained or yellow (owing to the chromic acid solution) in another. Such variations in color occur even in distinctly waxy fibres, some of which are deeply carminized, others again appearing unstained. Since hæmorrhage had taken place at the moment of excision of the muscle and a subsequent saturation with the blood plasma must have occurred, the differences in the reaction to this staining reagent would, it appears to us, in our case be deserving of but little attention.

From the appearances thus far described, as seen with low powers, it follows that a plastic or formative inflammation has invaded the muscle, being most conspicuous through the augmentation of the interstitial connective tissue, more especially of that of the perimysium externum. This process is known under the term of hyperplastic perimyostitis, a process very prominent in our case of pseudohypertrophy from which microscopical studies were presented to this Association last year.

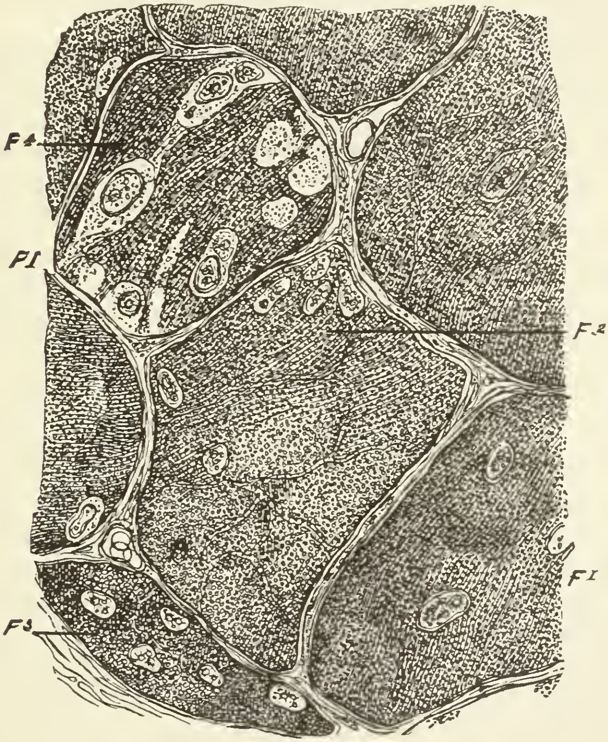

FIG. II.—Transverse Section x 800.

F.1. Muscle fibre with central nuclei.

F 2. Muscle fibre with augmented peripheral nuclei, showing indication of breaking up into muscle plates.

F,3. Muscle fibre, holding five central nuclei, each one corresponding to a muscle plate, the whole resembling a myeloplax. The sarcous elements enlarged and crowded.

F.4. Muscle fibre transformed to a great extent into partly nucleated, partly non-nucleated protoplasm.

PI. Perimysium internum almost unchanged.

At the same time marked changes must have occurred in the muscle fibres themselves, leading to a breaking up of the individual fibres, their contractile matter into lumps, and terminating in waxy degeneration—a process which is recognized as a parenchymatous one since Virchow, and meriting the name of "myositis."

The question now arises, What relation in our case does the myositis bear to the perimyositis? In other words, how much of the process is interstitial and how much parenchymatous?

This question can easily be settled by resorting to higher powers (500 diameters). The first feature which impresses the observer is the augmentation of the nuclei. In the normal muscle, nuclei are seen at the periphery of the muscle fibre close beneath the sarcolemma, but not in large numbers. In our case, muscle fibres are seen holding sharply defined nuclei scattered throughout the body of the fibre without regularity, and surrounded by a light rim, possibly due to shrinkage; whereas the contractile matter is apparently little changed and still exhibits the granulation produced by the sarcous elements.

As central nuclei, in the fully developed fibre, are known to occur in human beings only in the muscle of the heart, the presence of a larger number of nuclei within the fibre, as such, would indicate a pathological process. When the nuclei are much increased in number, the transverse section of the muscle fibre may assume the aspect of a so-called giant cell, whereby the regular arrangement of the sarcous elements is lost and the granulation has become irregular. In this case the sarcolemma sheath is still present and still allows the boundary of each single muscle fibre to be easily defined. In the next stage the muscle fibre breaks up into a number of indifferent or medullary corpuscles, many of which still contain nuclei, either singly or in groups. The boundary of a muscle fibre has in this stage to a great extent lost its sharpness; and since the adjacent perimysium is largely composed of similar bodies not markedly nucleated, no strict boundary line can be drawn between the muscle and the perimysium. The inference, however, almost forces itself upon us, that the original muscle tissue, after having split up into medullary corpuscles, loses its specific structure and becomes transformed into fibrous connective tissue, adding, as it were, a certain amount to the hyperplastic perimysium itself. This inference impresses itself upon us all the more strongly when we see fields of fibrous perimysium with traces of previous muscle fibres composed of indifferent corpuscles, and finally fields of considerable extent between slightly changed muscle fibres, embracing fibres in waxy degeneration.

Still more instructive are for our purpose muscle fibres which in part are unchanged, in part, on the contrary, are transformed into a coarsely granular mass, which has entirely lost the regular arrangement of the sarcous elements, and is provided with a varying number of nuclei. The presence of the sarcolemma is a positive proof that the morbid changes have taken place in a portion of the contractile substance of a single muscle fibre only. Such a partial transformation is by no means a rare occurrence.

The appearance of a number of nuclei within the muscle fibre is not the only way in which the morbid changes are inaugurated. Sometimes a muscle fibre breaks up partly or *in toto* into clusters and lumps, apparently due to an increase of the contractile matter and its grouping into homogenous masses. Such lumps may likewise grow up to the size of nucleated medullary corpuscles, indicative perhaps of a more acute course of the process. This process we must define as a myositis, a process leading first to a reduction of the muscle fibre into its embryonal constituents, the so-called muscle plates or sarcoplasts. One of the terminations of myositis has already been described, namely, transformation of the contractile matter into fibrous connective tissue, or perimysium. In addition to this termination we find two others present in our case, namely, fatty and waxy degeneration of the muscle fibres.

Here and there transverse sections of muscle fibres are met with, which contain vacuoles in their centres, varying from one to five in number, and enclosed by a sharply defined capsule. Such vacuoles differ in appearance from empty spaces by dropped-out nuclei. Although the fat is mostly extracted from the specimen, owing to the use of alcohol and ether for the purpose of embedding, the comparison with analogous formations in the perimysium admits the diagnosis of the formation of fat in the middle of the muscle fibre. It is also this treatment with ether and alcohol which renders it impossible to say whether or not fibres lacking the carmine stain, being yellow, highly refractive, and split up into clusters by irregular crevices, are in fatty degeneration. We know, however, that the sarcous elements may be directly transformed into fat granules, by the coalescence of which, appearances are produced identical with those seen in these specimens.

The second and more common termination of the myositis process is waxy degeneration. In almost every bundle we find one or more muscle fibres transformed into a highly refractive mass, known under the name of "waxy" or "amylaceous." The manner in which this metamorphosis is produced can easily be traced. At first we notice a breaking up of the muscle fibre into a number of medullary corpuscles, whereby the sarcolemma sheath remains unaltered. Such corpuscles are saturated with or infiltrated by a peculiarly changed blood serum, which renders them refractive, glossy, and homogeneous. Often a number of medullary corpuscles coalesce into a lumpy, homogeneous mass, in which faint traces of the boundaries of the previous medullary corpuscles are discernible. In the highest degree of waxy degeneration we see the homogeneous mass almost continuous, holding in its interior star-shaped or oblong protoplasmic bodies not in waxy degeneration, so that the appearance of cartilaginous tissue is brought forth. If the number of protoplasmic bodies prevails over the waxy basis substance, the latter may produce a framework similar to a honeycomb, the sarcolemma under these conditions being entirely lost.

Longitudinal sections of the muscle of the leg show all the changes described above as present in transverse sections (Figs. III. and IV.). Especially conspicuous is the small size of all fibres, these at the same time being split up lengthwise into minute fibrillæ, and only exceptionally exhibiting a distinct transverse striation. The size of the sarcous elements is strikingly small. The augmentation of the number of the nuclei is obviously less noticeable in the longitudinal than in the transverse sections, but here and there chains or rows of nuclei are seen.

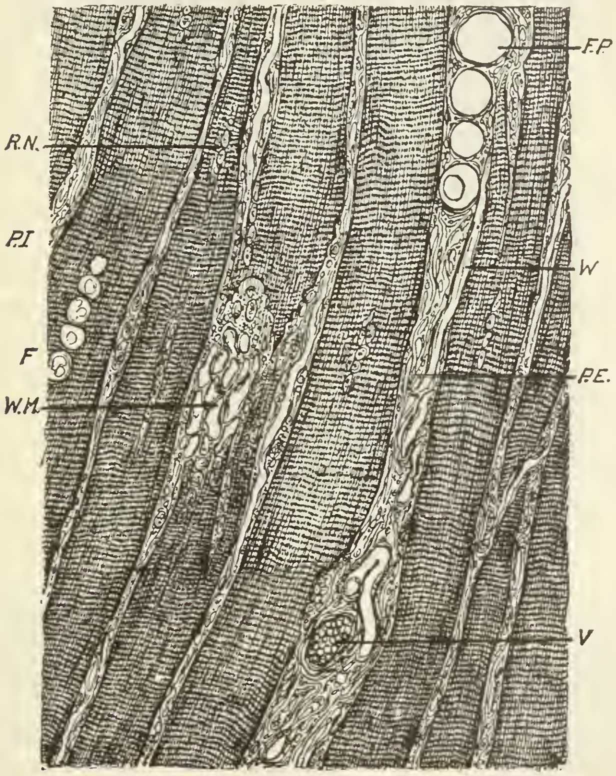

FIG. III.—Longitudinal Section x 200.

Pe. Perimysium externum, considerably augmented; composed of coarse bundles of fibrous connective tissue.

V. Vein, engorged with blood.

F. P. Fat globules in external perimysium.

P. I. Perimysium internum, slightly augmented.

R. N. Rows of nuclei.

F. Row of nuclei.

F. Row of fat globules (?) in centre of muscle fibre.

W. M. Clusters of medullary corpuscles in waxy degeneration. In the vicinity of this cluster, the gradual transformation of the muscle tissue into inflammatory corpuscles and the destruction consequent destruction of the muscle tissue is marked.

W. Peripheral portion of the muscle fibre in marked waxy degeneration.

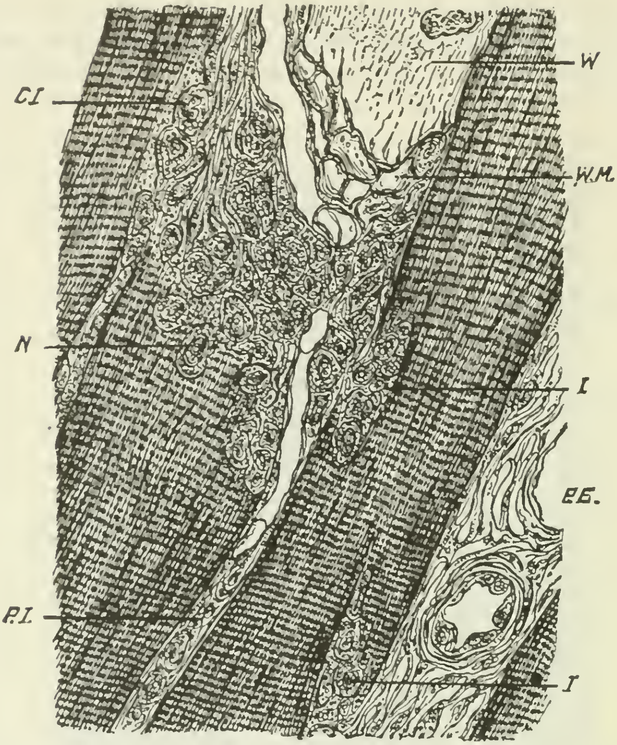

FIG. IV.—Longitudinal Section x 800.

P. E. Perimysium externum, broadened, composed of coarse bundles of fibrous connective tissue freely vascularized.

P. I. Perimysium internum, transformed into inflammatory or medullary corpuscles.

I. I. Groups of inflammatory corpuscles, obviously arisen from previous contractile tissue

N. Nucleated inflammatory corpuscles imbedded in muscle tissue.

C. I. Clusters of inflammatory corpuscles in bay like excavations of the contractile tissue, in part spindle shaped, in transition to fibrous connective tissue. The medullary tissue transversed by a large, probably newly formed capillary blood-vessel.

Both the perimysium internum and externum have increased in size, especially the externum. That this increase is not a mere augmentation of the fibrous connective tissue, but is the result of an active participation of the contractile tissue, can plainly be demonstrated in longitudinal sections. Here we not infrequently encounter muscle fibres whose body terminates almost abruptly, the fibrillar structure being lost and replaced by medullary tissue. It also happens that only a limited portion of the muscle fibre appears transformed into rows of medullary corpuscles, blending with the contractile substance proper. This contractile substance first shows a breaking up into the muscle plates, and subsequently into rows or clusters of medullary corpuscles. Such clusters are seen to appear in the middle of a muscle fibre, being then bordered by an almost unchanged muscle tissue. The medullary tissue assists in the new formation of both external and internal perimysium, whereby the bulk of the muscle fibre is diminished and that of the fibrous connective tissue augmented.

Waxy degeneration is a marked feature both in the perimysium and in the muscle fibres. In the former, even the middle and adventitial coats of the arteries and the adventitial coats of the veins are subject to waxy degeneration. In no place of the muscle tissue itself has this degeneration attacked the muscle fibre in toto. Lumps showing the characteristic high refraction, as a rule are seen only in the axes of muscle fibres, as though representing prolongations of them. This fact, more particularly however the appearances seen in transverse section of the muscle, forces upon us the conviction that it is the contractile tissue itself which has become waxy, after having first been broken up into medullary tissue.

Numerous medullated nerve fibres are visible in longitudinal as well as in transverse sections. These are seen singly as also in bundles. Many of these nerves appear to be unchanged, whereas in others an increase of the nuclei of Schwann's sheath can be seen, and in still others the perineurium internum appears to be more or less crowded with medullary or inflammatory corpuscles, features which we are accustomed to see in neuritis and perineuritis.

How many medullated nerve fibres have been destroyed by being transformed into fibrous connective tissue, we are unable to say. The large number of unchanged nerve fibres, however, strongly points towards the view that the process of neuritis and perineuritis is not the primary one, but altogether secondary to the myositis and perimyositis. In the same manner as a certain amount of contractile tissue is transformed into fibrous connective tissue, with the intervening stage of inflammatory or medullary tissue, so also the nerves are at least in part changed, first to medullary, and finally to fibrous connective tissue.

The muscle of the arm shows changes identical with those observed in that of the leg, the only difference between the two being that the process is less extensive and less intensive in the former than in the latter.

In summing up the results of this examination, I would condense them as follows:

1. The process is an acute myositis and perimyositis, grafted on a chronic plastic or formative process.
2. The plastic process has led to a new formation of connective tissue of the perimysium externum as well as of the perimysium internum. This process became progressive by repeated recurrences of myositis, leading to a diminution of the contractile, and to an increase of the fibrous connective, tissues.
3. The result of the chronic process is fatty and waxy degeneration. Both have invaded the perimysium, as well as the muscle tissue proper.
4. Neuritis and perineuritis are secondary to myositis and perimyositis.

Before entering into consideration of the case itself, if we now take a brief review of that which has heretofore been done in acute disorders of the muscular tissue, we will see that, notwithstanding inflammation of muscles ought *a priori* to occur as easily as inflammation of other tissues, as a matter of fact, it was universally supposed that such inflammation of the muscles themselves was impossible, until Virchow,[1] in his remarkable thesis of 1852, showed this view to be erroneous. Even at present there are many observers to whom the existence of a primary myositis savors somewhat of a myth. This condition of affairs can only be due to the fact that, although inflammation of the muscles, be it due to operative measures or traumatism of various kinds, or to propagation of the inflammatory process from neighboring parts, is often produced, nevertheless the myositis itself rarely comes under the observation of the physician. The slight forms cause so little inconvenience to the patients, and the severe forms are of so rare occurrence, that even when these severe forms are encountered *intra vitam*, a disunity of opinion as regards the diagnosis and character of the affection has been the result. Our knowledge of the microscopical changes produced by the process has, with perhaps the exception of the more minute changes, remained the same as it was left by Virchow. According to this observer, the pathological changes occur either in the muscular fibre itself, in the interstitial connective tissue, or in both, and it is often impossible to determine from which of these the process primarily originated. The contractile substance becomes involved in a number of regressive changes—hyperæmia, turbid swelling, change in color, loss of longitudinal and transverse striation, then fatty or waxy degeneration, or, in very acute cases, destruction of the fibrillæ and softening.

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[1] *Virchow*, Ueber Parenchymatoser Entzundung, Archiv. f. Patholog. Anat. und Physiologie. 1882, Vol. 4, p. 261–323.

The processes occurring in the interstitial connective tissue are of a more active nature ; here we have the formation of pus, granulation and fat tissue, or tendon and bone tissue, which substances were supposed to force themselves between or supplant the contractile elements. Thus, according to the tendency of the changes of the medullary corpuscles, we have either a fibrous, a purulent, or an ossifying myositis. The category in which our case belongs, microscopically, is, as already stated, that of a parenchymatous myositis.

Upon entering upon the clinical aspect of this case we are confronted by an important question. Is the affection here a primary or a secondary one ? Of secondary affections of the muscles not due to local causes, we have pain and swelling due to muscular rheumatism. Etiologically and anatomically, we know nothing as to the character of this affection. Clinically, we only know that beyond the pain and swelling there is no analogy between the two affections.

So also after polyarticular rheumatism occasionally, many muscles are affected in a similar manner ; but in our case there is no history of joint involvement at any time.

After variola, Wagner[1] has seen similar occurrences.

The muscular implications occurring in consequence of scorbutus, glanders and syphilis need only be mentioned to be disposed of. Not so is it with the muscular changes occurring during typhoid fever. To these more than a passing notice must be given. Changes in the muscles of typhoid fever patients, resembling very much the changes described by Virchow as above stated, were observed by Zenker[2] in 1864. He found degeneration of the striated fibres in the most varied groups of muscles, and as has been shown by later observers these changes are almost as characteristic as the changes of the intestinal mucous membranes. The adductors of the thighs and the recti abdominis are the muscles chiefly involved. A medium or pronounced degree of waxy degeneration is here found, sometimes associated with granular degeneration. A certain number of fibres still retain their normal transverse striation. The extraordinary fragility of the muscles is shown by the frequency with which transverse ruptures are encountered. Microscopically, Zenker differentiates the granular and waxy degeneration, the first produced by interspersion of finest molecules in the contractile muscular substance, the latter consisting of a transformation of the contractile substance into a waxy, homogeneous, colorless, shining mass, with complete disappearance of the transverse striæ and destruction of the muscular nuclei, the sarcolemma alone remaining intact. In addition Zenker also noted degeneration in the form of discoid separation and fibrillary splitting. The connective tissue, vessels and nerves show no changes, but in many cases a proliferation of nuclei takes place in the perimysium. Although the comparison of the microscopical findings in our case with those of Zenker show many points of identity, still it is evident that there are also points of difference. But even were the results of the microscopical examination absolutely identical, we would not be justified in considering the processes as clinically analogous. Indeed we have long believed and have publicly expressed the opinion that from an examination of muscles alone, it is impossible to decide to which clinical variety of muscular disorder a given case belongs.[1] This idea also receives support from Waldeyer,[2] who in myositis, mechanico-experimentally produced, found the same histological changes as in typhoid myositis. Therefore it is necessary in a review of conditions analogous to those presented in our case also to give particular attention to the clinical aspects.

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[1] Archiv. der Heilkunde, 1872, p. 107, Vol. xiii.

[2] *Zenker*, Ueber der Veranderungen der Wilkurlichen Muskeln in Typhus Abdominalis, Leipzig, 1864.

The clinical symptoms differ materially in our case and in typhoid myositis. In the latter the course is of a light character with slight pains and slight tenderness to pressure, whereas in our case the other extremes were noted. We have no hesitancy in excluding the fact of our patient having had typhoid fever over a year prior to the occurrence of the described affection, as entirely free from any etiological significance.

The next class of cases which bear a certain analogy to ours are those described by Scriba.[3] The principal point emphasized by these cases is that we may have an acute myositis due to infectious inferences. Here the infection was caused in three cases by a furuncle and in one by a carious tooth. The fifth case is valueless for comparison, as it did not present a pure myositic picture. These cases presented slight fever, severe pain in the muscles affected, with swelling. The affected muscles were greatly increased in size and were of a bony consistency to the touch, and could be traced from origin to insertion, like a plaster cast. The process was always limited to isolated muscles and the contiguous muscles were soft and painless. The cases all ended in complete cure in several weeks, either with or without suppuration. In the second case microscopical examination of a teased preparation of a piece of muscle was made. The transverse striation was found to be almost entirely absent. The sarcolemma were from two to three times larger than is normally the case. The perimysium externum was slightly infiltrated with medullary corpuscles and the perimysium internum was only very slightly implicated. Scriba considers the affection very rare even in Japan, and was unable to obtain the history of a single similar case from other physicians. In Europe, also, the affection was entirely unknown prior to this publication. The points of variance between these cases and ours are also evident.

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[1] Discussion, N. Y. Neurological Society, March 5, 1888.

[2] *Waldeyer*, Ueber die Veranderungen der Quergestreiften Muskeln bei der Entzundung und dem Typhus Process, etc., Virchow's Archiv., Vol. 34.

[3] *Scriba, J.*, Myositis Acuta, Deutsche Zeitschrift f. Chirurgie, Leipzig, 1885, p. 497–502.

This completes the review of the acute secondary inflammatory muscular disorders. Of the acute primary or so-called idiopathic disorders still less is known.

Under the name of acute and subacute myositis, a class of cases has been described by various observers, Strauss,[1] Clark,[2] Treves,[3] Devis,[4] Walther,[5] Hepp[6] and others, giving as causes cold, blows and muscular over-exertion.

Nosologically this affection stands in the same rank as acute periostitis and osteomyelitis, with which it has in many cases been confounded. It is undoubtedly an acute infectious disease with a tendency to suppuration differently localized according to the preceding infection. All the muscles of the body become affected. A typical course cannot be given, but it is undoubtedly a serious disease, beginning with dull, increasing pains, which render the use of the affected muscle impossible. The muscle swells, becomes hard, abnormal positions ensue (due to approximation of the attachments), suppuration takes place, causing single large or several small abscesses, and the severe cases end fatally, the lighter ones recovering.

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[1] *Strauss*, Jaccond's Dictionnaire, Vol. xxiii., p. 362.

[2] *Clark, F. W*, British Med. Journal, 1887, ii., p. 69.

[3] *Treves*, Brit. Med. Journ., 1886, ii., p. 1215.

[4] *Devis*, Tr. Pathol. Soc. London, 1881, p. 273-75.

[5] *Walther*, Deutsche Zeitsch. f. Chirurgie, 1885, p. 285-87. Gives table of 19 cases, of which cases 12 (*Maas*) 15, 16, 17 and 18 (Scriba) do not belong here, but must be classed as secondary affections.

[6] *Hepp, Paul*, Ueber einen Fall von acuter Parenchymatoser Myositis, welche Geschwulste bildete und Fluctuation vortauschte, Berliner Klinische Wochenschrift, 1887, p. 389.

Only one more class of cases remains to be described, and to this class, of which only three cases have been thus far reported, our case belongs. As these cases are of such rare occurrence, and they all have been described within the last two years, I may be pardoned if I enter upon them somewhat closely. A few cases have been described, prior to these, nnder other names, which probably also belong to this class.

These cases clearly show the existence of an acute inflammation, rapidly affecting nearly all of the voluntary muscles, characterized chiefly by pain and swelling, and ending in death within a more or less short period of time. They show us that we may have an acute primary progressive inflammation of the muscles, the chronic form of which alone has been hitherto known. The first case is that of Wagner.[1] Female, æt. 34. Years ago had typhoid. Now tuberculous. Since end of June, without exposure to draught or cold, pains in joints, back, neck, shoulders, and legs. Received in hospital, July 19th, 1886. Well nourished, strongly built. Muscles of neck painful to pressure. Stiffness in both shoulder joints, but freedom from pain. Slight œdema of back of hands and forearms as well.

July 24th.—Paræsthesia in both forearms. Œdema the same. Nerves not sensitive to pressure.

July 28th.—Arms also swollen, forearms most. Skin tense, so that contours of muscles could not be distinguished. Muscles feel doughy. Movements impeded.

August 7th.—Swelling has decreased, Erysipelatous redness on flexor surface of left arm.

August 17th.—Redness has spread.

August 24th.—The thighs have become affected. Cough and dyspnœa.

August 29th.—Disorders of deglutition and spontaneous attacks of dyspnœa. Death.

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[1] *Wagner, E.*, Ein Fall von acuter Polymyositis, Deutsches Archiv. f. Klin. Medizin, Vol. 40, 1887, p. 241.

Autopsy.—Extensive muscular disease. Slight œdema of both upper extremities. Slight tuberculous phthisis of lungs. Diffuse bronchitis. Severe emphysema. Hyperæmia of brain and cord. The muscles which were microscopically changed were the biceps, triceps, and brachialis internus. These were of a peculiar, stiff consistency, but at the same time more fragile than normally. In the forearm the flexors are chiefly affected, but irregularly, spots and stripes of very pale appearance alternating with darker parts. Nearly all the muscles except the glutei, muscles of the calf, and the abdominal muscles, are affected. Brain, spinal cord, and peripheral nerves normal. The majority of specimens for microscopical examination were taken from the biceps. Serous infiltration, fatty and waxy degeneration of the muscle fibres, were the characteristics most marked. The interstitial connective tissue showed proliferation of nuclei, and single multinucleated giant cells were also observed. Processes of new formation were found in the muscular substance, as well as in the perimysium. Only in a few places was the perimysium internum increased. The adventitia of the small arteries was somewhat rich in nuclei. The nerve endings in the muscles were not examined.

The second case is that of Unverricht.[1] The patient was a male, æt. 24, who had been well until two weeks prior to presentation. Etiologically nothing. For a few days dragging pains in arms and legs, which increased in severity until walking was impossible.

Examination showed a medium-sized, well nourished individual, of good muscular development. Organs of chest and abdomen normal. Complains of pains in all extremities and back. Motion of extremities intact, but painful; force normal, no paræsthesia. No fever, nor disorders of digestion. After a week, slight swellings of the extremities and also of the face. Increase of the swelling. Muscles painful to pressure. The joints free from exudation, and painless. At this stage the extremities were swollen to thick, formless masses, whereas the body was not affected. Later, the thorax became painful to pressure, and swollen. Three weeks after entrance to hospital, pains in swallowing; fluid food alone could be taken. Thoracic muscles very painful, breathing abdominal, pneumonia, death.

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[1] *Unverricht*, Polymyositis acuta progressive, Zeitschrift f. Klinische Med., 1887, p. 534.

Autopsy showed brain and medulla normal. The muscles showed extensive and remarkable changes, chiefly limited to certain groups. Color in general, dark reddish brown, but upon section many muscles are found colored in irregular spots. Pale, somewhat transparent, light gray strips alternate with dark red spots. All these muscles are swollen and fragile. The changes are most marked in the extensors of the extremities, whereas the flexors are almost entirely unaffected. The pectoralis major affected in parts. The deltoids extensively, biceps slightly. Extensors of forearms very much, triceps free, muscles of ball of thumb free. Extensors of thighs and toes very much, but flexors of thighs and legs only slightly affected. Abdominal muscles, muscles of back, and intercostals slightly implicated. Muscles of tongue, eyes, and diaphragm not affected. The idea of trichinasis had been expressed, but not a single trichina was found.

Microscopically, the results corresponded to the macroscopical examination. The interstitial connective tissue was infiltrated with medullary bodies. Muscular substance in all stages of degeneration, some places showing normal muscle, and the immediately contiguous place being severely degenerated. Loss of transverse striation; hygroscopic; granular and waxy degeneration. In other places fatty degeneration was found. Regeneration, and increase of muscular nuclei not present. Spinal cord and peripheral nerves were unchanged.

The third case is that of Hepp.[1] Patient, female, æt. 36. Family history good. Previous history unimportant. Sick since March, 1886; did not feel well during two to three weeks; then eruption on face of red, thickly settled, elevated and non-itching spots. Next day spread to back, neck, and mucous membrane of mouth. After a week, disappeared. Then increasing pains in neck and back; legs swollen, but not the thighs. Took to bed. After eight days, swelling of face and right arm, then, after three days, of left arm. Swelling of face disappeared, that of arms increased. Spontaneous pains rare, but very severe upon slightest motion. Then stiffness of extremities; could not bend or extend arms; legs not so bad.

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[1] *Hepp, Paul,* Ueber pseudo Trichinose, eine besondere Form von acuter parenchymatoeser Polymyositis, Berl. Klin. Wochenschrift, 1887, p 297.

Examination shows slight redness of face and chest. Œdema of face, extremities, and chest; feet, ankles, and hands free. Arms swollen to a hard cylindrical mass; pectoralis major in contracture; hard, stiff, and non-elastic. Muscles of neck and lower extremities the same, but not so marked. No pains when quiet. Joints free; passive movements free and painless; extended movements difficult on account of contracture of muscles. Active movements not possible. Facial muscles and tongue not affected. Voice, pupils, sensorium, special senses not affected. Tendon reflexes absent. Affected muscles do not react to either electrical current. Increase of disorder, affection of muscles of deglutition, death on May 22d. Anatomically, affected muscles yellowish and pale. Everything else normal.

Microscopical Examination.—Absence of trichinæ. Waxy or hyaline degeneration near apparently normal fibres; others in which transverse striation indistinct, and in still others entirely absent. Others showed all stages of hyaline transformation, from the cylindrical hyaline fibre to complete disintegration into granular detritis. In no place granular or fatty degeneration. Spinal cord normal.

The following two cases have been published under other names, but probably also belong in this category. First, by Wagner.[1] Woman, æt. 43. Well until a few days prior to entrance into hospital.

Status, Oct. 17th, 1861.—Badly nourished, pale, small. Both arms very much swollen up to elbows. Forearms to hands less. Reddened, œdematous ; great pain on pressure. Lower extremities not swollen, but have a feeling of tension. Difficulty in swallowing fluids.

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[1] *Wagner, E.*, Fall einer seltenen Muskelkrankheit, Archiv. der Heilkunde, 1863, Vol. iv., 282 and 83. Additional remarks in the article on Polymyositis by the same author. Loc. Cit.

19th.—Swelling of upper extremities less.

20th.—More swelling.

21st.—Arms very much swollen. Death occurred during the following night.

Microscopical examination showed intense and extensive changes in all the muscles of the upper part of the body. Partly fatty metamorphosis, partly colloid degeneration. In many places both forms of degeneration were found in one and the same fibrillæ. Numerous diffuse, very small foci of pus corpuscles, and increase of the nuclei. The intermuscular and subcutaneous connective tissue did not show any proliferation of connective tissue bodies or pus.

Second Case.—Marchand.[1] Patient male, æt. 23. Sick since five weeks. Began with gradually increasing muscular pains and corresponding disorder of function. At the same time œdematous swelling without albuminuria occurred. Diagnosis of trichinosis. The respiratory muscles became affected and death ensued. The autopsy revealed an intensely diseased condition of the muscular system, not equably distributed, but affecting principally the extensors of the extremities, partly also the muscles of the chest and neck as well as the adductors and flexors of the thighs. The muscles were increased in size. No trichinæ were found. Microscopical examination showed granular and fatty as well as hyaline degeneration of the primitive fibres. Spinal cord and peripheral nerves normal.

Now, having found the proper place for our case, there are two questions, applicable to this entire class, which must be answered before any final conclusion as regards the position if any which it bears to the chronic forms of primary dystrophies can be arrived at. The one is the question of trichinosis in the production of analogous conditions, and the other is that of the relation which multiple neuritis bears to these cases. In all the cases above cited as being cases of polymyositis, the similarity of the symptoms to those observed in cases of trichinosis is marked, and all of the authors with the exception of Wagner, in his first case (1861), had even more than a suspicion that trichinæ were the cause of the severe muscular disorder. Also in two cases reported by Kussmaul and Maier,[1] which although accepted by others as cases of polymyositis, I have purposely excluded, this conviction is expressed. Hepp even goes so far as to give the name "pseudo-trichinosis" to the complaint, a name which has received its well-merited criticism and censure from others. In none of the cases, however, were trichinæ found in the muscles, except in Wagner's second case, and here in about three hundred muscular sections three trichinæ were found; these were encapsulated and estimated to be about four months old, so that also in this case their influence upon the production of the myositis must be excluded.

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[1] Breslauer Aerztliche Zeitschrift, 1880, p. 264.

In our case the question is effectually disposed of, not only by the absence of trichinæ from the microscopical specimens, but also on account of the long duration of the disease.

That multiple neuritis may produce symptoms similar to those observed in our case cannot be gainsaid. But if we take into consideration the facts, that during the entire course of the disease there was freedom from spontaneous pains; there was no pain produced by pressure over the nerve trunks; there were no disturbances of sensation; there was no actual paralysis of any muscles, the inhibition of movements being due entirely to the pain produced by them; there was no reaction of degeneration in any of the muscles, and, finally, considering the intense swelling of the muscles, we believe that neuritis as the primary affection may be unhesitatingly excluded. All of these negative symptoms taken together are valuable, a single one by itself of course proves nothing.

Owing to the changes found in a few of the intramuscular nerves it is to be greatly regretted that pieces of the larger nerves could not be obtained for examination, although, as above expressed, it is evident from the microscopical examination that the neuritis is secondary to the myositis.

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[1] Ueber eine bisher nicht beschriebene Erkrankung (Periarteritis nodosa) die mit morbus Brightu und rapid fortschreteinde algemeiner Muskellahmung einhergeht. Deutsches Archiv. f. Klin. Med., Vol. 1., p. 484.

The frequently cited cases of combined neuritis and myositis described by Eisenlohr,[1] as well as the recent article of Senator[2] upon the same subject, go far to prove that the inflammatory process, whether it primarially originates in the nerves or in the muscles, does in many cases not remain confined to the primarily affected tissues, but secondarily involves the muscles if the nerves, the nerves if the muscles were originally involved. In Eisenlohr's and Senator's cases the presence of disorders of sensation with paralysis of the muscles, in addition to the other symptoms, make the primary involvement of the peripheral nerves probable.

Another question, and one which cannot be disposed of so easily, is the possible relationship between our case and cases like ours, and primary progressive muscular atrophy; in short, is our case perhaps a case of progressive muscular atrophy in which the inflammatory stage is more marked, clinically, than usual, due to the myositis being more of a parenchymatous character? Would we not possibly, if we examined our cases of the chronic forms with a view particularly to the history of the initial stage, find analogous conditions to those presented by our case, or at least a history of muscular rheumatism so-called? In other words, are not some cases of primary progressive muscular atrophy the resultants of light cases of polymyositis parenchymatosis? We believe that there is considerable evidence in favor of these views, and we do not stand entirely alone in possessing this opinion. Wagner insists and Unverricht admits the possibility of their cases being acute cases of primary progressive muscular atrophy. Cases, in fact, of the acutest possible kind, Wagner's cases showing a duration of three and eight weeks respectively, and Unverricht's a duration of six weeks.

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[1] *Eisenlohr*, Idiopathische subacute muskellahmung und atrophie, Centralblatt f. Nervenheilkunde, 1879, p. 100, Vol. ii.

[2] *Senator*, Acute multiple myositis bei neuritis, Deutsche Med. Wochenschrift, 1888, p. 449. Discussion in Verein f. Innere Medizin, meeting May 28, 1888. Berl. Klin. Wochenschrift, p. 493, 1888.

None of these cases however, showed any atrophy, but it cannot be said that this might not have developed if the life of the patients had been more prolonged. At any rate the proof of a relationship between these very acute cases and the chronic ones, would be a very difficut one to furnish, unless an intermediary can be found which will serve as a connecting link. This link we believe to possess in our case.

We admit that if we compare this case with typical ones of primary progressive muscular atrophy, the disparity is evident, but we must not forget that we are dealing with an unusual case, the assignment of which to any of the well known types of disease meets with difficulty. But let us compare the two affections, and the following points of similarity and dissimilarity will become apparent. The first objectionable point in our case is the late occurrence, namely, at the age of 32. Still, we know that rare cases of the pseudo-hypertrophic form do not begin until the eighteenth or twentieth year, and that Schultze's,[1] which, although a very unusual case, he classes under this form, did not begin until the twenty-seventh year, we also know that in the primary atrophic form the occurrence may take place as late as the sixtieth year.[2] We here speak of the pseudo-hypertrophic and atrophic forms, 'almost in one breath, because we all know that the separation of the two forms is not absolutely certain on account of intermediary cases, which cannot easily be placed in one or the other category.

The second objectionable point is the mode of origin. This in all cases of polymyositis, as also in ours, was acute, a mode decidedly at variance with that of progressive muscular atrophy. But even here, exceptions to this rule are furnished by the so-called rheumatic forms, a number of cases of which have been compiled by Fredreich,[1] who has also added one of his own.

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[1] *Schultze, F.*, Ueber den mit Hypertrophie Verbundenen progressiven Muskelschwund, Wiesbaden, 1886.

[2] *Gowers*, Manual of Disorders of the Nervous System, 1888, p. 395.

The duration of the affection in the cases of Wagner, Unverricht and Hepp, was so short, that the main stumbling block for comparison seems to lie there. In one case the duration of two and a-half years is also short, but still it is a step towards the shortest duration of authentic cases of progressive muscular atrophy (8–10 years). As worthy of note is the fact that in these short cases death has generally been due to involvement of the respiratory muscles, as it was also the cause in ours.

Now the points of complete similarity may be mentioned.

(1.) There was in our case, the same symmetrical distribution of affected muscles as is found in cases of primary progressive muscular atrophy.

(2.) The degeneration always affected single muscles and parts of muscles (degeneration individuelle), and not entire muscular masses (atrophie en masse). This was very characteristic microscopically ; here this *dégeneration individuelle* could be traced down to the single bundles, where single fibres in each bundle were always seen to be affected, and not the entire bundle.

(3.) Sensory disorders were absent.

(4.) Reaction of degeneration was not present.

An endeavor to class the case among any of the so-called "types," taking into consideration the distribution of the atrophy, would be entirely fruitless, as the patient died so soon after the onset of the atrophy that the possible ultimate distribution of the same cannot even be surmised. Furthermore, we believe that the lines separating the various known groups are entirely too tightly drawn, which is clearly shown by such cases as Barsickow[2] and Zimmerlin.[3]

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[1] *Freidreich, N.*, Ueber Progressive Muskelatrophie, Berlin, 1873.

[2] *Barsicow*, Zwei Familien mit Lipomatosis muscular, progress. Diss. Halle, 1872. In one of these families the affected patients generally showed involvement of the muscles of the legs and back. but in one case the muscles of the *arms* and *back* were affected. In the other family the legs alone were usually affected, but in some members of the same family the shoulder muscles became affected first and then the muscles of the legs became involved. How are these cases to be classified ?

[3] *Zimmerlin*, Mendel's Centralblatt, 1885, No. 3. In one family, two cases of juvenile form and third beginning in face, with pseudo-hypertrophy of lower limbs.

Microscopically the results of the process in our case was seen to be the same as those in progressive muscular atrophy, namely, destruction of the muscle by inflammation as well as by degenerative processes. While last year, in our paper on pseudo-hypertrophy, we took occasion to express surprise at the complete similitude between the results of the microscopical examination in that case and those obtained by Friedreich[1] in a case of spinal progressive muscular atrophy, we this year must call attention to the great analogy presented microscopically by our case of a year ago, with the one of to-day. Without again entering upon the details of either case, we beg to note the following quotations from the paper on Pseudo-hypertrophy. "I am convinced that in my patient the disease is essentially a chronic inflammation invading both the perimysium and the muscle fibre." "I may here say that I cannot agree with Gowers and Buss, that the proliferation of connective tissue is the primary, and the disease of the muscular tissue is the secondary process. Either the reverse of this is true or the process occurs simultaneously both in the muscle and the perimysium."[2] These citations sufficiently show the similarity of the findings in the two cases. From the arguments here adduced we can come to no other conclusion than that in this case of polymyositis progression we are dealing with a form of primary myopathy, closely allied if not identical with some forms of primary progressive muscular atrophy.

The following paper was read:

PROGRESSIVE MUSCULAR DYSTROPHIES: THE RELATION OF THE PRIMARY FORMS TO ONE ANOTHER AND TO TYPICAL PROGRESSIVE MUSCULAR ATROPHY.[3]

BY B. SACHS, M D.

The diseases to be discussed in this article have passed under so many different names that it will be necessary, first of all, to state what shall, and what shall not, be understood by the term "Progressive Muscular Dystrophies."

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[1] *Friedreich*, Loc. cit., case 10, p. 37.

[2] *Jacoby, G. W.*, Microscopical studies in a case of pseudo-hypertrophic paralysis, Journal of Nervous and Mental Disease, Vol. xiv., 1887, Sept., Oct.

[3] This article, with full references, was published in the *N. Y. Medical Journal*, December 8 and 15, 1888.

This term is intended to designate those forms of disease in which a primary progressive wasting of some or all of the muscles of the body is the most characteristic feature, and in which this wasting (atrophy) may or may not be associated with true or pseudo-hypertrophy of some muscles. These primary progressive dystrophies are our chief concern; we have nothing to do with muscular atrophy following cerebral, myelitic, or peripheral nerve disease. One form of disease, however, which is undoubtedly due to changes in the spinal cord we must draw into the discussion. I refer to the typical progressive muscular atrophy. This must, in fact, be the basis upon which our discussion shall proceed, for a very large number of the cases and different forms of disease which we shall have to consider, were once classed under this term. "Progressive muscular atrophy" was for many years, and with many authors still is, a mere clinical designation, just as locomotor ataxy was a mere clinical term until the pathological anatomy of the disease was established, and the term was finally restricted to cases of tabes dorsalis.

Duchenne distinguished two forms—progressive muscular atrophy of the adult, and progressive atrophy of infancy. The latter will come up for consideration together with the new type of muscular atrophy which Landouzy and Déjérine have described and advocated. The former type remains almost in all particulars as Duchenne described it. Modern authors, including Charcot, Leyden, Strümpell, Hammond, Gowers, and others, have been able to add but very little to Duchenne's original description. The chief characteristics of this form are as follows:

Progressive Muscular Atrophy (type Aran-Duchenne).—This form begins in a large majority of cases with an atrophy and corresponding weakness in the small muscles of the hand (thenar and hypothenar). The atrophy spreads from muscle to muscle ("atrophie individuelle"). Beginning as a rule with the adductor pollicis longus, it involves next in order the opponens pollicis and deep muscles of the thenar; from these it extends to the hypothenar, the interossei, the flexors and extensors in the forearm. At this stage the disease may remain stationary or it may spread to the flexors in the upper arm, to the deltoid, possibly the triceps, and finally to the muscles of the trunk, the shoulders, and the back. Duchenne recognized the fact that the atrophy may begin in exceptional cases in the trunk, in the shoulders, or in the legs. Certain it is that in those cases in which the atrophy begins in the hands, the legs are not affected until very late in the course of the disease. One marked exception to this rule has occurred in my own practice in the case of a woman aged 40, in whom the atrophy attacked almost simultaneously the small muscles of the thenar and the anterior muscles of the thigh. This case had all the other symptoms of typical progressive muscular atrophy.

The atrophied muscles in progressive muscular atrophy exhibit fibrillar contractions and for a long time retain their faradic contractility. There may be a diminution of faradaic or galvanic excitability proportionate to the wasting of some muscles, and a complete or partial reaction of degeneration may be present in other muscles. The march of the disease is steadily progressive. Heredity is a strong factor in the disease, as is shown by the remarkable series of cases published by Naunyn and Eichhorst in the *Berliner klinische Wochenschrift*, and by the account of the Weathersbee family given in the later editions of Hammond's treatise, although the latter cases probably belong to the peroneal type to be discussed later on. Osler's cases also give strong proof of heredity.

With the exception of the factor of heredity, all the clinical features as given above were known to Duchenne. For many years, too, the clinical features of progressive muscular atrophy were beyond question. All discussions that followed related to the question whether this disease was of spinal or peripheral origin. Duchenne first regarded the disease as of peripheral origin, but in his third edition retracted this view, convinced, as he says, by the pathological and anatomical facts gathered by Charcot and Joffroy, Lockhart Clarke, Hayem, and others. To Clarke, and above all to Charcot and his school, we owe the advances made (in the years 1860 to 1870) in our knowledge of the pathology of progressive muscular atrophy.

The main changes found are these: a sclerotic and pigmentary atrophy of the ganglion cells of the anterior horns; inflammatory changes in the neuroglia; increased size of the blood-vessels, and proliferation of the cellular elements. In fresh preparations granular corpuscles are found, and according to the degree and stage of the disease the anterior gray cornua are reduced in all diameters, and the ganglion cells either atrophied or entirely lost. The anterior nerve roots are affected secondarily to the lesion of the gray substance. The nerve fibres are not all destroyed, a number of them remaining intact. Those that are destroyed exhibit the appearances of simple atrophy—a point to which Charcot alludes as distinguishing these cases from infantile spinal paralysis.

The theory of the disease was and is, that the inflammation spreads slowly from the ganglion cells of the anterior horns along the anterior nerve roots, without destroying as many of these fibres as is the case in infantile poliomyelitis. The atrophic changes in the muscles are, on this hypothesis, the direct result of the irritation which begins in the cells of the anterior horns and is propagated thence through normal or only half wasted nerve roots to the peripheral muscular fibre.

The earlier pathological investigations erred in various respects; first of all that changes in the spinal cord were not noted, the white columns of the cord were not carefully examined; in consequence of this inadvertence in the examination of pathological specimens and on account of insufficient clinical description many cases of amyotrophic lateral sclerosis were recorded as cases of progressive muscular atrophy. It is Charcot's great merit to have done pioneer work in this, as in so many other neurological problems. In France, Charcot succeeded in making his *tephro-* (*polio-*) *myélite chronique parenchymateuse* the anatomical substratum of Duchenne's progressive muscular atrophy.

From this time onward, German investigators play a very important rôle in the solution of the problem under discussion, attacking the problem both from the pathological and from the clinical standpoint. Bamberger and Recklinghausen published two cases of Duchenne's atrophy in which no changes could be found in the spinal cord post-mortem, but it was not until the appearance of Friedreich's great monograph that the possible peripheral origin of progressive muscular atrophy was again pushed into the foreground.

Friedreich claimed that the changes found in the anterior nerve roots and in the anterior cornua, in cases of progressive muscular atrophy, were secondary changes, and to this he allowed no exception. According to Friedreich's views, progressive muscular atrophy is a primary chronic myositis which is followed in due course of time by secondary changes in the nervous system. The intermuscular nerve filaments are the first to be affected, and from these nerve filaments an ascending neuritis travels along the peripheral nerve trunk to the anterior roots of the spinal cord segment; the neuritis of these anterior nerve roots may spread to the cord and here set up chronic myelitic changes which will vary greatly in degree and distribution; the extent and character of the changes will, according to Friedreich, depend upon the extent of the muscular affection. The changes in the peripheral nerve fibres and in the ganglion cells of the anterior horns are the result of the impaired motor functions of the affected muscles (op. cit., p. 118 and 124).

On this theory alone, Friedreich insisted, can we explain why in certain cases a widespread muscular atrophy is associated with changes in the cervical segment only, as in the cases of Dumenil, of Lockhart Clarke and Gairdner, in the cases of Clarke and Cooper, Clarke and Johnson, and others, in which changes were found in the spinal cord, and none in the nerve roots. Friedreich claims that the nerve roots were not properly investigated; on the other hand, the cases of Recklinghausen, of Friedreich and Cruveilhier, of Trousseau and his own cases

(Nos. 4 and 21), proved to *him* that changes may occur in the muscles themselves, or in the nerve trunks and anterior nerve roots, and not in the spinal cord ; but Charcot (op. cit., p. 209,) very correctly protests that all these cases upon which Friedreich's proof rested were examined before the present successful histological methods for staining the spinal cord had come into vogue, and that they, therefore, prove nothing.

While Friedreich's judgment unquestionably erred in regard to many of these cases, the error can be explained, since many of the cases upon which he based his views are now known to belong to other forms of muscular atrophy in which there is *no* accompanying change in the spinal cord. As regards typical progressive muscular atrophy the investigations of later years have put the spinal origin beyond question, although as Schultze has shown in his excellent monograph there are but two cases of Duchenne's atrophy (cases of Pierret-Troissier and of Strümpell in which the anterior gray matter was the *only* part affected and alone responsible for the widespread muscular atrophy. To this last we might add the case of Wood and Dercum, if the clinical history were not unsatisfactory. Schultze arrives at his conclusions by excluding even those cases in which the nuclei of the medulla had become involved by extension of the process. Without wishing to depart from the subject before us, I may intimate that these pathological researches prove that although progressive muscular atrophy is of spinal origin, and is a distinct clinical entity, it is not necessarily a morbid entity, and in most cases represents an early stage of one of several spinal cord diseases.

It is now time to retrace our steps and note the development of our knowledge regarding pseudo-hypertrophic muscular paralysis.

The history of this form can be related in few words. The clinical features as laid down by Duchenne, Griesinger, Seidel, and others have been universally accepted. These authors and all who followed them fastened upon the increase in the size of some muscles as the characteristic symptom of the disease, and have largely disregarded the widespread muscular atrophy which is present in many cases of pseudo-hypertrophy.

The earliest cases of pseudo-hypertrophy of muscles were described by Meryon in 1852. Similar cases had been described by Charles Bell in 1830, but were not valued at their true worth, and Meryon even claimed that his cases were intimately related to Cruveilhier's (Aran-Duchenne's) atrophy. Oppenheim in 1855 published a thesis at Heidelberg on progressive muscular atrophy in which he reported a number of cases of pseudo-hypertrophy, without, however, making a distinction between these cases and Duchenne's type. It was Duchenne again who, in a paper (22) published in 1861, first called attention to the increase in the volume of certain muscles as the important feature in the disease, and in his "Electrisation localisée" established this type of disease for all times. Since that time innumerable cases have been published, enabling Gowers in 1879 to base his studies upon a series of 220 cases; some of these, however, evidently belonging to other categories. The clinical features have been verified so many times over that we need not in this paper analyze all the cases, but can without hesitation present the general features of the disease.

Pseudo-Muscular Hypertrophy, or pseudo-muscular sclerosis (Jaccoud), atrophia musculorum lipomatosia (Seidel) is a disease of early youth, the vast majority of cases beginning before the age of six. Boys are affected somewhat more frequently than girls, and there is good proof of heredity. The disease, although largely affecting boys, being most frequently inherited through the mother. Meryon's cases appeared to form an exception (vide Gowers, op. cit., p. 24). The first symptoms are a weakness in the muscles of the leg, a waddling gait, and an apparent increase in the size of some of the muscles of the leg. In many cases the calves only are hypertrophied, in others the calves and thighs, and in rarer cases, like one now under my observation, the disease is limited to, or at least begins in, the thigh muscles.

Author's Case I.: (Pseudo-Hypertrophy).—A.K., aged 10; mother has six children; one died of "brain fever," and one of croup. Four living; one older than patient; all healthy. No history of heredity. Patient, a stout child, a newsboy, had first teeth at four months; when one year old began to walk. At one and a half years showed weakness and could not walk alone, was provided with some sort of machine with which he learned to walk. Was treated for rickets. Youngest sister has distinct rickets at present. No change until last December, when parents noticed that he was getting lamer. Mother states that thighs were always large; had difficulty in finding trousers that would fit the boy in the thighs. Boy could never walk as other children did and could never run after others. He now complains of great fatigue and when walking throws himself down on the grass from mere fatigue. Examination shows increase of volume of anterior thigh muscles of both sides, most marked in the middle portion of the vasti. Calves not hypertrophied; no other atrophy anywhere except in the serratus anticus of the right side. Grasp of both hands normal; knee jerks present; all electrical reactions normal. With the assistance of Dr. Peterson I excised two pieces of muscle from the left vastus externus, which will be referred to in a later section of this paper. The wound healed readily, boy complains of greater weakness in the leg from which pieces of muscle were removed.

Duchenne made out three stages of the disease. In the first, difficulty in standing and walking, and weakness of muscles of lower extremities and of sacro-lumbar region. In the second stage the hypertrophy becomes the prominent feature, spreading to various muscles of the body, and in the third stage there is increased feebleness of the muscles of upper and lower extremity of the trunk. Other authors recognize a weakening of the sacro-lumbar region and in a general way a weakness of the upper extremities, but in view of Erb's recent studies it is due to Gowers to state that he called attention to the fact that in many cases of pseudo-hypertrophy the "infraspinati and deltoids are often increased in size. . . . The latissimus dorsi is commonly much wasted, and so also is the lower (sternocostal) portion of the pectoralis major. . . . The forearm muscles are rarely affected."

To complete the clinical picture we must in addition refer to the lumbar lordosis (probably due to the weakness of the extensors of the hip), to the occasional presence of contractures, and to the peculiar difficulties in rising from the ground (the patient climbing up upon himself) which are present in some cases, but not necessarily in all, and to which Gowers attaches too much importance in making it the cardinal symptom of the disease. My patient has distinct pseudo-hypertrophy, but rises from the floor with the greatest ease. In a general way it is to be noted that there are no fibrillar contractions in the affected muscles, no changes in the electrical reaction, except diminished excitability to both currents, no sensory disturbances, and the patellar reflex may or may not be present. As a typical example of pseudo-hypertrophy and for some special reasons I will cite the following case now under my observation:

Author's Case II.—M. K., girl, aged 12½; mother has one other child living and healthy. One son died at age of 24 of meningitis. Patient first seen by me two years ago. History showed that child had severe fright at age of ten months. Child has always been very nervous; learned to stand and walk at usual age, but had diphtheria at age of four, since when the disease has become much worse. Legs first grew thin. The calves increased in size about four years ago. Child has always had characteristic difficulty in walking and rising from the floor. Examination shows decided weakness in posterior group of leg and thigh muscles; calf and thigh muscles distinctly hypertrophied. Nerves and muscles of lower legs react well to faradic current, much more readily on indirect than direct excitation. No atrophies anywhere in the body, none around shoulder girdle, hands normal. Child has difficulty in getting upon a chair and in descending comes down with a bound.

Thigh, left, $13\frac{10}{16}$ inches; right, $13\frac{3}{16}$ inches.

Calves, left, $10\frac{1}{4}$ inches; right, $10\frac{5}{16}$ inches.

Examined the child again after two years; found condition very much the same. Thighs, left side, 16 inches; right, $15\frac{1}{2}$. Calf, left side, 11 inches; right, $10\frac{1}{2}$, showing that the growth of the calf muscles has not kept step with the growth of thigh muscles. Muscles of calf and anterior thigh muscles still appear large. Resistance to passive movements very much diminished, particularly in extensors of thighs. Atrophy of sternal portion of the sterno-cleido-mastoid, left shoulder stands out more prominently than right, but shows no hypertrophy. All arm and forearm thin, distinct atrophy in the muscles of the interosseous spaces, grasp very weak, right 18, left 18. In walking, both feet assume valgus position. Arms are in marked contrast to legs. Length of arms, 25 inches; length of legs, 28 inches. Electrical examination: All muscles respond promptly to faradic current, except interossei and vasti of both sides, which require very strong currents. Galvanic response diminished in interossei and in muscles of thenar but formula not altered.

Having agreed to accept the foregoing description and histories as typical of what is ordinarily called pseudo-muscular hypertrophy, we must now devote a little more attention to the pathological anatomy of the disease. Cases of pseudo-hypertrophy with autopsies are relatively few, and for that reason the evidence must be carefully sifted.

Middleton, in his very carefully prepared paper, collected seventeen cases of pseudo-hypertrophies with autopsies; one of these must be excluded from the list as being a clear case of amyotrophic lateral sclerosis. Schultze (op. cit ., p. 36,) has added to this list the two cases of Middleton, one by Berger, two cases described by Günther, one by Pick, and one by Friedreich (op. cit., p. 347), making twenty-three cases in all.

Of these twenty-three cases, those of Friedreich, Meryon (case 2), Kesteven, Baeg, Brigidi, Ross (case 1), and of Günther must be excluded, either because the spinal cord was not examined microscopically or because the examination was not properly made. Of the fifteen remaining cases, *the spinal cord and anterior nerve roots were found absolutely normal* in ten, and in five others the changes that were found could not be held responsible for the changes in the muscles. These ten cases are unobjectionable in every point; their clinical histories are very similar in every respect and are sufficient proof of the fact that pseudo-hypertrophy of the muscles is *not* dependent upon changes in the spinal cord.

In the endeavor to increase this list, I have carefully searched for earlier cases with autopsies, in our own literature in particular, which might have escaped Schultze's notice, and have furthermore endeavored to collect cases which have appeared since the publication of Schultze's monograph, but the total increase is not great.

First of all, attention should be directed to Gibney's case, which was presented to the American Neurological Association two years ago. The history of the boy, aged 16 at death, who had been under observation for ten years, is a typical one of the disease. There was first distinct enlargement of the calves, followed later on by atrophy. A brother is affected in the same way. Dr. Amidon, who examined the cord, reports: "The only lesion appeared to be in the ganglion cells of the anterior horns. . . . About one-half of the cells seemed to have disappeared, leaving no trace. The remaining ones are poorly defined, small, and in many instances processless. . . . Lesion more marked in the dorsal than in lumbar region."

Through the kindness of Dr. Amidon, I have been permitted to re-examine the specimens, and I hope he will permit me to say that the case may be used to show that there are no *serious* cord changes in pseudo-hypertrophic paralysis. Processless ganglion cells mean as little in the spinal cord as processless pyramids mean in the cortex; and a diminution in the relative number of cells in any one section is a point exceedingly difficult to determine, and, if present, is more apt to be a secondary than a primary affair.

I hope that both Drs. Gibney and Amidon will agree to this view of their case.

The only other cases of pseudo-hypertrophic paralysis *with autopsies* which I have been able to find, were these: Westphal reported the cases of two sisters, both affected with pseudo-hypertrophy, in the one case characterized by unusual increase in the volume of many muscles. Westphal found *no changes whatever either in the cord or in the peripheral nerves*. Coming from so distinguished an author, these facts deserve the greatest consideration.

Middleton has described another interesting case with enormous pseudo-hypertrophy and a wide-spread atrophy, including even the masseters; but the cord did not harden well, and a microscopical examination could not be made. The case is, therefore, useless for our present purposes.

Further autopsies on typical cases of pseudo-hypertrophy are extremely desirable; but Westphal's cases, together with the others analyzed above, place the non-spinal origin of pseudo-hypertrophy beyond question.

These facts do not appear to be properly appreciated as yet, for we find that Dr. Inglis very recently reports several cases of pseudo-hypertrophy, and assuming that all pathologico-anatomical facts point to the spinal cord as the seat of the disease, Dr. Inglis gets over the discomforting negative facts by stating that "the cases in which the post-mortem examination shows the cord visibly intact do not invalidate this idea (the spinal origin of pseudo-hypertrophy); and that the defect in the distal ends of the motor fibres, while not in every case accompanied by atrophy of the central cells, is yet the indication of an impaired activity of those cells."

It is more surprising still to find Hammond disregarding the evidence of the last ten years, and adhering to the spinal theory of pseudo-hypertrophic paralysis, and even going so far as to entitle the disease "pseudo-hypertrophic *spinal* paralysis." Hammond's conclusions are based on cases of Barth (40), Müller, and Lockhart Clarke. Barth's case is one of amyotrophic lateral sclerosis; Muller's case was complicated by cerebral disease, and therefore useless for the determination of the anatomical lesion; while Lockhart Clark's case showed changes which are not primary, and which Gowers, whose case this was, acknowledged (in the *Lancet* for 1879) to have been possibly due to the paralysis of long standing and to the frequent pulmonary troubles.

At this stage of our studies let us note that careful clinical investigation and post-mortem examinations have shown, among other facts, that a wide-spread atrophy is common to progressive muscular atrophy, type Aran-Duchenne, and pseudo-hypertrophy; but that the absence of all changes in the central nervous system, the absence of fibrillar contractions, and the absence of reaction of degeneration, in cases of pseudo-hypertrophy, separate it widely from the former disease. Later on we shall see that a very intimate relation exists, however, between pseudo-hypertrophy and certain other forms of muscular dystrophy which were formerly included under the general heading of Progressive Muscular Atrophy.

The process of distinguishing these forms from progressive muscular atrophy was of slow development, and with the steps of this process we shall become best acquainted by alluding to a few excellent articles published between the years 1870 and 1880.

Lichtheim was one of the first to take up the cudgels for Friedreich's theory of progressive muscular atrophy. In 1878 he published a paper on a case of "Progressive Muscular Atrophy without Disease of the Ganglion Cells of the Anterior Horns."

This case of Lichtheim was followed up by one of Erb and Schultze and one of Kahler. The former authors endeavored to disprove Lichtheim's case by a case of typical progressive muscular atrophy with changes in the cord. Erb's criticisms were quite severe, but they have lost all of their force since Schultze showed in later years that the changes which he and Erb found were not sufficient to account for the muscular changes, the cells that were atrophied being now known to be in no physiological connection with the muscles that were atrophied; and, furthermore, Erb has since decided that Lichtheim's case, though a very important one, belongs to the type which Erb (34) first described a few years later. And to this most important class of cases we must now devote our attention.

Erb's Juvenile Form.—Erb described this new form of disease in his "Elektrotherapie," but sufficient attention was not paid to this juvenile form until Erb again called attention to it in a lengthy article on the subject published in 1884.

The following is a typical case of Erb's juvenile form, the history of which will bring out clearly enough the differences of this form and typical progressive muscular atrophy.

Erb's Case I.—Male, aged 46. No hereditary history, no syphilis; several acute diseases in childhood. At the age of 15 noticed that the right arm was weaker and thinner than the left. No pains or paræsthesiæ. Trouble did not grow worse until about the age of 40; at that time the legs and left arm became involved; no sensory, vesical, or sexual disturbances.

Examination revealed changes in the following muscles:

Wasted: Both pectoralis major and minor, both trapezii, latissimus dorsi, serrati ant. maj., rhomboids with exception of upper portion of right rhomboid superior, both sacro-lumbalis and longissimus dorsi, deep neck muscles, levator anguli scapulæ right > left, brachialis anticus right > left, supinator longis (both sides), triceps right > left, gluteal muscles right > left, iliopsoas right > left, quadriceps, tensor fasciæ; anterior leg muscles weak with exception of tibialis anticus; abdominal muscles, diaphragm paretic.

Normal: Sterno-cleido-mastoid, levator anguli scapulæ, dexter, coraco-brachialis, flexors and extensors of forearm, thenar and hypothenar, adductors, flexors of leg, calf muscles, small muscles of foot.

Hypertrophied: Deltoid left > right, infraspinati muscles, both teretes.

Not ataxia; patellar reflex present; no fibrillar contractions; diminished electrical excitability of muscles, but no trace of reaction of degeneration.

The other cases of Erb resemble this one in every respect, except that in at least one of his patients a later examination revealed an incipient hypertrophy of the calves.

Erb has taken the trouble to hunt through the older literature and proves very conclusively that similar cases have been described by Aran, Duchenne, Friedreich, Ross, and others, either as cases of progressive muscular atrophy or of pseudo-hypertrophy. Erb thus summarizes the chief features of this juvenile form: It is a progressive wasting with weakness of certain groups of muscles, beginning either in childhood or early youth, involving as a rule the muscles of the shoulder girdle, the upper arm, the pelvic girdle, the thigh and the back; the forearm and leg muscles remaining intact for a very long time. The atrophy may be associated with true or pseudo-hypertrophy of some muscles. Fibrillar contractions and reaction of degeneration are never present. No sensory or visceral disturbances. He adds that the wasting is distributed in a typical manner. The pectorals, trapezii, latissimi dorsi, the serrati, the rhomboids, as well as most of the upper arm muscles and supinators are apt to be wasted, while the deltoids, supra and infra-spinati are either normal for a long time or hypertrophied. The preservation, furthermore, of the hand and forearm muscles give a very striking clinical picture.

This disease Erb has chosen to call the juvenile form of progressive muscular atrophy—a very unfortunate term, since many of the cases exhibited no symptoms until the patient was well advanced in years, and others again began in early infancy. Erb's description has been accepted by Nothnagel, Schultze, Charcot, Eulenburg, Remak, Gowers, and many others.

Upon the exact distribution of the atrophy and hypertrophy as demonstrated by his cases, Erb lays the very greatest stress. According to his view, well-preserved forearms, atrophied upper arms, hypertrophied deltoids, and efficient scapular muscles would be almost sufficient for a diagnosis of his special form. In the lower legs an almost analogous wasting occurs: thighs and glutei well wasted, while leg muscles and calves are well preserved.

The question arises, whether Erb did not attach too much importance to this exact topographical distribution of muscular atrophy and hypertrophy. He claims perfect identity between his juvenile form and pseudo-hypertrophy; page 518 he says: "If this disease occurs in earliest childhood and is not associated with any considerable lipomatosis, the disease is what has been termed hereditary muscular atrophy. If it happened to be associated with early developed and excessive lipomatosis, particularly in the lower extremities, it is synonymous with so-called pseudo-hypertrophy." "But all of these forms are identical with one another and merely represent different manifestations, different march of the disease (Verlaufsweisen) and varying degrees of intensity of the same disease."

The relation to hereditary muscular atrophy I will discuss later on, but as for its relationship to pseudo-hypertrophy, is it not curious that Erb's form is so far less frequent than the ordinary pseudo-hypertrophy? To be sure, this might be explained in a number of different ways. First, the accuracy of description has been at fault in many cases. Most authors have had the hypertrophy, and that only, in mind, and have not, with the exception of Friedreich and Gowers, paid much attention to the atrophy in the upper extremities; and if detected, most authors have described the atrophy so poorly that a clinical picture such as Erb discovered cannot be made out from their description. This is true not only of older writers, but also of those that have written since the appearance of Erb's paper. I have analyzed all recent cases of pseudo-hypertrophy for the purposes of clinical differentiation, but in the fewest cases have even the functional motor disturbances been stated with sufficient clearness to permit an inference as to the wasting of certain muscles, and definite statements with regard to the atrophy of this or that muscle are entirely wanting in the majority of cases. I wish incidentally to remark that every case of pseudo-hypertrophy should be examined with the greatest care regarding the condition of the upper extremities and the smallest amount of atrophy or hypertrophy of any muscle should be distinctly noted. I have found a slight change in the faradic response of symmetrical muscles a valuable hint in getting at an incipient wasting with corresponding paresis. Such a condition would, in at least one case, have escaped my notice if I had not examined both pectorals and had found that the one gave a much more lively response to the faradic current than the other did.

And yet, allowing for all these possible errors, an examination of American cases, for instance, has convinced me that Erb's juvenile form is very much rarer in this country than typical speudo-hypertrophy is. In England, Ormerod Ross and Dreschfeld are the only ones who have described cases resembling Erb's form, and Ormerod's case contains several atypical features. In this country none have to my knowledge been published as cases of Erb's juvenile form, though as Seguin has pointed out Mastin's cases of hereditary ataxia may be cases of Erb's form. I have not been able to get at the original paper of Mastin.

During the past two years I have waited patiently for an example of Erb's form to turn up, without, however, meeting with a single one. This disease may be as much less frequent in America, as the Landouzy and Déjérine type is less frequent in Germany than it is in France. Furthermore, the thought naturally occurs to one that Erb's special form may represent in many instances a late stage of pseudo-hypertrophy, and that the majority of cases of this disease dying at an early age never reach this stage. And yet we must not forget that Erb has described several cases of his typical form beginning at a very early age; and, on the other hand again, we well know that cases of typical pseudo-hypertrophy may be associated with atrophy in the upper extremity, without this atrophy assuming Erb's characteristic distribution, as proved by my own case (M. K.) cited above.

· In view of such cases as this one and the reasoning followed above, it seems to me that the topographical distribution of the atrophy or hypertrophy cannot be depended upon to prove the close relationship between pseudo-hypertrophy and the juvenile form. And that for the present pseudo-hypertrophy deserves the rank of a special form. Their relationship seems to me, however, to rest upon several cardinal symptoms.

First. Upon a progressive wasting beginning in early life associated with hypertrophy at any time during the course of the disease.

Second. Upon the entire absence of fibrillar contractions.

Third. Upon the absence of the reaction of degeneration.

Fourth. Upon the absence of changes in the spinal cord, the autopsy in Lichtheim's case going to prove this last statement.

Fifth. Upon the occurrence of both forms in various members of a single family.

These cardinal symptoms several other forms of muscular atrophy have in common with the two forms just discussed.

We have now to turn our attention to another type, to the so-called hereditary form of progressive muscular atrophy. This type was created by Leyden and warmly advocated by Moebius. According to Leyden this form is characterized as follows:

The Hereditary form of progressive muscular atrophy attacks several members of the same family. It appears at an early age, as a rule between the eighth and tenth year, in one case not before thirty. Males are more apt to be attacked than females (the elder Eulenburg, however, described the affection in three sisters of one family). The disease begins invariably with weakness in the back and lower extremities and in these regions a wasting of the muscles is first observed. After a lapse of years the muscles of the upper extremities may be involved. Occasionally the patient may attain to an old age. Atrophy may become so extreme that the patients are absolutely helpless. The march of the disease is steadily progressive. Electrical reactions normal; no fibrillar contractions. The atrophy is associated with hypertrophy, particularly of calf muscles. No sensory disturbances, no disturbance of speech, of deglutition or ocular movements.

Leyden records the case of a man thirty-seven years of age, who had trouble in walking from early childhood on, and who had decided atrophy of back and thigh muscles, with vast increase of calf muscles, without any involvement of shoulder and arm muscles. The general symptoms were of the kind stated above. Leyden counted among this class of cases a well-known one of Meryon, the cases of Oppenheimer, Hemptenmacher, of Bernhardt and of Eichhorst; but all of these cases have been considered by most other and later authors to belong to the type of pseudo-hypertrophy. Leyden has been followed by Moebius, by Zimmerlin, by Landouzy and Déjérine, by Schultze, and others, in the description of this type; but of these Moebius and the French authors alone can be said to be advocates of this special form.

In my opinion, there is not sufficient reason to create a separate type of disease on the points laid down by Leyden. First, all forms of muscular atrophy may be and often are hereditary. This is particularly true of pseudo-hypertrophy. Second, cases with distinct heredity often start in the upper extremities, and, third, all cases beginning with weakness and atrophy in the back and leg muscles are not necessarily hereditary, as we shall see when we come to the consideration of the peroneal type of progressive muscular atrophy.

As regards the first point, in the cases of Oppenheim, Freidreich, and Hemptenmacher, the disease began in the muscles of the back, but spread to the upper extremities instead of the lower. Barsikow has described a number of cases occurring in two families. In the members of one family the disease attacked the back and leg muscles, in the other family the spreading of the atrophy was not uniform, attacking the leg muscle in one member and in another the shoulder first and then the leg muscles.

Zimmerlin (loc. cit.) published seven cases, four in one family and three in another. In one family the four cases are distinctly of the juvenile type, while in the second family the two cases began in the upper extremities, leaving the legs intact, while in the third case there was involvement of upper and lower extremities and even involvement of face muscles—an approach to the type Landouzy-Déjérine.

Schultze (loc. cit.) describes the cases of two brothers, one affected with typical pseudo-hypertrophy, and the other with a general wasting of the upper and lower extremities. In this country, Harrington has reported seven cases, in which the onset was in the legs in some, in others in the legs and arms simultaneously, and in still others the legs were affected first, and only a year later the arms. Ormerod's cases of muscular atrophy in three children after measles, might be used to show the same differences in the mode of onset.

We have therefore good reason for insisting that Leyden's hereditary form is not entitled to rank as a special type of progressive muscular atrophy; that pseudo-hypertrophy and Erb's juvenile form are distinctly hereditary, and furthermore that cases with a distinct heredity are by no means necessarily characterized by an atrophy first attacking the muscles of the back and thighs. All of Leyden's cases would properly come under the head of pseudo-hypertrophy or of Erb's juvenile form, and the peroneal type.

The next type of progressive muscular atrophy, the type fascio-scapulo-humeral, type Landouzy-Déjérine, the infantile progressive muscular atrophy of Duchenne, cannot be disposed of so easily.

Cases of progressive muscular wasting with involvement of face muscles, have always been considered rare. Duchenne described several, Remak Mossdorf, Bernhardt, Kreske, and Westphal, have each described one or two cases; but Landouzy and Déjérine (loc. cit.) have succeeded in calling renewed attention to this form, have made the most careful examinations, and have obtained a post-mortem examination in one case. For this reason it is just to refer to the features of this type as laid down by Landouzy and Déjérine, who have seen more cases than all other recent authors taken together.

The Type Landouzy Déjérine.—This form of progressive muscular wasting begins, as a rule, in early life, and in the majority of cases in the muscles of the face, giving rise to what the authors term the "*facies myopathique.*" The lips are considerably thickened, and constitute the "*bouche de tapir.*" Great stress is laid upon this tapir-mouth appearance. Later on in the course of the disease the atrophy spreads to the shoulder and arm muscles; the supra and infra spinati, the subscapularis, the flexors of the hand and fingers remain normal. The muscles of deglutition, mastication, and respiratory and laryngeal muscles, as well as the ocular muscles, remain normal. In exceptional cases the disease may begin in the shoulder or arm muscles or even in the lower extremities. The disease is distinctly hereditary. Fibrillar contractions and reaction of degeneration are never present.

In their first paper, Landouzy and Déjérine published cases occurring in two different families; in the first the disease could be followed up through five generations. Cases that are described relate to a father and four sons, five other children not having been affected. The history of one son is characteristic.

The trouble began at the age of three with atrophy of face muscles; no other symptoms observed up to the age of 17. From that time on, atrophy was noticed in the muscles of the shoulder and arm, spreading to the trunk. At the age of 21, atrophy had become extreme—"nothing but skin and bone"—*facies myopathique* and *bouche de tapir.* Sensation normal, sphincters also, patellar reflex lost, electrical excitability diminished in proportion to the wasting, but no reaction of degeneration. At the age of 24, death of phthisis.

Autopsy.—Atrophy determined as follows: frontalis, orbicularis palpebrarum, zygomatici, orbicularis oris, and buccinator of both sides (levator anguli oris normal), trapezius, deltoid (infra and supra spinati, subscapularis, teres major and minor normal) biceps, brachialis internus, and coraco-brachialis, triceps, supinator longus and extensor radialis (supinator brevis, flexor digitorum sublimis et profundus normal), extensor pollicis longus and extensor indicis (extensor digitorum communis, extensor digiti minimi, extensor ulnaris normal), abductor longus and extensor pollicis brevis slightly wasted, abductor brevis pollicis wasted, other thenar and hypothenar muscles normal. Lumbricalis distinctly wasted and interossei slightly wasted, pectorals wasted, serrati and sacro-lumbar normal. Lower extremities not so carefully examined; glutei were atrophic; no marked lipomatosis anywhere; no changes in the nervous system. Diseased muscles revealed simple atrophy of primitive muscular fibres; slight traces of increase of interstitial connective tissue and of fat. No increase in muscular nuclei.

The histories of the cases of Remak and of the other authors quoted are very similar. In some the atrophy set in in the extremities first, and in the face later on. In Remak's case both sides of the face were involved; in Kreske's the one side only.

The similarity between this form and Erb's will be apparent to every one at a glance; it is practically Erb's form plus involvement of face muscles. Erb never observed this complication in his own cases, and Landouzy and Déjérine argue that their cases are different on account of the absence of lipomatosis and the presence of facial symptoms. As for Erb never having observed the facial atrophy in any of his cases, it is worth noting that in a later paper Landouzy and Déjérine publish a case (No. 6) of their form, in which the face muscles appeared normal during life, but on post-mortem examination revealed decided morbid changes. It is possible, therefore, that the changes were present in some of Erb's cases without so excellent an observer as he being able to detect them. If this is allowed (and the French authors themselves admit the possibility of this), there is no just reason for making a separate type for such cases as they describe. They deny the resemblance between the two forms in consequence also of the invariable absence of lipomatosis; but Westphal again seems to have found a decisive case which shows that the face muscles may be associated with typical pseudo-hypertrophy, and it must be remembered that Landouzy and Déjérine grant that they have found hypertrophied fibres in some of the muscles. We cannot, therefore, see the propriety of creating a separate type such as Landouzy and Déjérine have described. There is a slight difference between their cases and those of Erb in the topographical distribution of the atrophy, and even this is doubtful; while their cases resemble Erb's form in the involvement of the upper arm and shoulder muscles chiefly, in the presence of hereditary influences, in the absence of fibrillar contractions, and absence of reaction of degeneration.

I wish, however, to enter a special plea for the recognition of still another type—*the peroneal type of progressive muscular atrophy.*

This form was first described by Charcot and Marie and, independently of them, by Dr. Tooth, of England, in a Cambridge thesis. Charcot and his associate reported five such cases, Tooth four cases, and Herringham has recently reported one case in a family in which various members in successive generations have been similarly affected. To this list I am able to add one case of considerable interest, and similar cases, although not designated by this title, have been described by Hammond (Weathersbee ail), by Ormerod, by Schultze, and no doubt some other of the cases of hereditary muscular atrophy would more properly belong to this class.

This special form of progressive muscular atrophy begins in early youth, or may, as in one of Charcot's cases, attack a person beyond the age of puberty. It shows distinct family inheritance. According to Herringham, as a rule, boys inherit the disease through the mother, as has been shown to be the rule in cases of pseudo-hypertrophy. The atrophy begins in the lower extremities, first attacking the extensor hallucis longus, then the common extensors of the toes, and then the peronei; the small muscles of the foot may be affected as well. The calf muscles atrophy a a little later, while the muscles of the thighs offer greater resistance and do not undergo atrophy until the disease has well run its course. Several years after the onset of the disease in the legs, the hands become involved; the interossei, the muscles of the thenar and hypothenar, as well as the muscles of the forearm become wasted; the muscles of the shoulder, of the neck, trunk, and face remain normal. The atrophy need not be entirely symmetrical. Fibrillar contractions occur occasionally; the reaction of degeneration is present in some muscles; the skin reflexes remain normal.

My own case is as follows:

R. J., a Russian girl, aged 12, was referred to my department at the Polyclinic by Dr. Gibney. She is the third child of healthy parents; two born later died, one of diphtheria, and one of cerebral trouble after a fall. No disease similar to the one from which this patient suffers has been known in any branch of the family. While carrying this child, the mother was considerably troubled with swollen feet and legs, possibly of nephritic origin, but is now a healthy, stout woman. The child was asphyxiated when born; no doctor in attendance. Patient began to walk at nine months; had a slight fall at the age of ten months without doing any injury to herself. At the age of three, mother noticed that there was something wrong with the right knee, and in the hospital at St. Petersberg a plaster of Paris splint was put on. This the child wore for seven weeks. She could walk perfectly well after that, played as well and ran as fast as any child. Has had a number of diseases—measles at the age of one year, small-pox at the age of four, scarlet fever at six, typhoid fever at six and a half. In spite of all, recovered and walked perfectly well. Came to this country one year ago; nine months ago fell on left hip, and for some weeks had pain in left hip. While recovering from this fall, she noticed that she had difficulty in moving the toes of the right leg. This is now five months ago. The impairment of motion gradually grew worse until the child was not able to move the toes at all. Never had pain on her right side. Her present manner of walking developed very slowly. At first sight her gait seemed to be characteristic of poliomyelitis. Child complains of fatigue, particularly in mounting stairs; no other special symptoms. Patient was a bright girl; no hysterical tendencies. The history shows that the present condition of paresis developed slowly and was not preceded either by convulsions or fever; furthermore, that there was no pain accompanying the paresis at any time. Has distinct feeling of movement under the skin.

Examination.—Girl of medium size. Upper extremities, good grasp with both hands 43. Forearm muscles and hand muscles well developed; supinators, also biceps and deltoids well marked, the latter not hypertrophied. Trapezii and rhomboids of normal strength; right pectoral a little thinner than left. Right shoulder blade shows slight winged appearance. Right serratus slightly weakened. Distinct wasting of the right leg, thigh, and of the right gluteal region. The leg muscles of the right side more distinctly atrophied than the thigh muscles. The child cannot lift toes of right foot while resting the heel on the ground. The same movement can be performed fairly well on the left side. Cannot raise herself on tiptoes on the right side, but can do so with the left foot. Posterior surfaces of thighs proportionately less developed than anterior surface. Right extensor quadriceps very weak; left weaker than normal, but stronger than on right side. Evident atrophy, therefore, of anterior tibial and posterior tibial group of right leg, of posterior thigh muscles and the glutei muscles of right side. In the lying position, 10 centimetres below lower edge of the patella, right leg, 23½ cm.; left leg, 24½ cm.; 18 centimetres below iliac, right thigh, 37 cm.; left thigh, 39 cm.

Knee-jerk absent on right side; on left side it was impossible to obtain the knee-jerk for several weeks; it is now present, however, and very lively. Occasional fibrillar contractions. No sensory disturbances anywhere. No rectal or visceral symptoms. The triceps tendon reflex present on both sides, but weak. Occasional fibrillar contractions have been noticed.

Electrical Examination.—Faradic examination of all nerves and muscles gives satisfactory responses except in the case of the right peroneal nerve which exhibits diminished faradic excitability. On faradic excitation of peroneal nerve, tibialis anticus muscle contracts very feebly. Serratus also responds more powerfully on the left side than on the right to current of moderate strength. Left pectoralis major does not respond as well as right to faradic current. Galvanic examination satisfactory. The following alone need be mentioned. Examination with the 10 ctm. square electrode: Right peroneal nerve, KCC, 2¼ ma.; AOC, 3½ ma.; ACC, 6½ ma. Left peroneal nerve, KCC, 2½ ma.; AOC, 3½ ma.; ACC, 6¼ ma. Right tibialis anticus muscle, direct examination, KCC, 7 ma.; ACC, 8 ma. Left tibialis anticus, KCC, 4½ ma.; ACC, 6 ma. Electrical examination thus shows a decided diminution of response to the faradic current, and to the galvanic current as well in the tibialis anticus of the right side, the KCC being almost equal to the ACC. Ormerod would have said that the peroneal nerve showed reaction of degeneration with regard to the anode, but this, I insist, is nothing morbid. We have, therefore, slight electrical changes in a single muscle; the other muscles of the peroneal group respond normally.

The diagnosis in this case could have rested only between acute anterior poliomyelitis, a peripheral neuritis, or this form of progressive muscular atrophy. The mode of onset, gradually and without pain, without fear or convulsions, argues against a poliomyelitis anterior acuta, as well as against peripheral neuritis. The atrophy, too, is not as great as we would expect in a case of spinal infantile palsy. All of the symptoms—the paralysis proportionate to the wasting of the muscles, the absence of the knee-jerk, and the slight changes in electrical reaction can be best explained by the diagnosis we have made. Furthermore, the disease is not retrogressive as poliomyelitis acuta would be, but gradually progressive, and the slight indications of this progression in the muscles of the trunk lend further support to the view of a progressive muscular atrophy, which is strengthened still more by the occasional presence of fibrillar contractions.

The diagnosis in such cases as these must be made with the greatest care, but I have no doubt that some of the cases which have hastily been put down as cases of peripheral neuritis will prove to be cases of this type. From poliomyelitis anterior acuta it will not be difficult to differentiate this disease, nor from neuritis. It will be more difficult to distinguish between these cases and those of a widespread atrophy following traumatic joint lesions, in which, as I have seen a number of times, the atrophy may spread with surprising rapidity and may affect the entire extremity. We must, therefore, either rely upon the history in these cases, upon the presence or absence of fibrillar contractions, or must exclude a purely traumatic atrophy in case the atrophy jumps from the affected part to some other portion of the body.

In many cases of progressive muscular atrophy of the typical form, the histories show that the disease was first noticed after some accident. The question, therefore, arises, whether it may not be possible for a typical progressive muscular atrophy to develop after a joint lesion in a subject predisposed to this disease.

We have now to consider the relations of this peroneal form of progressive muscular atrophy to the other primary dystrophies which we have discussed.

It will be seen at once that the anatomical distribution is entirely different from the four forms of primary myopathies discussed above. If the atrophy spreads to the upper extremities, it involves the muscles more after the fashion of a Duchenne's atrophy than after the fashion of a pseudo-hypertrophy or an Erb's form of atrophy. The analogy to Duchenne's form becomes still closer when we consider that this peroneal form is distinguished from the other myopathies by the occasional presence of fibrillar contractions and by alterations in the reaction of degeneration. The spreading of the atrophy from the muscles of the big toe and the small muscles of the foot to the muscles of the legs and thighs reminds one of the manner in which the atrophy spreads in the upper extremities in cases of typical progressive muscular atrophy. There seems therefore, to be good reason to separate this form from the simple muscular myopathies and to make it a subdivision of typical progressive muscular atrophy. This form might be properly entitled the leg type, in contradistinction to the hand type which would represent the ordinary form of Duchenne's progressive muscular atrophy.

If the ordinary progressive muscular atrophy is a poliomyelitis anterior chronica cervicalis, the leg type might represent a poliomyelitis anterior chronica lumbalis. But this is speculative pathology and needs corroboration, as indeed all the clinical and anatomical features of this form do.

In the preceding pages I have given an account of the commonly received forms of progressive muscular wasting. Some cases will surely be found that cannot properly be classed under any one of these heads. Schultze's case, for instance, had some of the features of pseudo-hypertrophy, some of those of Erb's form, and in the presence of the fibrillar contractions and reaction of degeneration in some muscles approached to the type of typical progressive muscular atrophy. I have had occasion to observe one case in a child about seven years of age in which there was a general wasting of all the muscles of the body excepting those of the head. The power of the legs and arms was weak, without there being any actual paralysis. There was a winged appearance of the scapulæ, but there were no other disproportionate atrophies or hypertrophies anywhere in the body. The wasting was an entirely uniform one. Such a case as this one is mentioned by Charcot in his recent volume and by Gowers in his text-book. Baeg (loc. cit.) and Oppenheim have reported cases with involvement of the face, tongue, laryngeal and ocular muscles, which it is impossible at present to classify under any of the ordinary forms of progressive muscular atrophy.

There is good reason, therefore, for allowing that there are mixed cases of progressive muscular atrophy, and that the exact rank of these cases cannot be determined at present, except that according to their cardinal symptoms, they should be classed either with .the spinal or primary myopathies.

The study of the histological changes in the various forms of progressive muscular atrophy is omitted in this abstract.

From the survey of the histological researches in various forms of muscular atrophy, we conclude that an examination of muscular changes may help us to differentiate between typical progressive muscular atrophy and the primary myopathies; and again, if Hitzig be correct, between pseudo-hypertrophy and Erb's juvenile form. There does not, however, appear to be a marked distinction between Erb's juvenile form and the remaining primary dystrophies, the histological changes in the peroneal form being still undetermined.

The argument which has been held throughout these pages leads to the following conclusions:

1. Progressive muscular atrophy, type Aran-Duchenne, is due to spinal cord disease. The peroneal type of progressive muscular atrophy bears close resemblance to this form and may possibly have a similar pathology.

2. Duchenne's type of progressive muscular atrophy might be termed the hand type, while the peroneal form would represent the leg type.

3. Pseudo-hypertrophy is not of spinal origin. Lipomatosis is a mere incident in the course of the disease, and is associated with wide-spread atrophy in various parts of the body.

4. There is a close relationship between pseudo-hypertrophy and Erb's juvenile form of progressive muscular atrophy, but not an absolute identity. This close relationship is marked by the onset of the disease at an early age, by its occurrence in various members, of a family by the entire absence of fibrillar contractions in both forms, by the absence of reaction of degeneration, and by the occurrence of lipomatosis some time during the course of the disease. They differ from each other in the distribution

of atrophied muscles and possibly in their histological conditions.

5. Hereditary muscular atrophy does not deserve the rank of a separate clinical entity, all forms of primary myopathies being occasionally hereditary.

6. The type Landouzy and Déjérine is closely related to Erb's form, the additional involvement of the face muscles not being a sufficient basis for a marked clinical differentiation.

7. Pseudo-hypertrophy and Erb's form should be regarded as the two representative forms of primary progressive dystrophies.

8. Primary progressive dystrophies are distinguished from spinal progressive dystrophies by their cardinal symptoms—the onset at an early age, the occurrence of true or false hypertrophy, the absence of the reaction of degeneration, and the absence of fibrillar contractions.

This paper cannot be properly closed without reference to the subject of classification. The term "progressive muscular atrophy" has been variously used both to designate the fact of a general and progressive muscular wasting, and also as to the proper name for Duchenne's type of atrophy. This has led to great confusion, and it would be well if the term "progressive muscular atrophy" were to be used in a generic sense merely, and if some other name were found for Duchenne's type. Erb's suggestion seems to me to be a good one, and I therefore propose to designate the type Aran-Duchenne as spinal progressive amyotrophy.

If my argument against the validity of anatomical distribution of atrophies or hypertrophies as a basis for classication be accepted, the classification of muscular atrophies could be reduced to the following simple form:

1. Amyotrophia spinalis progressiva;
    a. Hand type;
    b. Leg type = peroneal form.
2. Primary progressive dystrophies;
    a. Pseudo-hypertrophy;
    b. Erb's form.

DISCUSSION ON DRS. JACOBY AND SACHS' PAPERS.

DR. P. C. KNAPP distinguished between the primary and the secondary myopathies. There was hypertrophy of the fibres in the former and atrophy of the fibres in the latter. The primary process in primary muscular dystrophies was not the chronic interstitial inflammation. The increase of nuclei in the fibre indicated an inflammatory process, and it was more in accord with our present idea of these degenerations to suppose that this was primary and the interstitial change secondary. This would reduce the process to the type of similar degenerations in the liver and kidneys and in the cord, in tabes, Similar degenerations might affect the cortex, perhaps; the spinal motor tract, possibly; the peripheral motor nervous mechanism, certainly; and also the muscles. The primary myopathies of the muscles were analogous to the changes in other parts of the motor tract.

I think that we could yet divide clinically cases into those of peripheral or central origin. Dr. Sachs had said that there were no fibrillary contraction in the former. When the speaker returned to New York he would be glad to show him a case in which there were such fibrillary contraction. Even pseudo-muscular hypertrophy was mixed with atrophy. He thought it wiser to acknowledge no conclusions rather than to adopt false conclusions, even though they should be those of Dr. Erb.

DR. W. A. HAMMOND, in reply to Dr. Sach's criticism of his book stated that the first edition of this book was written eighteen years ago, and the last edition four years ago. It was therefore hardly fair to bring him into court for any conclusions therein contained. He did not agree with the trenchant manner in which the author had dismissed the harpoon. All we knew upon the subject had been obtained by means of the harpoon. It was easier to criticise than to experiment. Later he might accept the classification presented, but for the present he would not change, but would sustain the position of Dr. Gray.

DR. G. L. WALTON referred to a case of the peroneal type and yet myopathic in origin. Heredity was traced into the beginning of the seventeenth century. The patient was 62 years of age, and had been affected for twenty years. Her mother had died at 87, having been affected since her thirty-fifth year.

DR. J. J. PUTNAM reported a case of hereditary dystrophy, in which there were no gross signs of pseudo-hypertrophy but the microscope showed hypertrophic as well as atrophic fibres. Specimens were exhibited.

DR. SACHS reminded Dr. Hammond that he had been appointed to open the discussion, and hence had had to assume a critical attitude. He had only intended to make the broad distinction between spinal and primary. The presence or absence of fibrillary contractions and of the reaction of degeneration were important and had distinct bearing upon the differential diagnosis.

Dr. GEORGE W. JACOBY.—In discussing this subject of primary myopathies, a subject which during the last few years has received so much attention at the hands of competent observers, and of which the present state of our knowledge has been so thoroughly and ably set forth today, it would almost seem that personal views and opinions only, can be given by each participant, as American literature upon the subject is very scant, this being due to a great extent to the rarity of certain "types" in this country. This, at any rate, has been my own experience; for, outside of cases of pseudo-hypertrophic paralysis in its various stages, and some cases in which the myopathic origin was doubtful, I have seen little.

I can only consider it a misfortune that the tendency has for so long a time been to subdivide the muscular atrophies more and more, not resting content with a simple division into spinal and primary muscular form, but also to subdivide these latter, until every deviation from the so-called typical course must needs receive a special designation and be looked upon as a special form of muscular disorder, with the gratuitous hypothesis by some that each special form is dependent upon a special cause. This tendency has happily during the last few years, owing to the efforts of Charcot and Erb, received a decided check, so that to-day only can we say that we are on the right path towards a correct comprehension of the subject. How much still remains to be decided is shown by the fact that even the fundamental division into spinal and muscular forms cannot in all cases be sharply made. The possibility of this division has been considered unassailable; and, although it cannot be denied that there are many cases in which we can at once say that they are purely spinal, many others that are purely muscular, still there are two classes of cases which under certain circumstances may make the differentiation as to their spinal or muscular character impossible during life. The cases to which I refer are, first, those in which there are no distinct nervous systems, in which there is no heredity, which show neither hypertrophy nor pseudo-hypertrophy, begin in the shoulder muscles, and occur in adults. The second class consists of the type described by Charcot and Tooth as peroneal, and here I include also the cases of Schultze published in 1884. Schultze is perhaps more justified in considering his cases peripheral than is Tooth; for, in the cases of the former observer, fibrillary twitchings were absent. Whether these cases are really of peripheral origin, as assumed, is a question upon which I have my serious doubts. The difficulty in separating the just-mentioned forms becomes apparent when we consider that fibrillary twitchings and reaction of degeneration have also been found to be present in certain purely muscular forms.

If then we have trouble at the very beginning of our classification, how much more difficult will it be to prove that the various forms of primary myopathic disorders are really different "types," and not simply variations in the localization of one and the same process. It seems to me, from all we now know, that if we take into consideration the published cases of the juvenile and of the pseudo-hypertrophic form, each with involvement of the facial muscles; Duchenne's infantile forms now considered neuropathic, varying to such an extent that they as often show the juvenile as the pseudo-hypertrophic character; cases of pseudo-hypertrophy which later show a complete picture of Erb's juvenile form; cases of pseudo-hypertrophy occurring in adults, and classed under the juvenile form by Erb; finally, the various forms occurring in one and the same family (Zimmerlin, Barsickow),—considering these facts, I believe that we must admit a clinical entity, that all these so-called types are simply fortuitous localizations of one affection, no matter in what light the process itself may be considered. The difficulty in understanding these myopathies is, however, not solely due to the varying clinical characteristics, but also to our lack of definite knowledge regarding the minute muscular changes,and on account of the darkness surrounding their pathogenesis. Microscopically, we find a coincidence of appearances which can only fortify our position as regards the unity of the various forms. Muscular examinations have been confined chiefly to cases of pseudo-hypertrophy, under which I include Schultze's very complete examination of his atypical case, and the case of infantile muscular atrophy by Landouzy and Déjérine (Revue de Méd., 1888). In addition to this, muscular examinations of the juvenile form have been made by Erb and Hitzig during the last few years. In all these examinations attention has been mostly given to the changes found in the connective tissue, and the changes in the muscles themselves have been very much neglected; the only differences found in the various forms being that now the formation of fat or proliferation of connective tissue preponderates, now there is simple atrophy in the muscular fibres, and again, in other cases, waxy, fatty, or fibrous degeneration.

More or less the same changes are always seen by various observers in the various cases, but the interpretation of that which is seen is not always the same. It would seem that the only reliable differential point, microscopically, between muscles from the spinal and those from the muscular forms lies in the presence or absence of hypertrophic fibres. That is to say, the presence of hypertrophic fibres always excludes the spinal origin of the disease, the absence, however, of such fibres not proving anything, as shown by my case of pseudo-hypertrophy published last year. I myself do not believe that this test is always reliable, particularly in view of a case recently published by Hitzig. This case was one which began with paræsthesias and was followed by loss of use of the upper extremities through atrophy in a year and a quarter. Finally symptoms of severe irritation occurred, cramps and extended muscular twitchings with increased reflexes. The periosteal reflexes of the lower extremities and the patellar tendon reflexes were very much increased. This case, which I think we all would diagnose as spinal, Hitzig diagnoses as muscular, for the only reason that he found hypertrophic fibres present microscopically.

I think that, allowing the diagnosis between the spinal and muscular seat of the affection to depend entirely upon a single symptom, is going entirely too far, particularly when the clinical symptoms are at variance with that which we are accustomed to see. Now, as regards the microscopical changes found in the muscles, we must, I think, differentiate between the processes which seem to occur either alone or together. These processes are primarily degeneration and inflammation, processes which, in my opinion, to a great extent determine the clinical symptoms and course of the disease. It would appear, *a priori*, that, in examining muscular tissue microscopically, there ought not to be any difficulty in deciding what is inflammatory and what is degenerative. As far as the changes produced by acute inflammation are concerned, I think no one will doubt the possibility of diagnosing this process microscopically. My paper on polymyositis presented to-day clearly illustrates that. In my specimens of this case we can trace the process almost step by step. We have here, referring to the muscular fibre itself, a proliferation of nuclei in the body and at the periphery of the fibre, without any change being observed in the surrounding contractile matter. These nuclei increase until we have the appearance of a giant cell, the sarcolemma being still present. Then the muscle fibre breaks up into indifferent or medullary corpuscles, some of which are still nucleated. The sarcolemma sheath is now indistinct, and it is difficult to clearly separate the muscle fibre from the adjacent perimysium, which also is largely composed of similar bodies. In other places we see the process commencing, not by the appearance of nuclei in the body of the fibre, but by a breaking up *in toto* of the fibre into clusters and lumps, these then growing up to the size of nucleated medullary corpuscles.

These are changes which we see in all tissues when acute inflammation occurs, as also in secondary inflammation of the muscles from the various causes enumerated in my paper.

But when we come to the later stages—the products of the acute inflammation—then it is truly difficult to say whether we are dealing with products of inflammation or with primary degenerative processes. Every one will, however, admit that if by the side of the acute inflammation I see changes which are similar to degenerative changes, I am warranted in concluding that these changes are not primary, but are the results of the inflammation.

Unfortunately all our cases of chronic primary myopathies show little or nothing of the acute stage when the microscopical examination is made, and therefore we stand before such processes as "proliferation of connective tissue," "simple atrophy," "waxy degeneration," "fatty degeneration," without being able in each individual case to say, Is this a primary process, or is it the result of an inflammation? While now I would not go as far as Friedreich and consider every case inflammatory because an increase of nuclei is present, still I think that if I am able to show that an acute inflammation of the muscles can produce changes identical with those observed in the chronic myopathies, it is not unreasonable to suppose that some of these myopathies may be of inflammatory origin. Clinically, also, cases have been described of pseudo-hypertrophic paralysis with cure, which, in my opinion, can only be understood upon the hypothesis of an acute myositis (Donkin, Brit. Med. Jour., 1882, I., p. 537; Fawsitt, Lancet, 1887, II., p. 158). Particularly is this the case in Fawsitt's case, which presented muscular pains preceding the symptoms of the pseudo-hypertrophy.

Even Gowers concurs in the opinion that this is a true case of pseudo-hypertrophic paralysis. Now, I personally cannot conceive of a complete regeneration in a primary degenerative process, but I can easily believe that we have here an inflammation which has stopped short of degeneration.

It seems to me probable that these primary muscular affections, including progressive myositis, myositis ossificans, and also Thomsen's disease, are all due to an embryonal malformation (shown in my case to-day by the small size of all the fibres), a malformation of the muscle plate rendering the future muscles particularly susceptible to pathogenic influences. What these influences are can of course not be stated; they need, however, not be physical in character, but may be chemical, due to atmospheric influences, etc. This tendency to disease will in one patient or family localize itself in that group of muscles which is most inhibited in its development. That even gross defects in the development of the muscular system occurs is shown by the fact that the latissimus dorsi and lower half of the pectoralis are sometimes congenitally absent in cases of pseudo-hypertrophic paralysis. What form the disease will take will, in my opinion, depend entirely upon the cause of production; and if this be chemical or atmospheric (infectious), the probabilities are that it will begin as a myositis; if due to other causes, dependent perhaps upon an altered influence of the nervous system, then we will have a primary degeneration. I cannot conceive of a primary degeneration independent of nerve influences. Any further remarks which I might be inclined to make in this direction would only be a repetition of statements already made in my paper on polymyositis.

Dr. L. C. GRAY stated that, while pseudo-muscular hypertrophy was probably of peripheral origin, he thought the question was still open to doubt. He referred to Gibney's and Amidon's cases.

Miscellaneous Notes.

NATIVE MINERAL WATERS IN KIDNEY TROUBLES.

By Dr. WILLIAM C. WILE.

Not a few of the mineral waters of the United States have become noted for their value in various diseases of the kidneys. The most prominent of these which have proved of greatest value are the Buffalo Lithia Water of Virginia and the Bethesda Water of Wisconsin. Both are warmly endorsed by the leaders of the profession of this country, the first especially in those conditions where a slushing out of the kidneys is desirable, and in renal calculi; the latter in diabetes and Bright's diseases. Of both we have had ample and extended experience, the results valuable to ourselves and patients.

In a recent outbreak of nephretic colic in our own person, the attack under the Buffalo Lithia Water, of the Buffalo Lithia Springs of Virginia, was speedily cut short, the stones quickly passed, and the debris which followed showed a thorough cleansing of the kidneys and bladder of all foreign substances. All of the reflex symptoms and sequelæ were promptly relieved, and we feel under a deep debt of gratitude to this most excellent water for wonderful relief from suffering and disease.—*New England Med. Monthly*, Dec. 15th, 1888.

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TO MEDICAL MICROSCOPISTS.

In behalf of "The American Association for the Study and Cure of Inebriety," the sum of one hundred dollars is offered by Dr. L. D. Mason, vice-president of the society, for the best original essay on "The Pathological Lesions of Chronic Alcoholism Capable of Microscopic Demonstration."

The essay is to be accompanied by carefully prepared microscopic slides, which are to demonstrate clearly and satisfactorily the pathological conditions which the essay considers.

Conclusions resulting from experiments on animals will be admissible. Accurate drawings or micro-photographs of the slides are desired.

The essay, microscopic slides, drawings, or micro-photographs are to be marked with a private motto or legend, and sent to the chairman of the committee on or before October 1st, 1890.

The object of the essay will be to demonstrate: first, Are there pathological lesions due to chronic alcoholism? secondly, Are these lesions peculiar or not to chronic alcoholism?

The microscopic specimens should be accompanied by authentic alcoholic history, and other complications, as syphilis, should be excluded.

The successful author will be promptly notified of his success, and asked to read and demonstrate his essay personally or by proxy, at a regular or special meeting of the "Medical Microscopical Society" of Brooklyn. The essay will then be published in the ensuing number of *The Journal of Inebriety* (T. D. Crothers, Hartford, Conn.) as the prize essay, and then returned to the author for further publication or such use as he may desire.

The following gentlemen have consented to act as a committee:

Chairman—W. H. BATES, M.D., F.R.M.S., Lond., Eng.,
(President Med. Microscopical Soc., Brooklyn,)
175 Remsen St., Brooklyn, N. Y.

JOHN E. WEEKS, M.D.,
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Original Articles.

THE WHITECHAPEL MURDERS: THEIR MEDICO-LEGAL AND HISTORICAL ASPECTS.[1]

By E. C. SPITZKA,

NEW YORK.

IT is when approaching the consideration of this repulsive subject that we appreciate the old writer's definition: "Man is an ape without the tail, who walks on his hind legs, is gregarious, omnivorous, restless, mendacious, thievish, salacious, pugnacious, capable of many arts, the foe of the rest of animate creation; the worst foe to his own kind."[2]

The original Bluebeard of history far exceeded in destructiveness the Whitechapel assassin. Like Tiberius the Roman emperor, he sacrificed hecatombs of children to his lusts, in a solitary chateau in France, whose ruins still mark the site of this horrible blot on humanity. He was of the family of the proud Montmorenci, a companion in arms of the Maid of Orleans, and in good repute as a soldier. The following extracts from his manuscript confession I cite from Julian Chevalier:[3]

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[1] Remarks made in discussing Mr. Austin Abbott's paper on the same subject before the Society of Medical Jurisprudence, New York.

[2] "Simia homo sine canda pedibus posticis ambulans, gregarius, omnivorus, inqiuetus, mendax, froax, salax, pugnax, artium variarum capax, animaluim reliquorum hostis, sui ipsis irimicus teterrimus."

[3] Inversion of the Sexual Sense, quoted in *Amer. Journal of Insanity*, 1887, p. 360.

"I do not know how, but of my own self, without counsel of any one, I concluded to act thus, solely for the pleasure and luxury it afforded me. In fact I found incomparable delight in murder, doubtless by the instigation of the devil. It is eight years now since this diabolical idea came to me. One day, being by chance in the library of the castle, I found a Latin book describing the lives and customs of the Cæsars of Rome. It was written by a learned historian of the name of Suetonius. The said book was adorned with pictures very well painted, which showed how these pagan emperors lived ; and I read in this beautiful history how Tiberius, Caracalla,[4] and other Cæsars slaughtered children, and took pleasure in torturing them. Upon this I determined to imitate the said Cæsars, and on that very evening I commenced to follow up in earnest and carry out the text and the pictures in the book. . . . . I abused these children for the ardor and delectation of luxury which their sufferings caused me. Afterwards I caused them to be slain by these fellows.[5] Sometimes I made them cut the throats of the children, severing the heads from the bodies. Sometimes I crushed their skulls by blows of a heavy stick. Sometimes I removed their limbs ; removed their entrails, hung them on iron hooks to cause them to languish, and while they were languishing in death, I had connection with them. Sometimes I did the same after they were dead. Oh, I had great pleasure in seeing the most beautiful heads of these children after they were bloodiest. . . . . As to those slain, their bodies were burned in my chamber, except some very beautiful heads which I kept for relics."

In this same confession he begs the king abjectly to spare his life, and allow him to expiate his crimes by retiring to a monastery, in connection with the statement, that he had retired from the king's service, as otherwise he would have been unable to resist the same furious impulse to slay the young Dauphin of France, the son of the king (Charles VII.).

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[4] An error ; he probably meant Caligula.

[5] Henriet and Ponton, his assistants, who were also executed.

Whether this latter statement was false and made to work on the king's feelings, it is now impossible to determine. It assuredly conflicts with his former statement that he owed his inspiration to Suetonius. The whole tone of the confession, the frequent involuntary exclamation of "beautiful," "luxury," and such like terms, when describing the revolting and horrible, indicate to my mind, that he was nothing more nor less than a degenerated voluptuary. Certainly he was not an impulsive lunatic, for he had assistants, and continued his excesses for eight years, entrapping, outraging, vivisecting and destroying, according to his own confession, over nine hundred children of both sexes, or one every third day. I am not inclined to credit his statement about the Dauphin. If he had had the "furious impulse" at that time, who can doubt that in that age he could have gratified it over and over again on others, as he in fact did later on. It is inconsistent with what I can find in the records of similar cases that he should have had an impulse to destroy the Dauphin, without an accompanying sexual motive. It is improbable that he should reach the age of thirty-six before yielding to the impulse. It is more likely that his earlier excesses had led to a condition in which, as Shakespeare has it, "desire outliveth performance;" that he exhausted all artifices to stimulate his weakened powers; and coming across the work of Suetonius, it exercised a horrible fascination over him. No one will claim that his associates in crime were insane. But the very fact that it is more difficult to conceive how two persons could be found so brutalized and callous as to do for money or interest what de Retz did in obedience to the most powerful passion—however perverted—shows how careful we must be in allowing the horrible nature of a crime to rank as a proof of insanity. From those men to whom "the shriek of torture" of the violated virgin "is the essence of their delight," and "who would not silence by a single note the cry of agony over which they gloat," exposed in the *Pall Mall Gazette*;[6] from the country boy who, after excessive self-abuse, developed a *penchant* for intercourse with ducks, geese and other animals, his gratification being exalted by their dying agony,—there is an unbroken chain of cases, showing how the acts of Gilles de Retz and the Whitechapel murderer may evolute on a basis of voluptuous exaltation associated with sexual failure. There is the case of the Russian physician who when sober was sexually normal, but when intoxicated could obtain gratification, accompanied by ejaculation, only as the blood flowed from wounds made with a lancet on the buttocks of his mistresses.[7] Numbers of women found dead with evidences of violation, exhibit injuries pointing to an association of murderous, or shall I call it "wild-beast" instinct, with the libidinous motives of their destroyer. Thus Mrs. Ebenbauer was found with the vagina torn apart by fingernails and the left nipple bitten off.[8] The body of a young girl was found in South Carolina, about 1865–1866, frightfully hacked about the vulva, and three negroes, discovered to be the perpetrators, were lynched after confesfession. Even the pæderasts, at the height of their disgusting and forced orgasm, are guilty of similar acts. Thus the "passive" pæderast, Richeux, had had his throat cut by his "active" partner, and the dead body was then placed in the attitude of the antique statue "Hermaphrodite." Letellier was similarly murdered by Pascal, and Binel by another. Both victims showed extensive scrotal ecchymoses, proving that the murder had been preceded by violent manipulation of the genitals.[9] Indeed, it is asserted that the "active" pæderast delights in grasping and clasping the throat of his partner. Frank,[10] after carefully studying the case of a brakeman who had been committed to an asylum after having knocked down a girl, cut open her genitals to permit his entry, and failing, cut her throat "without knowing why," decides that he was of limited intelligence and not insane. In the case of Tirsch,[11] who, after a life history of insubordination, theft, and immoral attacks, animated in his fifty-fifth year by a hatred of the entire female sex because one of that sex had rejected his proffers of marriage, solicited, forced, and killed an old woman, robbing her clothes and money, and cutting off the breasts and genitals, which he boiled and ate. The subsequent history did not reveal the existence of any pronounced form of insanity, but rather an angry disposition culminating in periodical outbreaks of fury, as to whose exact nature the reporter appears to have been in doubt. In the case of a Parisian military officer, who being enamored of a woman, applied leeches to her anus and vulva, so that the flowing blood might incite his passion, sequestration was followed by furious mania, and later by dementia which continued until his death.[12] Here, as in the cases where sexual perversion and cannibalistic propensities marked the incipient development of senile or paretic dementia, the morbid nature of the act was proven by its close chronological association with marked insanity. The same cannot be said of two cases which have, I think, undeservedly been held up as reproaches to French medical jurisprudence: Menesclou and Leger. Menesclou[13] was guillotined in 1880 at Paris, after having been pronounced of sound mind by Motet, Lasègue, and Brouardel. Aged only nineteen, he had violated a girl of four, choked her, and cut the body into pieces. At the anthropological laboratory both frontal lobes, the two upper temporal, and part of the occipital gyri were alleged to have been "softened." Such extensive softening it is impossible to conceive in a young person without accompanying motor and sensory symptoms that would have placed his irresponsibility beyond a doubt. Their absence militates against the genuineness of the record. ·Leger at the age of twenty-four left home to seek a situation; instead he wandered about over a week in the woods, overcome by a desire to eat human flesh. He captured a girl aged twelve, violated her, mutilated the sexual organs, tore out the heart, ate it, drank her blood, buried the body, and denied his act when captured. He was guillotined, and Esquirol found adhesions between the pia and dura.[11] Unfortunately but little of the life history of either Leger or Menesclou can be utilized in determining what relation the post-mortem findings could have had to their sexual aberrations. This brings me to the consideration of the case of a French nobleman, who wanted but the opportunities he would have had in the middle ages to have graduated into another Marshall de Retz or a Whitechapel assassin.

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[6] Modern Minotaur, in "The Maiden Tribute," 1884. Fielding, in his "Jonathan Wild," speaks of one of his characters as "well knowing that there are certain dispositions so brutal that cruelty adds a great savour to their pleasures." Montaigne says: "Lust seeks stimulation in pain."

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[7] Tarnowsky, Die krankhaften Erscheinungen des Geschlechtsinnes. Berlin, 1886, p. 61.

[8] Tardieu, Attentats contre les meurs. 3d edition, p. 116, German translation.

[9] Tardieu, *Op. cit.*, p. 168.

[10] Vierteljahrshchrift f. gerichtliche Medizin, xlvii., p. 200, 1887.

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[11] Maschka, Prager Vierteljahrschrift, 1886, i., p. 79.

[12] Brierre de Boismont, cited by Lunier: Annales Medico-Psychologiques, 1850, p. 107, from Gazette Medicale de Paris, July 21. 1849.

[13] Affaire Menesclou, Annales d'Hygiene publique et de medicine legale, 1880, p. 439.

Donatien Alphonse François, Marquis de Sade, was born 1740. His father was distinguished as a literateur. He entered a regiment as a cadet, and was expelled on account of his immoral life. In 1772 he was sentenced to death *in contumacium* for sodomy and attempted poisoning. He evaded the death penalty by flight; but returned to France, and probably with the aid of assistants carried out a number of excesses, undiscovered, similar to the following. The strollers on one of the Parisian streets, one evening in 1777, heard groans as if from a remote apartment in a deserted house. The door having been forced, other doors were encountered which were forced in turn; and in one room a nude female was found tied down on a table and pale as death from loss of blood. There were two wounds, one at each bend of the elbow, as if made by a phlebotomist, one in each breast, and corresponding ones on the vulva. Her story was as follows: In the ordinary course of her "profession" she had been solicited by the Marquis, who invited her to sup with him and his associates. After the supper she was tied down, assured that only a little blood would be drawn, and that a surgeon, or at least one who used surgical instruments, had made the wounds in question. As soon as the blood flowed freely he threw himself on his victim. She however alarmed, cried out, and the Marquis and his accomplices fled on hearing the doors forced. Otherwise it is probable the wounds would have been stanched and the victim silenced by the means usually effective with her class. He was arrested, tried, convicted, and imprisoned in the Bastile. From here he was transferred to the asylum at Charenton for alleged insanity. In 1790 he regained his liberty, and published several prurient books.[15] It was his attempt to issue a collected edition of these works which led Napoleon I. to order his rearrest and confinement in Charenton, where he died, leaving several posthumous manuscripts of a like character.

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[11] Georget. The cases of Leger, Feldtmann, etc. Translated into German by Amelung. Darmstadt, 1827.

In his case neither periodical impulses, epileptiform nor vertiginous seizures existed. His perversion ran like a red thread through his entire mental organization. Yet I believe that a consideration of the case of the Roman emperors, whose example had so marked an effect on De Retz, Sade's prototype, will show that unlimited indulgence and absence of responsibility are competent to make sexual monsters out of mere voluptuaries. Tiberius after excessive normal indulgence retired to Capri, where he finally employed persons nicknamed Spintrii to devise modes of sexual pleasure. It is difficult to indicate in decent language the successive steps by which he reached the murderous acme. It may suffice to say that in the early part of this phase of his career an aspirant to his favors offered him the choice as a present between Parrhasius' painting of "Atalante exciting Meleager *per os*" or one million sesterces, and that Tiberius chose the former; that the performances of the "animal with three backs" was devised to stimulate his desires when they waned, the "little fish boys" when they were extinguished; and that then he entered on a course of horrible butcheries of the youth of both sexes, which served as a model to De Retz.[16] Nero, after indulging in the worst incests, dressed the emasculated Sporus as empress, and he himself acted the part which Sporus acted to him, to his "husband" Doryphorus, imitating the while the cries of a ravished virgin. He committed pæderasty with young Anlus Plantius prior to having him executed;[17] and, finally, having had men and women tied to stakes, and himself clothed in the skin of a wild beast, he threw himself on them with fierce cries to bite off their genitals.[18] A long series of these debauches could be named, who like their *numerous* modern imitators delighted in epilating the mons veneris of their concubines.[19] Epilepsy, and the form of insanity rendered popularly familiar through the case of the late King of Bavaria, were rife in the family of the Cæsars. Caligula, who had the bloodiest propensities of all, who anticipated Ludwig's architectural craze, was a true epileptic, and nothing served him better to convince him that a child he had by Cæsonia was really his than its wildness, for it bit, scratched, and tortured all other children who came near it.[20] He himself cut down an assistant at the sacrifices, laughing out wildly, as he did on another occasion among his senators, when, being asked the cause of his merriment, he replied, "because a single word from me and all these throats are cut." The existence of neurotic taint in the family of the Cæsars does not prove that their sexual aberrations were necessarily due to insanity. It shows that they are more likely to assume certain guises in the insane than in the sane. Tiberius was not a lunatic nor an epileptic, and his excesses more nearly resemble those of De Retz than do those of Caligula and Domitian. As I hinted before, example, opportunity, and license operating on a mind not strong, turned by flattery, and meeting temptation at the hands of hordes of willing purveyors, will sink into loathesome luxuriousness even when not insane. Thus Elagabalus,[21] coming from Syria, "abandoned himself to the grossest pleasures with ungoverned fury, and soon found disgust and satiety in the midst of his enjoyments. The inflammatory powers of art were summoned to his aid; the confused multitude of women, of wines, and of dishes, and the studied variety of attitude served to revive his languid appetites. . . . . A long train of concubines and a rapid succession of wives, among whom was a vestal virgin ravished by force from her sacred asylum, were insufficient to satisfy the impotence of his passions. The master of the Roman world affected to copy the dress and manners of the female sex,[22] . . . . and dishonored the principal dignities of the empire by distributing them among his numerous *lovers*."

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[15] "Justine. ou Malheurs de la vertu," Paris, 1791; "Pauline et Belval," and "Juliette," 1798.

[16] Suetonius, translated by Stahr, p. 239.

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[17] *Ib.*, p. 403. [18] *Ib.*, p. 403. [19] Domitian, *Ib.*, p. 548. [20] *Ib.*, p. 291.

[21] Gibbon, "Decline and Fall of the Roman Empire," i., p. 100.

To come down to the case of the Whitechapel assassin, there are very few cases in the literature of the subject that nearly approximate his case. None are exactly like it. Long series of murders on women, done in the same manner and committed from evidently similar motives are on record, but they were all committed in comparatively deserted localities. Only one was continued after the murderer knew that the hue and cry had been raised and skilled measures adopted for his capture. But while he mutilated his victims in the same way as the Whitechapel unknown, Bertrand selected bodies of the dead and not the living. At Gainesville, and near Austin, Texas, ten murders, terribly similar in every detail, were committed in 1887. The first blow was with an ax, and afterwards the bodies so mutilated that they fell apart on being lifted up. The killing was uniformly done in bed, the victim was, as a rule, dragged into the yard and there hacked to pieces. Most of those destroyed were colored servants. In his tenth case he failed to complete his task, the victim escaping with her life. The perpetrator has not been discovered. The cases of Andrew Bichel[23] and Bertrand resemble the Whitechapel one in the fact that both revelled among the intestines, the former of living, the latter of dead subjects. Both describe their *penchant* as irresistible and the delight they experienced as indescribable, and probably the Whitechapel fiend experiences the same. Bertrand had a periodical fever accompanied by headache, which was relieved by his violation of sepulcher, and followed by a sense of lassitude. During his fury he bruised and lacerated his hands without feeling it. The discovery of his penchant was made by himself in the following way. A young girl having been placed in the grass, the diggers were driven away by a thunderstorm; before their return he went to the grass, and, as if at random, beat the body with a switch. This gave him such pleasure that he returned two days later, dug up that body, cut it into pieces, and reburied them. In this case, as in the others, he long denied the very feature which might have convinced his physicians that he was insane, namely, the sexual motive. He had his full consciousness during his escapades. For two years he violated bodies in various cemeteries around Paris, escaping sentinels, repeatedly fired at, dislocating an infernal machine which had been set at the place where he usually cleared the walls, but finally blown up by one, escaping, and discovered by coming to have his wounds dressed. Experts who have described his and similar cases are inclined to regard them as a case of periodical mania in the guise of necrophilism[24] or sexual furor. At the same time it is admitted by the best authorities—Westphal, Krafft-Ebing and Tarnowsky—that the mere existence of anthropaphagy, necrophilism or sexual perversion when unaccompanied by other evidences of nervous or mental disease, is not sufficient proof of insanity. What shall we say of the prominent clergyman who has a prostitute chalked, so as to resemble a corpse, placed in a shroud on a catafalque, and the room hung in black; who then recites the offices of the dead, and when arrived at a certain point—permit me to draw the curtain[25] here; our record is already overloaded with bewildering horror!

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[22] Like Lord Cornbury, Colonial Governor of New York, nephew of Lord Clarendon; see Spitzka : "A Historical Case of Sexual Perversion." Chicago Medical Review, Aug. 20, 1881.

[23] Feuerbach, cited by Krafft-Ebing, Arch. f. Psychiatrie, vii., p. 302.

It is not and cannot be disputed, that the impulse to perform murderous acts may be pondered and debated over in the mind of the assassin, obscurely (to us) associated with sexual motives and finally yielded to. The case is on record[26] of a young man who, seized with the desire to associate sexually with and murder a woman, went with a prostitute, accomplished the first part of his purpose, but reflecting that it would be disgraceful to be reported and convicted as the murderer of a prostitute, he deferred the latter part. He then went to a restaurant, wrote and transmitted a letter to the police acknowledging that he had this impulse, and, indeed, before the authorities arrived, he stabbed one of the waitresses. On examination, it was found that he was subject to vertigo and to fainting seizures. Le Grand du Saulle and Falret pronounced him irresponsible on the ground of epilepsy. Laségue assumed the existence of periodical mania. I must admit that the history of this, as of similar cases, is defective in the exhibition of the *raison d'être* of the impulse, an admission which is unfortunately to be made with reference to many other sensational and intrinsically interesting and startling records.

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[24] For details see Lunier, Ann. Med. Psychologique, 1849, p. 351.

[25] La prostitution contemporaine, by L. Taxtil, p. 171.

If the inscription on a window-shutter, stating that he had twenty to kill and would then surrender himself, signed "Jack the Ripper," be really the writing and signature of the Whitechapel assassin, it may put an entirely different aspect on the case. If it be a genuine expression of intention it is impossible to account for it on the theory of impulsive,periodical or of epileptic insanity. It is not inconsistent with sexual perversion, that he might have written this to mislead. Indeed, it would not surprise me if this person were an acquaintance of an author of eminence, unbosomed himself to him, and thus utilized in a sensational tale. It would not be the first time that a subject of sexual perversion had entered the lists as a writer,[27] and no artifice that ingenuity could devise or industry execute would be too cunning for one of this class. I look upon the revelation of his identity with the highest degree of curiosity, and I am prepared to learn that, like the Texas and Westphalian assassins, he may discontinue his work and remain forever unknown. Such a mind is not immune to the influence of fear and the necessity of caution, and as regards the last phase in the history of the Texas and Westphalian assassins it may remain an unsolved alternative between latency of the impulse and suicide of the assassin. Strange motives crop out among impulsive lunatics. Singular antipathies, romantic notions of revenge, pseudo-philanthropic ideas, mysterious associations of certain numbers, may all bear a part in the horrible scheme to which the Whitechapel fiend appears to have devoted himself, if paranoia be one of his mental loads. If so, we may look for peculiarities in dress, peculiarities of writing, and peculiarities of countenance in him. But whatever theory we indulge in this wide field of speculation, one prominent fact remains as the most unprecedented in the history of murderous sexual frenzy. Newly married husbands, as in the case cited by Mead, have in this state torn their wives to pieces, and been found with the bleeding entrails wound round their hands or strung about the neck of the victim. But in addition, in the Whitechapel case, the uterus was uniformly found missing, and in one case the left kidney. What did the assassin remove these for? The greater probability is that he devoured them; the lesser that he preserved them as De Retz preserved the heads of the murdered children, as the *frotteurs* of Paris preserve the handkerchiefs they steal from the women whose odor attracts them, and as the stealers of aprons, of women's shoes, and of drawers exposed on wash-lines, establish a collection of these objects. It is less likely that he removes them for some superstitious reason,[28] or utilizes them in the preparation of some nostrum.

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[26] Annales d'Hygiene publique et de Med. legale, 1875.

[27] See the cases of Numa Numantius and the Marquis de Sade.

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[28] I have seen it stated in the Newgate Calendar quoted in Lichtenberg's descriptive text of Hogarth's works, that many English murderers believed that the devouring of the heart of an innocent child would render them immune to discovery and punishment by earthly justice. In a letter received while this article is going to press, Dr. Richard Gundry of the Catonsville Asylum referring to the witch-candle superstition of the Middle Ages, cites the following startling case of this singular belief which appears to crop out at "strange times and strange places," to use my correspondent's words. He writes: "A legal friend gave me a sketch of a trial in old slavery times of an old half-witted negro for the murder of a woman after consulting a white fortune-teller, who told him that if he melted together the uterus, one ovary, half the vulva and one mamma of a white woman, then making a candle of it that it would render him invisible when he had it during his stealing excursions. He was convicted and hanged on the Eastern shore of Maryland. The fortune-teller—as the testimony of a black was not then admitted in evidence—escaped scot-free." Of this case Dr. Grundy has promised further details.

I do not believe that the ten Whitechapel murders are the only acts of the kind of which the unknown has been guilty. Either he has performed similar acts on the living in deserted localities, where the cunning he has since exhibited so manifestly would aid him in obliterating every trace of his deed, or he has served an apprenticeship on the dead body, be he butcher, medical man, or amateur. It is not easy to remove the human uterus without a fair knowledge of pelvic topography, and he who endeavors to expose and remove that organ on the strength of an experience acquired among the lower animals need well be a good homologist.

Finally I would suggest that not the least probable theory is that the same hand that committed the Whitechapel murders committed the Texas murders. We can well picture the man to ourselves: of Herculean strength, of great bodily agility, a brutal jaw, a strange, weird expression of the eyes, a man who has contracted no healthy friendships, who is in his own heart as isolated from the rest of the world as the rest of mankind are repelled by him. Perhaps some other part of the world is destined to be startled by a series of similar butcheries, and his discovery and apprehension (the latter a task of great difficulty I imagine) will permit us to study with more satisfaction than in its present hypothetical condition this singular subject.

The English medical and secular journals have been strongly censured for attributing the Whitechapel murders to an American. Undoubtedly they did this on absurd grounds and in a cockney spirit; but to any one familiar with the Texas homicides of a year ago, the theory that both acts were committed by one and the same person does not seem unreasonable. At the last meeting I noticed among the audience two men who were undoubtedly cases of sexual perversion, who came to hear Mr. Abbott's paper; and there have been stranger freaks in history than would be the fact of the Whitechapel murderer sitting among us at this very moment.

We must not forget, in estimating the true nature of the murderous impulse, that among animals such impulses are often associated with sexual life. The female mantis religiosa devours the head-piece of her mate, while the abdominal segment (apparently undisturbed) completes the marital act. Certain female spiders devour their mates *post-coitu*. Again, the gorilla—according to evolutionists, a near relative of our species—when he has disabled a foe, delights in tearing out his entrails and revelling in massacre. In the sacking of cities, infuriated soldiers have frequently been guilty of combined acts of butchery and rape. The wild beast, as stated at the opening of this article, is slumbering in us all. It is not neccessary always to invoke insanity to explain its awakening.

NOTE.--Countless instances are on record, showing that bloody propensities and mutilation are apt to be directed against the sexual organs. The beasts who disgraced, and eventually aborted the French Revolution, after hacking the body of the beautiful and virtuous Princess Lamballe to pieces, mounted her head, hands, feet and vulva on pikes, and paraded these parts before the prison windows of her whom they styled Madame Veto (Marie Antoinette).

Society Reports.

PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, November 26, 1888.

THE VICE-PRESIDENT, CHARLES K. MILLS, M.D., IN THE CHAIR.

DR. MILLS made some remarks on the classification of

DYSTROPHIES,

arranging them into cases of muscular, neural, spinal, and cortico-spinal origin. He referred to the classifications of Erb, Gray, and Sachs. Speaking of dystrophies as complicating or added affection, he said that, in a considerable list of cases, a dystrophic condition is a marked, but not the only feature, of certain well-known nervous diseases, as, for example, glosso-labio-laryngeal or bulbar paralysis, ophthalmoplegia externa or progressive paralysis of the external ocular muscles, and true poliomyelitis in its various types—acute, subacute, and chronic.

Atrophy of the tongue is occasionally seen as an isolated affection, but more commonly in association with other diseases, such as ophthalmoplegia externa, glosso-labio-laryngeal paralysis, or regular or irregular forms of sclerosis. Whether an isolated affection or simply on incident or another disease, it may be unilateral or bilateral. Raymond and Artaud have recorded a case of unilateral degeneration of the hypo-glossal nucleus in tabes, and other cases with acute apoplectic onset have been reported. Westphal demonstrated a similar interesting specimen from a patient who had ophthalmoplegia externa and complete paralysis of both eyeballs, and atrophy of the antero-lateral portion of the left side of the tongue.

With almost every form of sclerosis described as a separate disease, a dystrophy may, at times, be associated, as his own experience had shown. In posterior spinal, lateral, amyotrophic lateral, or disseminated sclerosis, early or late may develop progressive muscular atrophy of one or more extremities; or a bulbar paralysis of either the glosso-labio laryngeal, or of the external ocular type. Such cases are included in the list to be presented at the present meeting. It might be worth while to discuss the question of the identity or not of progressive muscular atrophy and amyotrophic lateral sclerosis, some authorities denying the justice of any distinction. Charcot regarded the degeneration of the pyramidal tracts as primary, and the affection of the horns as secondary, and hence the name given by him.

Besides the joint affections which occur in the course of posterior sclerosis and other spinal diseases, cases are sometimes seen in which widespread disease of the joints is associated with equally widespread atrophy to which Gowers devotes a few pages, and which has been discussed by Duchenne, Vulpian, Paget, and others. The atrophy which commonly attends inflammation of the joints is not to be altogether explained by disuse. The joint disease may be the cause of the atrophy, or the two may be associated and due to a common cause. Paget speaks of these cases as reflex atrophies due to disturbance of some nutritive nervous centre, irritated by the painful state of some of the sensitive nerve fibres. In some of the cases of widespread arthritic atrophy the joints are extremely painful, and in some not.

Dr. Mills presented notes, and exhibited patients, illustrating some unusual forms of dystrophies.

CASE I. *Progressive muscular atrophy of traumatic origin.*—J. G., aged forty-four years, white, born in Germany, laborer, was admitted to the Philadelphia Hospital, March, 1884. In June, 1876, he fell from a scaffold and sustained severe injuries in the cervical region, for which he was treated at the Pennsylvania Hospital for two years, when he was discharged, able to do light work. In December he resumed work as a stonecutter, and considered himself well up to February, 1884, when he fell from a scaffold, and again hurt his spine, this time in the dorsal and lumbar region. During five weeks after the accident he lost all control of the bladder. On several occasions after his admission he passed blood with his urine. Four weeks after the accident he felt two constricting bands, one at the level of the nipple, and the other just below the umbilicus. The upper band tightened at the least movement of the arms, almost preventing respiration, and on attempting to rise, the lower band tightened. He complained also of a burning sensation in the soles of his feet.

In 1884 it was noted that he was able to walk; but had marked atrophy of the muscles of the shoulder and arm. The thumb was strongly flexed, and the fingers were contractured. He also had some atrophy of the muscles of the lower extremities. Recent examination shows extreme wasting of the muscles of the upper half of the body, at least as far as the face. Most of the muscles of the neck are visibly atrophied, but the trapezius and sterno-mastoid are in good condition. The muscles are uniformly atrophied in the upper half of the body. On tapping the pectoral muscles over the ribs, local elevations occur. No fibrillary tremors are noticeable. There are irreglar contractures of the hand and a striking appearance of the thumbs. The second phalanx is flexed, and the first drawn backward at a right angle. The muscles of the legs are atrophied to a less degree. Knee-jerk is exaggerated and ankle clonus marked, most on the left side. Faradic contractility is retained to a current of moderate strength. Partial degeneration reaction with galvanism is present.

CASE II. *Progressive muscular atrophy of traumatic origin.*—N. S., aged thirty-four, white, born in Germany, a sailor, during his infancy was sickly, but after the age of two years was strong and healthy. He denied any specific history. He had malarial fever in the summer of 1884, for three weeks, but recovered perfectly so far as he knows.

About three years ago while working in a rolling-mill, a ball of red hot molten iron was dropped into water and exploded, and one of the fragments struck him in the right forearm, inflicting a severe wound; the bones were exposed and the tendons laid bare. The wound healed slowly, and he was under treatment for eleven weeks. His arm was not quite healed for nearly fifteen weeks, although he was able to do some work with it. He went back to the mill and for about six months his arm gave him no trouble, when he began to notice gradual wasting and loss of power in it, which slowly extended to the shoulder, the other arm, and later to the neck, etc., as now observed.

He presents advanced atrophy of the muscles of the forearm and shoulders of both sides. The muscles of the neck generally are atrophied. When the patient bows his head, after reaching a certain point the head suddenly falls forward, and in lifting the head the muscles of the back and neck are brought into strained and unnatural action. He can only keep his head erect by resting the occiput on the back of his neck. The atrophy has not yet spread much over the lower half of his body.

He has fibrillary tremors, especially in the muscles of the chest and neck.

Both knee-jerks are exaggerated, and he has slight ankle-clonus. He complains of considerable pain in the legs and arms. His pupils are normal.

Farado-contractility is retained, partial degeneration reaction.

CASE III. *Diffused sclerosis, chiefly amyotrophic lateral, with bulbar paralysis.*—J. S., sixty-six years old, has been in the Philadelphia Hospital for many years. His mental powers are generally enfeebled, but his memory for past events is fairly good.

He has considerable tremor; his head sometimes shakes, and sometimes his trunk and entire body. Both hands are very much atrophied, the thenar and hypothenar eminences, interossei, etc., wasted. He has an apparently double wrist-drop. His fingers are usually a little flexed. He can elevate his arms, but they are weak; the muscles high up in the limbs are slowly getting worse. The left upper extremity is weaker and more wasted than the right. While his hands have the appearance of double wrist-drop, they are not completely helpless; he can with a strong effort extend his hands and fingers. The thumbs are usually carried across the palms.

Both legs are in a spastic condition, having a tendency to remain contracted in extension. The feet assume a slightly varus position. There is a spasm of the adductors of the thighs. The muscles of the legs respond to faradism, but it requires a strong current, which is probably due to the condition of the skin. His senses of touch and pain are retained. He has incontinence of urine. Both knee-jerks are exaggerated, and muscle-jerks marked.

The right pupil is larger than the left. The iridic response to light is diminished. He has no true facial paralysis, but poor control of the muscles of expression; he cannot with facility draw his mouth to one side or the other, and he has little power in the oral muscle, as in whistling. The general bulk of the tongue is small. The contours of the face are more strongly marked on the left side than on the right. He complains of difficulty in swallowing. Testing with water and bread, the difficulty seems to be in the constrictor muscles of the pharynx. His method of speaking is peculiar. He speaks with great effort, the difficulty being in phonation, as well as articulation. At times his speech is explosive or stormy in character; sometimes it is hesitating, but it is not distinctively of this character. Smell and taste are preserved.

Farado-contractility in the muscles of the forearm is retained. When a strong current is used on the extensor muscles of the forearm, while these contract, the flexors contract so much more strongly that the movements of the extensors are obscured, an observation which would seem to show that when one group of muscles are much atrophied and weakened, extra-polar diffusion of the current to antagonistic muscles might lead to the mistake of supposing that the muscles tested did not respond.

CASE IV. *Unilateral atrophy of the tongue in an old man, with senile dementia.*—J. C., aged eighty-six, a laborer, had an insane grandfather; has had acute rheumatism, and malarial fever, has been a moderate drinker, but denies venereal disease. For several months he has suffered from incontinence of urine and feces, and has had some trouble in micturition.

He has a peculiar defect of speech; when he talks his articulation is a little indistinct and tremulous. His tongue is distinctly atrophied on the left side. The imperfection of speech appears to be due to this lingual atrophy, and weakness of the oral muscles. He has some tremor of both hands and arms. He appears to be weak on the left side, but there is no well-defined paralysis. He is irascible and irritable, and it is hard to fix his attention. At times his mind seems to wander, and he is often querulous and worrying. The arcus senilis is highly marked, and his blood-vessels are extremely atheromatous.

CASE V. *An irregular form of sclerosis with hemi-atrophy of the tongue.*—J. W. J., thirty-six years old. About sixteen years before coming under observation had a chancre, and since then has had various attacks of sore throat. Ten years ago he first noticed slight numbness in the left foot beginning in the toes. This feeling extended slowly, so that the entire left side of the body became weak in about two years. He retained power, however, on that side for nearly three years, when the left arm began to fail. He began to lose power in the right leg about three years after the first attack of numbness in the left foot. In about two years later he became almost helpless in the right leg. The right arm has not been affected. He began at a date he could not fix to have tremor of the tongue, which wasted on one side. His bladder has never been affected, except that he has sometimes suffered pain during micturition. For nearly two years he was so helpless that he could not leave the house. Under specific treatment he got well enough to go out, and for five or six years has kept about the same.

Examination shows no headache. He has abundant secretion of saliva, and has fair control over the facial muscles.

The right half of his tongue is practically normal. The left half presents a remarkable appearance; it is much smaller than the right, and is irregularly atrophied, so that its border presents an unevenly eroded, or corrugated look. It is in constant tremulous motion.

The left hand always feels cold to the touch, but has a flushed appearance. Sensation in the right leg and both arms is well preserved for touch, pain, and temperature. The left hand is numb and sensation is diminished. Sensation in the face is preserved. The left knee-jerk is exaggerated; the right well-marked; ankle clonus is decidedly on the left, slight on the right; muscle-jerk is decided on the left. He walks with a cane, dragging the right leg.

Both faradic and galvanic contractility are retained. At times he complains of dizziness. When he lies down in any position but upon the right side, he feels as if he would fall face forward.

CASE VI. *Simple muscular atrophy associated with old joint disease.*—W. C., aged thirty-two, white, is very pale and emaciated. Atrophy of his legs is especially marked, in the right leg and thigh more than in the left. Foot-drop is marked on the right side. Patella reflex is retained in both legs, and about normal; ankle clonus is absent. The legs seem stiff, and the patient cannot move either of them. He is totally unable to walk or stand. The paralysis of the right leg is more complete than the left. He does not appear to be able to move leg, foot, or toe. His right leg is extremely atrophied or emaciated from the hip down. The right hip-joint shows signs of old inflammation with adhesions, and other secondary changes, so that the thigh is absolutely immovably fixed to the pelvis. On handling the hip-joint no pain is experienced. The left leg is thin, but shows much less wasting than the right. The middle of the right thigh measures 11½ inches; of left thigh, 13⅛ inches; middle of right leg, 8½ inches; of left leg, 9¼ inches. The right leg from the anterior superior spinous process to the internal malleolus measures 28½ inches; the left leg, 32½ inches. Sensation appears to be perfect. Farado-contractility and galvano-contractility are normal.

CASE VII. *Idiopathic muscular atrophy.*—D. S., aged thirty-one, white, born in Pennsylvania, is one of twelve children, six of whom died of convulsions. One sister is hysterical, and all of the others are subject to fits. The patient has two older brothers, aged respectively forty and forty-two years, affected like himself. The disease came on them in childhood, as in his own case. He had spasms from infancy until he was twelve years old.

At age of eight years it was noticed that he could not go up stairs without putting his hands on his knees. He continued slowly to get worse, soon walking with a cane. He learned barbering at sixteen, and was able to work at it for ten years. At twenty-three he had to use crutches. Until 1882 the weakness was confined to the legs; it then began to involve the arms, which pained him when he raised them to shave.

He has no difficulty in speech or swallowing. His sight is good. The face shows very slight smoothing out, and weakness of the right side. The irides respond to light.

He exhibits universal, or almost universal, wasting, although in varying degree, of the muscles below the head. His upper extremities are very thin, particularly the upper arm and forearm. His hands are also wasted, but the thenar and hypothenar eminences and interossei muscles are not so much wasted comparatively, considering the stage of the affection, as the muscles of the forearms, hands, and trunk. The deltoid muscles are also not absolutely wasted. The left deltoid is less wasted than the right. Marked atrophy of the trunk muscles is well shown when he attempts any movements of the trunk, for which he is incapacitated except to a small degree. The latissimus dorsi and the pectorals are in an advanced stage of atrophy. The muscles of the lower extremities show wasting as above—the right muscles probably in the most advanced stages. The patient thinks his disease began in the thighs. In the lower extremities are well-marked vaso-motor changes. The feet and legs are purple or reddish in color, and colder than they should be, this mottling being most marked in the region of the knees and thighs.

The knee-jerk and muscle-jerk are abolished. Faradic and galvanic contractility are retained.

Sitting, this patient can, by an effort of the will, cause the muscles of the thigh to contract almost like fibrillary or involuntary contractions, and yet he cannot use the same muscles for their usual physiological purposes. He cannot, for example, kick out, or cross the legs, and yet he can produce by a willed effort waves of muscular movement in the quadriceps and other muscles.

DR. J. P. CROZER GRIFFITH reported a

CASE OF ARTHRITIC MUSCULAR ATROPHY.

S. K., twenty-two, single, American. Father died of consumption, and a paternal uncle has had rheumatism for the last ten years. In other respects the family history is negative. The patient had typhoid fever when about three years of age. Some time in his fourth year he had a fall, injuring his back; and soon after this a posterior curvature developed, which progressively increased up to the age of fifteen, since which time it has remained stationary. About two years after the commencement of the curvature he became unable to walk, and remained so for two or three years; after this time, however, he was able to walk perfectly well, and to attend to his business, which was that of a huckster. The present illness began about a year ago with a rheumatic inflammation of the left knee, for which local remedies were employed, and which did not hinder him from going about. In a few months, however, all the larger joints grew swollen and painful. Six months ago he became unable to walk, and went to the Hospital at Scranton, where he seemed to get better for about two weeks, but after this little improvement could be noticed. While in this hospital he began to waste, and says that in about two months he was as much atrophied as when Dr. Griffith first saw him. He states distinctly that on entering the hospital, his limbs were not at all wasted.

The following brief notes were made upon his case at the time of his admission to the University Hospital, June 13, 1888:

The patient complains of constant pain in the larger joints, increased by motion. He is anæmic, and his face is thin and has a delicate appearance. The arms are much wasted, and are held flexed, being more comfortable in this position. The elbows are swollen and painful, and there is a tendency to abrasion from pressure on the condyles. There is some degree of contracture of the flexors of the forearm, especially on the right side. Motion of the shoulder-joints is painful. There is extreme kyphosis, occupying all the lumbar and most of the dorsal regions. The vertebræ are not tender on pressure. The hip and knee-joints are held flexed, but are not fixed. The left knee-joint is somewhat swollen, the right little if at all so. The legs and thighs are greatly atrophied ; the thigh measuring only seven and a half inches at its upper third. The ankle-joints appear nearly free from disease. The patellar reflex could not be obtained. The examination of the thoracic and abdominal viscera was negative, except for a few râles in the lungs, and a faint systolic murmur in the heart.

The course of the case while in the hospital is interesting, inasmuch as the various plans of treatment tried during the four months appeared to have little or no influence upon the disease.

The diagnosis of the case was, however, of great interest, for atrophy developing so rapidly and reaching such a degree, with the coexistence of a posterior curvature of the spine, raised the question whether the disease of the joints might not be of the nature of a spinal arthropathy. Yet, the long immunity which the patient had enjoyed from any evidences of disease of the nerves or cord, and the manner in which the affection of the joints had developed and progressed, rendered it much more probable that it was of a rheumatic nature, and that the case was a well-marked instance of arthritic muscular atrophy, *i. e.*, as Paget explained it, "a reflex atrophy, due to the disturbance of some nutritive nervous centre irritated by the painful state of the sensitive nerve fibres." Nevertheless, some doubt is thrown upon the genuineness of this case by the presence of decided wasting on the distal sides of the affected joints, and by the absence of patellar reflex, both of these being rather more characteristic of a neuritis, while the reflexes are usually increased in arthritic muscular atrophy. It is possible, however, as Bury has pointed out, that in addition to the joint inflammation, or independently of it, there might have developed a rheumatic neuritis of certain nerves.

DR. F. X. DERCUM reported

A CASE OF ARTHRITIC MUSCULAR ATROPHY OF GONORRHŒAL ORIGIN.

H. L., a man aged thirty-six, of average stature and rather slight build, presented himself at the University Hospital with the following history. Fourteen years ago he had a small venereal sore which healed rapidly, was not followed by secondary symptoms and was probably benign in character. Previous to and since this time he had been entirely well. In the middle of April last (1888), however, he contracted gonorrhœa. The attack does not seem to have been of more than ordinary severity and ran an average course. Three weeks after its commencement he noticed great pain and some swelling on the right elbow. One week later the right knee became similarly affected and he was obliged to take to his bed. One after another the various joints of the extremities were attacked though the patient no longer remembers their sequence. He simply tells us that shortly after the appearance of pain in the right knee, both ankles, the opposite knee, both shoulders, and the joints of the left arm were affected.

Four weeks after the involvement of the right elbow, wasting of the muscles of the upper arm on the same side was noticed. Next the muscles of the opposite arm and of the legs were observed to be growing smaller. Gradually in an order which the patient unfortunately cannot recall, the bulk of the muscles of the upper and lower extremities were involved.

He was confined to bed some eight weeks, when the pain, in a measure, subsided. It persisted, however, and is still marked at the present time. At first the weakness of the muscles was so great that even walking was impossible. In the beginning of July, however, he had improved so as to be able to take a few steps. Since that time he has gained sufficiently to walk short distances and to ascend a stairway, though the latter is still a difficult feat.

Though the pain in the joints had for a long time been insufficient to interfere with movement, his arms continued until lately to be almost useless. A constant attendance of a relative was necessary to assist him in eating, dressing, and the ordinary acts of life.

His present condition is as follows: All of the muscles of the upper and lower extremities and some of the back appear to have suffered. The wasting is most marked in the shoulders and upper arms. The face has not been attacked. The affection is quite symmetrical except in the back, where extreme wasting of the lower portion of the trapezius and of the rhomboid muscles of the right side is contracted, with but slight wasting of these muscles upon the left. Fibrillary tremors are readily observed in various situations. No change is noted in the myotatic irritability, unless it be that the knee-joint is slightly increased. Electrical examination shows that no qualitative change has taken place. Slight diminution to both currents is, however, noticeable. If the statements of the patient are to be trusted, he is undoubtedly improving. He tells us, for instance, that he has had a distinct gain in the arms during the past month.

DR. F. X. DERCUM also exhibited a patient with

A SUBCUTANEOUS CONNECTIVE TISSUE DYSTROPHY.

This case is one which has been described in full in the *University Medical Magazine*, and it is therefore unnecessary to give the details of it here. It is a case of dystrophy of the connective tissue. Here we have enormous hypertrophy of the connective tissue which is in an embryonal state; with this there are associated many of the symptoms of myxœdema. The woman noticed some three years ago that the arms were increasing in size. Some six months later she began to have pain in the right arm, and this symptom has been confined almost entirely to the right side. The pain is not in the nerve trunks alone, but diffused through the mass of tissue. Upon the right side there are also some anæsthetic patches, variable in extent. These are present both on the arm and leg. There are other sensory symptoms, such as impairment of vision, diminution of hearing, and decided impairment of taste and smell. These are all most marked on the right side. The muscles of the palm are a little wasted. There are qualitative changes in the reaction to the galvanic current.

The bulk of the enlargement is due to mucous tissue. She is the subject of crises of pain which are similar to those found in myxœdema, and these attacks are attended with hardening of the part where the pain is located. There is at present a little mass in the posterior cervical triangle of the right side, which forcibly reminds one of the supraclavicular swelling described by writers on myxœdema. I cannot feel the thyroid gland. While there is marked dystrophy of the subcutaneous connective tissue and some changes in the muscles, the skin is not involved. This is a distinguishing feature between this case and myxœdema proper. In the early history, however, sweating was scanty or absent. Slowness of thought or slowness of movement are not marked. There has, however, been decided speech involvement a number of times, which has appeared to be associated with the crises. Not only would the neck and arms swell, but the tongue and soft palate would also swell. There has also been bleeding from various mucous surfaces, as the mouth, throat, bronchial tubes, and stomach. The case, therefore, presents many of the symptoms of myxœdema. My explanation of the peculiar hardening of the tissue is that there is some obstruction to the lymph outflow. Punctures made during the attacks of pain resulted in the appearance of lymph-like fluid.

Dr. H. C. WOOD said that the wasting of muscles about an inflamed joint has long been noted. It is seen in almost every case of chronic rheumatism. Charcot has pointed out that this is independent of the extent of the joint inflammation. It may occur after very slight injuries. It is almost always the extensors that suffer, as was illustrated by the case shown by Dr. Dercum. He is thoroughly in accord with those who insist upon the necessity of amalgamating the various so-called nervous diseases. He thinks that there are very few, possibly not more than eight or nine, organic diseases of the nervous system. We make one disease of chronic inflammation or degeneration of one tract of the cord, and when the same change is found in another tract it is given a different name; or if two regions happen to be affected together, we have still another disease. We must, of course, for the sake of convenience, talk about locomotor ataxia and lateral sclerosis, etc. But, in doing so, we must understand that these are not, properly speaking, distinct diseases—but simply clinical groups of cases, each group characterized by certain symptoms due to the original position of the lesion—but the lesion identical in character, and occasionally so situated as to make cases whose clinical features partake of those of several groups.

Dr. WILLIAM OSLER regarded one of the cases of Dr. Mills as a characteristic example of amyotropic lateral sclerosis. The spinal form of progressive muscular atrophy in its later stages, usually shows signs of lateral sclerosis, and the picture presented by the patient of wasted arms and spastic legs is extremely common. In chronic cervical pachymeningitis the clinical features are very similar. The condition of the neck is no evidence that there is anything the matter with the cervical vertebræ.

There is one practical point in regard to the arthritic atrophies. He has frequently seen good results follow the use of massage, electricity, rubbing, and the like, if used early. If, however, the condition is allowed to go on for months, it may, and often does, result in permanent disability and uselessness of the joint.

It is not improbable that the case presented by Dr. Dercum may be allied to reported instances of neuritic and spinal trouble following gonorrhœa. Many years ago, Gull and others called attention to spinal troubles to which they gave the names of reflex paraplegia, in association with genito-urinary disorders. Many of these cases have been shown to be forms of myelitis, such as occur in other microbic affections.

Dr. F. X. DERCUM regarded the case which he presented as one of atrophy following gonorrhœal rheumatism. He thought that we must agree that we have two forms of inflammation or degeneration in the cord, one which originates in the connective tissue, and one which originates in the nervous tracts. We are so in the habit of speaking of the inflammation of the columns that we are apt to forget that these affections are really forms of degeneration. Doubtless in these tract degenerations the essential factor is frequently a feeble vitality impressed upon the parts during the development period. We must, he thought, make a distinction between the diseases affecting the sensory and those affecting the motor tract, but whether we make a distinction between degeneration of the upper and of the lower segment of the motor tract, seems to be immaterial. In tabes dorsalis we have degeneration of the sensory tract, and in myelopathies degeneration of the motor tracts.

Dr. JAMES HENDRIE LLOYD said that Dr. Osler had referred to the view he took of one of the cases presented by Dr. Mills, a traumatic focal lesion with secondary degeneration. If we are to assume that this is a case of amyotrophic lateral sclerosis, it differs from many cases of that affection which we have seen, in its etiology, at least. In this case there was a distinct history of traumatism, the patient being thrown by an explosion upon a pile of iron, striking the back of his neck and his arm. This was followed in a few months by rapid atrophy of all the muscles of the shoulder and arm on both sides. Later, this was followed by paralysis of a peculiar character of the neck muscles. This is followed by a descending degeneration, evidently in the lateral tracts, shown by exaggerated patellar reflex, distinct ankle-clonus, and rectus-clonus, without atrophy of the muscles of the legs.

His view is, that there was a local injury of the cervical portion of the cord, causing atrophy of the cells in the anterior cornu, and that possibly from the injury he has had a descending lateral degeneration through the motor tract. There is at present another case, very similar to this one, in the Philadelphia Hospital. It is the case of a man who fell from a scaffold, and has never walked since. He has progressive atrophy of the neck and upper extremities, with the symptoms in the legs of lateral sclerosis. The cause, in his case, is distinct, and can scarcely be denied.

TRANSACTIONS AMERICAN NEUROLOGICAL ASSOCIATION.

FOURTEENTH ANNUAL REPORT.

Continued from November Number.

A CASE OF FOCAL EPILEPSY SUCCESSFULLY TREATED BY TREPHINING AND EXCISION OF THE MOTOR CENTRES.

By JAMES HENDRIE LLOYD, M.D.,
AND
JOHN B. DEAVER, M.D.

Medical Report by Dr. Lloyd.

The following case was admitted into the nervous wards of the Philadelphia Hospital under the writer's care, early in the past spring:

J. W. G., aged thirty-five years, American born. Mother died of phthisis, father of paralysis. Patient has had the usual diseases of childhood. He denies positively ever having had any venereal disease. When fifteen years old he was struck on the head with a ball-bat, from which blow he became unconscious and was confined to bed for one week. Further details of his condition at that time are not obtainable. His fits did not begin until six years after. Fourteen years ago he had his first seizure while asleep. In this he bit his tongue. The question arises whether this was his first fit, or whether really it was not rather his first *discovered* fit by reason of the wound of his tongue. Probability is lent to the latter supposition by the fact that many of his seizures have been nocturnal. Nine months after his first discovered fit he had his first seizure during the day. After this time he had them varying in number and intensity until admitted to the hospital. He described his seizures as follows: He would have a decided sensory aura commencing in the fore and middle fingers of the left hand, extending up the arm, through the neck to the left side of the head, when the convulsion would begin. He has stopped the aura at times, and thereby the fit, by tightly compressing the wrist. The aura lasted quite an appreciable time, and gave him ample notice of the explosion.

During the time of the patient's early sojourn in the hospital his seizures were mostly nocturnal. He was conscious of many of these. He said they lasted but a short time, involving, as a rule, only the left face and arm, and that he did not always lose consciousness. He also said that he has had occasional attacks involving both sides of the body, but his accounts of these were not clear, and it is probable that his consciousness was lost or obtunded in these greater attacks. The few minor attacks, which happened in the daytime, occurred during the absence of any trained or intelligent observer, but several of his fellow-patients confirmed in the main his own account.

In order to render the diagnosis more positive and the description more exact, Dr. F. W. Talley, resident physician, began a systematic nocturnal watch upon the patient, without the latter's knowledge, sitting up in constant vigil several nights in succession. During the first night nothing was observed, although the patient said in the morning that he was sure he had had one or two slight seizures. In the second night Dr. Talley succeeded in observing a characteristic attack, which he describes as follows:

The fit commenced in the left arm. The fingers were flexed over the thumb, the hand flexed at the wrist, the forearm flexed upon the arm. The head was drawn over to the *right* side, the right arm and leg then became rigid. The head soon began to rotate to the left, the fingers of the left hand relaxed, the mouth opened and was drawn to the left side with the right angle depressed. As soon as the face reached the median line a series of clonic spasms began in the left arm and left side of the face. (In two of his most severe attacks clonic spasms were observed in his right arm.) The pupils were widely dilated and fixed. Consciousness appeared to be preserved, partially, at least, throughout. The spell was of very brief duration. Following the fit there was well-marked paresis of the left arm and left side of the face.

These memoranda by Dr. Talley very faithfully describe the main features of the attack. The frequency of the seizures, on account of which the patient had applied to the hospital, increased, and they occurred both day and night, so that they were soon observed by the nurses, members of the resident staff, and by several of the neurological and surgical staffs, who were called in consultation. The greatest number of seizures recorded in one day was twenty-eight, at which time the patient seemed to be passing into a veritable epileptic status, being confined to bed, and becoming very dull and altered in his mental condition.

The paresis of the left face and arm at this time began to be very noticeable. The face was relaxed, the angle of the mouth depressed, and the right or sound side drawn over perceptibly. The orbicularis palpebrarum muscle was not involved. The tongue was not paralyzed (?). The pupils were equal and responded to light and accommodation. The arm was perceptibly weakened, especially in the flexors of the fingers and wrist, the biceps, and the deltoid. These muscles were not wasted, and did not present any reactions of degeneration. On those days when the patient's fits were infrequent this paretic state of the muscles improved considerably in the longer intervals, and was most marked just after a seizure. There was no alteration or retardation of tactile sensibility. An examination of the eye-grounds at this time by Dr. de Schweinitz revealed nothing indicative of organic cerebral changes.

The onset of these seizures, upon which special stress was laid both in the diagnosis and subsequent surgical treatment, was always the same, and verified by numerous observations. The left hand, especially the two fingers, was the seat of the signal symptoms, both sensory and motor, and, however varied the extent of the convulsive wave in different seizures, there was never any variation from this constant initiation. The convulsive area varied considerably, from a slight twitching of the affected face and arm, with no apparent loss of consciousness, to an almost universal bilateral convulsive explosion, always worse, however, on the left side, with decided obscuration of consciousness. This loss of consciousness was not always as great as it appeared, for once after a severe seizure, during which I asked him some test questions, he answered them correctly as soon as he regained control of his muscles. The patient complained but little of headache and said it had never troubled him ; the slight degree of it from which he suffered in the hospital appeared to be an effect of his rapidly increasing seizures. He had no gastric irritability whatever.

It seemed very evident to my mind in studying this case that we had a focus of discharge in the region of the junction of the middle and lower third of the ascending frontal convolutions on the right side, possibly involving also contiguous portions of the ascending parietal convolutions in which experiment seems to have demonstrated centres for the hand and wrist. The nature of this irritative lesion did not appear very clear to me, although I was inclined to think it might be old scar tissue and thickened membranes, the results of his injury. I considered the long duration of his affection to contra-indicate a tumor, especially as he had neither headache, vertigo, vomiting, nor changes in his eye-grounds ; although the focal nature of the discharge and the more or less constant paresis of the convulsed muscles were very suggestive of a new growth. I saw no reason to doubt the man's sincerity on the subject of syphilis, but I classed him with the rest of mankind and gave him the benefit both of the doubt and the iodides. He did not improve. A consultation was held with my colleague, Dr. John B. Deaver, of the surgical staff, and an operation discussed. At a subsequent consultation with Drs. Deaver and Sinkler the operation was decided upon, with the concurrence also of Drs. Mills, Dercum, and de Schweinitz, who kindly saw the case by invitation.

On the 12th of June Dr. Deaver operated in the presence of the above-named physicians and with the assistance of Dr. J. William White. The details of the operation and the surgical aspects of the case will be narrated by Dr.

Deaver. It had been decided beforehand that the incision should be simply an exploratory one in case nothing was discovered in the membranes or cortex, unless by faradic stimulation we should succeed in locating the irritative area in the cortex, in which case it should be cut out. By following Reid's and Horsley's lines, Dr. Deaver exposed, with an inch and a half trephine, an area which appeared to include both sides of the central fissure (Rolandic) in the region of the junction of the lower and middle thirds of the ascending convolutions. This area was afterward much enlarged, especially in the anterior direction, by the Hopkins' modification of Rongier's forceps. Nothing abnormal whatever was discovered in the bone, membranes, or cortex by gross inspection. The difficulty of identifying the parts was so great that exploration was soon begun with a faradic current, and with very gratifying results. Upon faradizing a point back of the fissure of Rolando, more properly the wrist centre, according to Ferrier, muscular contractions occurred as follows: turning in of the thumb on the palm, flexion of the fingers, flexion of the wrist, extending to flexion of the elbow (biceps action). I cannot say that it was verified topographically—*i. e.*, by appearance of fissures and convolutions seen in the wound, what exact centres were here excited. It was behind what appeared to me to be the Rolandic fissure. The difficulty of identifying fissures and convolutions in a small trephine wound appears to me to be extraordinary. What is of greater importance was, however, here accomplished; the reproduction of the exact muscular movements which occur in the fit. At a point farther front and below, and in front of the fissure seen in the middle of the wound (Rolandic?), faradic stimulation caused marked contraction of the face-muscles of the affected side. The mouth began to contract, and was drawn toward the left side with a tremulous motion, and soon the tongue began to protrude toward the left corner of the mouth. Soon the left thumb began to be contracted and adducted into the palm; the fingers were contracted into the palm and about the same time the face muscles began to contract more actively, while the head was drawn to the left side, and the left eyelid began to work. At the same time the hand was gradually closed, and contraction of the forearm and arm began, while the latter was drawn from the side to an angle of forty-five degrees (deltoid action), and contractions of the biceps occurred. At no time in the course of the faradic applications, anywhere within the area exposed by the trephine and forceps, did any contraction of the leg muscles occur.

I observed especially, in making these applications of faradism to the cortex, that considerable areas of it did not appear excitable at all to the strength of current employed, at least did not give muscular response anywhere, while the two comparatively narrow points above mentioned reproduced almost exactly the muscular contractions of the epileptic seizures, and seemed to stand for more "centres" than the diagrams of those who have experimented would allow to any such narrow areas.

In the absence of any visible organic lesion it was decided to excise these portions of cortex. The possibility of a sub-cortical tumor was not ignored, but there was absolutely no evidence of such in any alteration of the vascular supply or of the consistency of the brain tissue. The parts did not bulge into the wound, nor was the color of the gray matter in any way changed. Accordingly, Dr. Deaver excised from the region back of the central fissure a portion about twelve millimetres square, carrying the incision well down to the white matter. Two small portions were removed from the excitable region anterior to the central fissure. Further exploration by means of these incisions failed to detect any tumor. My attention had not been called at that time to the distinction which Franck[1] makes between the faradic excitability of the gray and that of the underlying white matter. This distinction is that the gray matter gives rise to a series of clonic spasms in the related muscles, epileptiform in character, continuing even after the faradism is withdrawn, while the white fasciculi, when faradized, cause a tonic contraction which ceases at once on withdrawing the poles. I am quite positive that the contractions caused in our patient by stimulating the gray matter were epileptiform—and if my memory serves me, after this lapse of time, the white fasciculi at the bottom of the wound were also touched and caused but a momentary tonic contraction.

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[1] Lecons sur les Fonctions Motrices du Cerveau, etc., par le Dr. Francois Franck, Paris, 1887, p. 107.

The patient's condition after the operation may be briefly epitomized as follows, prefacing with the remark that he was watched by competent observers day and night and the nursing records kept in a book.

It was observed from the first that he slept with his left eye partly open. The legs moved freely and were never paralyzed. The left arm was markedly paretic, lying quite flaccid by his side; he would occasionally raise it by taking hold of it with his right hand. His left face was also paretic. Late on the first night he had his first convulsive movement; it was only a slight twitching of the left side of the mouth which was thus drawn to the left side. These twitchings of the face, accompanied occasionally by twitching of the left hand and forearm, continued at intervals during the first six days, when they ceased, and the patient has never had any convulsive movement whatever since. They were not so severe as before the operation, nor so widespread. About the third day there was some stiffness of the fingers, which may possibly be explained by irritation of the white fasciculi during the process of healing of the cortical wound. There was at this time, according to the nurse's records, a difference in temperature of the two sides, the left axilla being from one to one and a half degrees higher. After one of his twitching spells the patient spoke of the spells returning, but he never mentioned his aura.

On the fifth day his muscular condition was as follows: The flexors of the wrist and fingers were almost quite paralyzed. The biceps was much weakened. The pronators and supinators were paretic. When told to raise the arm he would reach for it with his sound hand, and when restrained in this he would raise the affected arm with a sort of fling and evidently with the aid mostly of the shoulder and chest muscles. All his attempts to move the paralyzed muscles, especially to close his fist, were accompanied by analogous movements of the right arm. All the muscles of expression of the left face were affected, as well as the left side of the occipito-frontalis. He had control of the orbicularis palpebrarum. When he laughed the muscles of the paretic side appeared to respond almost as well as those on the sound side; which seemed to show that a cortical paralysis is not absolute as far as bilaterally associated movement is concerned. The patient is right-handed, and has never been aphasic.

From about the sixth until the eighteenth day the patient cannot be said to have been at any time in his normal mental state. He became dull, then lachrymose and incoherent, and for a part of the time had marked maniacal delirium with hallucinations of sight and hearing. The surgical condition did not seem adequate to account for this. The operation and subsequent treatment had been conducted with strict antiseptic precautions, and the patient never had a serious rise in temperature. There appeared to be headache at times, as he frequently attempted to pull off his dressings. There was at this time much œdema of the scalp. While he was at his worst there was some priapism, and one of the resident physicians was confident that the patient had masturbated. I doubt if the patient in his condition at the time was conscious of it. The pupils were dilated and the eyes expressionless. There was one involuntary passage of urine. During his most delirious and restless stage it was thought that he did not move his left leg as much as the right, but if so, this was the only time the leg was affected. His left face became much more flushed than the right. From this ominous condition he began gradually to improve toward the end of the third week, until he could sit up, and so gradually began to get about. By the end of the fifth week he was practically well, and had recovered some of his lost muscular power.

The following memoranda have been made quite recently (three months after the operation) of the patient's condition. He has had no convulsive seizures whatever since his convalescence.

Sensory condition. (Patient blindfolded). In the left, or affected hand, he feels the slightest touch with the blunt points of an æsthesiometer. There is no retardation. On the forefinger he does dot discriminate the blunt points one inch apart, but he tells the sharp points one-quarter inch apart. In the other fingers and on the right hand he discriminates better. With weights varying from *two to twelve* ounces, patient is able to tell the heaviest by cutaneous pressure as well on affected as sound side. (The paralyzed hand has a more delicate skin from disuse.)

The patient is not able to distinguish form when an object is placed between his forefinger and thumb; thus he appears quite unable to tell a small square object, a silver quarter, a silver dollar, a small flower, or a penknife. It is evident, however, that this is not a sensory but a muscular defect, because his fingers are still so paretic that he holds these small objects in the most awkward way, and cannot move or twist them about in his fingers; hence he is not able to bring his sensory nerve endings in rapid contact with the outlines of these things. This cannot, therefore, be quoted as a proof that muscular sense is in the motor cortex. His sensation to pain and heat is perfect.

Motor condition. With a dynamometer his right hand registers 130, his left hand 20. He makes a great effort, straining even with his facial muscles. The paretic face is slightly flushed. He says there is no difference in the sweating. In the left face the tactile sense is quick and perfect. He cannot close the left eye by itself, but closes both together—a further evidence that bilaterally associated movements are not lost in cortical paralysis. The left face is still markedly paretic and the tongue deviates to the left. The muscles especially paralyzed in the arm are the flexors of the fingers. The forefinger and thumb are notably weak and awkward. He has good control of the flexors of the wrist. The biceps contracts firmly. He says he has a feeling of weakness about the shoulder, and his arm moves awkwardly, but the deltoid and individual muscles are apparently about normal. The regain of power is rather greater than was expected.

Dr. Allen J. Smith makes the following report of the appearances of the excised tissue:

"Three pieces were referred to me for examination; one governing arm alone and the other two arm and face movements. Stained by Weigert method. Those sections from piece governing arm alone (post to fissure of Rolando), each showed numerous foci of infarction, apparently recent and possibly due to some violence to the tissue during operation. There was possibly some degeneration in the cortical substance, but at most very slight. In the large pieces governing arm and face (taken from anterior to fissure of Rolando) there is a distinct degeneration of the large multipolar pyramidal cells, with the same foci of hemorrhage as in the smaller pieces. A number of these large cells seem to be in a condition approaching fatty metamorphosis, and small granular bodies, like fat drops make up the bulk, which is less than usual, and in most cases shrunken away from the walls of tissue about the cells. These degenerated cells refuse to take the stain as well as their comrades that are undegenerated."

In closing the account of this case it seems proper to offer a few special observations. As far as I am aware, there have been two cases operated on in which no discoverable lesion was present and in which the irritable area was mapped out with faradism and removed. There may, of course, be others. The two to which I refer are one of Mr. Horsley's cases,[2] and one operated on by Dr. Keen, of Philadelphia. The propriety of the operation is to be decided upon in individual cases, and cannot yet be made the subject of a general rule; it must depend largely upon special features, as, for instance, the strictly focal character of the fits, their severity and frequency, and the extent to which they destroy usefulness or jeopardize life. Macewen[3] discusses the propriety of removing large wedges of brain-cortex, and lays much too great stress, it seems to me, upon the evils of producing hemiplegia in trying to cure fits—to which it may be said, in the light of this case that, first, in curing focal epilepsy it may not be necessary to cut out such large wedges as to produce hemiplegia, and, second, the evils of a partial monoplegia are certainly not to be compared with the direful effects of frequently repeated epileptic seizures.

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[2] British Med. Journal, April 23, 1887.

[3] British Med. Journal, August 11, 1888.

SURGICAL REPORT BY DR. JOHN B. DEAVER.

J. W. G., on June 11, 1888, the day previous to the operation, had his bowels moved freely with a saline purgative ; his urine carefully analyzed and examined microscopically, showing it to be normal; and his chest examined with negative results. He was given a warm water bath, followed by a boric acid bath, then the entire scalp was closely shaved, washed with turpentine and scrubbed with soap and water, then washed with ether and alcohol, when it was enveloped in a towel wrung out of 1 : 1000 solution of the bichloride of mercury. Here, I feel justified in saying that part of the success of all operations is attributable to the careful preparation of the patient. During the operation the following day, June 12th, the most strict antiseptic precautions were observed.

Operation, June 12th, 11 A. M.—The patient was placed on the table for operation. A hypodermatic injection of one-quarter grain of sulphate of morphia was given immediately before the anæsthetic was administered, the object being to contract the arterioles and thus lessen the amount of bleeding. Chloroform was administered until the patient was fully under its influence, when sulphuric ether was substituted and continued throughout the operation. In the presence of the neurological staff of the hospital, and assisted by my colleague, Dr. J. William White, I first mapped out upon the scalp of the right side of the head, the seat of operation, the fissures of Sylvius and Rolando by using Reid's lines (see *Lancet*, 1884, p. 359), which I will describe. First, draw a line, which runs from the lower border of the orbit through the centre of the bony meatus of the ear. To find the fissure of Sylvius, draw a line from a point one and one-quarter of an inch behind the external angular process of the frontal bone to a point three-quarters of an inch below the most prominent part of the parietal eminence. Measuring from before backward, the first three-quarters of an inch of this line will represent the main fissure and the rest of the line the horizontal limb. The ascending limb starts at the point indicating the termination of the main fissure—*i. e.*, two inches behind the external angular process, and runs from this vertically upward, for about an inch. The fissure of Rolando is found by drawing two lines from, and perpendicular to, the base line to the top of the head, one passing through the depression in front of the ear and the other through the posterior border of the mastoid process. The fissure of Rolando is now represented by a line drawn from the point of intersection of the posterior vertical line with the line connecting the nasal eminence with the external occipital protuberance, indicating the great longitudinal fissure, to the point of intersection of the anterior vertical line with the line representing the fissure of Sylvius, upon either side of which are the ascending frontal and parietal convolutions containing the centre we wished to remove in this case.

I prefer Reid's lines to Broca's, Lucas Championnière's, Hare's, or Wilson's method of locating the fissures, as I have proven them upon the cadaver to be quite as correct as any of the others, and I think simpler and more comprehensible ; and again, as they map out more fissures than do the others, as brain surgery advances they will be more useful. Over and a little in advance of the middle third of the line representing the fissures of Rolando after all the layers of the scalp, including the periosteum, had been dissected up by making a large horseshoe-shaped flap, with its convexity downward and forward, thus favoring drainage, a trephine one and a half inches in diameter was applied to the skull and a section of bone corresponding in size to that of the trephine removed. Thus far both the soft parts and the bone were perfectly normal, there being not the slightest evidence of depression of the latter. The dura mater, which now presented at the bottom of the wound intact and normal, was incised and reflected, thus laying bare the arachnoid and pia mater, both of which membranes to the naked-eye appearances were healthy. Before incising the hemisphere (to make sure we were over the proper area) Dr. J. Hendrie Lloyd applied electrodes which had been wrapped with sublimated cotton, and which was lying in a 1 : 1000 solution of the bichloride of mercury, to the surface of the brain thus far exposed, with the result of bringing about movement of the fingers and wrist but not of the forearm, when I, with a pair of Hopkins' modification of Rongier's forceps, cut away several small pieces of bone from the anterior margin of the opening made by the trephine, Dr. Lloyd again applied the electrodes when the forearm was flexed and supinated, the angle of the mouth elevated, and the face muscles contracted. A saturated solution of boric acid containing four per cent. of hydrochlorate of cocaine was now applied to the arachnoid and pia mater to contract the blood-vessels of the latter membrane. With an ordinary sized scalpel, held perpendicularly, three pieces of brain tissue, each three-quarters of an inch in depth, were removed, one-half an inch square in size, back of the fissure of Rolando, and two smaller portions anterior to the Rolandic fissure.

The cut vessels of the pia mater were ligated with fine juniper-oiled catgut, and hot water applied to the surface to check the oozing; the latter proved to be very efficient. A few strands of heavy juniper catgut were placed in the bottom of the wound and the flaps of the dura mater approximated over it and sutured with catgut. Again, a few strands of heavy juniper catgut were placed in the wound, resting on the sutured dura mater, the skin flaps approximated and sutured with silver wire. The wound was dressed antiseptically (bichloride of mercury being used), and the patient sent back to the ward.

The temperature of the patient after the operation was 97° Fahrenheit; in the evening of the same day 99°; pulse 98; respiration 15. Ordered milk and lime-water.

June 13.—Temperature 99⅕°, pulse 94, respiration 16. Dressings not soiled; bowels moved slightly. Ordered potass. bromide, gr. xx, every four hours.

14th.—Dressings slightly stained; wound dressed, when found to be completely sealed. No discharge. Pulse, respiration, and temperature normal.

15th.—Dressing not disturbed. No pain. Pulse, respiration, and temperature normal.

16th.—Dressings slipped. Wound had to be dressed. No discharge.

17th.—Patient not quite so well; is restless, showing some evidence of cerebral irritation. Complains of some pain in the head. Pulse 84, respiration 16, temperature 100° Wound dressed and found healthy. No discharge. The scalp behind the wound is œdematous. Ordered ice-bag to the head, and calomel, $\frac{1}{4}$, with Dover's powder, gr. ij, every three hours.

18th, 11 P. M.—Patient, while asleep and dreaming, tore off his dressings. Wound dressed, when the inner dressing alone was found slightly stained with bloody serum, otherwise healthy. Scalp still œdematous. Patient's general condition much better. Bowels were moved after the administration of a simple enema.

19th.—Wound dressed, six sutures removed, allowing three to remain. The points from where the sutures were removed were touched with solid stick of nitrate of silver.

21st.—Patient more restless than the day previous. Pulled at the dressings, necessitating a redressing of the wound, which was found free from discharge, and healthy. Pulse, respiration, and temperature normal.

25th.—Patient attempted to remove his dressings, but was not successful. The dressings were removed, when the wound was found to be healed. The three remaining sutures taken out, and the points corresponding to the site of the sutures touched with solid stick of nitrate of silver. The part of the scalp covering the trephine opening was quite prominent, and upon palpation fluctuation was detected. I made an incision into the scalp here at two points, evacuating bloody serum only. I then introduced a small rubber drainage tube and dressed the wound. Pulse, respiration, and temperature normal. Patient complains of no pain; tongue dry; calomel and Dover's powder stopped. Ordered whiskey half an ounce, two grains of quinine every four hours, and also three drops of turpentine, in emulsion, every six hours.

27th.—Wound dressed, drainage tube behaving nicely, very little discharge.

28th.—Bowels were moved after an enema had been given.

29th.—Wound dressed. Still some little serous discharge through the drainage tube. Stopped emulsion of turpentine, but continued with the quinine and whiskey.

July 1.—Bowels moved twice during the night. Patient comfortable and doing well in every respect.

2d.—Wound dressed. Drainage tube removed.

3d.—Three weeks since the operation, patient allowed to sit up.

4th.—Bowels moved.

6th.—Wound dressed, very little discharge of serum from tract of drainage tube.

12th.—Wound dressed. Still a little discharge of serum from tract of drainage tube. No pain or tenderness on pressure. The pulsation of the brain at the centre of the flap covering the trephine opening in the skull was very marked.

17th.—Wound all healed. No further dressing applied. Patient entirely well. Walks about the hospital.

The deductions which I would draw from this case are that this, as well as other cases, proves that excision of parts of the brain can be done with, I may say, perfect impunity; therefore, in the case of a lesion the nature of which is doubtful, and which in a short time will destroy the patient's usefulness if not his life, why not here, as well as in the abdominal cavity, make an exploratory incision? I think our success is due, largely, in these cases to the precaution taken in regard to strict cleanliness.

Since Mr. Macewen has practised putting back the button of bone removed in trephining and obtaining union, you may ask yourselves, Why did I not likewise? Notwithstanding I had subjected the large button of bone, as well as the small pieces removed in my case, to the proper treatment, preparing them to be reposited, I do not think it worth while to place back so large a piece, as I had seen this done in the practice of two of my friends, and in both cases it necrosed and had to be removed; neither did I have at hand the proper instrument with which to divide the large piece of bone into small pieces or resolve it to bone-dust. Had I done the latter and placed it in the wound, it would not have been safe, owing to my not having absolute apposition of the flaps of dura mater, in which event, the brain would have been subjected to irritation, from the presence of the small particles of bone. The last examination made of this case, September 14, 1888, by Dr. Lloyd and myself, shows the opening, with the exception of a point at its centre, a quarter of an inch square, to be filled in with bone. At the point referred to very slight pulsation of the brain can be detected. Here we have had regeneration of bone from the periosteum, therefore, I am now well satisfied with the course I pursued and feel sure before long the entire opening, made in the skull at the time of the operation, will be closed by bony plate.

DISCUSSION OF DRS. LLOYD AND DEAVER'S PAPER.

DR. DAVID FERRIER, of London, congratulated the gentlemen upon the success of the operation. It was perhaps, however, a little early to say that the case was cured. In several cases of his own of the true Jacksonian type of focal epilepsy without loss of consciousness he had excised and the patient had not been cured. He referred particularly to the case of the son of a medical man in whom after a blow on the side of the head there had been epileptic seizures, beginning in the left hand. Lister had trephined, expecting to find a spicule of bone; but the skull was not even thickened. Horsely had subsequently repeated the operation, but the attacks had not subsided. Even though begun as a local lesion, the removal of this lesion does not always effect cure, the system having apparently become habituated to the attacks. The sooner such cases were operated upon the better. The motor paresis in Dr. Lloyd's case favored the assumption of an organic lesion. Cases having an organic lesion were more apt to recover. He inquired whether the paresis referred to continued between the intervals of the attacks.

Dr. Lloyd replied that it was diminished during the intervals, but that it never entirely disappeared.

Dr. Ferrier referred to the symptoms of postep[illegible]c hemiplegia as possibly of the same character. Cases of operation for traumatic lesion with thickening of the membranes do well. He referred to the case of a young man who received an injury over the posterior extremity of the superior frontal and the ascending frontal convolutions when eight years of age. Ten or twelve years had elapsed before the operation, when he was having as many as three hundred fits in fourteen days. The cicatrix and thickened tissue was removed. Three years had since elapsed and the patient remained so much improved as to be able to earn his living.

The case of Dr. Lloyd was interesting also as a physiological experiment. He understood that there was paralysis after the operation while the tactile sense was perfect, any difficulty in distinguishing objects being evidently due to impairment of the mechanism upon which associated movements depends. The speaker inquired whether Dr. Lloyd had tested the sensibility by passive movements. The case was interesting as a proof that after the removal of the motor centres the tactile sensibility was preserved.

Mr. Victor Horsley, F.R.S., of London, referring to the question of recurrence in epilepsy, cited a case in which the facial region was excised after stimulation with faradism. Lingual equilibrium was obtained, which was not present before the operation and the fits were absent for three weeks, when they returned. The speaker referred to another case, in which after excision of a focus there had been no fits for two years, and to another, operated upon twenty-three months ago, in which there had been no fits since. He would not, however, venture to say that the case was cured. In his opinion five years should first have elapsed. To free the patient from epileptic attacks for six months or two years would, however, restore mental power and was no insignificant result. He agreed with the previous speaker that the operation should be done as early as possible. The faradization of the cortex for the purpose of diagnosis was a step in advance. He had himself irritated the corpora quadrigemina in one case without unfavorable symptoms.

Referring to the muscular sense, the speaker stated that it had two phases, that of moving the segment and that of moved it. In some cases the last was impaired without the impairment of the first. He referred to Mr. Steadman's method of gauging the appreciation of muscular sense by seizing the segment by the side.

In regard to the surgical aspect of the operation, drainage did not require that the convexity of the flap should be downward. In his second case he had sloughing of the dura from division of the temporal artery by this procedure. He now cut the convexity of the flap upward and backward. With the patient lying, full drainage was secured. The speaker used the spray for the purpose of continuous irrigation. He had tried intermittent irrigation, but found that it did not do so well.

DR. W. W. KEEN stated that he found irrigation unnecessary, washing with sponges being all that was, in his opinion, required.

DR. GRAY was glad to hear so conservative a rule as five years for determining the cure of focal epilepsy. Even in idiopathic epilepsy the duration of the intervals between the attacks varied enormously at different times. In several cases in his experience the fits had been absent for three or four years. In one case, that of an intelligent man, there was cessation for ten years. In regard to the question of cure in reflex epilepsy, he referred to a case of petit mal, in which the cure of a vaginitis was followed by recovery, also to a case of the laryngeal type in which there was cessation for several years after the removal of the growth. He also referred to a case in which the convulsion was limited to the hand. Frank W. Rockwell had cut down, incised the dura, and found a growth of a dark color, supposed to indicate a gliomatous formation; he did not excise, but the convulsions were absent for four months following. Now, however, they were as bad as before. In another, a case of intracranial syphilis, the convulsion was limited to one upper extremity, and operation was determined upon. The operator, however, not believing in antisepsis, the patient died from violent acute encephalitis, the brain being honeycombed with pus.

Dr. Dana had understood Dr. Lloyd to state that there was some disturbance of the sensibility in the fingers ; he had also stated that there had been a loss of the sensory aura present before the operation. It was hardly fair to say that the cutaneous sensibility was not disturbed. Further, the part excised was small, the sensory centres occupied a space larger than those of the motor centres.

Dr. Mills shared Dr. Ferrier's impression that true cutaneous sensibility was not destroyed in this case. The method of examination with the sharp and blunt points of the æsthesiometer was defective. Great variations would be found even in the present company in regard to these tests. The personal equation made a great difference. Blindfolded, this patient would instantly detect the gentlest impression, the lightest touch or breath upon the skin. When given objects of peculiar shapes, he would fail to recognize them, apparently because unable to run the fingers over them as in the other hand. His difficulty lay chiefly in motor inability to apply the tests.

Mr. Horsely stated that so far as he knew he had been the first to propose the blindfold test, the patient with his forefinger indicating the point touched. He referred, too, to the fact that if the representative of a whole segment was removed with marked loss of tactile sensibility, recovery proceeded from the proximal end downward, the same as the anæsthesia of hysteroid cases.

Dr. Seguin would place upon record the fact that the patient operated upon by Dr. Weir last year is now in a fair condition of recovery. During the summer he had had a few convulsions, affecting the right hand and cheek. There is no headache or choked disk. There is more paresis of the right arm than in the summer. There was unquestionable anæsthesia of the cheek, hand and forearm to contact tests as well as to the esthesiometer. The patient does not feel slight contact tests unless there is some indication in the temperature of the object applied. He estimates small differences in weights. There is also anæsthesia of the lower part of the face, the lips, and the inside of the cheek.

DR. GODFREY, of Bridgeport, has recently found that the patient does not taste sugar upon the right side of the tongue. The wound in this case involved a piece of the cortex and adjacent white matter one inch in diameter and of considerable depth, as the growth had to be scooped out.

DR. FERRIER asked what was the character of the sensibility in the leg and trunk.

DR. SEGUIN replied that in the leg it was normal. In the trunk he could not say.

DR. FERRIER asked what was the situation of the tumor.

DR. SEGUIN replied that it occupied the caudal extremity of the second frontal, extending partly under the præcentral, covering the face and arm centres.

DR. FERRIER asked whether it extended to the longitudinal tissue.

DR. SEGUIN replied that it was situated two-thirds ot the way down to the fissure of Sylvius.

DR. FERRIER asked what was the size of the tumor.

DR. SEGUIN replied that it was about eighteen mm. across and almond-shaped. It was a sarcomatous growth.

DR. LLOYD stated that he had not referred to the pathological examination in his case, because not yet complete; but he had been told by the pathologist this morning that there was evidence of degeneration in the large pyramidal cells. When he had said that the patient was cured, he had simply meant, that so far the patient had been relieved. Previous to the operation he had been having as many as twenty-eight convulsions in a day, and since the operation he had not had one authentic attack. If in the future recurrence takes place, he would not neglect to report it. While the tests for muscular and tactile sensation were not very exact, perhaps, he thought that the patient did have some difficulty in locating the point of contact, he would mistake one finger for another. It seemed to the speaker that the fact of a sensory aura in the case showed that sensation was inherent in the cortex.

A CASE OF ACUTE FATAL NEURITIS OF INFECTIOUS ORIGIN; WITH POST-MORTEM EXAMINATION.

By JAMES J. PUTNAM, M.D.,

OF BOSTON.

The following case is one of generalized neuritis, of inflammatory and disseminated character, involving the nerves to a greater or less extent from their roots to their terminations, and associated with changes in the muscles on the one hand, and with alterations in the central axis on the other, which may have had a slight share in producing the symptoms.

Death occurred from asphyxia on the seventh day; and at the autopsy, besides the signs of neuritis, the lungs were found crowded with small nodular hæmorrhages, and the spleen enlarged.

The patient was under the care of Dr. M. A. Morris, of Charlestown, who kindly asked me to see him in consultation. This I did once only, and that at the beginning of his illness.

The notes which Dr. Morris has kindly placed at my disposal, supplemented by my own, cover most of the essential points. I did not have conveniences for making an electrical examination, and was partly deterred by the agitated, restless state of the patient from investigating certain symptoms as thoroughly as I ought to have done.

At that time, however, while the diagnosis seemed clear, the fatal issue of the case was not anticipated.

The patient was a man twenty-eight years of age, in good health in all respects, and free from constitutional disease of every kind. He was of a highly nervous temperament, and his father and mother are reported as having been also of nervous temperament, but otherwise well. The patient himself had been formerly laid up with what was called nervous prostration, but at the time of his illness was in his usual health. He did not use liquor to excess.

On Friday evening, November 28th, 1886, he rode from Boston to his home in Charlestown on the front platform of a horse-car, in a heavy rain storm, and got thoroughly drenched. Before morning he awoke with pain in the left shoulder and across the back.

On the following day he complained of a feeling of stiffness in his muscles all over the body; his gait was weak and unsteady, and he felt a general sense of feebleness in all his movements.

The next day he was only with difficulty able to stand or walk. In the evening of this day (the third of his illness), he was first seen by Dr. Morris, who found him complaining of pain, not only in the left shoulder, but also in the anterior muscular mass of both thighs and of numbness in the toes of both feet. He had also noticed that he had had no early-morning erection of the penis since his illness began, which had previously been habitual.

I will here remark that the patient had been excessive in sexual intercourse,during his married life of several years duration, but not so for the period shortly preceding his illness.

There was no pain in the back, nor girdle sensation, nor any weakness of the sphincters; the pupils were normal; his gait was very unsteady, the legs tending to cross one over the other, so that he nearly fell to the floor; the heart was beating regularly; temperature and respiration were normal; heart sounds normal. Dr. Morris ordered 5 grs. of sodium salicylate every two hours, for the relief of the pain.

The next morning he found the patient perspiring freely, and without pain, except in the hip-joint when he rolled over in bed. He was unable to walk, falling forward on the floor when he attempted to do so. The numbness of the feet had increased, but there was no noticeable loss of sensation to ordinary tests; there was pain on pressure over both sciatic nerves and also on deep pressure over both post.

tibials. The pulse was 80, temperature normal, respiration 20.

On the 29th (the fourth day of his illness), the symptoms had still further increased, but were of the same character as before. The pulse was 80, temperature 99° F., respiration 20. The patient was feeling anxious and restless.

On the following day the pulse and temperature were still nearly normal. The patient found it difficult to raise his legs from the bed, but could with some effort draw them up and push them down.

On December 1st (the sixth day of his illness), the pulse was 84, temperature 99.3° F. The patient had been unable to draw the legs up since the previous night; he could not raise his left hand to his head, but could raise the right with moderate effort; the grasp of the left hand was much weaker than that of the right; he could flex both arms; the calves of both legs were tender to deep pressure; there was some strangling on attempting to drink, and slight cough with expectoration of frothy mucous; the patient was talkative and restless.

On December 2d the pulse was 86, temperature 99.2° F. The patient had been delirious the night before and had not slept; he coughed and raised a great deal of frothy mucus; coarse moist rales were heard over both upper fronts; there was complete paralysis of both legs; pain on pressure over both facial nerves; the conjunctivæ were congested; he had a good deal of difficulty in swallowing fluids, but took, on the whole, a good deal of nourishment.

During the afternoon of this day (the seventh of his illness), the paralysis of the upper extremities increased notably; the cough and expectoration and the injection of the conjunctivæ also increased; swallowing became very difficult.

At about seven in the evening, while propped up in bed and taking some nourishment, he suddenly began to cough and strangle, and became insensible. His wife, who was feeding him, thought that a small bit of soft bread soaked in chocolate which he was trying to swallow might have entered his trachea. On Dr. Morris' arrival, forty-five minutes later, the pulse was found to be 120, quite strong and regular; respiration barely perceptible, slow and regular; tongue swollen and discolored. There was no evidence that any foreign body had entered the trachea. He died half an hour later, to all appearance from paralysis of the respiratory functions.

I saw the patient on the fourth day of his illness, and obtained essentially the same history as has been given.

The sequence of symptoms was reported to be as follows:

On the night following the exposure, he had pains in thighs on motion, pain in the left shoulder and back, numbness of the toes of both feet.

The next day he found it difficult to raise the left arm at the shoulder, and had weakness in walking. The day following he began to have numbness in the fingers. At the time I saw him all motions were possible, but most of them very feeble. He could still raise the foot about six inches from the bed with the leg extended, but only by the aid of a sudden impulse. The movements of extension at the knee were fairly good. The muscles of the legs were flabby and the patient was unable to stand alone, but from weakness rather than loss of co-ordinating power, and closure of the eyes did not increase the difficulty. Deep pressure in the regions indicated was painful, but there was certainly no marked loss of sensibility to touch or pricking, though only rough tests were used, the patient's restless condition not inviting to more critical investigation.

In view of the paræsthesia, local tenderness, steady increase and wide-spread bilateral distribution of the muscular symptoms, and yet the absence of complete paralysis, the diagnosis of multiple neuritis seemed to be justified, but there was no indication at the time of my visit of paralysis of the heart or lungs.

The autopsy was made on the day after death, December 3d, by Dr. R. H. Fitz, who has kindly given me the following notes:

"Right side of heart distended with liquid blood; both lungs injected and moderately œdematous; punctate ecchymoses throughout the lungs in every part, spleen enlarged to nearly three times the normal size, soft, injected; liver and kidneys deeply injected; nothing abnormal in the appearance of the brain or spinal cord.

"Pathological diagnosis; nodular, pulmonary hæmorrhages; acute splenic hyperplasia."

A portion of the anterior crural, the iliolumbar, and vagus nerves, and part of the left axillary plexus, a portion of the diaphragm with filaments of the phrenic, part of the deltoid muscle, and a piece of one lung, were removed for subsequent examination.

It is to be regretted that these nerves and others were not removed in their whole length, especially in view of the fact that in the closely similar case reported by Rosenheim, which came to my notice within a few days after this examination, localized hæmorrhages were here and there found in the course of the nerves.

The appearances, however, in such portions as were removed, leave no doubt as to the general character of the process.

The nerves were examined both fresh, in osmic acid, and after hardening in Müller's fluid. The same pathological appearances, though varying greatly in degree, were found in all; but the best specimens were obtained from the axillary plexus and the anterior crural, and the description will therefore be based mainly upon these, so far as the examination of the hardened specimens is concerned.

The vagus nerve was not examined after hardening. In the fresh state the most marked appearance was a strikingly beaded arrangement of the myeline, due to an accentuation of the markings of Schmidt. I should hesitate to regard this as certainly pathological, were it not that it occurred in connection with further changes.

The myeline was eaten out near the annular constrictions of Ranvier, but this may have been a purely passive, postmortem change (v. below). Here and there the myeline was swelled and had wholly lost its characteristic markings, and in spots there was an infiltration of cells such as will be described further on.

Of the phrenic, only a few terminal filaments could be examined, and these looked perfectly healthy. Had a more extensive examination been practicable, changes would doubtless have been found, because a certain proportion of the muscular fibres of the diaphragm had wholly lost their transverse striation, and looked lustreless and granular.

The osmic acid appearances in the other nerves were of the usual kind, but the larger number of the fibres examined looked fairly healthy.

Sections of the anterior crural nerve, obtained after hardening and stained with carmine, showed under a low power a streaked or mottled appearance, as if a number of nerve tubes here and there had been blotted out and a new formed substance, taking the carmine stain, had taken their place. When the sections were examined with higher powers, the outlines of nerve fibres were seen more or less altered in the affected parts ; yet, nevertheless, the spots seemed of a uniform red color.

The axis cylinders in many of the nerve bundles were to all appearance normal, or nearly so, except in the spots above described ; but in some bundles, on the other hand, the same series of striking changes had taken place, which will be described more at length in connection with the axillary plexus.

Here and there were foci of cell formation or infiltration, but on the whole the changes were less marked than in the left axillary plexus.

The lesions met with in the axillary plexus were of the following character:

1. Infiltration of small round cells with granular contents, together with an admixture of cells of other kinds, especially a number having a nucleus of about the size of a leucocyte, and granular contents, but with a distinct body of a pale, homogeneous protoplasm, giving to the whole cell a spherical or oblong shape.

These were by far the most numerous toward the periphery of the nerve bundles, and especially between the sheath and the fibre, and next, around the blood vessels, where they often formed a well-marked ring, spreading thence outward among the nerve fibres. They were also met with, however, remote from either sheath or vessels, following the course of the nerve fibres themselve ; so much so, that I have some preparations of isolated fibres surrounded, at a part of their course, by quite a mass of these cells. Sometimes they were collected into columns which lay between and parallel to the nerve fibres.

I was not able to make out that any of these cells lay actually inside the nerve sheath, except possibly in one or two instances ; nor did the nuclei of the nerve fibres appear to be increased in number. Some of the cells were evidently in process of multiplication, the nucleus being divided by a sharp line into two parts. Of these I saw perhaps, three or four well-marked instances.

The degree of this cell-infiltration varied greatly in diferent sections from the same nerve.

Besides the cells described, there were numerous spindle-shaped cells belonging to the connective tissue, whether increased in number or not, I am not prepared to say, and also here and there larger granular masses which may have been protoplasmic with particles of myeline, or may have been simply altered masses of myeline.

There were also large and highly granular cells, with a large nucleus and irregular, oftentimes flattened border, which I took to be endothelial cells, normal or more or less altered.

I searched for so-called " Mast-zellen" with various ani line colors, as indicated by Rosenheim, but succeeded in finding only one or two that seemed to be characteristic.

2. The next most striking change affected the axis-cylinders, which were in some places greatly enlarged, in others more or less atrophied, in other, again, entirely destroyed.

The distribution of these changes was largely by nerve bundles ; that, is, one bundle might show nearly normal axis-cylinders, while in the next bundle they were greatly altered. In some sections there were whole (secondary) nerve bundles, in which scarcely a single axis-cylinder was to be seen ; in others all the different changes were represented, showing that the swelling, atrophy, and disappearance were parts of the same process of destruction.

The changes were also much greater in some strands of the axillary plexus than in others.

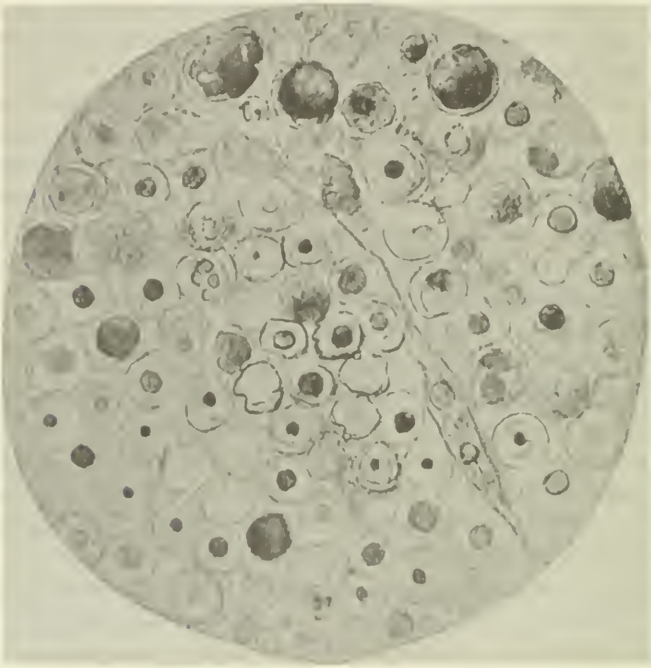

PLATE I.

Section from axillary plexus, illustrating the alteration and destruction of axis-cylinders.

Some of these swelled axis-cylinders occupied the entire thickness of the nerve tube, and even the nerve tube itself seemed sometimes to be distended. In other cases, the sections of the swelled axis-cylinders appeared not round but crescentic, occupying half or two-thirds of a nerve tube.

In order to study more closely the position and character of these changes in the axis-cylinder, I made a series of longitudinal sections, and also of isolated nerve-fibre preparations. Through these it became evident that the swelling occurred here and there in the course of the fibre, and that its most common position was at or near the "annular constrictions" of Ranvier (or "connecting disk" of Schiefferdecker).

In some cases it was obvious what had taken place. The axis-cylinder had become swelled into a bulbous en-enlargement, and this had finally burst on one side, leaving half of the shell to give rise to the crescentic sections. In other cases the appearance suggested more or less liquefaction of the altered axis-cylinder (perhaps the myeline as well); for the disk at the annular constriction and the ad-

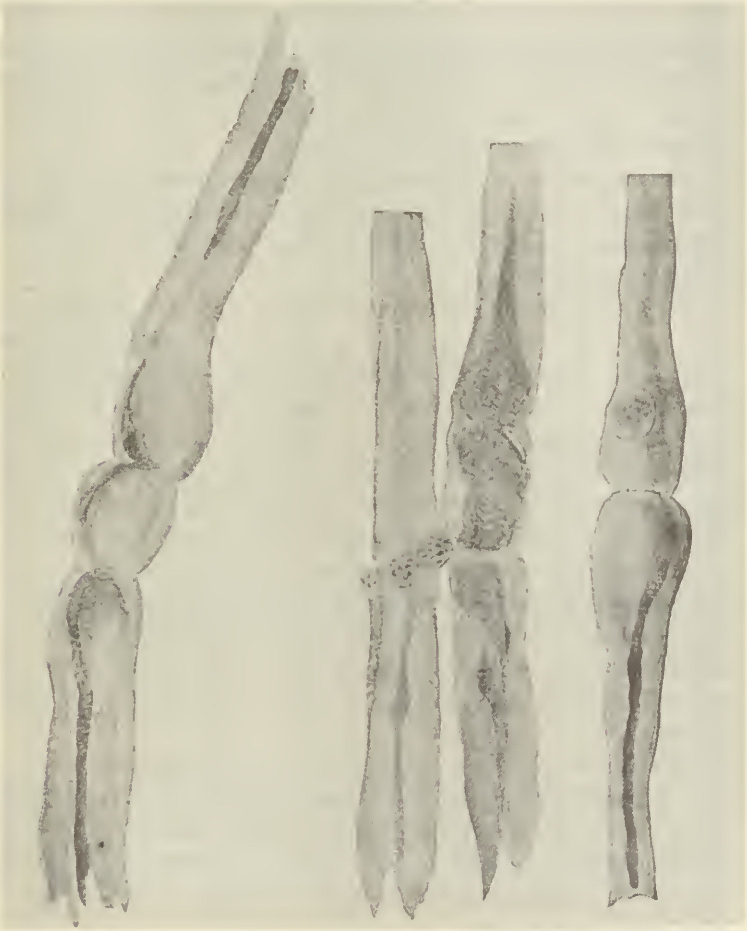

PLATE II.

Nerve fibres with alterations in the axis-cylinders near the constrictions of Ranvier. Hartnack Immersion, one-tenth.

joining walls of the nerve tube seemed to be, as it were, plastered with a substance coloring strongly with carmine, and evidently formed of the products of degeneration. It is interesting to observe that, as a rule at any rate, the disk had not been broken through, but remained clear and distinct, although, as is shown by the experiments of Boll, Hesse, and Schiefferdecker,[1] on the action of water and other substances upon the fresh nerve, the osmotic or capillary current setting through the nerve fibre is quite competent to break down the connecting disk.

It is worthy of note that these changes of the axis-cylinder, though almost always near the connecting disk, are not always most marked exactly at that point, but often at a little distance back.

The question arises, Were these changes which have just been described wholly or in part post-mortem in character? In one sense I think this question can be confidently answered in the affirmative. That is to say, it is highly probable that the swelling, etc., occurred after the death of the particular nerve fibre; but, on the other hand, it is equally probable that they did not occur in the process of the hardening of the preparation. My reason for that conclusion is, although the same kind of change—that is, swelling and vacuolization—is said sometimes to take place to a certain degree during the hardening of healthy nerves in solutions of chromic salts, yet I have never seen nor read of any change approaching to this in degree, from that cause.

On the other hand, Schiefferdecker describes, as the result of the treatment of fresh nerves with water and dilute acids, a localized swelling of the axis-cylinder, and eventually bursting of the relatively fluid contents through its envelope, which seems to be quite analagous to that which has here taken place.

It is probable that the swelling observed in such cases as this, is of similar origin with that seen in acute inflammation and acute anæmia of the spinal cord.

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[1] Arch f. Micr.sc. Anat., 1887, Bd. xxx., 435.

The character and position of the myeline sheath were also of interest.

In some of the cross-sections, stained with picro-carmine, the nerve tubes could be seen to be still filled with the remains of myeline, even though no axis-cylinders were visible. At times the myeline seemed to have been changed, so that it took up the coloring matter of the carmine to some extent, and it was doubtless in part to this change that the mottled appearance of the cross-sections was due.

Here and there a tube would be entirely empty of myeline for a considerable distance, both in those cases where the axis-cylinders were preserved and in those where they had been destroyed. This may have been partly due to mechanical violence, or to changes during hardening, but it seems hardly possible that it should be entirely accounted for in this way.

The myeline at the annular constriction was, in the bichromate of potash preparations,almost always absent for a certain distance, and this, also, was partly coincident with the alteration in the axis-cylinder at that point, and partly independent of the latter change, occurring in some places where the axis-cylinder ran through the constrictions, as it sometimes did, uninterruptedly and with clear and parallel outlines. So marked was the displacement of myeline at these points that some of the longitudinal sections seemed to be dotted over, under a low power, with vacuole-like spaces.

It will be remembered that Neumann, in his classical paper upon nerve degeneration, points out that the neighborhood of the annular constriction was one of the places where the degenerative changes in the myeline were earliest observed.

Dr. Webber, of Boston, has noted the same fact in some unpublished experiments of his own.

To a certain extent the changes in the myeline at the annular constrictions are probably of post-mortem origin.

It is well known that Ranvier long ago pointed out that when nerves were exposed to the action of water and other fluids for an hour or so after death, the myeline on either side of the connecting disk would be found rarified and apparently eaten away, and that he considered this change to be an evidence of the fact that nutritive fluids probably enter the nerve at these points.

Schiefferdecker, in the interesting paper above alluded to, while expressing his agreement with Ranvier as to the fact that coloring matters, and probably nutritive fluids, find their way into the axis-cylinders at these points, does not admit that the myeline is dissolved out by these fluids as Ranvier suggested, but considers that it is displaced by the slight mechanical violence in removing the nerve from the body, or of putting it slightly on the stretch, as Ranvier was in the habit of doing as a preliminary to the immersion in osmic acid.

The reason that the displacement of the myeline took place at these particular points is believed by Schiefferdecker to be because the delicate membranous sheath of the nerve, which follows the outline of the fibre and dips down into the narrow portion at the annular constrictions, exerts a lateral pressure where the angle occurs, when it is put upon the stretch.

In other words, the stretched membranous sheath tends to assume the form of a cylinder, the end of which is as large as the connecting disk, but not larger. Consequently, that part of the myeline which occupied the neighborhood of the tapering end of the cylinder is compressed and displaced.

This explanation entirely concurred with the results of some experiments which I had been making, and which will be given elsewhere in detail.

In order to test the point further, I made a number of careful observations upon the nerves of a frog, stretching some of them with a weight of three grammes, and leaving others unstretched.

The results of the experiments were such as to entirely confirm the view taken by Schiefferdecker.

The unstretched nerves, if removed with care, did not show these changes in the relation of the myeline to the connecting disks, either after one hour's immersion in water or other fluids, or on exposure to the fluids of the body after death for twenty-four hours; whereas the nerves stretched with a weight of three grammes showed the changes very clearly, as Ranvier described them.

Occasionally, even in the unstretched nerve, a fibre is seen in which these changes are observed, but not with sufficient frequence, I think, to invalidate the explanation offered. I believe that there is also reason to think that the putrefactive changes which go on during the twenty-four hours or so after death may make this change occur more rapidly, but this point is still under investigation.

The nerves in the present case were not exposed to any special stretching other than was necessary in their removal, and it is therefore probable that the results were partly the effect of pathological change, and only in part of mechanical violence.

I have, however, seen a similar change, though not nearly to the same extent, in a healthy nerve removed from the body at an autopsy and treated with the same reagents that were used in this case.

One other point should be mentioned in this connection, namely, that the membranous sheath in the neighborhood of the connecting disk, as seen in the hardened specimens, looked as if it had been exposed to pressure from within, making it bulge slightly outward. I have no explanation to offer of the exact manner in which this effect was brought about; but this influence, whatever it may have been (possibly the result of decomposition), may have had its share in the displacement and destruction of the myeline as well.

Changes in the Deltoid Muscles and Diaphragm.—Pieces of the deltoid muscle were examined, both in a fresh state, with and without osmic acid, and after hardening in Müller's fluid. In the fresh specimens the only change observed was that now and then a fibre was seen that had entirely lost its transverse striation, the rest of the fibres being apparently perfectly healthy. The examination of the hardened specimens was more fruitful. The cross-sections showed the size of the fibres to be uniform and normal. There was no trace of the vacuolization or so-called serous atrophy, and apparently no deposition of fat either within or between the muscular fibres.

The morbid changes were the following:

First, loss of transverse striation, limited in extent, sometimes occupying only a small part of a fibre, the appearance presented being that of fine granulation, sometimes with traces of transverse striation here and there in the midst of the altered substance.

Second, marked infiltration of cells in the connective tissue and around the vessels.

Third, a localized increase in number of the muscle-nuclei, which sometimes, but not always, appeared to be more marked at the place where the transverse striation was wanting.

Fourth, the intra-muscular nerves, as far as could be judged from the few which appeared in the sections, were almost entirely destroyed.

Out of the whole number of fibres making up a small nerve bundle, one or two atrophied axis-cylinders might be seen, as dark, shining points, in picro-carmine sections, the rest of the bundles being represented by altered fragments of myeline with numerous granular round cells lying amongst them.

Fragments of the diaphragm were examined, fresh and with osmic acid, and granular fibres without transverse striation were here and there noted.

Of the central nerve system, the medulla and spinal cord were examined. The brain was preserved, but has not been examined.

In the medulla and spinal cord the following changes were observed: First, in the membranes and in many of the nerve roots, both anterior and posterior, there was an infiltration of round cells both around the vessels and amongst the fibres, and in other respects the nerve roots were more or less changed, the degree of alteration being less than in the peripheral nerves.

This infiltration was rather greater in the lower dorsal region of the cord than in the lumbar or cervical region, or most parts of the medulla.

The blood vessels, both of the membranes and of the central axis, were everywhere crowded with blood. This I take to have been, in part, the result of the asphyxia with which the patient died ; in part the sign of an inflammatory process.

Within the substance of the cord the vessels were surrounded here and there with a moderate number of cells contained in the peri-vascular sheath, and the central canal was filled with similar cells.

The nerve cells, so far as I could judge, were essentially normal. Here and there was one with a shrunken or otherwise altered nucleus ; but there was nothing, in my opinion, that might not be accounted for by post-mortem changes. The only other pathological appearance in the cord was that here and there at the periphery, especially in the lateral column near the post. cornua, and in the ant. column in the nerve root zone, greatly enlarged axis-cylinders staining feebly with carmine.

The condition of the medulla was carefully examined, in the hope of finding a satisfactory cause for the multiple pulmonary hæmorrhages. There was a general filling of the vessels, large and small, and here and there an accumulation of lymphoid cells in the peri-vascular sheaths, and occasional slight hæmorrhages from the capillaries and smaller vessels.

These disturbances of circulation were, in most of my sections, more marked in and near the sensory nucleus of the vagus and glossopharyngeus than elsewhere, and the hæmorrhages were in fact only seen in this neighborhood. It could not be asserted, however, that the nucleus appeared to have been materially injured.

The vagus nerve roots were affected in varying degrees, and one section, shows a more excessive infiltration than perhaps any other nerve root that I have seen.

It is easy to arrange this case in its proper pathological position up to a certain point. It evidently belongs with such cases as were reported by Eichhorst in Virchow's Arch., vol. 69, 1876, and Rosenheim in the Arch. f. Psych., vol. 18, which the latter has discussed so ably ; and with the other acute, fatal cases of multiple neuritis, none of which, I think, have been of shorter duration than this.

In Rosenheim's and Eichhorst's cases, to be sure, hæmorrhoids existed in the nerves, visible to the naked eye, indicating a more intensely active process than here. On the other hand, nothing could be more intense than the congestion in this case; and the evidences of minute hæmorrhages and cellular infiltration were more extensively present than in most of the other cases, involving the muscles, spinal roots, membranes, and even the central nervous axis to a certain extent, as well as the nerves.

It would be interesting to know whether, if the patient had not died, the spinal changes would have assumed a greater prominence, and a poliomyelitis or a diffuse myelitis have developed itself.

Certainly the topography of the lesions suggests this possibility; but it is to be noted, as regards the question of poliomyelitis, that the posterior nerve roots were quite as much affected as the anterior. The fact that the changes in the white columns (enlargement of axis cylinders) were mainly confined to the periphery, and more marked in the neighborhood of the nerve roots than elsewhere, but they did not seem to occur in the posterior columns. The amount of accumulation of lymphoid cells in the peri-vascular sheaths of the central gray matter was perhaps not great enough to count as the first step in an inflammatory process, though it may have been of that character; but, to say the least, one can hardly doubt that it would have taken little more to precipitate such an event, especially in view of the fact that in the medulla minute hæmorrhages had actually occurred.

The questions of chief importance in connection with this case are: first, as to its etiology, and especially if we can gain through it any light upon the supposed toxic origin of generalized neuritis; next, as to whether these acute, fatal cases exhibit clinical features which will enable us to detect them at their onset.

With regard to the first point, the reasoning, as clearly expressed by Rosenheim, and endorsed by Leyden in his recent address before the Militär-ärtzliche Gesellschaft of Berlin, is that the great majority of these cases are truly of toxic origin, the source of the poison being the bacteria of tuberculosis or other constitutional disease, or some mineral or other poison. The bacteria are, however, not supposed to themselves be present in the nerve, but only the poisonous substances to which their growth gives rise.

In this way the distinctly infectious cases are brought into a parallel with the cases due to metallic poisoning.

Strümpell, moreover, in a recent paper upon degenerative changes in the spinal cord, although not speaking especially of neuritis, suggests another method in which these processes of poisoning may be started or propagated, namely, through the action of the products of decomposition of the nervous tissues themselves. Dr. Spitzka suggested last year a similar cause for the outbreak of delirium grave.

In Rosenheim's case it was believed that the primary source of the infection was tuberculosis, with which the patient was affected, although at the time of his attack in good nutrition; and he further remarks that, in his opinion, scarcely a case of multiple neuritis has been observed in which no infectious constitutional disease was present. With this view I cannot wholly agree; nor is it maintained, so far as I can see, by Leyden, who himself reports the onset in one of his rapidly fatal cases as being apparently due solely to a condition of exposure and fatigue.

In my case the patient was not the subject of any contagious disease, so far as could be ascertained, and the wetting to which he was exposed was the only cause which could be discovered. Nevertheless, the pathological signs of infection are even stronger in my case than in Rosenheim's and others. In them those signs consisted almost solely in the acute onset and generalized character of the disease, together with the fact that hæmorrhages were present in the nerves. In my case, a marked hyperplassia of the spleen was also present, such as has been observed in several cases of Laudry's disease, but not often and never to this extent, so far as I know, in cases of multiple neuritis. In Rosenheim's case the spleen was reported as measuring twelve centimeters in length, and being soft in consistency.

To what cause to attribute the multiple and nodular hæmorrhages scattered through both lungs, in the present case, I am unable to decide. In view of the absence of hæmorrhages in other regions of the body it would be hardly probable that those were due to the local action of the toxic agent, although this is not to be set aside. It seems more probable that they were of the same origin with the multiple hæmorrhages described originally by Brown-Sequard as due to certain injuries of the medulla, and probably of vaso-motor origin. It is possible that the neuritis of the vagus nerve may have given rise to them, although, so far as I know, such a result has not previously been demonstrated.

It is noteworthy that in this, as in the other important cases of similar kind, the toxic agent, whatever its character, has not given sign of its presence in a diffuse way, but only here and there in foci of limited extent. This is very likely due to the fact that the means of examination at our command do not enable us to detect the first signs of toxic influence. Even if we had any such evidence as that afforded by the examination in Laudry's disease, which seems to be universally considered as most probably of infectious origin, to show that loss of function may precede noticeable alteration in structure, yet we should still, from abundance of facts, be ready to consider that this was possible.

The fact that such a large proportion of the nerve fibres in the affected districts preserved a healthy appearance cannot be taken as a proof that they were performing their functions in a normal manner.

It would not be out of place, before leaving the subject of infection and its possible sources, to refer to the group of symptoms characterized as a new infectious disease and described recently by Weil and others in the Deutches Arch. f. Klin. Med., 1887, 1888.

Taken as a whole, this is not to be confounded for a moment with neuritis, the high temperature and the jaundice characterizing it as belonging in a different category. It is, however, noticeable that in several of the cases, severe muscular pain and other signs of alteration of the peripheral nervous system were present.

As regards the clinical aspects of the case, I would only mention one or two points.

In the first place, the absence of fever throughout the sickness is noteworthy. This was also noted in Rosenheim's case, and referred to by him as being sometimes present and sometimes absent in the other cases that he describes.

The mode of death seems to have been by respiratory, rather than cardiac paralysis. At any rate, the respiratory symptoms were the striking ones, and although shortly before death the pulse had risen to 120, it is reported by Dr. Morris as having been full and regular, while the respiration was scarcely perceptible.

The distribution of the muscular symptoms deserves a moment's comment. Pierson has advanced the opinion that the paralysis of the cranial nerves is characteristic of the acute cases. This, however, is not endorsed by Rosenheim, who points out, very properly, that these paralyses are seen also in chronic cases, and not necessarily in acute cases only, although when present they indicate a wide generalization of the disease, and may be of bad import. In the present case, the signs of paralysis of the cranial nerves which was noticed, consisted in the difficulty in swallowing and the tenderness over both facial nerves.

The fact that the left shoulder muscles were so early and so severely affected was to me of special interest, because I have been recently collecting cases in order to test the diagnostic value of this distribution of symptoms. There seems to be no doubt that although the extremities are usually the first to be involved, in the multiple neuritis of every cause, yet the larger muscles uniting the limbs and trunk are sometimes affected very early.

Finally, I would call attention to a point which should, perhaps, have guided us to the recognition of the serious import of the case, which was early presented, namely, the restless, anxious and agitated mental condition of the patient, which seemed at first out of proportion to the severity of his symptoms.

NOTES.—1. Valuable remarks relative to the significance of this acute swelling of the axis-cylinder, may be found in the Arch. f. Psychiatrie, etc., 1887, p. 263, (Auhang), Zur activen Verand, der Axen-cyl., bei Entzundungen, Dr. M. Friedmann; and Bd. XCVI. der Sitzb.der Kais. Akad. der Wissensch. III. Abth. Nov. Heft. 1887. Ueb. die Verand. am Ruckenmark n. Zeitweiser Verschliessung der Banchaorta. J. Singer (Reprint, p. 12).

2. Otto Dees, (Arch. fur Psych., etc , 1888, p. 97), the latest writer on the anatomy and physiology of the vagus nuclei, mentions, incidentally as his opinion that the large "dorsal" nucleus is vaso-motor in function. It cannot, unfortunately, be stated whether in the present case the fibres and ganglia of the sympathetic system were healthy or diseased.

DISCUSSION.

Dr. Webber had four years ago had a large number of cases under observation in the Boston City Hospital, with three deaths. The history of Dr. Putnam's case was similar to that of these fatal cases. The changes about the constriction of Ranvier noted by Dr. Putnam he had found, especially in the earlier stages of the disease in animals, his experiments were upon rabbits and guinea pigs. In one fatal case he had found but little change about the node of Ranvier; he could not say whether there was traction used in removing it or not. Even where the nerves were extensively altered, there would be some fibres intact. The process seemed to affect the coarser fibres rather than the finer. At the time when there had been so many cases in the course of a few months, there had also been prevalent a sickness among horses attended with weakness and temporary paralysis. He had tried to obtain nerves from the horses, but did not succeed.

Dr. Pye Smith, of London, referred to a case of his own, in which both alcoholism and gout were excluded. He inquired in regard to the position of gout in the etiology of the affection here. He understood that gout was rare in the States, yet in speaking with Dr. Welch, of Baltimore, he had learned that the metatarso phalangeal articulation of the great toe often showed evidence of gout on autopsy.

The speaker inquired also in regard to the prevalence of that enigmatical affection called Landry's paralysis. In Landry's original paper there was but little to correspond with the superstructure which had been reared upon it. Landry had stated that there were no post mortem changes. The speaker thought that it must be a very rare condition. It would be especially interesting to know whether cases examined by modern methods of investigation gave equally negative anatomical results.

DR. VAN BIBBER referred to a case of his own, in which the facial nerve had been affected. The attack had passed off, and the patient recovered.

DR. DANA had not met with or found reported a case in which gout had given rise to multiple neuritis. Local neuritis was not uncommon from gout. He had seen one and perhaps two cases of multiple neuritis from rheumatism. In one case there had been for several years attacks of rheumatism each fall. One fall, in place of the usual attack, the patient had an attack of typical multiple neuritis.

DR. PUTNAM inquired whether any gentleman had met with the multiple pulmonary hæmorrhages present in his case.

DR. WEBBER stated that in the fatal case which he had examined there had been no such hæmorrhages, but in this case the vagus had not been affected, while in Dr. Putnam's in was.

DR. PUTNAM said that true gout was rare, at least in Boston. Neuritis did accompany rheumatism, however, both as a local and a general disease.

In regard to Landry's paralysis, there was, he thought, such a group of symptoms not due to multiple neuritis, and not due to spinal trouble.

DR. SEGUIN presented Dr. Shaw's regrets at being absent from the meeting, and exhibited for him three slides, representing a case of locomotor ataxia.

Miſcellaneous Notes.

The Paris correspondent of the *Wiener Freie Presse* quotes the following regarding the critical analysis made by Dr. Fauvel, the noted Paris laryngologist, in reference to Mackenzie's book "Frederick the Noble":

"* * * That which most surprises me is the fact that medication played a secondary *role* in the management of the case. I would have recommended the employment of *Coca Mariani* to rouse the flagging energies of the patient." * * * "My investigations, dating back to 1865, establish the fact that Coca is a potent agent in combating debility. I have also shown that the injection of concentrated Coca (Thé Mariani), has a salutary influence on the laryngeal mucous membrane, alleviating pain and congestion. In Europe this remedy is relied on in cases of debility and where pain is a prominent symptom." * * * * "As further proof, the case of General Grant is cited, in which Drs. Fordyce Barker, Geo F. Shrady, J. H. Douglas, and Sands, were active. Coca was employed in this case with success (the preparation exhibited being Thé Mariani), and it was stated by the attending physicians that without the use of this drug the General would not have been physically able to undergo the strain incidental to the work of finishing his Memoirs."—*Berliner Tageblatt.*

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