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Original Articles

A STUDY IN TACTUAL LOCALIZATION IN A CASE PRESENTING ASTEREOGNOSIS AND ASYMBOLIA DUE TO INJURY TO THE CORTEX OF THE BRAIN.*

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of Boston,

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James C., about 40 years of age, as the result of a blow from the corner of a carpenter's spirit level which fell upon his head, received a circumscribed compound fracture of the skull over the left parietal region. The wound was treated on modern surgical principles. After recovering from the immediate effects of the injury there remained a marked spastic paralysis of the right arm and a slight paralysis of the right leg not sufficient to prevent its use for ordinary purposes of locomotion. Beyond the condition of sensibility, to be presently described, and the location of the injury, there was nothing remarkable in the case.

Location of the injury: Over the parietal bone is a marked depression, slightly oval in shape and slightly larger than a quarter dollar. On mapping out on the cranium the fissure of Rolando, it is found that the center of this depression lies directly over the line of this fissure at about its middle third. This is shown in the accompanying photograph. It will thus

*Read at the thirty-third annual meeting of the American Neurological Association, May 7, 8 and 9, 1907.

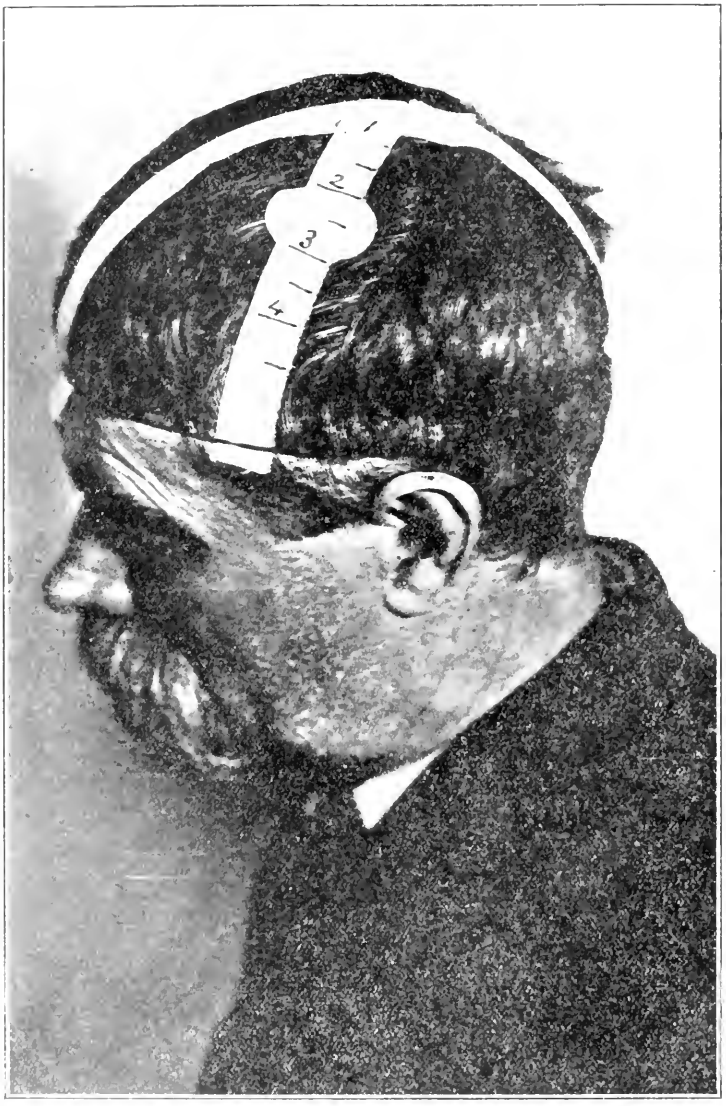

Fig. 1. Showing Location of Injury.

be seen that the depression corresponds very accurately with the arm center and with what should be the location of both the post and pre-central convolutions, and although in the absence of autopsy it is impossible to determine the extent of the underlying cortical lesion, yet the character of the blow was not such as to cause an injury of wider extent.

Sensibility: The tactile sense was found to be absolutely unimpaired in the affected right hand. This sense was tested by the von Frey method; that is, by means of single hairs of different stiffness and also with the camel's hair brush and cotton wool. No difference whatsoever between the sense of tactile sensibility in the normal left hand and affected right hand could be recognized. The slightest touch that could be perceived in one hand could be perceived in the other, and the patient himself could recognize no difference in the tactile perceptions whether tested by a single hair or by the other tests.

Temperature sense tested by test tubes filled with warm and cold water, and pain sense tested by the prick of a pin were also found to be normal in the affected hand.

Bone conduction tested by the tuning fork was rather better appreciated in the affected hand than in the normal hand.

Pressure sense was tested to determine qualitative but not quantitative difference in the two hands. For this purpose, objects of different weights were placed on the palmar surface of the fingers and on the palms, and pressure was exerted by the examining finger. There was found to be a very marked difference between the two hands as shown for instance by the fact that an iron paper weight weighing approximately two pounds pressing by one corner on a circumscribed spot, felt light to the right affected hand, but heavy to the left hand. Pressure exerted by the examining finger until it was forcibly hyper-extended over the dorsal of the phalanx was not felt more forcibly than very light pressure similarly applied to a corresponding spot on the left hand.

Perception of posture of the fingers was practically lost. They could be flexed, extended, abducted and adducted, at the different joints without the patient being aware of the position which the fingers assumed. In fact, the patient said with his eyes shut he had no idea where his fingers were. The

wrist, elbow and shoulder joints were similarly affected, but to a less degree than the fingers. The impairment of perception of posture of these joints diminished in intensity as one proceeded proximally to the shoulder joint, which was only slightly affected in this respect.

The perception of movement and direction of movement was also much impaired. Quantitative measurements showed that any movement under approximately ten degrees was not felt at all; when movement was extensive enough to be appreciated direction of movement was not recognized at all.

Localization: Two methods of testing were employed; First, the "naming" test; that is, directing the patient to name the finger, or segment of the finger, touched with a camel's hair brush or pencil point while the eyes were closed. Second, the Volkmann method, or so-called "looking procedure" (Spearman). This consists of touching a spot on the skin with a pointed object while the patient's eyes are closed; then, after waiting a second or so for the indented skin to resume its normal appearance, having the patient open his eyes and point out the exact spot touched. (It was found that Weber's or the "groping" [Spearman] test was inapplicable as the patient having lost all sense of position of his hand did not know, with his eyes closed, where it was. The method consists in having the patient point out the spot touched while his eyes are closed.)

In the first series of experiments the tests were applied to the palmar surface. By the two tests employed, the "naming" and "looking" tests, it was found that by both tests there was great loss of perception of localization over both surfaces of the fingers, and to a less degree over the palm of the hand. The patient by the naming test as often as not did not know which finger was touched or which segment of a finger, but as will be described later in connection with a third series of tests, localization by the "looking" test was more accurate than by the "naming" test, particularly in regard to the segmental error, even when the wrong finger was pointed out.

About two weeks later, March 6, after there had been considerable improvement in sensibility, the perception of localization was tested by the spacial method, often wrongly called a test of the "spacing sense." There are two methods: the

one is the compass method by which two points are touched at the same moment; the other consists in touching the two points successively instead of contemporaneously. (It is generally overlooked that the recognition of two points contemporaneously does not necessarily mean the recognition of the separation of those points.) The second method was employed,

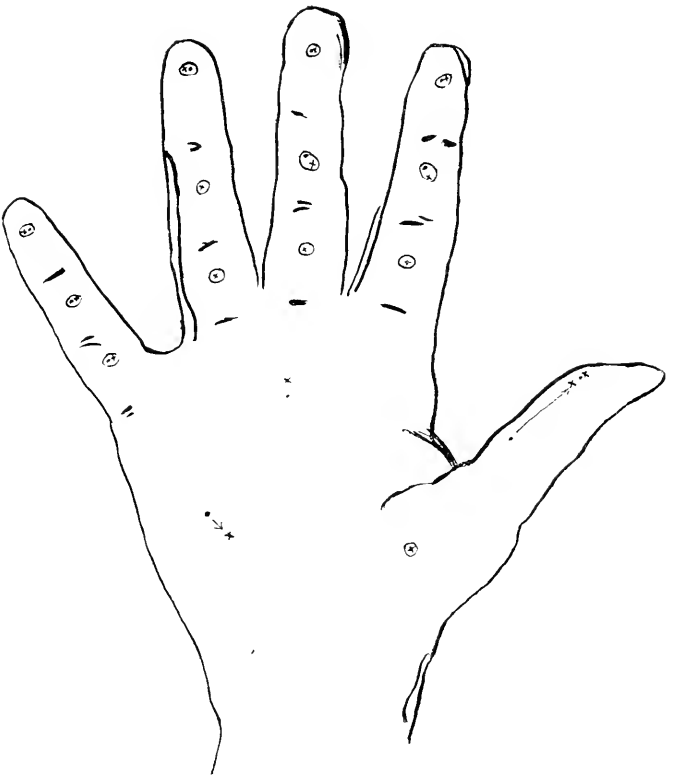

Fig. 2. March 22. After improvement; palmar surface; details same as in diagram of dorsum. Localization by the Volkmann method practically normal. By the "naming" test on this date patient often could not localize correctly segment or finger.

the subject being directed to say whether the point touched was nearer the shoulder or tip of the finger. By this method it was found at this time that the recognition of the perception of localization was still not as good on the palmar surface of the right hand as on the left. He was still unable to recognize form or the nature of objects.

The condition of sensibility in the hand continued to improve and it was found two weeks later (March 22) that the perception of localization over the palmar surface of the hand,

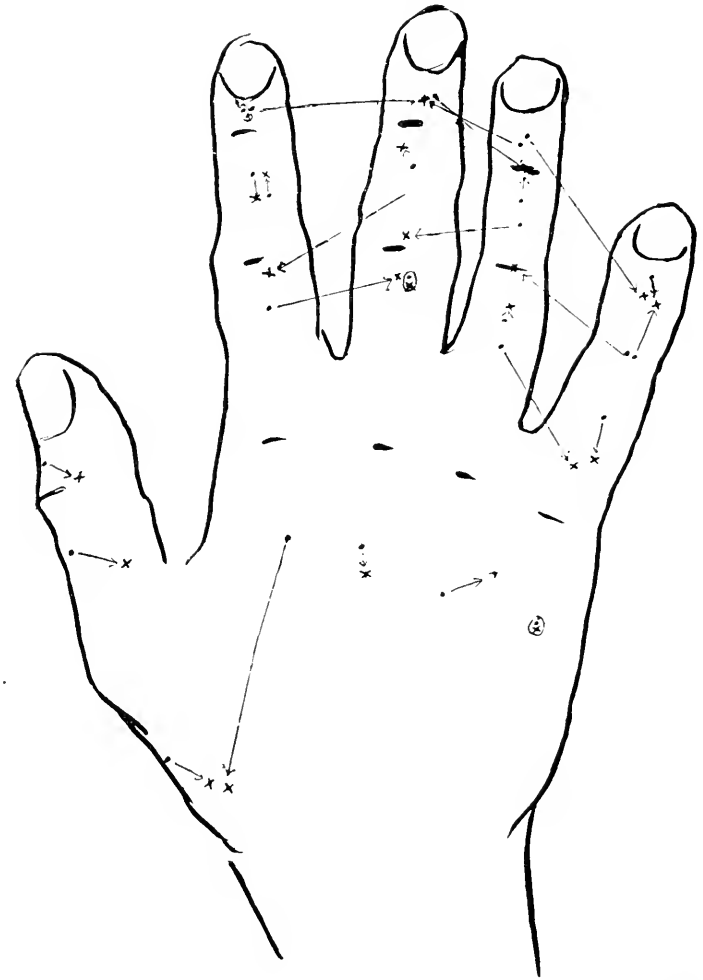

Fig. 3. March 22. After improvement. Localization by the Volkmann method, dorsum of hand; dots = test points; x = localization by subject; arrows = direction of error; circles = correct localization within normal limits.

tested by the Volkmann ("looking") procedure, was practically normal. (See Fig. 2.) On the dorsum of the hand,

however, there continued to be marked errors in localization but errors were post-axial and pre-axial rather than segmental, although there were some segmental errors. (See Fig. 3.) That is to say, it was the wrong finger rather than the wrong segment that was localized. When the "naming" (eyes screened) test was employed, results that were widely divergent from these were obtained. Now on the palmar surface of the hand, where the patient by the previous (Volkmann) test was able to localize normally, there were numerous errors in localization both as respects the finger touched and the segment, and the errors were nearly as numerous and extensive as on the back of the hand where by the "looking" procedure, as above stated, there had been marked axial errors. On this (dorsal) surface of the hand the errors now by the "naming" test were also segmental. Further the localization of the correct finger on the palmar surface was better than on the back, but the segmental errors were quite as numerous as on the back.

I would emphasize here the importance of these results as showing the influence of the form of the test in determining the presence of the loss of the perception of localization. A patient may be able to localize by one test and not by another, according to the kind of sensibility that is impaired;[1] that is to say, whether it is sensibility of the impressions from the joints, skin, movements, etc. The recognition of this fact acquires particular importance in the study of asymbolia and astereognosis. Tactual localization plays a very large part in the judgments involved in the perceptions of form, stereognosis) and recognition of objects (symbolia). That the capacity to make these judgments may be lost, although simple tactile sensibility is retained as in the above case, is well known, but the failure to recognize the fact that localization may be impaired when tested in one way and not when tested in another has led, it would appear, to the error of postulating stereognosis and symbolia as specialized faculties or "senses," rather than as intellectual judgments of the tactual impres-

[1]For a thorough analysis of the fundamental conditions underlying localization compare "Localization in a case of Brown-Séquard paralysis," C. Spearman. British Jr. of Psychology, January, 1905.

sions on which they depend, and localizing them in special regions of the cortex. Before such a conception can be entertained entire freedom from defects of the tactual senses essential for such judgments must be shown. But the proof of such freedom depends upon the tests employed. Different tests are required according to the kind of tactual defect present. At one stage in the above case, while astereognosis and asymbolia were present, localization by the Volkmann test on the palmar surface was normal, but by the "naming" test (with the eyes closed, *i.e.*, the condition essential for the test of the recognition of form and objects) there was marked defect of localization.

Perception of form (tactual stereognosis) and of objects (tactual symbolia): The patient was unable at all periods to recognize form of any kind. Not being able to designate whether small objects, like a pencil and coin, or large objects, like a blackboard eraser and pin-cushion, were long and narrow, round and flat, large or small, he had absolutely no idea of the shape. He was, therefore, unable to recognize and name them. In all these experiments control tests were made with the sound left hand. It was found, for instance, with this hand, that he could recognize the slightest motion given to the fingers both in direction and extent, and he could recognize form and shape and name any objects with which he was familiar.

It will thus be seen that the location of the bone lesion is entirely in accordance with the view of Campbell that the various sensibilities making up the so-called muscular sense have their location in the posterior half of the post-central gyrus, but not with his view that the tactile, pain and thermal senses are located in the anterior half. In this connection it must be kept in mind that while Campbell brings forward evidence to show that the post-central convolution is sensory, this evidence does not, as he fails to see, bear upon the character of the sensory functions here located, *i.e.*, whether they are tactile or so-called "muscular" senses, etc.

Foot: The results of the examination of the foot will be referred to in a subsequent paper on astereognosis and asymbolia. I will here merely state that no distinguishable differ-

ence in the tactile sense was discoverable between the perception of the right foot and of the left. Localization was only slightly affected, but more on the dorsal than on the plantar surface.

Experiments were now made to test the direction of the segmental error in the perception of localization. It will be remembered that Russell and Horsley have maintained that in errors of localization the error is always proximal and not distal; that is to say, the patient always locates the sensation in the segment nearer the root of the limb rather than in the more peripheral segment. Yet the authors referred to record, by the way, two observations where the error was in the distal direction. The results in my case did not confirm these findings; on the contrary, the error in the great majority of cases (in every instance but one) was distal, excepting that in five instances where the last phalanx was touched the error, if any, was necessarily proximal. The results of the tests in detail will be seen in the following table. (Those giving the finger alone without the segment are a few illustrative of the many that were made.) It will be seen that the segmental error was distal in fourteen instances, proximal in six, and in five of these six it was the last phalanx that was tested. Therefore, as I have said, the error in these five, if any, was necessarily proximal.

"NAMING" TEST

PALM OF THE HAND

Finger touched (Eyes closed, or screened)	Answer
Thumb	Middle finger
Fore finger	Fore "
Little "	Middle "

BACK OF HAND

Ring finger	Middle finger
Fore "	Fore "
Fore "	Middle "
Fore finger	Little "
Little "	Could not tell
Ring "	Little finger

BACK OF HAND

(Eyes open, but screened)

Ring	finger	Don't know	
Fore	"	Fore	finger
Ring	"	Ring	"

BACK OF THE HAND

Finger and segment touched (Eyes open, but screened)			Answer			Direction of error
Little	1st	phalanx	Don't know			.....
Fore	3rd	"	Fore	1st	phalanx*	Proximal
Middle	2nd	"	Middle	1st	"	Proximal
Ring	2nd	"	Middle	3rd	"	Distal
Thumb	1st	"	Thumb but more distal	1st	"	Distal
Thumb	2nd	"	Thumb	2nd	phalanx	.....
Ring	2nd	"	Fore	2nd	"	.....
Ring	3rd	"	Fore	2nd	" *	Proximal
Ring	2nd	"	Middle	2nd	"	.....
Ring	1st	"	Middle	3rd	"	Distal
Little	1st	"	Ring, between 2nd and 3rd			Distal
Fore	1st	"	Fore	2nd	phalanx	Distal
Middle	3rd	"	Middle	3rd	more distal	Distal
Ring	3rd	"	Ring	3rd	phalanx	.....
Fore	3rd	"	Fore	3rd	"	.....
Fore	3rd	"	Ring	2nd	" *	Proximal
Fore	3rd	"	Ring	2nd	" *	Proximal
Fore	2nd	"	Ring	2nd	"	.....
Fore	1st	"	Fore	1st	"	.....

PALM OF THE HAND

Middle	2nd	phalanx	Middle	3rd	phalanx	Distal
Ring	1st	"	Ring	3rd	"	Distal
Little	1st	"	Ring	3rd	"	Distal
Fore	1st	"	Fore	3rd	"	Distal
Thumb	3rd	"	Thumb	3rd	"	.....
Ring	3rd	"	Ring	3rd	"	.....
Fore	1st	"	Fore	2nd	"	Distal
Fore	2nd	"	Fore	3rd	"	Distal
Middle	2nd	"	Middle	2nd	"	.....
Ring	2nd	"	Fore	3rd	"	Distal

Ring	2nd	phalanx	Middle	2nd	phalanx	.....
Little	2nd	"	Ring	3rd	"	Distal
Little	3rd	"	Ring	2nd	" *	Proximal
Ring	3rd	"	Ring	3rd	"	.....
Fore	1st	"	Fore	1st	"	.....
Thumb	3rd	"	Thumb	3rd	"	.....

*Error on the third phalanx necessarily had to be proximal.

TACTILE STEREOGNOSIS AND SYMBOLIA; HAVE THEY LOCALIZATION IN THE CEREBRAL CORTEX?*

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Before discussing the localization of stereognosis, we ought to have an understanding as to what we are talking about.

The earlier writers on astereognosis (for instance, Hoffmann, Sailer, Dercum, Walton and Paul) commonly identified this defect with the failure of recognition of objects, and the practical test usually employed to determine its presence was to place objects in the hand of the subject and ask him to identify them. This conception is still largely and rather blindly, as it would seem, adopted by many recent writers (Mills and Weisenburg, Campbell, Russell and Horsley, Starr, Cushing and others). Although stereognosis, strictly speaking, means the perception of solidity or form only, it might be insisted that the question of the use of the term is one of definition, and that there is no particular harm, beyond the fact of a bad terminology, in using stereognosis for tactile recognition of objects so long as it is merely a matter of description of a syndrome. When, however, it comes to a question of localization in the cerebral cortex of what these writers have been pleased to call the stereognostic "sense," at once a confusion of ideas as well as errors in our scheme of cerebral localization arise. From the very first, when astereognosis came to be studied as a syndrome, it was realized that the recognition of objects by tactile perception was a very complex affair and depended not only upon the correlation of a large number of subsidiary sense impressions, which included tactile, position, localization, movement, pressure, temperature, impressions, etc., but upon the perception of form and the interpretation of the whole

*Read at the thirty-third annual meeting of the American Neurological Association, May 7, 8 and 9, 1907.

through higher intellectual processes which again included visual and other memorial images of the objects, as well as memories of past experiences. In other words, it is an intellectual process; yet all this, while thoroughly appreciated, it should be said, Walton and Paul, for instance, proceeded to call a stereognostic *sense* and to localize in the Rolandic area.

Now, properly speaking, stereognostic perception is the perception of form, alone, in three dimensions and though this again is itself a complex conception, and not a *sense,* it is far less complex than the *notion* of the nature of an object.

This distinction between stereognostic perception and the recognition of objects by touch is now clearly recognized by some recent and more precise writers (Raymond and Egger, Dejerine, Grasset, Claparde), and has led to some interesting observations on the defects of form-perception and object-perception and their interpretation (agnosia, asymbolia, tactile aphasia, etc.), which I shall presently refer to.

As to object-perception, aside from the theoretical unlikelihood of a complex intellectual process being localized in a small focal area within one or two convolutions, an improbability that is based in part on the absence of all analogy with anything that is known of intellectual processes, there is absolutely no clinico-pathological evidence that requires such a cortical localization. The localization of such an intellectual process is at best only an interpretation of the clinical findings and these, if carefully studied, will be found, I believe, to be insufficient for an interpretation of this kind. For as the intellectual notion of an object depends upon the integrity of the subsidiary sense perceptions, and as in every case where sensibility has been thoroughly tested in all its forms and where there has been loss or impairment of the *notion* of objects, there has been also more or less impairment of the subsidiary sense impressions, the loss of the notion can be equally well and more logically interpreted as due to insufficient information from the sensory loss. All that the clinico-pathological evidence justifies is the localization of the subsidiary sense impressions. The practical importance of clinically distinguishing between form-perception, or stereognosis proper, and object-perception is conclusively shown by two cases recently reported: one by Raymond and Egger[1] and one by

[1] Un cas d'aphasie tactile; Rev. Neurol., April 30, 1906.

Dejerine,[2] in both of which there was preserved the ability to recognize form of objects in three dimensions with entire inability to recognize the nature of the objects. From the fact that their patient was unable to *name* the test object placed in the affected hand, Raymond and Egger interpreted the syndrome as one of "tactile aphasia," analogous to word-deafness; but Dejerine, who promptly brought forward a similar case, argued with convincing logic that the condition was not one of aphasia, as the question was not one of *naming* only, but of *recognition;* for the patient had no notion of the nature of the test object, and of course could not name it. As an interpretation, he pointed out that the failure to recognize the object could be explained by the impairment of certain subsidiary sense perceptions, for although the tactile and various other senses were preserved, yet there was marked impairment of the localizing perception (one of the most important), and some of the knowledge of the position of the fingers; and though what remained might well be sufficient for form-perception (stereognosis) it was not sufficient for object-recognition.

I would also point out in corroboration of this view (what seems to have escaped notice) that the answers of both subjects (Raymond and Egger's and Dejerine's) to questions regarding the form, though showing a perception of three dimensions, was far from sufficient to allow a notion of the object to be gained. For instance, who could recognize from the following answers, showing form, the nature of the test object:

OBJECT.	RIGHT (AFFECTED) HAND.
Thimble.	It is small. I have my finger in it. I do not know what it is.
Porcelain button, flat, with four holes.	It is a small object which has a bottom. It is a thimble.
A large key.	It is of steel. It is longer than my hand. It is a handle. [At the moment when the thumb enters the ring] It is a pair of scissors.
Dessert spoon longer than the hand.	It is smooth. It is of metal. It is longer than my hand. It is a pair of scissors.
Orange.	It is large. It is round. It is rough. It is hard.
Pocket handkerchief.	It is soft. It is wrinkled. It is not of cotton or paper.

[2] Considerations sur la soi-disant 'aphasie tactile'; Rev. Neurol., July 15, 1906.

Surely, even with the preservation of most of the tactual perceptions, unless the patient has more conception of form than that given by three dimensions, and has various tactual sensations, he can not recognize the nature of the object. Touch, too, must not only be quantitatively but qualitatively preserved. As Dejerine well says, therefore, Raymond and Egger's patient and his own were unable to name the objects from lack of information, not from tactile aphasia. The condition he terms agnosia.

Claparède,[3] discussing the above papers, has suggested the following terminology: Astereognosis is equivalent to a trouble of what Wernicke has called "primary identification"; what may be called *simple perception* or *sensory recognition*. It is, therefore, a trouble of the perception of the form of objects. The notion of an object is intellectual recognition. He proposes the term "Asymbolia" for a trouble of this process. Both troubles, that of primary identification and that of secondary identification, are forms of Agnosia. Agnosia may, therefore, be subdivided as follows:

Agnosia.—(a) Trouble of the primary identification; primary agnosia. (b) Trouble of the secondary identification; asymbolia.

As these terms are also applicable to recognition through other senses, it logically follows that in the domain of touch the primary identification would be called "tactile astereognosis"; while the trouble of secondary identification would be called "tactile asymbolia." There would naturally be numerous transitional forms.

Claparède, however, goes farther and holds that inasmuch as asymbolia is an intellectual defect, and in his view due to a defect in the cerebral association connections, that it may exist without loss of either stereognosis or sensory troubles. This interpretation, however, he admits is theoretical and not based on actually observed data.

The intellectual element in perception of form and recognition of objects is brought out in testing those faculties in the use of the toes. Walton and Paul called attention to the inability of normal people to recognize form and objects with the feet, but their tests were insufficient, as they consisted simply in

[3] Agnosie et asymbolie; Rev. Neurol., Sept. 15, 1906.

pressing an object against the sole of the foot. The limited information furnished by such a method would be insufficient even in the hand, as may be easily shown. The only proper test is to allow a person to palpate, i. e., grasp the object with the toes. Tests made in this way with the patient whose case is reported by me in the present number of THE JOURNAL[4] gave some instructive results. The case was one of hemiplegia, the leg being slightly affected. In the right (affected) foot there was no loss of tactile sense, as tested with the camel's hair brush. The localizing perception similarly tested showed only few mistakes on the plantar surface (more on the dorsal). The perception of *movement of* the toes of the affected foot was moderately impaired both for movement itself and direction, the big toe, which was unable to recognize large circles of circumduction, being most affected. The same was true for posture.

Now when the perception of form was tested by allowing him to palpate the objects with the toes and roll them about against the floor, it was found that form was about as well recognized with the right foot as with the normal left, as shown by his answers, although subjectively it was apparently better recognized on the left, the patient asserting that this appeared to him to be the case. The difference was not remarkable. In correctness the answers varied considerably as respects both feet, but apparently perception depended upon the nicety with which the object could be grasped. Once the perception on the right was substantially correct when a flat circular tin ointment box about one and one-half inch in diameter and one-fourth inch deep was described as "round (i. e., circular) and flat on top, one and one-half to one and three-quarters inches in diameter"; but on repeating the test it was described as "not round," as was the case on both occasions on the left.

A small bottle.—Right, "round like a ball" Left, "like a penknife as I roll it over." Repeating test on right, he gave the same answer as on the left, but thought he palpated the bottle differently from the first time.

Square box.—Right, "round like a ball." Left, same.

Piece of erasing rubber.—Right, "long, something like your finger" (correct); "bevelled edges" (wrong). Left, same.

Small glass tubular bottle.—Right, "like a pencil." Left,

[4] A Study in Tactile Localization, etc.

"larger" (correct). (Thought it elliptical in diameter owing to a ridge made by paper label becoming creased when rolled under foot).

The patient found it difficult to give a more extended description than that contained in the above answers. But as to the *recognition of the object,* whether by the normal left or by the right foot the patient had not in a single instance the remotest idea of what it was. A little consideration will make clear that this is what might have been anticipated, for however near his recognition approached to a true perception of form in three dimensions there was plainly insufficient information for recognition of the object itself, for he could not follow the details of form nor make more than a guess at the material. The fact that the failure of object recognition was due to failure of sufficient information to form an intellectual judgment, and not to the absence of a cortical center for tactile symbolia, is supported by the following interesting detail: When the subject was shown the test-objects (seven in all) before the tests were made, he was able to name the object in nearly every instance, making only two mistakes with the *right* foot and one with the left: viz., on the right a ball was called a "short bottle," and on both sides the necked-bottle was called the tubular bottle, the patient being unable to detect the change of form at the neck and therefore recognized only the tubular form.

When asked to analyze the subjective perception and describe how he identified the objects after having become visually familiar with them, he said there came into his mind a visual image of the object which he could "see under his foot." In other words, it would appear that, now knowing what sensations ought to go with each object, out of such sense impressions as were furnished he could construct the object subjectively and identify it. This is supported by the fact that he continued to see the necked-bottle as a tube-bottle even after knowing its true nature, for, as he said, the feeling was that of a tube without a neck, and this raised the image of that kind of a bottle.

By contrast [with the hand where there was profound alteration of various forms of sensibility (localization, movement, pressure)] he remained still unable to recognize objects after he had been made familiar with them by sight. That is to say,

the sensory impressions from the hand were not sufficiently precise to allow him to identify them with the memorial images of those previously experienced. This interpretation is supported by the following observation: After considerable improvement had taken place in the sensibility of the palmar surface of the hand it was found that the perception of localization, when tested by Volkmann's method, was practically normal. This method, called also the "looking" procedure, consists in allowing the patient to open his eyes after a spot on the skin has been touched and directing him to point out the spot touched He thus has the actual visual image of the hand to guide him in finding the spot. Now, when a screen was placed so that he could not see his hand and he was directed to mention which finger and which segment of the finger were touched, he was all at sea in his answers. The errors in localization were very wide and consisted in guessing the wrong finger as well as the wrong segment.

The explanation which the subject gave of the difference in the results of the two procedures I employed was as follows: By the "looking" method the visual image of his hand enabled him to recall the normal sensations belonging to each segment, and by comparing these memorial images with the sensation awakened by the test he could locate the latter. When, however, he was unable to see his hand, then he had no memorial images with which to judge and compare the test sensations which in themselves must have lacked the quality of "thereness" necessary to enable him to locate them without comparison. It is of course under the latter conditions (without vision) that stereognosis must necessarily be tested, and the observation shows how the perception of localization varies according to the method by which it is tested. This has been well brought out by Spearman[5] in his excellent analysis of the various procedures.

Mills and Weisenburg[6] in a recent paper on "The Subdivision of the Representation of Cutaneous and Muscular Sensibility and of Stereognosis," meaning symbolia in the cerebral cortex, have maintained that the stereognostic zone is independent of the cutaneous and muscular sensibility and also of the motor area, and, following Mills' previously expressed views,

[5] Analysis of localization, etc. Br. Journal of Psychology, January, 1905.
[6] The Journal of Nervous and Mental Disease, October, 1906.

that the streognostic (i. e., symbolic) area is located in the posterior parietal region. (What they mean by stereognosis is not perfectly clear, as they do not definitely distinguish between stereognosis and the intellectual recognition of the object; but their language and the tests employed identify the two and make astereognosis the equivalent of asymbolia.)

They report three cases, in two of which their so-called astereognosis was confined to the three outer fingers of the affected hand. In the third case, while there was general asymbolia of the whole hand, they note what would appear to be a significant fact, to be later referred to, that there was loss in these same fingers of the perception of angularity of objects. From these findings they draw the conclusion that there is a subdivision of the stereognostic perception in a definite cortical area of the brain which they localize as above mentioned. A careful study of these cases, however, shows that they prove too much for this interpretation.

In the first case there was at one time impairment or loss of the important tactual perceptions (including that of position) in the whole affected hand and with this defect intellectual recognition of objects (called by the authors "stereognostic conception") was also lost in the *whole* hand. Later the loss of sensibility largely cleared up in the thumb and forefinger, where the "sense of position" became normal and with this clearing up the patient regained the ability to recognize objects between these digits, but could not do so with the other three fingers where sensibility remained impaired. Later, when loss of sensibility again extended as before over the whole hand, he was again unable to recognize objects over the *whole* hand. With partially lost sensibility there was partial asymbolia; with completely lost sensibility, complete asymbolia.

In the second case Spiller had found loss of "stereognostic perception" apparently in the whole affected hand, corresponding to loss of perception of position, localization and movement (Spiller does explain what is to be understood by stereognostic perception). Later Weisenburg found that the loss of sensibility had largely cleared up in the thumb and forefinger, but persisted in the three ulnar fingers and correspondingly, be it noted, that he could recognize objects with the thumb and forefinger, but not with the others.

It seems to me these findings are pregnant with meaning, not as showing the subdivision of the "stereognostic conception" center in the cortex, but as showing that with the subdivision of information given by the tactual senses there is a subdivision of the intellectual ability to recognize objects. If the "stereognostic conception" had a special center of its own and particularly if, as the authors believe, separated from the tactual centers, *we should not expect this recession and expansion of the "stereognostic conception" pari passu with recession and expansion of the tactual perceptions.* We should expect that they would not be absolutely *co-extensive* as they are, but that stereognostic loss from lesion of one center would at times exceed, or be present without loss of, sensibility due to lesion of another center. Tactual defects always occur with tactile asymbolia.

Stereognosis is plainly an intellectual process and to attempt to localize it is to do that for which there is no analogy in the scheme of cerebral localization. Intellectual judgments are not thus far, according to data at hand, localizable nor likely to be. The nearest approach to such a scheme is that of Marie's new theory of aphasia, according to which aphasia is an intellectual defect and localized in Wernicke's zone. This, to my way of thinking, is the weak point in Marie's theory. It is true that Marie defines the intellectual defect as a *special* intellectual function, but he fails to define what he means by a "special function," though holding that it is not one of special sensory images—visual, auditory, etc.

Though Marie may be quite right in his general theory, and in the localization of his intellectual aphasia in Wernicke's zone, his denial that the special intellectual defect consists of a loss of sensorial images may be wrong, and does not seem to be warranted by any evidence that he brings forward; and it may well be, therefore, that if his localization is correct, it is only a localization of sensorial images.

This denial of sensorial images in the mechanism of aphasia seems to weaken his theory rather than to strengthen it. Certainly, the localization of broad intellectual processes does not at present rest upon any sound basis of fact.

The only conclusive evidence for the localization of symbolia must rest upon clinical observations in which there is found to

be loss of power to recognize objects by touch, without loss of the subsidiary sensations and perceptions, and the final demonstration of focal localization in the cortex. No such case, at least no case exhaustively examined, so far as I have been able to find, with or without anatomical findings, has thus far been reported. In the absence of such a case there is absolutely no logical warrant in attempting to treat symbolia as a sense or concept or function and find for it a localization. Symbolia can only be spoken of and treated as a function symbolically, as a convenient expression to represent a complex process, and although it may be legitimate to do this for purposes of convenience, we are at once led into error when we attempt to localize the symbol. *In localizing symbolia, we are interpreting the evidence which in every case involves the impairment of different forms of sensation; and the interpretation which localizes the tactual impressions and makes the symbolia depend upon the loss of sufficient information for judgment fulfills all the logical requirements of the case.*

If the clinico-pathological evidence can not be interpreted as indicating the localization of asymbolia, the intellectual recognition of an object, can it be interpreted as indicating a localization of true *stereognosis* or form perception?

The tri-dimensional recognition of form is a complex process, though not so complex as that of identification of an object. It is dependent upon the integrity of the subsidiary sensations and perceptions. Though tri-dimensional perception, that is, of solidity (stereognosis), is not of itself sufficient for secondary (intellectual) identification, it must shade through intermediate forms into the latter as the perception of curves, angles and shape become added. Yet it is theoretically possible that in the simpler and in many of these intermediate forms, the subsidiary sensations and perceptions may become so organized as to permit of its definite localization as a quasi-function in a so-called cortical stereognostic center. A definite proof of this interpretation must, however, as with symbolia, depend upon the clinical demonstration of loss of the stereognostic sense coincident with *integrity* of the subsidiary tactual sensations and perceptions—touch, localization, movement, etc. In the absence of such demonstration and in the presence of impairment of one or more of the latter, we plainly are not justified in interpreting astereognosis

proper as a loss of a special function with a special center, but only as due to impairment of the subsidiary perceptions upon which our recognition of form depends; that is, to loss of sufficient information.

The above analysis and criticism of Mills and Weisenburg's two cases is also applicable to them in respect to the perception of form (stereognosis proper) which I think we may safely assume was lost in these cases because of the profound impairment of localization, etc. The third case bears distinctly on this point. Here was inability "to recognize an object placed in any part of the hand." Nevertheless, the patient could recognize "*angularity* of all objects with the thumb and forefinger," but not with the other fingers, and corresponding to this difference, sensibility was least impaired in the thumb and forefinger. Perception of angularity is of form, and this varied with the condition of sensibility. The sensory information from the forefinger and thumb, as in Raymond and Egger's and Dejerine's cases, was sufficient for the recognition of form but not for recognition of objects.

Whether or not then, stereognosis and symbolia are to be regarded as having a definite cortical localization depends primarily upon the interpretation of the clinical evidence. The localization of the subsidiary sensations and perceptions is all that this evidence warrants. Theoretically, it is not impossible that the association of the sensory perceptions involved in the recognition of form may become so highly organized that the correlated process may be localized in a definite center, but this is far from proved.

The terms "tactile asymbolia" and "tactile astereognosis" ought, I believe, either to be dropped from our symptomatology, or retained as symbols of complex tactual defects. We do not coin terms to define faulty judgments in other sensory fields; when vision is impaired so that objects are not recognized, we do not say there is visual asymbolia, and so with the other senses. If the terms are retained, they should be used only as convenient age careful examination of the various forms of tactual defects The disadvantage of such terms is that their use tends to discourage the careful examination of the various forms of tactual defects in individual cases and to confusion in cerebral localization.

GENERAL CONSIDERATIONS AS TO THE NATURE AND RELATIONSHIPS OF HYSTERIA

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Hysteria has long been called in the text-books essentially a mental disease, but this statement has been usually confined to the opening paragraph of the discussion and the clinical symptoms described without further reference to their mental origin. No effort has been made to show the relationship between the hysterical convulsion or contracture and the mental process. It was only realized in a vague way that the so-called physical symptoms of hysteria often arise in connection with mental stress; that agents operating directly on the mind were most effectual in removing them; that they characteristically lack evident organic foundation and come and go so quickly that they cannot be conceived as rooted in tissue changes more profound than those functional changes, whatever they may be, that are no doubt the concomitant of all thought. On the other hand the text-books on insanity have given hysteria but scanty consideration and have tended to regard it as a cause, or a condition accompanying insanity, rather than a distinct and definite mental disturbance. The psychiatrists have largely ignored its mental nature. Mental disease has been for them too much insanity—disease of the brain—a condition demanding confinement in an institution and totally abolishing responsibility; and they have overlooked the possibility of milder mental disorders only in aggravated instances becoming severe enough to require restraint; for, it may be said that hysterical insanity is only hysteria so aggravated in one or another of its mental features that the disturbance of the reason is the most prominent symptom, and often so aggravated that the condition is not recognized as hysteria at all.

Before proceeding to the study of hysterical insanity a definite idea of hysteria must be had. The idea of simulation

should first be discarded. Hysterics have poor memory, have the natural or even an exaggerated fondness for sympathy, are suggestible, develop the symptoms of whatever malady comes to their attention but they do this in general unconsciously, and with no more intent to deceive than the medical student who finds in himself the symptoms of all diseases on which his professor lectures. Fraud is not hysteria, though some hystericals may add fraud to their psychosis.

Some believe suggestibility the essential factor in hysteria. This seems the view of DuBois, who regards neurasthenia, hypochondriasis, melancholia and hysteria as mental disorders of similar character and not sharply differentiated from each other, and often present together in one and the same patient. Such of these patients as are pre-eminently suggestible he regards as hysterics. This no doubt is one symptom, but persons who have never developed what we would call an actual hysteria are suggestible, and other more definite phenomena in hysterics have been pointed out by others.

Sollier explains the symptoms of hysteria on the theory that all the brain functions, sensorial, emissive and elaborative, have their brain centers in the brain cortex. Their combined and harmonious activity constitutes the personality. Hysteria according to this view consists in an inhibition, the nature of which he does not know, but which arrests the function of one or more of these centers. He likens this arrest to sleep and correlates the usual sleeplessness of hysterics and the fact that often they cannot tell if they are asleep or not. He regards some part of the brain as asleep, and accordingly tries vigorously to wake up his patients just as if they were asleep in the ordinary sense, and finds thereby a great change in their behavior. If this sleep falls upon the motor centers of a limb the limb cannot be moved. If it falls on the receptive centers an anesthesia is produced, and if it falls where given memories are stored, they cannot be received. If it overtakes a center that is functionally active the activity remains as a fixed idea, or a contracture. The action of a number of centers without the others explains double personality. These conceptions, going as they do far beyond our anatomical knowledge, and unsupported in their details by much that we know of the psychology of hysterics cannot

be accepted as statements of probable literal fact but may furnish a useful point of view in passing to those who like a schematic arrangement of knowledge even though that involves unproven assumptions and will not include all the facts.

In hysteria the emotions are poorly balanced and some have found in this the characteristic feature. Kraepelin would speak of hysteria when there is "a chronic constitutional state with paroxsysm presenting throughout a condition of increased influence of emotional reactions on the body." Ideas have the force of sensations and he believes that in hysteria we have always to do with the involuntary effects of excited sensations referable to the patient's own body. But this view again does not cover all the facts, for says Pierre Janet, "It is known that in general hysterics are very indifferent; they are fond of repeating past emotions but are incapable of forming new ones in harmony with their present situation"; and says Sollier, "This indifference is not only in the intellectual, but in the moral order. Their families often wonder at their lack of demonstrativeness: they bear the announcement of the happiest events without ever manifesting any sign of happiness or sadness; equally indifferent to laughter and tears."

It is not then emotions as such that modify the hysterical mental state in such a peculiar way, but the failure of certain ideas, often ideas of great emotional import to be clearly brought into consciousness. Charcot first showed this in an analysis of the traumatic hysterical accidents; paralyses, contractures, mutisms and anorexias. He showed that fixed ideas, not conscious to the patient in the ordinary waking state, caused these conditions, kept them up, and by suggestions, also not conscious to the waking state, he removed them. Moebius applied this concept to the whole of hysteria, and considered hysteria a morbid mental state in which ideas produce bodily disturbances. Hysteria, according to Charcot and Marie, is less a disease "than a peculiar mode of feeling and reacting." Janet endeavors to point out what mode this is, and finds the essential feature of hysteria to be a tendency toward mental disintegration with "undoubling of the personality." In his work on "The Mental Status of Hysterics" he develops this thesis.

Following Janet we speak of hysteria as a tendency to re-

duplication of the personality, but most of these so-called personalities are very fragmentary. Rather than an organized personality it may be a confused delirium that alternates with the clear state of mind or periods of agitation and unrest, a convulsion, or hysterical crisis in which at each time certain acts are reproduced. These states have been analyzed by various observers (Janet, Breuer, Freud, and Prince), and shown to be due to subconscious fixed ideas, just as contractures and paralyses were before shown by Charcot to be due to the same cause. One of the best of these analyses that we have seen is that of Riklin, summarized by Dr. A. Meyer in the *Psychological Bulletin* of July 15, 1905. Riklin traced in one case many of these symptoms to their unconscious origin, and found that as fast as these unconscious sources were uncovered and the facts brought to consciousness, the hysteric symptoms dependent on them disappeared.

Janet finds the basic defect, the source of the difficulty in hysteria, to be a limitation of the field of consciousness, making it impossible to attend to all the elementary sensations at once, and as a result some of them do not reach consciousness. None of us attends to all of his elementary sensations at any one time, but the normal person as the necessities of psychic life demand shifts one and another into the center of consciousness. When through defective mental synthesis some impressions do not reach consciousness, anesthesias develop, gaps in memory occur, from lack of attention enfeeblement of motion or catalepsies develop, the will is weakened, intellectual development is arrested, and changes in character occur. That these are due to faulty attention and not to organic failure of the peripheral sense organs or of the brain, is shown by such facts as their variability in short periods of time, just as absence of mind varies; by their disappearance under hypnotism and in many delirious states; by their limitation in some instances to sensations related to some especially singled out experiences; and by the realization of the anesthetic parts in many of their every-day activities, as for instance: the avoidance of obstacles though the peripheral parts of the retina are hysterically blind, and especially by the demonstration of their reception by methods of distraction and by automatic writing. These symptoms are spoken of as "stigmata"

of hysteria. They are the clinical signs that some sensations do not become conscious and when found in a person otherwise receptive to stimuli they mark a division in consciousness. Such a division is the mental nature of hysteria. The stigmata then must assume great importance for this study, for it is by scrutinizing cases of insanity for such stigmata that a division of the mental content or hysteria may be most readily shown.

Theoretically at least, the stigmata are set off in sharp contrast to the "accidents" of hysteria, the peculiar symptoms that chance in any particular case to develop on the hysterical basis. Thus in one patient the idea of powerlessness arises and she develops paralysis; another develops a contracture because of the persistence of some idea held by the subconscious state that controls the limb; these ideas may give rise to automatic acts, to criminal attacks, to convulsions, to somnambulisms, which Janet considers the typical accident, and by which he means "a state in which the subject possesses particular recollections which he finds no longer when he returns to the normal state," and finally to deliriums, by which he means such disturbances of thought as to be clearly insane.

These views of Janet concerning personality and its relations to hysteria may be more clearly understood in the light of the dissociation concept. It regards the normal personality as made up of the sum of its experiences and their elaborations. All know that often there are remembrances really in the mind, but which cannot at a given time be recalled. This is in the widest sense of the term a dissociation. Ordinarily such memories come to mind on thought or spontaneously after an interval, and in either instance they are recognized by the mind as its own. Besides this natural and usual order there are other conditions wherein memories lost to the normal consciousness are recalled in second and alternating conscious states, neither of which is aware of the events of the other, and wherein if by any chance a memory of the second state arises in the primary mind, it appears foreign and is not recognized as the memory of a personal experience. In other words the dissociation is systematized and two more or less separate and independent minds are found in one body. Dis-

sociations of this character are found in hysteria; in hypnotism, as Charcot pointed out, and in mediumistic states as shown by Binet. Their extreme type is multiple personality. Spontaneous sleep walking; automatic writing as practiced by hysterics and mediums; crystal-gazing and automatic replies in states of distraction are of the same character. The recognition that all of these conditions are on a similar basis of cleavage in consciousness, gives a double insight, allowing each to throw light on the nature of the others, and in turn, through them to be better understood.

Janet uses the word hysteria to cover all of these conditions, and we shall, for the purpose of this study into the hysterical insanities, regard evidences of dissociation with a tendency to organize into states interrupting the usual stream of conscious mental activity as hysteria. Though it departs far from the etymology of the word, we use it as there is no other to cover this concept as a whole, and as hysteria has long been used to include the major portion of these dissociations, it is only extending and making its meaning more definite when it is made to cover the whole. That dissociation should play a prominent part in mental diseases seems a priori probable when we reflect how nearly universal is susceptibility to one of its types, hypnotism. At Nancy they hypnotize 90 per cent. of the subjects presenting themselves at the clinic, and believe that with time and patience most of the remaining 10 per cent. would succumb. Some persons and some families, are, however, specially liable to such a change in consciousness, and its most typical manifestations as hysteria are most often seen in women. We think that this readiness for dissociation constitutes what writers such as Kraepelin regard as the constitutional feature of hysteria, but we regard it rather as a peculiar aptitude or mental tendency, if you please, to be compared rather to mathematical or musical ability or to anomalies of memory, and would speak only of hysteria when as a result of the limitation of consciousness any of the systematized dissociations have appeared. In a similar manner we regard ordinary suggestibility as a trait of character, and one that is present to some degree in every mind, but regard it as an hysterical symptom only when it is the effect of a dissociation; though it is evident that a conscious state that is

without its usual criteria for the formation of sound judgments especially favors suggestion.

GENERAL CONSIDERATIONS CONCERNING HYSTERICAL INSANITY

Hysteria rarely appears as the diagnosis of an asylum patient's mental disorder, and even hysterical insanity is widely ignored and lost under the all-compromising heads of acute mania or melancholia. Thus in all the thousands of cases admitted to the New York State Hospitals whose diagnoses are published annually in the reports of the New York State Commission in Lunacy, no mention is made of hysteria. This is not because no cases are received, for as Clouston remarks, hysterical young women are often committed, but rather because of a vague idea that the hysterical symptoms are not important; that they are accidental complication of some real mental disease; while hysteria is not regarded as a psychosis in itself; and the text-books fail to reflect the present state of our knowledge.

Gressinger considers hysteria among the causes of insanity, but remarks in advance of many who follow him that in hysteria proper, only very seldom do the mental faculties remain free from disturbance. Hammond goes so far as to say that hysteria is not hysterical insanity; Clouston, Skitzka, Talcott and Connelly Norman in Tuke's Dictionary of Psychological Medicine speak of hysterical insanity or hysterical mania as an intensification of, or further development of, what they regard as hysteria proper, and further complicate the picture by including in their descriptions symptoms which almost certainly they must have observed in deteriorating psychoses. Kellogg and Bevan Lewis ignore the topic, though the former has a chapter on somnambulic insanity, and the latter quotes a case under "Epileptic Insanity," a case which he thinks presents both true epilepsy and hysteria, and in which from the descriptions given it would seem that the hysteria is better established.

The newer books do not help us much more. They no longer ascribe the symptoms to simulation, perverseness and a diabolic desire to deceive, but recognize that they pass beyond voluntary control. Berkley heads his chapter "Psychoses Accompanying Hysteria," and describes briefly many

of the mental disturbances which are found in hysterics; but shrinks from the inevitable conclusion that these disturbances are hysteria. He very justly doubts if true mania or melancholia ever develops on an hysterical foundation. Brower and Bannister indefinitely regard hysterical insanity as something growing out of hysteria. Their description goes little beyond what is spoken of as the hysterical disposition, and they regard somnambulistic states as allied to epilepsy and on the borderland of hysteria, and best considered with it; rather than as the essence of hysteria itself. Defendorf heads his chapter "Hysterical Insanity," but his description applies to the mental peculiarities of all hysterics. Church and Peterson describe it among the nervous diseases, but give a good account of the symptomology, making free use of the investigations of the French. Kraepelin, in his Clinical Psychiatry, presents a few clear cases of hysterical mental disorder and lays stress on his conception that "Hysterical insanity is the expression of a peculiar, morbid tendency, and can be brought to further development but not originated by external causes." Krafft-Ebing describes hysterical insanity under states of transitory insanity; protracted states of hysterical delirium and hysterical psychoses. Under the last head he refers to mania and melancholia and especially paranoia on an hysteric basis. Paton describes several states of hysterical mental abnormality with useful points in differentiation, but shows the confusion in which the subject is involved by quoting as illustrative cases of hysterical insanity two cases of egocentric disposition, lacking application, wilful, vaccillating and contradictory in their actions, one an inveterate liar, the other the author of shallow threats against her own life, but without presentation of the distinctly hysterical features or symptoms showing dissociation. In one case especially he seems to regard the presence of such a character of itself enough to show hysteria, and as confirmatory evidence adds only the efficiency of a placebo in the control of pain. Bianchi recognizes that all the phenomena of hysteria are "either purely psychic phenomena or intimately connected with them. He lays stress on a duplication of consciousness, but seems to mean by this phrase contradictory character and inconsistency in morals. One of his cases with a long history of delinquency is without stig-

mata characteristic of hysteria other than mental instability. Besides hysterical delinquency he describes hysterical stupor with stereotyped poses of sexual or hypochondriacal nature, hysterical delirium, and following Schüle, Mendel, and Krafft-Ebing, hysterical mania, melancholia, and paranoia. Janet gives a chapter to the deliriums, by which he means mental derangement, especially delusional states. His first type is "Exaggeration of Abulia"—mental confusion. The patients attend insufficiently, forget, and doubts and astonishment before new objects develop, but usually not amounting to a delirium. One patient walks about, answers wrongly and at random questions asked her; one cannot tell her name, and becomes angry, complains, "How dark my brain is."

He quotes at length the case of a girl with hysterical antecedents learning with great labor, millinery. She had an attack of 20 minutes' duration with contortions, lockjaw, and contractures. After an attempt to seduce her she became much agitated, talking wildly about the gentleman who wanted to run away with her. After some days' agitation she again had repeated attacks of convulsions and contractures. Two days later she looked more surprised and dumbfounded than delirious, and was entirely without stigmata. It was nearly 3 months before she began to have lucid intervals during the day, and with them anesthesia developed. He is inclined to think her stupor not very characteristic, and that such stupors may be met in many mental maladies.

His second form is "Exaggeration of Automatic Phenomena"—"maniacal delirium." He tells of a patient who automatically chatters the "Telegraph language of the maniacs" for half an hour at a time. It is guided by the little impressions that strike her senses and by her own associations, but apparently from his description no elation accompanies it. Another patient has attacks of delirium when she is constantly hallucinated, hears people insult her and sees men and horses fighting and bleeding. He quotes the case of a girl of seventeen with five insane or hysterical near relatives, who was said to have had hysterical symptoms, including hemianesthesia, at the start, but which judging from his description of the case as he saw her, we would call dementia præcox. Janet sees in

a vague way the preceding hysteria, but he says, "It indicates evidently a considerable transformation of the malady."

His third form is the "Exaggeration of Fixed Ideas—Systematized Delirium." First are quoted cases in which fixed ideas persist as depressing obsessions, producing sadness and despair. He mentions a case showing how extensive lying developed from trying to conceal a single fault; another showing how eroticism arose from a perfectly proper offer of marriage; he regards the hysterical anorexia as a delusional state, and gives instances of its development from fixed ideas, and shows that hatred and persecutory delusions may arise on this ground. He completes the topic with an account of a case beginning with hysterical symptoms and sexual perversions now changed to a system of delusions of persecution and grandeur.

In his fourth and last heading he considers the relationship of these mental disturbances to the other phenomena of hysteria. He cannot find in the hereditary antecedents or personal characteristics of hysterics with delirium or delusional states anything which marks them off from others in which delirium does not develop.

He regards hysterical insanity as one of the accidents; just as a subconscious idea paralyzes or makes rigid a limb, or just as the hypnotized subject becomes anesthetic under the influence of an idea of whose existence his waking state is not aware, so we believe (and many experimental examples are found in Prince's case) a hidden idea sets up delirium, melancholy or hypochondriacal feelings the patient does not adequately explain, states of perturbation and extreme unrest, paranoid delirium as well as states of stupor and lapses of consciousness.

We have collected from actual experience among patients committed as insane the case histories of several hysterical patients. They were observed in the wards of the Middletown State Homeopathic Hospital, and on those of the Manhattan State Hospital while working in the New York State Pathological Institute, and I am indebted to the superintendents of those hospitals, Dr. M. C. Ashley and the late Dr. E. C. Dent, for permission to avail myself of the material, and to Dr. Adolf Meyer for many helpful criticisms and suggestions.

In each case presented as an hysterical psychosis the diag-

nosis rests primarily on finding hysterical stigmata at one or another stage of the disease, but it will be seen that they are cases whose mental symptoms cannot well be forced into the symptom pictures of manic-depressive insanity, dementia præcox or involutional melancholia, which are now standard diagnoses among alienists, and to which one or another case will present some resemblances. These psychoses have been sufficiently studied to give fair working symptom groups, and when cases with hysterical stigmata are found, and further these same cases very uniformly lack the characteristic mental features of these more generally accepted psychoses, it is not only justifiable but necessary to consider these psychoses hysterical throughout. Other cases presenting very close resemblances to these hysterical cases, and not resembling other accurately described insanities, but without demonstrated hysterical stigmata are also found. These belong to the borderland and are subjects for profitable study. Some of them, with their relationships, will be entered here.

HYSTERICAL INSANITY AS ILLUSTRATED BY ACTUAL CASES

The first group consists of cases whose hysterical characteristics are so definitely marked and so clearly in evidence that there can be little or no question as to with what we have to deal. The cases are pre-eminently hysterias, and they are collected to show the usual symptoms of hysterical insanity.

Case No. I is a single woman of 23 admitted to the Middletown State Hospital on November 8, 1903. The families of both parents are humble and illiterate. Her father, aged 61, has been for 20 years or more an invalid, high tempered, nervous and said to be hysterical, and once for three days after a sunstroke, insane. He has always talked in his sleep, one of his brothers and a daughter of this brother walk in sleep. An aunt has convulsions similar to those of the patient, and the four grown-up brothers and sisters of the patient "holler and yell" if they do not get their way.

At the age of seven years she fell down stairs and her mother says was never well afterwards; she had jumping pain in her side and complained of her womb being out of place. She was a timid child, cried easily and slept with a light in her room; once at the age of 12 years she walked in her sleep. At 15 she had vomiting spells lasting about a week at a time, and was much troubled with prickling in the limbs like electricity. She stopped school at this age as the doctor said she

was too nervous and that she had too far to walk. Her education, received at country schools, is very meagre. For several years she was engaged to a young man and had sexual relations with him, but finally broke the engagement, and in October, 1902, he married another girl with her consent. There was, however, a third woman of whom she was jealous, and when she became pregnant in the spring of 1903 she alleged she was raped at the instigation of this woman. In May there was an abortion, and at each succeeding menstrual period until she was committed she had convulsive attacks, became short of breath, sighed and felt numb. She was admitted while in her fifth series of attacks. At home she strode up and down her room, tearing her clothing, talking incessantly, sometimes quietly to herself and at intervals breaking into loud cursing. The nurse who brought her to the hospital found that she had that morning covered her floor with bits of torn paper. She was crying and moaning. For two weeks her friends had been unable to keep clothing on her. She started willingly on the short journey to the hospital, then resisted on changing cars until forced to do so by two men; then she slept and finally brightened up, smiled and talked of her pains and of her expectations of getting well. The afternoon of her admission she had several seizures, and one each on the 13th and 14th of December. (Menses from Dec. 8 to 14.) Before the attacks she feels a blur before her eyes, her hands and feet get cold; perspiration starts; the arms and legs feel drawn; she feels something start from the pit of her stomach and come to her throat; she chokes for breath; sometimes she retches and is nauseated; her face bloats and becomes livid; eyes open and staring, hands clenched, thumbs outside; much borborygmus; a peculiar churning motion of the abdomen, and much tossing about in the bed. She does not hurt herself in falling, froth at the mouth, or bite her tongue. Single convulsions lasted as long as thirty minutes. She talked some during the attacks, but upon becoming clear professed amnesia for them and for the events preceding admission. Her physician at home regarded this as epilepsy.

No traces of deterioration or hallucinations were observed. In the intervals between the convulsions she was correctly oriented and natural in manner. The first two weeks she could not employ herself. Said her eyes hurt, but after that became industrious. Urine was abundant and of low specific gravity. There were fits of prolonged and unexplained coughing, with no cough in the intervals. No anesthesias were found except contracted visual fields, which were charted December 17, when a concentric contraction to about fifteen degrees was noted. At the same time using the finger as a test object a visual field extending to ninety externally could be

shown. January 4 this disturbance had disappeared. After December 14 no definite convulsive attacks occurred, though on January 29 her eyes became glazed and she said, "Oh, I feel as if I am going to have a spell." Menses in January were scant and caused no disturbance beyond talking in her sleep, and in February there was no incident, and after menstruating she was paroled home where three months later she remained well.

Summary—A young woman of hysterical antecedents, who for five successive months after an abortion developed at the menstrual period, convulsions. She screamed, cursed, cried and tore her clothing. For a few days following the convulsions contracted visual fields were observed, but no other anesthesias. Under hospital regimen she quickly recovered.

(*To be continued.*)

Society Proceedings

THE BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY.

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The President, Dr. Bullard, in the Chair.

TWO CASES OF FRIEDREICH'S ATAXIA.

By Dr. Knapp.

The man, aged sixteen, came to the hospital six years ago. The family history is negative, except that his paternal grandfather had partial paralysis and his mother's sister epilepsy. He was born at term, the labor was long and difficult, and he was cyanosed, not breathing for an hour when born. He walked at twenty-two months and seemed healthy. When between two and three years of age it was noticed that he spilled his milk and that his hands shook especially as he tried to move them. This has increased, the movements of the hands have become more ataxic and he stumbled in his gait. For the last year there has been some tremor of the head, and he has complained of cramps and loss of sensation in the left leg. The memory has failed somewhat of late, but before that he has showed normal intelligence. His speech is a little thick and he shows a very slight nystagmus, but the pupils and eye-grounds are normal. There is a moderate knee-jerk and Achilles reflex, but in the past the knee-jerks have been absent. When he approximates the knees the left leg shows a spasm and develops an equino-varus. There is a very marked ataxia and jerking movements of the hands, especially on intended movements.

The second patient is eighteen, and gives no history of any nervous trouble in the antecedents. An older sister, however, has had a staggering gait and intention tremor for four years, and a cousin, his father's sister's son, has had a staggering gait for four years. In both instances the trouble began at the age of seventeen. This patient was well until the age of fifteen when he was ill for a week with some infectious disease, called typhoid fever. On getting out of bed after it he was weak all over and has staggered ever since, but he notices no trouble in executing movements when lying down. He is liable to spill liquids and his head shakes at times. The gait is slightly ataxic, there are some static ataxia and choreiform movements of the head and trunk. The static ataxia is increased on closing the eyes. The eye-grounds are normal and the pupils a little sluggish. Knee-jerks and Achilles reflexes are absent. There is some jerky inco-ordination of the hands.

Sensation is apparently unaffected. There is some scoliosis and an apparent lack of development of the upper part of the trapezius, with an over-development of the scapular muscles, but there are no symptoms of muscular dystrophy.

A CASE OF SYRINGAL HEMORRHAGE COMPLICATED BY MENINGITIS.

By Drs. W. N. Bullard and E. E. Southard.

The case was that of a woman, fifty years of age, who had noticed, less than a year before her death, a numbness of the left foot with pain and tenderness. The numbness and tenderness increased and extended. Pains and tenderness appeared over the abdomen and chest. Weakness of the legs and incontinence set in and, four days before admission to hospital, paraplegia appeared.

On admission the patient showed complete flaccid paralysis of the legs with absence of knee jerks and plantar reflexes as well as loss of pain sensation as high as the groins and sacrum. Leucocytosis appeared and increased. The anesthesia extended first to the umbilicus and later on the right side to below the nipple. On the left side there was an area of intense hyperesthesia above the anesthetic area. A few days later the anesthesia retreated to the level of the umbilicus. She died without modification of symptoms after a gradual failure six weeks later, being semi-comatose thirty-six hours before death.

The autopsy gave evidence of a syringomyelia with gliosis in both posterior horns with an apparently total transverse hemorrhagic lesion in the lower dorsal region. There was a meningitis most extensive in the sacral region and gradual thinning out in the thoracic region. The pus contained numerous bacteria of various kinds. There was a bed-sore measuring 10x7.5 cm. in the sacral region. From the base of the bed-sore a probe could be passed into the spinal canal. The organs of the trunk showed nothing remarkable beyond bronchopneumonia with acute pleurisy.

The readers interpreted the data of this case as an illustration of the following series of events: I, Gliosis with cavity formation in the spinal cord; II, Hemorrhage into cavity with effects tantamount to those of transverse myelitis; III, Extensive decubitus with erosion of bone and exposure of spinal canal; IV, Ascending meningitis.

A CASE OF ISOLATED DEGENERATION OF THE POSTERIOR ROOT BUNDLES OF THE CORD, OCCURRING IN A CASE OF GENERAL PARALYSIS.

By Dr. H. A. Cotton.

The lesion involved the entering roots of the seventh cervical segment, and was limited to the right side of the cord. The degenerated root bundles were easily made out macroscopically, and by their pearly gray color were easily distinguished from the other healthy root bundles. Serial sections of the cord above the lesion were stained by

Weigert's method for medullated fibers, and the course of the degeneration could be made out as a narrow tract passing upward, and gradually approaching the column of Goll in its ascending course. The interest in the case was mainly anatomical, as the clinical features did not differ from those in an ordinary case of general paralysis. Such lesions are rare except when produced experimentally in animals. A similar case has been described by Schaffer (*Anatomisch-Klinische Vorträge* Jena, 1901.), who lays much stress upon such a degeneration occurring in the cord in general paralysis. This is a true exogenous degeneration of the posterior columns. It differs from a true tabetic degeneration only in its intensity and can be considered as substantiating the view that the degeneration of the posterior columns in general paralysis is identical with true tabes.

The histo-pathology failed to show just why the seventh cervical root should be selected, although Schaffer found an enormous increase in the connective tissue cells at the point of entrance of the posterior roots, also a marked thickening of the pia. Photographs of the segments above and below the lesion were shown to illustrate the degeneration.

A CASE OF CONTUSION OF THE BRAIN, AND A CASE OF BRAIN TUMOR.

By Dr. Baldwin.

Dr. Putnam said that he had been interested in Dr. Baldwin's statement that what might have been diagnosticated as a "cerebral concussion," proved actually to be a cerebral lesion, and that, too, a lesion remote from the seat of injury. In his opinion cerebral concussion considered as wholly independent of actual injury of brain tissue rarely occurs, except in the sense that a severe nervous shock may form a part of a traumatism or that the cerebral circulation may be very seriously demoralized under similar conditions.

Dr. J. J. Putnam called attention to Babinski's method of treating aural vertigo by lumbar puncture, which this distinguished observer had advocated on the basis of a large number of observations. He had recently used this method in two cases, and with great benefit. At the time of the operation both patients were suffering greatly from a constant series of attacks that had practically incapacitated them for work. One of them was a middle-aged woman, the other an intelligent student of the law. In the first case about ten cubic centimeters of fluid were withdrawn; in the second about fifteen cc. The second patient suffered from headache, felt only, however, when he was in an upright position, for a week or more after the operation; but both patients were relieved from their vertigo, and had no return of it, except for one or two trifling indications, during periods amounting in one case to about six months, in the other to about three.

In commenting on the probable explanation of these results, the speaker referred to Babinski's statement that the withdrawal of the fluid diminishes the pressure in the labyrinth of the ear and pointed out the fact that we see quite often results that seem permanent in the treatment of neurological conditions as following from influences that

would have been classified as temporary. Thus, in one very interesting case of radicular pain in both arms and shoulders, due apparently to the encroachment of an intra-spinal tumor, the speaker had seen permanent relief brought about by vertebral laminectomy which had caused the draining off of a large quantity of cerebro-spinal fluid. It is now ten years or more since the operation was done and the pain which had become unbearable has never returned.

THE MELANCHOLIA-MANIA GROUP OF PSYCHOSES.

By Dr. Cowles.

Dr. J. J. Putnam said that he had been greatly interested in Dr. Cowles's work for a long time past and that he believed it to be distinctly in the right direction. The direction, namely, of making clearer the fundamental physiological principles that underlie the symptomatology of disease. This is a path which Hughlings Jackson, in his earliest writings, entered upon with so much interest and intelligence. Indeed, it may perhaps be said that he was a pioneer in this field. Ostwald's elaboration of the doctrine of energies is certainly of great value, even though there are some of us who could not agree with him in his belief that any such physical theory could be made a substitute for philosophy and metaphysics.

Dr. Cowles's chief argument, as he understood the matter, is that there is no such antagonism between the states of so-called depression and exaltation as the dictionary rendering of those words would suggest.

Dr. E. E. Southard agreed with Dr. Cowles that the Wundtian psychology, especially as regards the feelings, could be applied with great difficulty to numerous diagnostic problems in psychiatry. He felt that a bipartite division of the mental life, perhaps on the lines of Brentano, was in some ways superior, for psychiatrical purposes, to the *knowing-feeling-willing* psychology. Where feelings and the will are not too sharply sundered for the purposes of classification, the practical worker can the more readily stick to the important facts, which are, of course, motor facts. In this way the inadequate schematism of Griesinger can be dissolved.

Modern emphasis on the motor, viz., the *accessible,* elements in the mental life is also shown by Dr. Cowles' endeavor to utilize the energy concept in constructing curves approximating the reduction of energy in individuals suffering from neurasthenia, followed by melancholia, later by mania, then perhaps by exhaustion, before an up-grade is estabtablished once more. Whereas Dr. Cowles' idealized curves represent the scale of energy *to spend* in the individual and suggest that the maniacal subject may have less to spend than the melancholiac, some of the conventional curves of the textbooks represent rather the energy *spent. Melancholia agitata* is an example of a field very worthy of graphic presentation on these lines.

On the histopathological side it seems difficult to obtain even a rough idea of the amount of energy engaged in the brain. Chemistry yields little as yet. We can obtain some hints from the deposit of lipochromes in certain cells. These may serve as indices of materials

held back in the cells of older subjects. We should study more carefully these older subjects, the presenile and climacteric disorders.

Dr. Channing said that he felt it a duty to express the debt of gratitude he owed to Dr. Cowles for his admirable paper. It indicated to his mind the change that had come about in the last few years in the study of mental diseases. Formerly the more elaborate a system of classification could be made, the greater authority its maker was supposed to be. Now the emphasis was being laid on the investigations of psychical conditions, or psycho-motor states in the earlier stages before disease was fully developed. If we are to make progress in treatment we must first of all understand the significance of underlying psycho-physiological processes. Classification had been given far too much importance. Dr. Carter Grey long ago had said that in his opinion they were of little value, and the speaker was coming to believe that they were "the last resort of noble minds." We had heard of "the passing of neurasthenia," and we might bye and bye hear papers read on the passing of melancholia, mania, paranoia and dementia præcox.

Dr. Abbot agreed in the main with Ostwald's theory of energetics as outlined by Dr. Cowles. All activity, whether physical or mental, means expenditure of energy. This expenditure must conform to all the laws of energy. He looked on mental action, whether in the intellectual or emotional or psycho-motor fields, as activities of the individual, and hence as modes of energy, requiring the expenditure or transformation of energy. And the diminution or loss of available energy may give rise to the symptoms which we see in the manic-depressive group of psychoses.

But he should take exception to the diagrams that represent the maniacal or excited phase as indicating a greater loss of energy than the depressive phase, for example. In any individual case the symptoms will depend quite as much on the physiological locus of the fatigue as on the degree of fatigue (using fatigue in the sense of diminution of energy, which in these cases extends to a pathological degree). That is, if the physiological seat of voluntary inhibition is especially fatigued, the patient will show symptoms of motor activity and flight of ideas. If the physiological locus of motor activity is especially fatigued, the patient will show motor retardation. The amount or degree of fatigue may be the same in either case.

For similar reasons he should take exception to the attempt to represent diagrammatically the emotional, motor, and intellectual changes in a given case by one curve. The diminution of energy which underlies the emotional changes is not always equal to that underlying the psycho-motor or the intellectual phenomena, and at least these three curves of energy should be drawn to represent the given case. These curves would almost never be parallel.

NEW YORK PSYCHIATRICAL SOCIETY.

May 1, 1907.

The President, Dr. Adolf Meyer, in the Chair.

RECOMMENDATIONS CONCERNING THE IMPROVEMENT OF MEDICO-LEGAL METHODS.

By Dr. Pearce Bailey.

The speaker said it was not difficult to point out glaring defects in our code of criminal procedure, judged by the standards of modern psychiatry, but legal, judicial, and even medical obstacles stood in the way of remedying these defects. Any change from existing conditions would be possible only after a severe and prolonged contest by concerted forces. The fundamental error in criminal lunacy cases was the test of responsibility; *i. e.,* the knowledge of right and wrong as to the act at the time of its commission. This had been condemned by medical writers as being no test at all. Should the letter of the law be strictly observed, no lunatic could, under it, escape the full punishment for his offense, unless it could be shown that, at the time of the commission of the act, he suffered from distinct clouding of consciousness. Under this law, nevertheless, many murderers had been acquitted by reason of insanity. A loophole had been left in certain States—not New York—by adding the "uncontrollable impulse" clause. When this clause did not exist the fate of the prisoner depended upon the construction which the jury saw fit to place on the knowledge of right and wrong as mentioned in the law. The question would naturally arise why, if so unsatisfactory, this test had stood so long with so little variation. The reasons were evident. Defective as is the knowledge of right and wrong as a test of responsibility, no better substitute had ever been offered. Another reason why this law had stood might be found in the fact that the matter is always in the hands of laymen to whom the science of psychiatry is practically unknown. This test would have to endure so long as the code of criminal procedure remained what it is to-day, for the reason that this test is as good as any single test of insanity might be. Modern psychiatry demanded that this test be, not replaced by another, but be done away with altogether. No criminal procedure could be at harmony with modern views of mental disease as long as there was no middle course between responsibility and irresponsibility, and no means of judging between them except those furnished by the knowledge of right and wrong. Nothing had been more plainly taught by recent advances in psychiatry than that different States make different degrees of responsibility. No law which failed to take cognizance of this fact could be satisfactory to alienists. Before undertaking any legislation in the line of amending the code of criminal procedure, the society should bend its energies toward the passage of a law which emanated from the society and which was in the hands of the committee at Albany. This law, if passed, would do away with many of

the present evils of court procedure. It was suggested that the model plan for procedure in criminal cases would be that the question of lunacy be referred by the court to a committee of three, at least two members of which should be medical men. These men should have been certified as to their moral character and should have passed an examination as to their qualifications to pronounce on the mental conditions. There should be several such experts appointed in every judicial district. The commission thus appointed should examine into the mental state of the prisoner and report to the court. They should determine the degree of responsibility; that is, they should determine the character of the mental disease, if any, from which the defendant was suffering, and report in accordance therewith that he was fully responsible, partially responsible, or irresponsible. Partial responsibility in capital cases should be sufficient to remit the death sentence, but no one who had committed murder and who was acquitted on the ground of partial responsibility should be restored to liberty under ten years. In other felonies the cases of partial responsibility should have the shorter sentence optional with the judge. The report of the committee should be handed to the court and become part of the evidence, and should be the only medical expert evidence.

Dr. William Hirsch, of New York City, was of the opinion that no law, however good, would remedy the present condition of affairs so long as we have not to deal with honest men. If it be possible for corruption or any kind of dishonesty to enter legal procedures, then the best law could not be carried out in an ideal way. On the other hand, laws, no matter how defective, could be so handled that they would answer the purposes of humanity and truth if we have to deal with honest men. This society should direct its attention to its own profession and endeavor to raise the standard of expert testimony to such a point that dishonesty and incapacity would not be brought to bear in criminal procedures.

Dr. William Mabon, of New York City, thought the present law unsatisfactory, but so many questions were involved that it would be difficult to formulate anything that would improve conditions as they exist. He advocated referring the matter to a committee who should investigate conditions not only in this country but abroad. Nothing could be accomplished until the legal profession could be brought into closer touch with the medical profession.

Dr. A. R. Dieffendorf, of Middletown, Conn., asked Dr. Bailey to state, in closing the discussion, just what standards physicians would have for determining the degree of responsibility. He believed that neither the legal profession nor the community would permit a commission of alienists to pass upon that matter without knowing the rules which the physicians applied. He asked further if Dr. Bailey, in looking up the matter, had been able to determine how the criminal procedure in Great Britain had brought about the remarkable condition to which he referred; viz., that there were just as many men found not guilty of first degree murder on account of insanity as were found guilty and hung.

Dr. L. Pierce Clark, of New York City, was heartily in accord with the suggestion made by Dr. Bailey, and favored the appointment of a committee to study the matter very thoroughly. The law in the main was far better than what is in force at the present time, but a proper statute could be worked out only after careful study from a legal as well as from a medical viewpoint.

Dr. William L. Russell, of Poughkeepsie, N. Y., was, on the whole, in

accord with the suggestions presented in the paper, and believed that a committee might, by prolonged study and the presentation of preliminary reports, bring out a discussion of the whole matter that would be very helpful. He referred to the committee appointed two or three years ago by the American Medico-Psychological Association to confer with the American Bar Association, and thought it would be well for the committee under consideration to confer with this and other similar committees. A special procedure might be instituted to determine the advisability of detaining those who have committed murder or homicide and who have been acquitted on the ground of partial responsibility.

Dr. August Hoch, of White Plains, N. Y., said it was not so much a question of making, or urging the making of a different law in regard to the matter of responsibility, as it was of urging that a better basis be furnished for forming a medical opinion. In most cases experts would not differ so much about a given case if all had the same facts, and enough facts. The divergence of opinion arose because, in many instances, the experts were working with an utterly insufficient knowledge of the case, so insufficient, very often, that in every-day practice no conscientious psychiatrist would think of forming an opinion upon it.

Dr. Allan McLane Hamilton, of New York City, thought there should be some *rapprochement* between the medical and legal professions, and to this end suggested that a committee be appointed to confer with the Appellate Division of the Supreme Court with reference to the question in hand. Dr. Bailey had been misinformed concerning English laws. In the question of murder the facts were left to the jury, which enquired into the matter, the question of insanity coming up later. Expert witnesses were not objected to, but the final decision was left to some representative of the crown. He agreed with Dr. Bailey concerning the ten years' confinement of individuals acquitted of murder on the ground of partial responsibility. In disputed cases, however, the time of confinement need not necessarily be ten years.

Dr. Charles L. Dana, of New York City, did not take quite so pessimistic a view of the matter as did Dr. MacDonald. The committee, if appointed, should confer with prominent judges and lawyers, particularly criminal lawyers. The question of partial responsibility was an important one, and if all alienists would accept the idea that there is such a thing as partial responsibility it would have its effect upon the method of procedure. It would be useful for the committee to undertake work along the line of devising a test for insanity which might be accepted by the legal profession.

Dr. Adolf Meyer, of New York City, said that in practically all European countries partial responsibility had been recognized in some form. The question was whether this could be so formulated as to be satisfactory to the community, for after all our laws were not so absolutely bad. There has been heretofore too much expression of mere opinion on the part of alienists without enough attention paid to the determination of the actual facts. The relative looseness in the determination of facts and the too liberal acceptation of hypothetical questions on the part of physicians had led to the unfavorable impressions existing to-day. It was extremely difficult to form an idea as to what should be considered sufficient facts to justify an expert in giving an opinion.

Periscope

Revue Neurologique

(XV Year, No. 5. March 15, 1907.)

1. A Case of Crossed Atrophy of the Cerebellum. ANDRE THOMAS and RENÈ CORNELIUS.
2. New Contributions to the Study of the Bone Reflexes. NOICA.

1. *A Case of Crossed Atrophy of the Cerebellum.*—The patient began having epileptic fits at the age of ten years, and after a particularly severe one at the age of forty-one years, he remained paralyzed on the left side of the body. At autopsy there was found a marked atrophy of the right frontal lobe, right parietal lobe and the second temporal convolution. The Rolandic region and the first temporal convolution were less affected. There was a marked atrophy of the left lobe of the cerebellum which affected more severely the quadrilateral lobe. Microscopic examination was made by serial sections of the brain and cerebellum.

2. *Study of the Bone Reflexes.*—Movements produced by percussing the schapoid bone, the internal malleolus and the posterior extremity of the fifth metatarsal in muscles innervated by the fifth lumbar root. In the upper limbs there are similar reflexes obtainable in hemiplegics and spastic paraplegics.

(XV Year, No. 6. March 31, 1907.)

1. Some Researches on the Transplantation of Nerve Ganglia. MARINESCO.
2. A Sign of Organic Paralysis of the Lower Limb: The Possibility of Elevating the Paralyzed Limb by Itself with the Impossibility of Simultaneously Elevating Both Lower Limbs. GRASSET.
3. Ocular Palsies by Arachnoid-labyrinthine Intoxications. BONNIER.

1. *Transplantation of Nerve Ganglia.*—Marinesco examined histologically nerve ganglia at various periods of time after transplantation, and found that within five hours the nerve cells showed signs of beginning degeneration, which progressed to complete disappearance. A few cells at the periphery of the ganglia may persist and even show regenerative changes, but later they also disappear. The cause of these regressive changes in the ganglia is the shutting off of the nutrition by interference with circulation of the blood and the severing of the connection of the cells with the centrifugal and centripetal fibers. The reason that the peripheral cells persist for a time is because they are more easily nourished by imbibition.

2. *A Sign of Organic Paralysis of the Lower Limb.*—The sign is sufficiently explained in the title of the paper. It is useful in differentiating organic hemiplegia from neuroses.

3. *Ocular Palsies.*—Apropos of a recent article in the *Revue Neurol.*, No. 3, 1907, concerning the production of abducens palsy by sub-arachnoid injections, Bonnier explains the mechanism of their production as fol-

lows: The sub-arachnoid space and the labyrinths are communicating and variations in pressure or chemical composition of the cerebro-spinal fluid affect the papilla of the labyrinth immediately. The most manifest symptom of labyrinthine irritation is oculomotor troubles. The most frequent of the reflex oculomotor troubles produced by irritation of the labyrinth are paralytic or spasmodic phenomena affecting the sixth pair of cranial nerves. In the cases referred to above, the injection of the toxic substance is followed by irritation of the labyrinth and reflex paralysis of the abducens.

C. D. CAMP (Ann Arbor, Mich.).

Psychiatrisch-Neurologische Wochenschrift

(June 16, 1906.)

1. The Question of Cost of Larger or Smaller Pavilions. DELBRÜCK.
2. Paresis in the Canton Luzern in the Period from 1873 to 1900. ELMIGER.

1. *Cost of Pavilions.*—After a somewhat lengthy mathematical and geometrical argument, the writer concludes that institutions should not be built solely on the basis of cost, but on the obtainable results of care from a psychiatric standpoint.

2. *Paresis in Luzern.*—The population of this canton was 134,000 in 1874 and 145,000 in 1900, mostly agricultural. During these twenty-eight years there were admitted to the asylum 91 paretics—74 men and 17 women. Of the men, only 6 were farmers. This is only a percentage of 3.8 as against 25 to 30 per cent. in the larger cities and industrial communities.

(June 23 and 30, 1906.)

1. Defects in the Prussian Statistics of Asylums for the Insane, Epileptics, Idiots, Weakminded and Nervous. GRUNAU.
2. Some Points about so-called "Moral Insanity." NÄCKE.

1. *Defects in Statistics of Prussian Asylums.*—(Continued.)

2. *Moral Insanity.*—The author believes that the cases of so-called "moral insanity" show defects in other spheres than the ethical. The symptoms are those of defect, and therefore the terms moral weakmindedness or idiocy are preferable to the term insanity. We should realize, however, that the condition is one of a disordered or disintegrated "psyche," showing the symptoms of moral defect in the foreground. He classifies the cases under three rubrics: 1. The weakminded; 2, the degenerate (superior degenerates), the largest group; 3, cases with mild, periodical anomalies of mood, the smallest group.

The author takes exception to the use of Lombroso's term "born criminal," and points out that man is a product of his make-up plus his environment, so we might as well speak of "born poets," "born directors," etc. He calls attention to the fact that the moral and the intellect in general develop together, but they often become dissociated, and the important problem of "moral insanity" is to understand the process of this dissociation as well as the "affect-disposition."

As to the treatment neither asylum nor prison is ideal. An asylum attached to a prison, or better yet a separate asylum is best. Castration he decries and calls attention to the possibility of causing thus perverse desires.

(July 7, 1906.)

1. The Application of Artificially Produced Venous Hyperemia of the Brain in the Insane. DR. NEW.
2. The Objects of the Aid Societies for Discharged Lunatics in Baden and the Means of Obtaining Them. DR. ARTHUR BARBO.
3. Defects in the Prussian Statistics of Asylums for the Insane, Epileptics, Idiots, Weakminded and Nervous. DR. GRUNAU.

1. *Artificial Hyperemia of the Brain.*—The method of producing cerebral hyperemia is the method of Bier's by the use of a restricting band about the neck, tightened enough to interfere with the return venous circulation. The author describes in detail the technic, and says the band is tight enough when the face becomes somewhat bluish and swollen. Trying it on himself, he remarks how surprised he was at the slight discomfort in relation to the amount of congestion as indicated by his appearance in the mirror.

Bier reports several cases, mostly epileptic, treated this way. Of 10 cases of epilepsy, 1 was made worse, 6 improved, and 3 remained without change.

The author experimented more with mental diseases and those cases that little was to be hoped from. He reports 23 cases—3 paresis, 3 epilepsy, 8 melancholia, 3 dementia præcox, 5 paranoia, 1 amentia. No case showed any unfavorable results. Improvement both physical and mental occurred in one case of beginning paresis. No noteworthy influence was demonstrated in epilepsy. Slight improvement was noted in all the cases of melancholia. Dementia præcox and paranoia were uninfluenced. The pain in three cases of anemic headache was lessened and one case of anemic vertigo improved. There were no complaints of insomnia.

2. *Societies for Discharged Lunatics in Baden.*—(Continued.)

3. *Defects in Prussian Statistics.*—(Concluded.) Of local interest only.

(July 14, 1906.)

1. Statistical Contribution to the Etiology of Epilepsy. (Continued.) DR. KARL SIEBOLD.
2. The Objects of the Aid Societies for Discharged Lunatics in Baden and the Means of Obtaining Them. DR. ARTHUR BARBO.

1. *Etiology of Epilepsy.*—(Continued.)

2. *Statistics for Discharged Lunatics in Baden.*—(Concluded.) Of local interest only.

(July 21, 1906.)

1. Occupation Psychoses. HELLPACH. (Continued.)
2. Statistical Contribution to the Etiology of Epilepsy. (Continued.)

(July 28, 1906.)

1. Occupation Psychoses.
2. Statistical Contribution to the Etiology of Epilepsy.

1. *Occupation Psychoses.*—(Continued.)

2. *Etiology of Epilepsy.*—The author sums up the results of his statistical inquiry as follows: (1) Of the 913 cases studied 59.2% were male, 40.8% female. (2) 83% were afflicted before the 20th year. In the first decennium the males, in the second the females are more numerous. (3) In a quarter of the cases no cause was ascertainable, in the remainder 55.2% were hereditarily afflicted; these suffer earlier in life than those

not so afflicted. The maternal inheritance is common and more dangerous than the paternal. (4) There was a neuro-psychopathic defect in 34.8%, ⅓ commoner in males than females (21.8% against 13.0%). Consanguinity of the ancestors (grandparents) occurred in 3%, and if we may draw conclusions from so small a number of cases (11) it would seem that blood relationship in the ascendants was more dangerous for the male than the female descendants. In 42% near relatives were affected, females more commonly than males. (5) Abuse of alcohol by the parents was proved in 18.8%, in the male progeny double as common as in the female. Alcoholism in the father as a cause of epilepsy is more important than acquired alcoholic poisoning (18.8% against 2.9%). (6) In 30% of the cases were found acquired causes; of this number 16% were the result of the infectious diseases. 10% followed bodily trauma, more frequent in males. Alcohol occurred as an independent cause in only an inconsiderable number, 2.9%. (7) The cause of the first attack was ascertained in 27.5%.

(August 4, 1906.)

1. Occupation Psychoses. (Concluded.)
2. Statistical Contribution to the Etiology of Idiocy.

1. *Occupation Psychoses.*—The author thinks this an important field of inquiry because of recent trends in psychiatry which make an effort to explain somewhat psychic symptoms by environment. He mentions the moral defect of the "collector," the liability to alcohol and drug addiction of the druggist, the hysterical colored psychic change of the actor, and the "one foot in the dream world" condition of the artist.

2. *Etiology of Idiocy.*—This is a statistical study based on 290 cases. The per cent. having hereditary tendencies shows unusually large, 46.9% for males and 55.5% for females. The percentages of bodily diseases acting as causes are also high, 12.6% for males and 21.6% for females, with an additional 8.4% and 21.6% respectively doubtful. Practically one half of the cases in early life had suffered from rhachitis.

WHITE.

Journal de Psychologie Normale et Pathologique

(Fourth Year, No. 2. March–April, 1907.)

There are no original articles in this number of the *Journal,* the space being occupied by the report of the January 11, 1907, session of the Société de Psychologie. In this report appears the history of an interesting case of Mnesic Negativism (Fixed Idea or Obsession of Forgetfulness) by Seglas. The adroitness of the examiner in eliciting the fact that the patient's forgetfulness is not due to a true amnesia, but to the obsessed idea that he cannot remember, is the particularly interesting point in the report. There are also two communications from Marie, one upon Some Cerebral Localizations in Aphasia, with report of cases, and another upon Eunuchism and Sexual Psychology.

(Fourth Year, No. 3. May–June, 1907.)

1. Certain Properties of the Mental Act. TASSY.
2. The Solar Plexus and Its Functions. (To be continued.) LAIGNEL-LAVASTINE.

1. *Certain Properties of the Mental Act.*—This is a study in normal psychology, wherein the author attempts to substitute for the ordinary mode of psychological analysis that of physics and physiology. He first discusses the *tendency of thought to realize itself,* thus suggesting the similarity of the psychological processes with those of the simple sensory-motor phenomena. The idea and its expression, he says, is a single phenomenon. If the expression does not realize itself or actually take place, it is because the idea is not sufficiently intense to overcome the inhibition exercised upon it by other ideas.

Under a second head he discusses Thought as a Form of Dynamism. It is an error to believe that thought is founded upon a special force so long as the fact is recognized that it rests upon forms of sensibility that differ in no way from all the ordinary forms of sensibility. The difference is only a functional one. The cerebral hemispheres are the collectors of all the activities of the organism and are the regulators and distributors of the force that is common to them all.

In a final section he explains and analyzes what he calls *mental sensation.* This is not the same as an ordinary sensation, but is an infinitely attenuated sensation, scarcely perceptible in the sphere of consciousness.

The conclusion of the whole matter is that this method of examining a mental and sensitive phenomenon permits one to attack the problem of the nature of a psychic act much farther back than is usually done—indeed, back to the point where it cannot be further reduced to any other known form of terrestrial activity. Showing in this way how a man is identified in and with nature, we are better prepared to find out wherein he is to be contradistinguished from it.

(Fourth Year, No. 4. July–August, 1907.)

1. The Affective Characters of Perception. WAYNBAUM.
2. The Solar Plexus and its Functions. (Concluded.) LAIGUEL-LAVASTINE.

1. *The Affective Characters of Perception.*—This is a study in normal psychology. The author analyzes the nature of the emotive perception as the perception that is always accompanied by more or less feeling. A large part of our perceptions are purely intellectual, while others, less numerous perhaps, are wholly affective. This perceptive affectivity, in its turn, can have an unintellectual or mere reflective origin. The affective perception occupies as important a place in the psychic life as does the intellectual. He subdivides the affective perception into two distinct species, each depending upon its respective composition or substance and physiological pathway for the transmission of the affective element. With the aid of a diagram the author indicates the manner of the transmission of these two forms of emotive perception.

2. *The Solar Plexus and its Functions.*—(Concluded.)—The author insists that the study of the anatomy, physiology and pathology of the solar plexus ought to be given at the present day as much dignity as the study of the brain and the spinal cord. A knowledge of it is of the highest value to the physician, alienist and psychologist. The article is too long and too full of detail for satisfactory abstraction.

METTLER (Chicago).

Revue de Psychiatrie et de Psychologie Experimentale

(August, 1906.)

1. Senile General Paresis. OLIVIER.
2. Obsessing Hallucinations. GIMBAL.

1. *Senile General Paresis.*—By the term senile general paresis the author means the sub-acute diffuse meningo-encephalitis of the aged. The domain of paresis has been very much enriched during recent years. Pseudo-paresis in particular has caused a considerable extension, while we have also the paresis of infancy and adolescence classed with the same inflammatory process occurring in the adult. The author will consider those cases of so-called general paresis (tardiva), which occur between the ages of 50 and 60 years. Some authors do not admit of the possibility that these cases are true paresis. The writer, however, proposes to justify the existence of the disease. He limits his observations to cases where the first manifestations have been in the neighborhood of the sixtieth year. The presence of general paresis in the senile rests upon five categories of proof which are as follows: (1) The affirmation pure and simple of certain authors; (2) statistics; (3) clinical observations without autopsy; (4) clinical observations with macroscopical observations; (5) clinical observations with both macroscopical and microscopical examination. The author considers the proof under each one of these categories and gives detailed accounts of a considerable number of cases. As a result of these studies he concludes that senile general paresis supervenes from the fifty-ninth to the sixty-fourth year, very few cases occurring after this epoch. As regards further etiological factors he has very little to say.

The symptomatology is very similar to that of paresis as usually observed. Quite rarely there is a congestive attack which opens the scene immediately by motor disturbances. In some cases there is a persistent headache. The simple demented form occurs nearly as frequently as the delirious forms. In none of the cases observed were there true arrests or remissions. The disease evolves progressively towards death, the mean duration being two years and five months. The principal difficulty in diagnosis is to differentiate this condition from atheromatous dementia. The author does not say much about the distinction between these two conditions, but says it has been conceded by Klippel that this condition of the vessels favors the development of a diffuse meningo-encephalitis. The author thinks this is an ingenious explanation but by no means has it been demonstrated.

2. *Obsessing Hallucinations.*—The author defines obsessing hallucinations as hallucinations accompanying the characteristic phenomena of obsession, that is to say, anguish, irresistibility, the physical signs of pathological emotivity, etc. In discussing these conditions he describes numerous cases and concludes as follows: (1) Nearly always obsessions provoke only mental representations, more or less vivid—pseudo-hallucinations. (2) Sometimes these representations in certain subjects are replaced by true hallucinations, variable as to form, single or multiple. (3) Some of these hallucinations are conscious, others unconscious, these last form the origin of deliria, of episodes more or less important, in the midst of deliria, the obsession participating in the constitution of delirium and united to it by common elements, by a hallucination having the same subject as the idea. (4) The characteristics of the obsessing hallucinations are those of the obsession, that is to say, irresistibility, anguish,

emotional phenomena, diminished consciousness, etc. These characteristics are very variable in intensity, in general the intensity of the obsessing hallucinations and lucid consciousness are related to one another in inverse proportion. (5) Obsessing hallucinations are dissociable into two elements of unequal importance, one primitive, antecedent is the obsession, the other secondary, and consequent is the hallucination.

(September, 1906.)

1. The Care of Moral Defectives. VIGIROUX.
2. New Researches of the Scotch School relative to General Paresis. MARIE.

Moral Defectives.—This article is a description of the legal status of defective children and their methods of care by reformatory and educational means as practiced in France. It is of little interest to American readers.

General Paresis.—This article is a review of the work of the Scotch school of the pathogenesis and treatment of paresis. It mentions Bruce's work on the febrile movement and leucocytosis, Ainslee's on the phenomena of endarteritis, Douglas MacRay, and Robertson on bacteriological investigations. The work of the last investigator in identifying a diphtheroid bacillus of the Klebs-Loeffler type is especially mentioned. This discovery of a bacillus responsible for the disease of course indicates a method of treatment.

(October, 1906.)

1. Psychotherapy, Total or Superior. GRASSET.

Psychotherapy: Definitions. General Considerations.—In addressing only the inferior psychism, the inferior psychotherapy—therapeutics by hypnotists—one can only attack a disaggregated polygon; the O is not at all addressed, the separation of the psychisms is rather accentuated than cured. The superior psychotherapy on the contrary, far from separating the psychisms in order to modify one of them, addresses itself to the *ensemble* of psychisms and fortifies their union and their collaboration, increasing the force and influence of the O on the entire life of the subject.

[Above statement is based upon Grasset's theory of inferior and superior psychisms and the O refers to his designation of the highest point in psychic life as illustrated by him by polygons representing the two psychisms. This article can hardly be understood unless Grasset's theory which has been described in this Journal before is understood.]

Limits of Action and Contra-indications of Superior Psychotherapy.—If the central O is profoundly affected and the patients believe absolutely in their delirium and admit the reality of their hallucinations, psychotherapy will accomplish nothing. If, however the hallucinations are not real and complete, if the central O is only too feeble to control the situation some result is to be hoped for. Thus superior psychotherapy is not the method of treatment for the true and complete psychoses. On the contrary it is the treatment of choice for what Dubois calls the psychoneuroses characterized by mental debility, facility of sub-polygonal disaggregation, the mental instability of Duprat, the easy abdication of the O and the frequent predominance of the polygon. There are even cases where superior psychotherapy not only becomes inefficacious but dangerous. For example, the bad reasoning of members of the family or people in general against certain classes of "cranks" may produce harm.

Method of Procedure.—Under this heading the author calls attention to the fact that the action of psychotherapy on motor and psychic phenomena can be readily understood by referring to his diagram and noting the controlling relation of the O. As to the action of this method on the idea he refers to Dubois who has set forth the particulars fully. We cannot proceed with these cases, however, as in hypnosis, by order, by injunction, the action must be indirect. The principle of this indirect psychotherapy is two-fold. In the first place we may create an idea, a sensation, an emotion, an involuntary habitual psychic state, in making the subject do an act conforming to this idea, sensation, or emotion. In the second instance we may fix, develop, or accentuate the idea by voluntarily making all the acts conform to this psychic state. In doing this it is necessary, *first,* To distract the patient from the pathological idea, sensation, or emotion, and never address his attention to it; *second,* It is necessary to distract the patient from the necessity of conforming his life in relation to these morbid ideas; *third,* The superior psychotherapy is absolutely based on the personality of the subject, on his intelligent cooperation, and in order to obtain results it is necessary to point out to the patient the importance of the end to be attained; *fourth,* When applying these divers psychotherapeutic processes it is necessary to supervise in every particular the *milieu* in which the patient lives.

WHITE.

The Journal of Mental Science

(Vol. LII., No. 218. July, 1906.)

1. Amentia and Dementia: a Clinico-Pathological Study. JOSEPH SHAW BOLTON, M.D.
2. On Instinct: A Psycho-physical Study in Evolution and Dissolution. W. H. B. STODDART.
3. The Clinical Significance of Indoxyl in the Urine. LEWIS C. BRUCE.
4. Industry and Alcoholism. W. C. SULLIVAN.
5. A Serum Reaction occurring in Persons suffering from Infective Conditions. LEWIS C. BRUCE.
6. The Clinical Measurement of Fatigue. WILHELM SPECHT.

1. *Amentia and Dementia.*—Continued article.

2. *On Instinct.*—The author attempts to show that in mental diseases in general and in general paralysis of the insane in particular, the symptoms shown can be correlated with a dissolution of volition, followed by a dissolution of instinct. From his own observations and from various other sources he has compiled in chronological order the appearance of the various instincts in infants and children. He maintains that there are two motor nervous systems. First, the cortico-rubra-spinal, representative of the pristine instinctive motor system. Second, the pyramidal tract which is the volitional motor system. Volition he assumes is developed later than instinct, and hence in mental disease it is the first to be affected by dissolution, and then follows the dissolution of instinct in the reverse order of their appearance. He cites general paralysis as an example of his theory, and maintains that it is applicable at least to most, and probably to all cases of mental disease. Here the author falls into the common mistake of attempting to explain all mental disease by the same rules that might apply to general paralysis or senility. He further expresses the hope that this view of mental diseases may prove a guide to the prognosis in indi-

vidual cases, by simply noting in which stage of dissolution the patient may be at a certain time.

3. *Indoxyl in the Urine.*—The author presents his conclusions based upon the examination of twenty-seven patients, thirteen of whom "presented the mental symptoms of depression." In eight, or 61.5 per cent., the urine showed an excess of indoxyl, while in fourteen cases "not depressed," only two, or 21.4 per cent., showed this excess of indoxyl. From the rather inadequate data he concludes that there is some relation between an excess of indoxyl in the urine and mental depression, and that the excess of indoxyl is a cause of the depression rather than the converse. In one case the result of the treatment suggested that indoxyl was the chief cause of the disease. The statement that the presence of an excess of indoxyl in the urine means a loaded alimentary tract, comes nearer to being a true explanation than any hypotheses regarding the relation of indoxyl to depression, unless more systematic examinations were made.

4. *Industry and Alcoholism.*—Here are presented quite adequately and concisely the effects of two opposed types of drinking, the convivial and industrial, based upon carefully collected statistics from various regions, where the occupation and habits of the people differ greatly. The industrial drinkers include men who do hard laboring work and drink according to the traditions of a certain trade. The dockers are given as a striking example of this group, often drinking six pints of ale a day, or more than four ounces of alcohol. The opposed type, the convivial drinkers, are mostly skilled workmen, who drink much less because they are either prohibited from drinking during business hours, or have seen that such indulgence is detrimental to them. They may occasionally become intoxicated, but statistics show that in regard to mortality and crime there is no comparison in this class to the industrial drinkers. The following propositions give his ideas in regard to the use of alcohol by these classes.

1. That in a large majority of individuals alcohol in moderate doses acts as a real psychomotor stimulant increasing considerably the output of muscular work.

2. That this stimulant action lasts only for a short period and is ordinarily followed by a phase of depressed activity, which more than compensates the initial stimulation.

3. That on sensory functions the action of alcohol is regularly a depressant causing a diminished acuteness of sensations, and a diminished quickness of perception.

An interesting table is appended which shows graphically the relation of convivial and industrial drinking to crime and morality.

5. *Serum Reaction in Infective Conditions.*—Observation upon the opsonic indices of the insane accidentally led the author to observe that red blood corpuscles of healthy persons were agglutinated by the serum of the insane. Further tests showed that the following psychoses gave this reaction, i. e.: Mania with or without confusion. Katatonia, hebephrenia, the depression of "*manic-depressive*" insanity, and epilepsy with excitement. These he considers due to bacterial infection, and hence infective psychoses. Other psychoses "due to causes other than bacterial infection such as melancholia of metabolic origin, systematized delusional insanity, and insanity the result of exhaustion," did not give the reaction. Experimental work on rabbits, by infecting them with various organisms, would tend to prove that the agglutinative reaction was due to infection. From

this the author divides insanity into two great classes—infective and non-infective. From what has been stated it is readily seen what psychoses belong in these two groups. It is difficult to see how this reaction alone can be made the basis for such an important classification, as thus far those engaged in the study of immunity and allied problems have found the agglutination of the corpuscles by certain sera, while an interesting phenomenon was of little importance as a diagnostic factor.

6. *The Clinical Measurement of Fatigue.*—Continued article.

H. A. COTTON.

Allgemeine Zeitschrift für Psychiatrie

(Band LXIII. Heft 3 and 4. 1906.)

1. The Influence of Psychical Processes on Metabolism. ROSENFELD.
2. Manic-Depressive Insanity and Arteriosclerosis. ALBRECHT.
3. Intermittent Insanity. GREGOR.
4. Case of General Paresis after Shock from High Tension Current. ADAM.
5. Combined Psychoses. GEIST.
6. The Danger to the Descendants of Psychoses, Neuroses, etc., in the Ancestors. TIGGE.

1. *Influence of Psychical Processes on Metabolism.*—The author after a review of the various experiments on this subject, the results of which unfortunately are little conclusive, relates his own investigations which he carried on during a period of about two years. As subjects he chose acute severe cases showing the clinical picture of katatonia, and presenting decided anomalies in the taking of nourishment. These cases he considered were specially suited for this investigation, as sudden changes of weight are common among them, and on account of frequent refusal of food forced feeding is often necessary and gives the opportunity of careful quantitative determination of the food ingested. He was careful also to choose only those who showed no tendency to excessive muscular movement, were clean and presented no bodily ailment, especially no disease of the digestive tract. The patients who answered these requirements and who had wholly or partly abstained from food for some time, were fed several times daily, with the sound, quantitatively determined food, and the resorptive power of the intestinal tract observed. By this method he hoped to gain information on the following points. Is it possible in these patients who so often show sudden loss of weight to favorably influence metabolism by the artificial introduction of abundant food, and if so to what extent? What factors hinder taking on weight? What is the effect on the psychical disturbance of long-continued over-nutrition? What is the sustenance ration of such patients? To what extent is retention or excretion of water responsible for the alterations of weight so commonly observed? Is the metabolism of these patients to be regarded as retarded? Can it be determined that there is change in composition of the urine in patients nourished in this manner? Does nitrogen equilibrium occur in the same manner as in normal cases? Is there toxic albumin decomposition in these people? Is the relation between body weight and urine in sudden withdrawal of food the same as in a normal man who after a period of full nutrition is suddenly deprived of food? In the four cases in which his experiments were carried out he has noted in tabular form, weight of the patient, food in calories, and

its nitrogen content, specific gravity, quantity and percentage of nitrogen in the urine, quantity of feces, their nitrogen and fat content. The urine was also examined daily for albumin, sugar and indican. There was practically always indicanuria in the cases artificially fed. Other abnormal constituents were absent except for an occasional slight alimentary glycosuria. While weight can be influenced favorably in some cases, that it is often steadily lost in spite of the ingestion of abundant aliment, was shown by his fourth case, which under an abundant artificial introduction of food gained in nine days 3.7 kg. From this time he steadily lost weight in spite of the continued and abundant food ingestion, having lost at the time of his death, two months later, 10.2 kg. An autopsy showed entirely normal organs and only a small amount of feces in his intestines. The inference that psychical factors have an influence on metabolism seems justified in this case. Long-continued tube feeding seemed to have at any rate no unfavorable influence on the psychical condition, though some patients deteriorate mentally in spite of gain in weight. He makes no positive statement as to the sustenance ration, although he says that he introduced daily food of the values of from 54 to 82 calories. That water content is responsible for sudden alterations of weight he thinks is shown beyond question. He succeeded in one instance in raising the weight of a fasting patient to the extent of 5.5 kg. in 9 days by the ingestion daily of 2000 c.c. of saline solution. At the end of this time slight edema was produced. In the next four days the weight fell 4.5 kg., although the water was introduced as before. He finds no evidence of any difference in the process of metabolism, in these cases, from that which occurs in normal people. With regard to nitrogen retention these patients acted like any normal persons who after long inanition periods are suddenly abundantly supplied with nourishment. Toxic decomposition of albuminoids was found in no instance. The article is carefully prepared and a long list of references is appended.

2. *Manic-Depressive Insanity and Arteriosclerosis.*—The author gives the outlines of the histories of 54 patients (19 males, 35 females) presenting the clinical picture of manic-depressive insanity, in whom he carefully examined the condition of the arteries, and took the blood pressures by the Gärtner tonometer. Their ages varied from 25 to 81 years. Out of this number 9 men and 9 women, or about one third of all the cases, showed distinct arterial disease. Examining a number of patients presenting other psychoses, those having histories of exogenic influences calculated to produce arterial disease being excluded, only 10% showed arterial sclerosis. He concludes that the relation between manic-depressive insanity and arteriosclerosis is not an accidental one, but that they are on the one hand due, to some extent at least, to a common cause, a congenital deficiency of the vascular system; senility tending in such cases to come on early and its onset giving an exciting cause for mental disturbance in a brain already predisposed. On the other hand, the sudden and violent changes in the affective state in manic-depressive insanity cannot but react unfavorably upon the tonus of the arteries, acting hence as an exciting cause of arteriosclerosis.

3. *Intermittent Insanity* (Gregor).—The author reports on the case of an officer thirty years old, who presented the clinical picture of katatonia with distinct intermissions, at times from day to day. He compares this case with the not very numerous instances of such an intermittent course which have been reported, and discusses the clinical position of cases of this group.

4. *Case of General Paresis after a Shock from a High Tension Current* (Adam).—The author reports the case of an electrical worker who one year after the passage through his body of an alternating current of 10,000 volts tension developed symptoms of general paresis and died a year later under the typical picture of this disease. An autopsy fully confirmed the diagnosis, and in the absence of any other ascertainable etiological factor, the author considers that the shock probably acted as an exciting cause. The vascular dilating and thermic effect of the strong current he thinks may have started up the pathological process in the brain.

5. *Combined Psychoses.*—The author, after calling attention to the fact that in the present state of our ideas with regard to forms of mental disease, it is not safe to predicate the presence of two dissimilar forms merely on account of the presence of symptom complexes or symptoms common to both, gives the clinical history of a man of 30 years of age, who after a railroad accident developed a condition presenting all the characteristics of a severe traumatic neurosis. This lasted for about six years, completely incapacitating the patient. At the end of this time the picture gradually changed to one of general paresis. The legs which had been apparently completely paralyzed were able to be used again, but reflexes were exaggerated, and anesthesia which had been present in the legs had disappeared. There had developed, however, tremor of the tongue, disturbed pupillary reaction and delusions of grandeur were present. The disease progressed in the usual manner and upon his death about two years later the brain showed the characteristic changes of general paresis. Discussing this case the author thinks that if the initial disturbance had been due to the outbreak of general paresis, it would have been very unlikely that the patient should continue for six years stationary in a condition entirely typical of a traumatic neurosis, and that at the end of this time the power should be regained in the paralyzed limbs, and the sensory disturbances should improve with comparative suddenness. Also the course of the disease over eight years without the production of extreme dementia and marasmus is hardly typical of general paresis. Viewing the case in its entirety he considers that it must be regarded as an instance of the supervention of general paresis upon an existing traumatic neurosis.

6. *The Danger to the Descendants of Psychoses, Neuroses, etc., in the Ancestors.*—A statistical study unsuitable for abstraction.

7. *The Risk, with Regard to the Outbreak and Transmission of Nervous and Mental Diseases, of Marriage.*—A general review of the subject of the influence of marriage upon the probability of the outbreak of mental or nervous disease in one or both of the contracting parties, and the liability of its transmission to the offspring, à propos a case in which the author was called upon to advise, as to the risk in contracting marriage with a young woman certain members of whose family were insane. Studying the histories of both parties to the proposed contract and of their families, he endeavored to put into percentage figures for his client, first, the danger to the woman herself of the development of a psychosis, and second, the liability of transmission of hereditary defects to the children. He gives many references on the influence of heredity.

(Band LXIII. Heft 5. 1906.)

1. The Role of Endogenesis in the Etiology of General Paresis. DREYFUS.
2. Psychoses after Eye Operations. LAPINSKY.

3. Experiments with Alcohol.
4. Cases of Family Microcephaly. Vogt.
5. The Intoxication Psychoses. Schroder.
6. Injuries at Birth and Epilepsy. Volland.

1. *Endogenesis in General Paresis.*—The author thinks that the organic character of general paresis by no means excludes the possibility of its endogenetic origin. To prove this he gives a statistical study of 268 cases of general paresis, and a number of case histories bearing on the point in question. Among the 268 cases of paresis he found certain heredity of nervous or mental disease in 31%, a figure not so inferior to that which he determined for a large number of other forms of mental disease, 38%. His case histories show that a large number of paretics presented various more or less marked mental abnormalities long prior to the beginning of the general paresis, often from childhood, and that in many cases absolutely no indication of former syphilis could be found. He thinks that the following conclusions are justified. From the fact that a person is a paretic we have no right to conclude that he has had syphilis. Neither are we justified in the hope that by abolishing syphilis, general paresis will be done away with. It is also not correct to postulate that other things being equal, the presence of strong hereditary predisposition to nervous or mental disease, would in doubtful cases weigh against the diagnosis of general paresis, or to think that the descendants of a paretic are endangered only so far as hereditary syphilis is concerned.

2. *Psychoses after Eye Operations.*—The author describes the case of a sixty-year-old man afflicted with arteriosclerosis and with severe digestive disturbance, who being kept in a dark room as part of the treatment for a corneal ulcer, developed an acute hallucinatory condition. In connection with this case the author reviews the subject of psychoses after eye operations.

3. *Experiments with Alcohol.*—Having had to examine a certain number of persons who were under observation as to their mental condition on account of illegal acts which in most cases were attributed to a pathological effect of alcohol, the author adopted the plan of giving to these people a certain amount of alcohol and careful watching its effect on the mental condition and general state of the patient. He gives the histories of seven cases in which this was carried out, stating the conclusions which he felt able to draw from the reaction of each individual to the drug. As to the propriety of this procedure he concedes that it may be open to question both on moral grounds, and on account of possible injury to the patient directly. It should not be resorted to, he thinks, without the consent of the subject of the experiment, but as in most of the cases where it is indicated, the patient has been more or less addicted to alcohol, and his liberty or even his life may depend upon the expert opinion, the disadvantages are outweighed by the advantage of securing by direct personal observation an idea of the reaction to alcohol of the person under examination. He warns, however, against laying too much stress on the result to the exclusion of all the other factors in the case.

4. *Cases of Family Microcephaly.*—The author considers the factors in the production of this condition, both endogenic and exogenic, and calls attention to the fact that its occurrence in several members of a family is not so rare. He quotes the reports of cases from the literature, and adds a short account of eight cases from three families coming under observation at Langenhagen.

5. *The Intoxication Psychoses.*—Leaving out of consideration the psychoses of alcoholism which have been pretty well worked up, also those occurring in connection with morphine, cocaine and other narcotics, which are most frequently complicated with other etiological factors, the author limits himself to the consideration of the mental disturbances which follow the action of poisons introduced unwittingly into the system, such as ergot, lead, carbonic oxide, or used for long periods by the physician as iodoform, salicylic acid, etc. Considering the symptoms of the psychoses developing in connection with these poisons, one is struck by their general similarity, confusional, stuporous, or delirious states, broken at times by episodal attacks of excitement or convulsive phenomena varying somewhat with the particular agent it is true. The author urges that sharp distinction should be made between the immediate, and the more remote effects of the poison. We are acquainted with a large number of substances which given once in a large enough dose are capable of producing mental disturbances not differing in kind from those observed in true insanities, but it is very doubtful if one dose of such a substance sets up a real psychosis. For this effect a chronic poisoning is generally necessary. In the course of such chronic poisoning we see: 1. That psychical disturbances occur after substances which are not known to produce any effect upon the brain in one, even large, dose. 2. That in the case of poisons which in one dose can influence the psychical functions, chronic intoxication produces entirely different mental disturbances from those seen after one dose. 3. The mental disturbances resulting from these chronic poisonings bear a strong resemblance to one another. These facts seem to the author to indicate that the disturbances observed are not so much due to the direct action of the poison, but that some intermediary is also in action, i. e., that the poison sets up some bodily changes which are at the basis of the disturbed brain action.

These bodily changes once produced are more or less persistent, and from the general resemblance of the symptoms of the different intoxication psychoses they would seem to be similar. There is considerable evidence going to show that the mid link in the etiological chain is the vascular system. That there are other factors is highly probable, but of them we have little positive knowledge. Since our classification in this instance rests entirely upon an etiological basis, it is well to be somewhat cautious as to what we include under the head of intoxication psychoses, and before placing any given case in this category, we should carefully consider all other possible factors.

6. *Injuries at Birth and Epilepsy.*—The role of disturbances in the normal mechanism of delivery, in the causation of epilepsy in the child, has had a different degree of importance assigned to it by different authors. As a contribution to the subject, the author looking over a material of 1,500 cases in the epileptic asylum of Bethel at Bielefeld, found 45 individuals in whom difficult labor was given as an etiological factor.

These cases he examined with regard to family history, course of delivery, development, time of onset of the epileptic attacks, mental and physical condition and character of the attacks. His results he exposes in a table. Among the 45 cases there were 7 patients who had no history of heredity of nervous or mental disease, but in whom gross brain disease appeared to be the cause of the attacks. For these he has constructed a table showing the particulars with regard to delivery, time of onset of the paralysis, and of the epileptic attacks and other special features. He last analyzes the histories of labors in 19 families in which one or more

of the children were epileptic or defective apparently from difficult birth, comparing the number of epileptic or defective children with that of the normal ones. He feels justified in drawing the following conclusions: Compared to the great frequency of epilepsy, in the etiology of this disease difficulties at birth play only a subordinate role. In a small number of cases, however, difficult delivery seems to be a predisposing cause of later epilepsy.

C. L. ALLEN (Los Angeles).

Monatsschrift für Psychiatrie und Neurologie

(Band XIX. January–June, 1906.)

1. Partial Pure Word Deafness. HENNEBERG.
2. A Case of Myasthenic Paralysis. BOLDT.
3. The Symptom-Complex of a Circumscribed Autopsychosis on Basis of a Dominant Idea. PFEIFFER.
4. The Disseminated Forms of Brain Syphilis in Combination with General Paralysis. STRÄUSSLER.
5. Contribution to the Knowledge of the Delusion of Jealousy. TÖBBEN.
6. Contribution to the Study of the Puerperal Psychoses. MÜNZER.
7. The Symptom-Complex of Primary Incoherence with Excitement. FELS.
8. Chronic Subcortical Encephalitis. BEHR.
9. Studies on the Brain Tissue of the Insane with Weigert's Neuroglia Method. KOLLER.
10. Diagnosis and Prognosis of Amentia. STROHMAYER.

1. *Partial Pure Word Deafness.*—Pure subcortical sensory aphasia is an extremely rare affection. A servant girl twenty-nine years old has slight facial paralysis, normal hearing and well-preserved intelligence. The understanding of simple words (especially names of objects) tolerably well retained, but complicated words or phrases cannot be understood. Very simple sentences occasionally comprehended. The ability to repeat words and to write to dictation is disturbed in consequence of the difficulty in understanding. Appreciation of melody and music lost. All of the other functions intact, except for a few paraphasic utterances in the spontaneous speech. Reading and spontaneous writing undisturbed. The literature contains only six cases of uncomplicated word deafness. The author feels that the whole question is unsettled and emphasizes the lack of anatomical material and our deficient knowledge of the course of the auditory tracts. The relation existing between ordinary hearing and the perception of words is not understood. In the case recorded the lesion is thought to be most probably a subcortical focus in the left temporal lobe. The possible presence of an intellectual disorder in Marie's sense was apparently not considered. It is difficult to see how the lesion indicated would explain the facial parèsis.

2. *Myasthenic Paralysis.*—In a case presenting a typical clinical picture, the autopsy revealed no lesion in the central nervous system, but in all of the skeletal muscles there were found between the muscle fibers infiltrating foci of lymphoid cells. Similar findings by Weigert were attributed to metastasis from a primary lymphosarcoma. The author thinks the disease cannot be of nervous origin, but considers it to be a peculiar condition of exhaustion resulting from the cell infiltration between

the muscle fibers. An autotoxic cause is the most probable, although the nature of the poison is unknown.

3. *Circumscribed Autopsychoses.*—Two cases are described in which a limited paranoic system developed on ground of a dominant idea connected in each case with a painful emotional experience. Explanatory delusions, ideas of reference and retrospective falsifications were all related to the one complex of ideas. In other respects the patients showed no defects of judgment; intelligence intact, demeanor unaltered. Of the two current views regarding the origin of paranoic states, viz., that the delusion is the expression of a mental weakness attending a general disorder of the brain or that it is a symptom of a partial disturbance in an otherwise normally acting consciousness, the author thinks his cases support the latter conception. The real cause of the dominant idea is found in the abnormally strong accompanying affect. Further elaboration and systematization is not a sign of progress of the disease, but it is rather a normal reaction from the patient's altered point of view. Recovery without insight has been reported in similar cases.

4. *Miliary Disseminated Form of Brain Syphilis with G. P.*—Two cases are reported which showed, in addition to the alteration of general paralysis, miliary gummata situated in the deeper layers of the cortex. The meninges of the cerebrum remained free from any syphilitic process. The miliary gummatous form is the most infrequent variety of brain syphilis and it usually occurs as a gummatous formation in the meninges accompanying a diffuse syphilitic infiltrative process or a widespread plastic exudate. The author's cases demonstrate the occurrence of a gummatous form of brain syphilis spread through the cortex and *independent* of meningeal involvement. Brain syphilis, in the form of isolated gummata or luetic vessel disease, has often been reported in connection with G. P. and makes little difficulty in the histopathological diagnosis. The diagnosis between general paralysis and brain syphilis has referred mainly to syphilitic meningo-encephalitis; in the author's cases the differential points given (Alzheimer) would naturally not suffice. Clinically the cases were fairly typical of paresis and no symptoms were present to call for any other diagnosis. The author is able to confirm Alzheimer's observation that in syphilitic vessel disease the infiltrate consists mainly of lymphoid cells, while in the G. P. process the infiltrate is made up almost entirely of plasma cells.

5. *The Delusion of Jealousy.*—The older view that the delusion of jealousy was a clinically well-defined form of disease originating almost exclusively on ground of chronic alcoholism is clearly controverted by clinical experience. The author reports four cases, three of whom took no alcohol. In seeking a psychological explanation of the origin of the jealous idea one should proceed by analyzing in each case the situation under which the delusion developed. In one of the cases reported the ideas of infidelity arose after the patient was stricken blind; in another case the patient, suffering from a stomach ailment, thought his wife was trying to poison him to get him out of the way, and on this basis the delusion of jealousy was formed. Alcohol is not a direct cause, but through its many harmful influences it predisposes to the development of jealous ideas by furnishing conditions favorable for the growth of the morbid suspicions.

6. *Puerperal Psychoses.*—This study is based on an analysis of 101 cases admitted to the Heidelberg Clinic, and includes all of the psychoses occurring during pregnancy, the puerperium proper and the lactation

period. According to the form of the psychosis the cases were distrib uted as follows: Dementia præcox, 53; manic-depressive insanity, 26; amentia, 6; general paralysis, 6; hysteria, 1; uncertain, 9. Features usually mentioned as prominent in the puerperal psychoses, such as erotic traits, aversion toward husband and children, etc., were not conspicuous. From a symptomatological view-point the cases in the various groups differed in no particular way from cases occurring apart from the reproductive process. The dementia præcox cases belonged largely to the katatonic form. The author concludes that we do not know a puerperal psychosis with specific features. Although all of the usual psychoses may arise in relation to child-bearing, this fact does not exclude the possibility of the occurrence of a specific puerperal psychosis being identified upon closer clinical observation. Attention is called to the predominance of depressed forms in the gravid period.

7. *Primary Incoherence with Excitement.*—Three cases are reported to establish a symptom-complex not hitherto described, viz., primary incoherence with excitement. The onset was fairly acute, all of the patients being in middle life. With good orientation there was exhibited a marked emotional lability. No fixed delusions. Some "katatonic" features appeared transitorily. These cases are believed not to belong with the excitements of manic-depressive insanity, dementia præcox, amentia, etc. The usual factors leading to incoherence, viz., hallucinations, memory defect and flight of ideas—were absent, hence the disorder in thought is called "primary." After over two years' duration no deterioration has resulted. Although the author believes that the incoherence was not of the nature of a flight and that the affect was not that of mania, the case-histories make these claims doubtful and the psychoses seem to tally in the main with Kraepelin's description of manic-depressive insanity. Two of the patients had passed through previous attacks and recovered.

8. *Chronic Subcortical Encephalitis.*—This disorder was first described by Binswanger, who uses the term encephalitis in the broad sense of brain disease. Clinically these cases may be very difficult to differentiate from general paralysis. The case reported had unequal and stiff pupils, diminished knee jerks, speech disorder, convulsions without localizing symptoms. In addition there was mental enfeeblement with euphoria. Duration sixteen years. The anatomical findings consisted of extensive destruction in the right hemisphere of the medullary substance and a great overgrowth of neuroglia in this region. The convolutions over the affected area showed some atrophy, but the structure of the cortex was little disturbed and none of the changes characteristic of general paralysis was present. In regard to pathogenesis the lesion is the result of an arterial affection, narrowing of the lumen and cutting off of nutrition, associated with a hyaline alteration in the vessel wall. According to Binswanger the clinical picture differs from G. P. in having a longer duration, a stationary stage may last for years, and the defect symptoms tend to remain circumscribed or partial in the various fields.

9. *Neuroglia Studies.*—The author, using Weigert's method, examined the cortex in a variety of psychoses. In periodic insanity, involution melancholia, chorea minor, brain-tumor and meningitis no alteration in the glia was found. In dementia præcox one finds a mild diffuse increase of the glia elements through the entire cortex. The changes found in epilepsy are apt to be localized. In senile dementia there is an increase in the marginal layer (Filz) in which amyloid bodies are found; the fibers are coarse and occasionally take a corkscrew form. In general

paralysis the marginal layer is thickened; fibers are increased and often zig-zag in form; the vessels are accompanied by nuclei and a glia network. Fibers can be followed deep into the cortex. Especially striking is the overgrowth of glia and increase in the vessels in the transition zone between the cortex and the medullary substance.

10. *Amentia.*—This is an excellent review of the amentia question and a valuable casuistic contribution. Under a variety of names there exists in psychiatric literature a well-recognized symptom-complex, the fundamental features of which is a state of mental confusion combined with a disorder in the association of ideas—a primary incoherence in thought. The original and decidedly heterogeneous group designated by Meynert as amentia has been gradually curtailed, and finally Kraepelin restricts the term amentia to a small group of psychoses arising in connection with some *external palpable cause,* the clinical picture being that of a dream-like hallucinatory confusion with excitement. The author uses the term amentia in a somewhat broader way and reviews the 110 cases of acute confusion received into the Jena Clinic during a period of seventeen years. From an analysis of this material he sketches the symptom-complex of amentia as follows: After an acute onset with excitement and hallucinations, often a violent delirium, the patient grows somewhat quieter and the fundamental symptoms are then perceived, viz., confusion and primary incoherence in ideation; in severe forms the stream of talk may be limited to fragmentary utterances or senseless sound associations. No fixed delusions are formed. No primary or stable alteration occurs in the emotional field, but the mood, speech and action of the patient all reveal a high degree of perplexity, one of the most characteristic symptoms. Hallucinations, not an essential symptom, are usually present and may be so abundant as to overwhelm the patient, adding a further element towards confusion and incoherence. A very characteristic feature is the persistence of the incoherence during remissions when the patients are relatively clear, and also its continuance into the convalescent period. Katatonic symptoms and negativistic traits may occur and do not render the prognosis more unfavorable. As to etiology, in 90 per cent. of the cases some harmful influence was apparent and over 60 per cent. of the patients were suffering from some somatic disorder. A large number of the women were in the puerperal period. Of the 110 cases, 21 died in the hospital; 66 were discharged recovered or improved; 23 passed into chronicity. Of the 66 recoveries it was possible to follow up 61, and 55 of these were found to have remained healthy. Of the 23 cases with a chronic course the large majority occurred in young individuals and proved to be dementia præcox. The author discusses the differential diagnosis and criticizes Kraepelin's views regarding the relation of dementia præcox and manic-depressive insanity to amentia. The picture of an hallucinatory confusion in a young person without adequate external cause should arouse suspicion of a beginning dementia præcox.

G. H. Kirby (Wards Island).

Book Reviews

THE PSYCHOLOGY OF DEMENTIA PRÆCOX. Dr. C. G. Jung, Privatdozenten der Psychiatrie in Zurich.

An important and invaluable contribution to psychopathology is Jung's book on the psychology of dementia præcox. The style of the book is clear and lucid. Although he handles an extremely difficult subject, yet one can easily follow him and fully comprehend his profound thought. Jung adopted Freud's methods of psychopathological analysis and as he remarks "when a superficial glance is cast on the pages of my work, it will be readily seen how much I am indebted to Freud's genial conceptions." The book consists of five chapters.

In the first chapter he gives a complete anthology of the literature on the psychology of dementia præcox. He discusses in detail the following theories advanced by various authors: Apperceptive dementia (Weygandt); Dissociation, Abaisment du niveau mental (Janet and Masselon); disorganization of consciousness (Gross); dissociation of personality (Neisser and others); incongruity between thymopsyche and noopsyche (Stransky); tendency towards fixation (Masselon and Neisser); emotional deterioration (Neisser). Freud and Gross have laid much stress on the importance of the dissociation complex and Freud was the first to demonstrate the "principle of conversion" in a case of dementia præcox. However, Freud makes no attempt to explain why dementia præcox exists and not hysteria. Jung hints at the theory of autointoxication and asserts that most probably somatic causes are responsible for the mechanism of dementia præcox.

In the second chapter Jung discusses the affective complex and its general influence upon mental life. The essential foundation of our personality is affectivity. Thoughts and actions are expressions of our emotions. The component elements of psychical life are sensations, concepts, and feelings, which form certain units in our consciousness. These units may be compared to the molecules in chemistry. "The ego complex is a psychological expression of the firmly associated paths of all the bodily subjective sensations." Each individual molecule of the complex takes an active part in our emotions; it can awaken our emotions and its complexes. The trend of our imagination can be interrupted by an affective idea. The acute process of the working of a complex may become chronic. This chronic complex, as a result of a marked impression, may either produce an everlasting continuous after-effect, or may be awakened by new stimuli. To the latter type belong the sexual complexes. Every new complex shows tendency towards perseveration and new associations may develop. The continuous existence of an emotional complex produces some influence upon the psyche. Numerous illustrations are given, among which the interesting and classical paradigm of a lover is cited. One who is not able to put himself in the position of the infatuated lover would surely pronounce him a hysteric or a præcox. In the case of the lover, the persistent activity of the complex leads to "a partial apperceptive dementia associated with lack of proper emotional

reaction to all stimuli not pertaining to the complex. Each complex must under all circumstances be extinguished. In many cases the sexual complex cannot be naturally suppressed, hence it wears off by indirect means.

In the third chapter Jung offers a brief discourse on the influence of the emotional complex upon association. He analyses in full a dream which was quoted by Freud in his book, Traumdeutung. He compares the mechanism of a dream to that of dementia præcox and shows how intimately they are related to each other. Jung remarks: "If we allow a person in dream to act and walk around as if he were awake, then we have a clinical picture of dementia præcox." He further adds: "In dementia præcox a preformed mechanism is loosened which normally and regularly functionates in a dream."

A comparative analysis of dementia præcox and hysteria is given in the fourth chapter. The symptomatology of these two diseases is thoroughly analyzed. There are essential differences between dementia præcox and hysteria, and they may be thus outlined. Hysteria is possessed by a complex which cannot be very well conquered. The "wish dream" and "wish deliria" of the hysteric are gratifications of the complex. There are several complexes in dementia præcox which have become permanently fixed and which cannot be suppressed. The causal relation between the complex and the disease in hysteria is conspicuous, but not well defined in dementia præcox. However, analyses of many cases of dementia præcox clearly show that their onset was caused by marked affectivity from which painful emotional states developed. In addition to the psychological action of the complex, it is also believed that toxius helps its further activity. The psyche cannot liberate itself from the complex and therefore personality becomes distorted. Jung says, "what Janet speaks of the functions of the real in hysteria may be applied to dementia præcox, too. 'The patient constructs in his own imagination small, very coherent and logical stories; that is when he comes in contact with reality he is no more able to effect attention than comprehension.'"

In the fifth chapter we find a complete analysis of a case of dementia paranoides. The semiology of this clinical picture is thoroughly dissected by means of his association methods. The last chapter is an extremely interesting and valuable one.

In summing up Jung's book one can do no better than quote Professor Adolf Meyer: "As a guide to some system in the sizing up of central complexes and the protean manifestations, and a most valuable extension of dynamic psychology, Jung's work is a great achievement." (Adolf Meyer's review on Jung's Psychology of dementia Præcox, *Psychological Bulletin*, June 15, 1907.)

MORRIS J. KARPAS (Ward's Island, New York).

HYGIENE OF NERVES AND MIND IN HEALTH AND DISEASE. By August Forel, M.D., Formerly Professor of Psychiatry in the University of Zurich. Authorized Translation from the Second German edition. By Herbert Austin Aikins, Ph.D., Professor in the Western Reserve University. G. P. Putnam's Sons, New York and London. 1907.

Forel's activities of late years have been devoted more or less to popular expositions of topics connected with the nervous system. This, his latest treatise, which appeared in both the French and German tongues, has been written for the intelligent layman and covers a field almost entirely neglected heretofore. After a preliminary canter in the fields of the

anatomy and physiology of the nervous system, which we believe, even the intelligent layman will find difficult to follow by reason of the interjection of trivial details, Forel takes up the study of the pathology of the nervous system. Chapter VI. contains an excellent summary of some general conceptions concerning abnormal mental processes, and is followed by a short chapter dealing with the actual disorders of mind, constituting the main outlines of psychiatry in which an abridged Kraepelian terminology is followed.

Part III takes up the hygiene of the mental life and of the nervous system, which cannot be fully discussed in this place since the general features are commonplaces to those dealing with this specialty.

From the popular point of view the work is excellent. We cannot commend it from the standpoint of literature as rivaling in any sense the work of Huxley, yet for its purposes we believe it will prove very effective. It has been well translated and is an attractive volume which can be recommended as a preliminary treatment in severe cases which need a knowledge of some of the foundations preparatory to a course of psychotherapy.

JELLIFFE.

TICS AND THEIR TREATMENT. By Henry Meige and E. Feindel, with a Preface by Professor Brissaud. Translated and edited with a Critical Appendix by S. A. K. Wilson, M.A., M.B., B.Sc., Resident Medical Officer, National Hospital for the Paralyzed and Epileptic, Queens Square, London. William Wood and Company, New York.

We have had occasion to review this work with much favor on its appearance in its original French dress. No one has done justice to these slight and yet often annoying affections until Meige and Feindel analyzed them so thoroughly.

The translation is most excellent and we are glad to welcome such an attractive volume in such an acceptable form. It should form a portion of the armamentarium of every practitioner interested in nervous affections.

HOWARD.

UEBER DIE ANLAGE ZUR MATHEMATIK. P. J. Möbius. Ausgewählte Werke, Vol. VIII. J. A. Barth, Leipzig.

In 1901 the first edition of this work appeared, then a comparatively small pamphlet. This edition is one of 264 pages and is accompanied by an excellent portrait of the author, which will be the more highly prized by reason of his recent death.

Möbius here discusses a number of interesting features. In the first place he goes over the work of the early phrenologists on the localization of mathematical faculty in which he gives considerable weight to Gall's generalizations. He gives a series of some sixty pictures of mathematicians and the like, and draws certain inferences regarding their physiognomy. Considerable space is devoted to the problems of inheritance of mathematical faculty and the relations that mathematical genius bears to other walks in life. It is a highly interesting and suggestive discussion.

JELLIFFE.

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Original Articles

THE FUNCTIONS OF THE CORPORA STRIATA, WITH A SUGGESTION AS TO A CLINICAL METHOD OF STUDYING THEM.[1]

By Charles L. Dana, M.D.,
of New York.

Anatomical and Physiological.—The corpus striatum consists of three parts, the caudate nucleus, the lenticular nucleus and the nucleus amygdalae into which the tail of the caudate nucleus runs. The caudate and lenticular nuclei are in relation to and form part of the cerebral cortex just at the anterior perforated space. The nucleus amygdalae lies in and under the cortex of the uncus. Thus the striatum is part of the cerebral cortex, though connected with parts that have no very important function.

The connections of the corpus striatum are not altogether satisfactorily known. Its most important fiber-system is the strio-thalamic. This consists of fibers that pass from the caudate and lenticular nuclei (putamen) to the thalamus, sub-thalamic ganglia and substantia nigra (Edinger). These fibers are efferent. They are in close relation with the lenticular loop.

There is a band of fronto-striate fibers connecting the frontal cortex with the striate body particularly (Marinesco) (reticulated cortico-frontal bundles of Obersteiner). These are probably fibers of association. There are also fibers which originate in the corpora striata, and which pass down into the anterior portion of the internal capsule and cooperate with the motor tracts (Wernicke). There are fibers from the cortex that pass through

[1] Read at the thirty-third annual meeting of the American Neurological Association, May 7, 8 and 9, 1907.

the corpus striatum, descending in course, and fibers from the lenticular loop pass from the thalamus up through the striatum.

Thus, in brief, the caudate nucleus is connected by association paths with the frontal cortex and the anterior portion of the internal capsule. The caudate and lenticular nuclei are connected with each other by association fibers, and both portions of the corpus striatum by a tract which passes to the optic thalamus and to the ganglia lying beneath it, as far down as the substantia nigra.

The corpus striatum is an early development from the wall of the anterior vesicle, and its cells are more of the associative and sensory type than motor. It is most closely linked to the optic thalamus and the subthalamic ganglia. It is a ganglion which is present in the lowest of the vertebrate orders, and seems to have functional importance in fishes and reptiles when the pallium is nothing but a layer of epithelial cells (Edinger). We may suppose it, therefore, to be an older portion of the hemispheres than the cerebral cortex of man. As the cerebral cortex grew in importance, in higher vertebrates, the corpus striatum became less important relatively, and while it, no doubt, has some functions in man, they are probably of an associative and automatic character, rather than independent and specialized.

It does not seem to have been proved that the corpus striatum has any motor function except that possibly it may take some part in peripheral speech and articulation and perhaps in more general automatic movements. There is no evidence that it contains thermic centers in man, but there is evidence as will be seen later that it is connected with vaso-motor control. Double lesions are thought also to cause loss of control of the bladder. The statement that double lesions produce bulbar palsy is confirmed by some autopsies but, on the whole, has not been satisfactorily established. Edinger thinks that it may have something to do with spontaneity of movements. There are recorded spasms of forced laughter and crying in lesions of these parts, but such phenomena occur from lesions in other parts of the brain also.

Some of the more recent studies of the functions of these ganglia are recited below.

Experimental and Pathological.—Sellier and Verger (1) made experiments on dogs by electrical stimulation, and found that

lesions of the caudate nucleus caused a hemiparesis of the opposite side, with sensory disturbances analogous to those caused by lesions of the cortex. There were transitory disturbances of tactile and muscular, but not of pain sense.

A. Schüller (2), experimenting on dogs, found that lesions of the caudate nucleus caused apparently a hemiplegia, but he attributed it to a lesion of the capsule.

Tounel and Raviart (3) report the case of a man, aged 45, who had a right flaccid hemiplegia, with dysarthria and dysphagia, and some psychic troubles. This had lasted for six years. They found a softening of the inner segment of the lenticular nucleus.

Stida (4), in the experimental study and review of the functions of the caudate nucleus, concludes that lesions there produce no optic symptoms.

Chailchevitch (5), experimenting on dogs with electrical currents, produced tonic tension of the muscles of the trunk and extremities of the opposite side, with some augmentation of muscular activity on the same side. Respiration was slowed and blood pressure raised.

Lo Monaco and Bellanova (6) experimented on 32 dogs, removing the caudate nucleus. Four of them lived and had hemiplegia, a slight degree of hemianesthesia, and loss of muscular sense.

In another series of experiments made two years later, they state that lesions of the caudate and lenticular nuclei cause a disequilibration of the motor centers.

Mingazzini (7), as the result of pathological observations, comes to the conclusion that the lesions of the lenticular nucleus cause hemiplegia, often very slight paresthesia, vertigo, pains in the limbs and loss of memory. These symptoms gradually improve and may disappear entirely. He also observed sometimes monoplegias, facial and facio-brachial. He thinks that the motor functions of the lenticular nucleus are only supplementary. If the lesions occur in the left lenticular nucleus, they may produce dysarthria, especially if the middle part is affected. The sensory disturbances are slight and rare. Rectal and bladder troubles slight and variable. Atrophy of paralyzed parts may occur. A pseudo-bulbar palsy may occur.

In a later communication, the same author finds that small lesions of the lenticular nucleus cause no symptoms. Lesions

often cause a flaccid paresis of the opposite side; when on the left side, dysarthric symptoms, when on both sides, pseudo-palsy. It is doubtful if lesions here have any influence upon movements of the eyes, reflexes, bladder or rectum.

Löwy (8) reports a case of softening of both corpora striata (caudate nucleus and outer limb of the lenticular nucleus). This caused a general rigidity, like that of paralysis agitans, without tremor.

Touche (9) reports the case of a man, 66 years of age, who suffered from an incomplete right hemiplegia, with paresis of the lower facial and of the tongue. There was a hemorrhage on the left lenticular nucleus.

Otto Marburg (10) reports the case of a double lesion (tumor), of the caudate nucleus, which caused a disturbance of the bladder function. He finds that double lesions of the putamen may also cause paralysis of the bladder.

E. v. Czhlarz (11) reports the case of a tumor of both caudate nuclei and putamen, with bladder disturbances. He thinks that there are three centers in the brain for the bladder:

One in the cortex for voluntary movements; one in the corpora striata for automatic movements, and one in the thalami for bladder disturbances connected with emotion. Lesions must be double to cause these disturbances.

V. Bechterew (12) reports the case of a man, 38 years old, who had a sudden paralysis of the right upper lid, turning of tongue to left, disturbance of speech and tonic contractions of the left upper and lower extremities. With this he had false sensations as to the position and movement of his left arm. Lesion in the striatum.

Franceschi (13) reports a case of double lesion of the lenticular nucleus, with some involvement of the adjacent parts, but not of the O. T. or posterior segment of the internal capsule. The patient had forced crying and laughing and dysarthria, symptoms of bulbar palsy, with double hemiplegia. He thinks that the lenticular nucleus has supplementary motor functions, and that through it run thalamo-cortical paths concerned in emotive expression.

Giuseppe Pagano (14) destroyed the caudate nucleus of dogs and got reactions of anger, fear and also sexual excitement. He thinks the caudate nucleus has to do with emotional activity.

Piazza reports case of double hemiplegia with (slight) dysarthria in a senile dement aged 74. Double softening of the putamen. He collects 22 cases, and concludes that lesions of the putamen, in its middle part, cause paresis of the limbs. If the lesion is in the left side there is some dysarthria. Lesions outside the median part may cause choreic and athetoid movements.

Clinical and Pathological.—I have the notes of four cases of hemorrhage or softening of the corpus striatum which are placed on record mainly because of the rather negative evidence of localization which they furnish. One might infer only that lesions of this region produce a profounder effect upon the strength and vitality of the patient than would be expected from such comparatively slight lesions, and that at times there are disorders of the motility, such as rigidity and spasm, which are rather greater than would be expected from the character and size of the lesion. In one patient gangrene of the lungs developed, a fact of some significance in connection with the gas-poisoning case. One patient had a decided dysarthria in connection with a lesion of the left lenticular nucleus. One patient had two successive lesions of the lenticular nucleus, but continued to do his work as a laborer. The cases show that the motor symptoms (paralyses and rigidity) in these lesions are probably due to pressure. The slight speech disturbance in one case might be due to other causes than simply the striate lesion. In two of the cases the lesion was double but there was no pseudo-bulbar palsy.

Case I. Female aged 70, temporary right hemiplegia, with dysarthria lasting during illness but improving. Death on thirty-seventh day. Lesion of left lenticular nucleus. No abnormal temperature changes.

Examination on Admission.—Patient was admitted on November 15, in a stupid condition. Did not answer questions. Incomplete hemiplegia of right side. Muscles of right side rigid. Biceps contracted. Face drawn towards the left side. Tongue when protruded, deviated to the right. Sensation diminished all over same side. Reflexes active. Respiration slightly stertorous. Five days later the patient had slowly and steadily improved. Paralysis less marked. Sensation improved slightly. Breathing was markedly stertorous at times. She answered some questions, but the dysarthria rendered the answers very indistinct. She swallowed and took food. Bed-sore discovered over the sacrum. By the ninth day patient had nearly recovered all her power and sensation in right leg and arm. Some facial paralysis still remained, the mouth being drawn to the left, and the tongue when

protruded, directed to the right. January 15, patient had very offensive expectoration. Coarse and fine râles over lungs. January 17, expectoration very offensive. Slight dullness and broncho-vesicular breathing, with slightly exaggerated voice sounds over part of right lobe, posteriorly. January 19, marked dullness and marked bronchial and bronchophony over upper part of right lower lobe. Expectoration so offensive that patient was removed to a retired corner of the ward. Temperature 101° to 102°.

Patient grew weaker every day. She died on the 21st, thirty-seven days after admission.

Autopsy. Brain.—Slight atheroma of arteries at base. Left corpus striatum contains a small reddish, fibrinous clot within a sac, and around this the brain tissue was indurated to a slight degree and of a yellowish-red color. The position of the clot was in lenticular nucleus of corpus striatum. It was of oblong shape, and slightly involved adjacent island of Reil and the tissue beneath the nucleus. The white matter of the capsule had not been lacerated by clot, but simply affected by pressure.

Lungs.—Right lung was consolidated throughout, of gray hepatization. Two spots of gangrene in upper and one in lower lobe. The dead tissue was not entirely separated, of a grayish-white color. All the spots were round, or wedge-shaped, and touched pleura. The pleura on this side contained about ℥ lxxx of serum which was mostly between upper lobe anteriorly and chest wall, the adhesions below preventing the accumulation below.

Heart.—Normal. *Liver.*—Slightly fatty, depressed and marked by a slight sac depression, producing an elongation of right lobe reaching nearly to crest of ilium. *Spleen.*—Small. *Kidneys.*—Nothing marked.

Case II. Male, 59, left hemiplegia with exaggerated reflexes. Gradual mental failure, death on twenty-seventh day. Lesion of right lenticular nucleus, most marked in putamen, but involving globus pallidus and internal capsule somewhat. Temperature normal.

Patient was admitted on October 23.

Family History.—Father was insane; otherwise negative.

Previous History.—Had syphilis twenty-four years ago, and had been a heavy drinker of brandy and whiskey for many years. Five years ago had rheumatism. Has otherwise been well except for present trouble.

Present Illness.—October 22, the patient felt strong and well when he retired, but in the morning when he arose, he felt a peculiar numbness in the left side, and on attempting to stand, he fell over on account of loss of power in left side. Says he has been unable to use the left arm or leg since and that his head feels peculiar. Thinks he can not speak as readily as before the attack, but can say what he wishes, and has no loss of memory of words. Feels more drowsy than he did before the attack.

Physical Examination.—Patient was well nourished, average size, not anemic, tongue moist, slightly coated. In both hands there was a fine tremor and some ataxia, the ataxia being the more marked on the left. There was marked loss of power in the flexor and extensor muscles of the left hand. The plantar reflexes were exaggerated, and there was marked ankle-colonus on the left side. The left patellar reflex was exaggerated; the right one normal. The skin reflexes were absent on the left side, except epigastric reflex which was diminished. The patient could not stand alone, but there was some strength in both the legs.

Urine, acid, 1024, amber, no albumin, leucocytes, uric acid, polygonal cells, sed. mucus.

November 15, patient has been gradually failing in strength since admission, had incontinence of urine and feces. November 18, patient died twenty-seven days after attack.

Autopsy.—November 21, rigor mortis fairly well marked. The dura mater was quite adherent to the cranial bones, especially at the posterior portion. The arachnoid was much thickened, and of a whitish color. The vessels at the base were atheromatous. The brain was then cut in transverse sections according to Pitres' method. In the outer segment of the right lenticular nucleus was a patch of softening, about the size of a bean. There were also many capillary extravasations throughout this nucleus and in the internal capsule. The left brain was normal. Both lungs were somewhat congested, the right being bound down by slight adhesions at both apex and base; otherwise they were normal. The heart was somewhat flabby and slightly fatty; otherwise normal. The liver was very firm, of about normal size, and had some excess of interstitial tissue. Spleen somewhat soft, but of normal size. Kidneys were somewhat diminished in size, capsules slightly adherent, markings indistinct, others dusky and of irregular outline. No fluid was found in any of the cavities, save the pericardial, and this could not be considered in excess of the normal.

Case III. Male, aged 43. Left hemiplegia continuing for a year, but permitting patient to work. Eight months later right hemiplegia, which was temporary, patient returning to work. Finally ventricular hemorrhage and death on same day. Cysts found in both lenticular nuclei, larger in right.

Family and early personal history not obtainable. About one year ago the patient had an attack of apoplexy, which left him with right hemiplegia for some time. He partially recovered from this, but had some loss of power persisting. He returned to work, but was obliged to accustom himself to work with the left hand. Four months ago he had another attack which resulted in paresis of the left half of the body. He entirely recovered the use of this side. For some weeks patient has had indefinite sensations of illness, such as formication paresthesia in different parts of the body, and has suffered from headache, but

he was able to continue his work. On the day of admission he was at work in the morning, but complained of weakness and mental confusion. He went into a beer saloon and drank one glass of beer; he remained there intending to rest himself. He was then seen suddenly to fall from his chair and become unconscious. He was immediately sent to the Reception Hospital, at 99th Street, and from there transferred to Bellevue.

Examination on Admission.—Patient was unconscious, somewhat delirious and restless. Pupils contracted irregularly, the right less than the left. Loss of power is noted in the left lower extremity; the other limbs are somewhat rigid, except when voluntarily moved by patient. Patient rolls the eyes upward and to the right, and the head is turned to the right, with rigidity of the cervical muscles. Urine is passed involuntarily, and the respirations are slow, with inclination to stertor. Temperature 102°; pulse full and about 85. Patient remained in the cells until the following morning, when he developed pulmonary edema, and was transferred to the hospital wards. A small quantity of urine was obtained by use of catheter, and was found to be loaded with albumin. The patient died on the night of admission.

Autopsy.—Dura mater thickened and adherent. Pia mater opaque in many places, and beneath it a large quantity of blood was effused, distributed over entire surface of brain, but chiefly seen at the base.

Brain.—An extravasation of blood was found in the lenticular optic region of the right side. The tubercula quadrigemina were somewhat torn, especially on the right side. The ventricles, lateral, 3d and 4th, were filled with clotted blood, and blood had escaped from these beneath the pia mater over the brain surface. The internal capsule was little injured, being slightly torn posteriorly. In the lenticular nucleus of each side were cysts, the remains of former apoplexies—that on the right side being much larger than that on the left.

Heart.—Left ventricle hypertrophied largely. A small calcareous spot existed at base of mitral valve.

Lungs.—Congested and edematous.

Liver.—Cirrhotic and fatty.

Kidneys.—Presented interstitial and fatty changes. Capsule separated with difficulty, leaving surface torn and granulated.

Case IV. Male, aged 73, admitted in a condition of semicoma, no distinct signs of paralysis, but a general rigidity and later convulsive movements, relieved by bleeding. Death on same day. Bilateral softening of both lenticular nuclei, especially involving the globi pallidi.

Patient was admitted to the hospital March 9. No history. He could be partly roused to a consciousness of his surroundings, but was unable to respond to questions. Face was flushed; breathing quiet. Head and eyes turned to the right. Pupils

moderately dilated and even. Marked rigidity of all the limbs, most pronounced in the left arm. The left hand and fingers are held rigid, the fingers being flexed at the first phalanx and the second and third extended. There was no distinct evidence of paralysis. He did not respond to cutaneous irritation, probably on account of the stupor. He was able to swallow. The heart action was weak; the urine showed specific gravity 1026, albumin 0.1 per cent., no casts noted, but phosphates and a few pus cells. Spleen and liver normal. Twenty-four hours after admission the patient had slight spasms, beginning with convulsive movements of the face and left hand, and including movements of face, head and both arms and slighter movements of the legs. They were considered uremic, though there was little albumin and no casts or edema. He was bled $7\frac{1}{2}$ ounces, and immediately after the patient seemed more conscious. The pulse continued weak. The spasms ceased. The record shows that after this the stupor gradually deepened again, and the patient became comatose, dying early the third day. The temperature record of the three days that he was in the hospital was taken in each axilla and in the rectum. The temperature was 101.2 the first day 100 to 99.8 on the second, 102 on the third.

Autopsy.—The full record of the autopsy was lost, but the notes made by myself record two spots of softenings, one in each lenticular nucleus, and especially involving the globus pallidus.

The Effects of Gas Poisoning on the Lenticular Nuclei.—It has long been known that a very common, if not almost universal, effect of sewer gas poisoning, is a softening of both lenticular nuclei. This may happen if the patient has been unconscious from the effects of gas for more than twenty-four hours.

It has seemed to me that a profitable method of studying the functions of the lenticular nuclei might be that of observing the physical and mental condition of patients who have suffered from severe gas poisoning.

Ziemssen first reported a case of gas poisoning with softening of the corpus striatum in his "Electricity in Medicine," in 1864.

In 1868, Th. Simon (15) showed that gas poisoning caused thromboses and softening of the brain and particularly of the lenticular optic region. He describes first, I think, bilateral lesions of the lenticular nucleus as a result of this condition.

A woman, aged 46, was found unconscious and asphyxiated from CO_2. The poisoning was very severe and consciousness was not restored for several days. She finally recovered and resumed her work for a few days, then developed symptoms of mental disturbance. She lay with limbs stiffly outstretched,

groaning and screaming, and had some convulsive attacks. Pupil, left eye, contracted to point, right eye gone. Pulse 88. Answered no questions, swallowed little, spit out food put in her mouth. She died in seventeen hours. Autopsy showed in the left side of the brain a spot of softening the size of a cherry in the middle of the corpus striatum which at the limit of the thalamus behind was more solid. A similar, but small lesion was in the right striatum. No other spots of softening.

In 1898, Kolisko published a series of articles upon the blood vessels of the brain. In this he particularly called attention to the existence of a special artery which is given off from the anterior cerebral, near the communicating branch. This artery is usually single, but sometimes double. It runs upwards and backwards, and it supplies the caudate nucleus, part of the anterior portion of the internal capsule, and part of the outer portion of the lenticular nucleus. Owing to the length and peculiar direction of this artery, the blood that passes into it has to flow rather against the normal current. The result is that the pressure in this artery easily becomes relatively less than that of the other vessels, and when the general blood pressure is very low, as in gas poisoning, a tendency to stasis and thrombosis occurs. This artery is the one, then, that is particularly selected as that in which thromboses occur, and Kolisko states that it has as much right to the name of "the artery of cerebral thrombosis" as the branch from the middle cerebral has to the name of the "artery of cerebral hemorrhage." It is an involvement of this artery on each side, which occurs in cases of gas poisoning, and which leads to the very common lesion of bilateral softening of the corpora striata in that condition.

The Syndrome of Lenticular Lesion.—Klebs (16) quotes a case. Man poisoned by gas and under its influence four days. Had paralysis of the right arm, edema and gangrene of skin. He had a sharply limited focus of softening in the corpus striatum. He also reports two cases of symmetrical softening of the caudate nuclei and the two inner parts of the lenticular nucleus and anterior part of the internal capsule in which vaso-motor and trophic disturbances of the skin occurred.

Kahler (17) reports a similar case and the findings are shown in Lesser's Atlas, II, Tafel xviii, case 7.

Poelchen and Kolisko (18) observed the same phenomenon.

The study of the cases reported shows that with the involvement of this region, there often occur edema and gangrene of the skin (Klebs, Kahler, Porechen, Kolisko). So that when after gas poisoning one finds the patient unconscious with these vaso-motor and trophic disturbances present, a diagnosis of lenticular lesion may be made—Kolisko did actually make such a diagnosis, verified by autopsy.

It may therefore perhaps be said that severe symmetrical softening of the corpus striatum has a "syndrome." If a person poisoned by gas is made unconscious for over forty hours and develops skin lesions, vaso-motorial and gangrenous, he has a double lesion of the corpus striatum.

Clinical Study of Recovered Cases.—A further and special object which I had in view in studying this subject was to find out the later history of severe gas-poisoning in which recovery took place. It may be that in these patients the lenticular nuclei have been injured and thus a pathological experiment is made. I have tried to get hold of all the recovered cases that had been brought into Bellevue Hospital in the past year, but I was not very successful, as only four patients were examined. Besides these I had under observation several years ago a victim of gas. In this case there was a prolonged period of amnesia, with the development of practically a double personality. The case was reported by me at length in the *Psychological Review*.

Another patient was well except for curious attacks of vomiting which would come on explosively, without any particular cause. The other two said that they were as well as they had ever been in their lives. There is in the literature of gas poisoning only some references to the fact that it leads to impairment of memory. My investigations so far have not been very fruitful, but it seems to me that a further examination of these cases by neurologists may lead to some interesting results.

It is well known, of course, that other and severe lesions of the brain may be caused by gas poisoning, but the most common and practically uniform lesion is that of the lenticular nucleus. This lesion need not be a very large one and in fact, one must examine closely sometimes to find it. I have photographs of a brain with lenticular softenings from gas poisoning furnished me by the kindness of Dr. Otto Schultz and Dr. James Ewing, upon whose authority I make my statements regarding this condition.

Summary.—The corpus striatum has not any independent or specific motor function. It probably has some supplementary motor function, and especially in connection with articulation.

It may have some control over the bladder (double lesions).

It seems to have some control over vaso-motor and trophic conditions of the skin (and lungs?).

It has no thermic center.

It may have some supplementary and associative psychic function so that lesions affect memory or initiative.

It is an organ of less importance relatively in the higher vertebrates.

In severe gas poisoning there is a double softening of the lenticular nuclei due to thrombosis of "the artery of cerebral thrombosis," and there result vaso-motor and gangrenous conditions of the skin, so that these conditions in connection with a history of coma from gas poisoning form a group of symptoms called "the syndrome of the corpus striatum."

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GENERAL CONSIDERATIONS AS TO THE NATURE AND RELATIONSHIPS OF HYSTERIA.

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(*Continued from page* 35.)

Case No. II is another hysteric who long passed as an epileptic. Her father was a drunkard and died shortly before her admission in 1896 at the age of 14. She is one of 14 children and several of her sisters by common report do not bear good reputations for morality. As a child she had fainting attacks, and after being run over by a wagon without special injury at nine years, she had convulsive seizures. They come usually in series, and in recent years at the menstrual period. She has had as many as 97 convulsions in a night. A few hours after the fits are gone she is again strong and clear mentally. With the convulsions complete cutaneous anesthesia and visual field contraction develop, but these subside in the interparoxysmal period, and she is intelligent and industrious, but usually short tempered.

She exhibits little of the hysterical tendency to many physical complaints in her intervals, but likes to receive attention and is easily disgruntled if she does not have her way, when she will walk about as if partially dazed and mutter to herself.

The onset of a series of convulsions begins with dreams at night; especially about her father, whose death is said to have materially aggravated her symptoms. She also appears dazed and dreamy in the daytime; walks about, frowns, mutters and pays no attention to her acquaintances. She hears at these times hallucinatory voices and ringing bells at night. In her convulsions she invariably tries to bite her own right arm, sees men in the room and talks of some one coming to take us away, but what she says is fragmentary. Some menstrual periods are passed without convulsions and she has been several times since her first admission, at home, either on parole or discharged, but in a few months each time it has been necessary for her to return, except that since March, 1905, she has been home and unusually well.

Summary—A young woman hysteric since nine years of age and perhaps longer, characterized by series of convulsions chiefly at night at the menstrual periods, with hysterical stigmata subsiding in the interparoxysmal period; unmanage-

able by her friends during the attacks and irritable in the intervals.

The two cases above must be regarded as hysterics. They are not properly to be thought of as cases of mania, though so many authors speak of all hysterical insanity as hysterical mania, for the symptoms of depression and anxiety are at least as prominent as this boisterous conduct.

Both have been regarded as epileptics because of the occurence of convulsions, yet the convulsions of each were accompanied by so many hysterical features as to leave no doubt as to their character. They will serve to indicate that the relations of hysteria and epilepsy need restudy. Before accepting automatisms and deliria as epileptic, which have been in the past too readily considered such, the whole case should have the most careful scrutiny for hysterical features. The case of Bevan Lewis has already been referred to and certainly any general views of epileptic automatism built upon such material are in need of revision.

Case No. 3 is that of a mulatto girl aged 15 years admitted to Middletown on September 7, 1905. It was her first attack. She was a strong, well-grown child, the sixth of nine children, and considered a good worker. One sister falls in a swoon whenever there is any excitement, and a brother is an alcoholic.

In April, 1905, over four months before admission, she complained for a day or two that her eyes pained, then for a day of pain in the left arm, and for two weeks the arm seemed completely paralyzed. At the end of two weeks her mother found her unconscious on her bed, a smell of chloroform was present, but probably only from the liniment used in bathing her arm. Her head was bathed, and after half an hour she regained consciousness and the paralysis had gone. She was dazed at first, but gradually became clear and complained of pain in this arm and in her head and back, but after three or four days in the early part of May convulsions developed. Her first convulsion came on while kneeling in bed. She suddenly threw her head back until her head and feet came together, and all subsequent convulsions are said to have occurred in bed and to have exhibited the same phenomena. Sometimes she would maintain this characteristic attitude two hours at a time, until the doctor came and subdued it by a hypodermic injection or the administration of chloroform. She has had about 50 attacks in all, they were more severe with the menses, and sometimes she was free from them two weeks at a time and did her work well. She has been known to bite her tongue and to bite her arm in the seizures.

After the seizures two months before admission she saw cats about her bed, said there was a big black woman in the

room, saw angels and called out "Mamma! Mamma! Ain't they beautiful?"

About two weeks before admission after being some time quite natural she was startled while sitting at the piano by a sudden clap of thunder; threw herself on the bed and had a series of convulsions lasting all one afternoon and night. For three or four succeeding days she was chloroformed three or four times a day to control the convulsions, and when they subsided she seemed not to know her relatives, and said when she saw a cousin in a white dress, "Oh my, there is a little black girl all dressed up in white." She insisted that she was going away, that she was not treated well; that her parents were not really such, and that she would jump in the pond. She did escape from the house and when brought back angrily attacked anyone who came near her. She was mute, and when she wanted anything pointed to it and made a queer noise.

The nurse who brought her found the house in disorder, the bed was broken and without sheets or pillow cases, and the piano protected with quilts to keep her from breaking it as she had threatened. On the way to the hospital and on admission she resented especially the presence of men. Looked tensely at them, blew out her breath with a hissing sound like an angry goose, and sometimes sprang at them.

The day of admission she had three convulsions such as already described, in the presence of one of the hospital physicians. She was then without reaction to pin pricks and without corneal or conjunctival reflexes. There were eight convulsions that night, but none afterward. The next day corneal reflexes had returned, and she felt pin pricks, but she had no recollection of coming to the hospital and did not know where she was. Conjunctival and swallowing reflexes were not elicited.

September 12, when the physical examination was made, there was no visual field contraction. September 29 she complained of a "sharp" headache such as she said had before preceded her spells. She passed a menstrual period without difficulty. She was amiable and industrious. At staff meeting she denied any memory of her hysteric episodes or of coming to the hospital. October 17, 1905, she was paroled home in good condition.

Summary—Hysteria in a girl of fifteen which began as far as known with complaints of pain in her eyes and left arm, followed by paralyses of the arm which disappeared after a period of unconsciousness, and gave place to convulsive attacks accompanied by hallucinations, delirium and delusional developments.

These cases, and especially No. 3, illustrate the chief characteristic features of the typical hysterical attacks. It is

marked by paralysis, convulsions, anesthesia, delirious actions, and a complete forgetfulness of all that occurred in her hysterical states. On coming to herself she preserved no conscious memories of the interrupting mental state, and a certain tendency to organization in this state is seen in the constant recurrence of her convulsion in the same form, her aversion to men, whom she attacked whenever one came near her, and her desire to destroy the piano at which she sat when her final exacerbation began. When this state was on her she did not know her immediate relatives, showing how little of the acquired knowledge of the individual may be at the service of the second state. Hysteria usually starts from some psychic stress. None is definitely known in this instance, but it is thought that she had a real rheumatism of the left arm at the start and the later course of the disorder was perhaps aggravated by a diagnosis of cerebrospinal meningitis then epidemic and very fatal in the neighborhood.

Case No. 4 is abstracted from the records of the Manhattan State Hospital. She was a newly married woman of twenty-eight years, the eighth of nine children. One brother is said to be of a very nervous temperament.

As a child she was delicate and nervous. Did very well at school and business college, but changed her occupation often because her work made her nervous. She was employed as a nurse in May, 1902, when she was quietly married without the knowledge of her family. Within a few days she had crying spells and complained of pain in her right side, especially if her husband made any advances. She became dissatisfied; and much annoyed by any noise. Four weeks after marriage one evening she became restless, cried, lay down, and threw herself about the bed. After a while had a convulsion lasting half an hour. Was given a hypodermic injection which quieted her. On coming out knew a good deal that happened during the fit. A few days later she was depressed, crying, restless; she said that she wanted to die; that her husband abused her; thought he and his family wanted to poison her, and that she was talked about. She was taken to her sister's house and there acted as before, but slept well and had a good appetite. There she became worked up one day because her husband did not come to see her, and had another convulsion not involving the face or causing frothing at the mouth. After it she was stuporous 2 or 3 hours, but as before, a hypodermic brought her out and she looked about and inquired why so many people were in the room.

The next day she was taken to a sanitarium, where she was highly nervous, excitable, lacked self-control, and had frequent crying spells. She seemed fearful and started with the least noise. Was very changeable and made many threats of

suicide. Some days said she was out of her mind, that her brain was gone, and at times she was in a sort of ecstatic attitude, and for two or three minutes could not be aroused.

When taken home she showed a strong dislike toward her husband and suspiciously thought everyone was trying to be rid of her, and on July 13, 1902, because she could not go home with a friend, who had called on her, became enraged, broke the dishes, fought her husband with a knife, twice tried to inhale illuminating gas, and was taken to Bellevue Hospital.

Upon admission to Manhattan State Hospital she was found poorly nourished and sensitive over the right ovary. There was "no hemianopsia" indicating no contraction of the visual fields. She was emotional and restless, apprehensive and suspicious. Referred everything to herself. The night following admission she thought that she had a baby. Said to the physician on July 19, with great vehemence, "You are bound to kill me, are you. Go away from here. I heard you talking through the wall, trying to kill a good woman. . . . What is the difference if I was out with men before I was married?" (Q.) "Who said anything about it?" "Why, he is out there talking to her about it. . . . They are trying to hold me here. Trying to make me bad." (With great emotion.) She says she does not remember when she came here. She was doped with morphine and chloroform. Thinks she has been here a week (2 days).

July 20. Resists the nurses, orders them from the room. Continued hallucinations of hearing. Says the physician operated on her last night—she did not feel it, but someone said so. Spoon fed.

July 21. She lay limp, moaning and whining with each breath. Told the nurse she was to die. Takes no notice even of deep pin-pricks, except to move her feet slightly, and cannot be aroused. Temp. 100. Tube fed.

July 22. Talked of being etherized for an operation, but later became mute and limp. She spit out her food. Left arm raised, she kept it up over 40 minutes. There was strong resistance at the elbow. None at the wrist and fingers. Insensible to pin-pricks, except for slight movement of the feet when the soles are pricked. Winks slightly from motions before the eyes.

The catalepsy lasted until July 25, when she used the left arm. She was found whimpering and repeating, "Oh, don't kill me." Says she awoke and found herself here this morning and remembers nothing since her third day in Bellevue.

The next day she monotonously repeated, "They are going to kill me." Hallucinations continued, but disappeared after her transfer to another ward on July 30. She still claimed to feel pin-pricks less on the right than the left side.

On August 2, when the hallucinations had disappeared, a mental status was made. She was still suspicious, and referred things to herself, easily made to smile or laugh, had frequent crying spells, was perfectly oriented and had a good grasp of her situation. Memory was good except for the amnesic period mentioned above. Calculation was unimpaired, but she lacked powers of concentration and concentration was an effort. She gave a coherent account of the development of the psychosis. She dreaded marriage and was told that she could not have children. She wished to douche herself, and her husband said she was killing children, "Jawed me although he knew it would kill me if I had a child." "I found a change in him. He would be rough and grew cold. It worried me. I sat and cried. Then at last I got so melancholy I didn't care to eat." Then she goes on to tell as facts of many hallucinations of hearing. She improved during August, but complained of hot flashes, cold feet, pain in her side where an enlarged prolapsed ovary was found, and she was inclined to think herself detained for a purpose. Early in September she was fault-finding, lacked self-control, had frequent quarrels with other patients, liked to talk of her sickness, talked of being weak, but went to the dances and took part. She did not, as far as noted, Aug. 30, recover remembrance of her amnesic period. On Oct. 10, 1902, she was discharged recovered.

Summary.—A woman of the hysterical disposition, bright but nervous and complaining, who following marriage and disagreements with her husband had crying spells; had two convulsions; became restless; wanted to die; thought people talked about her; and that her husband tried to poison her. Sanitarium treatment failed to benefit her and she was sent to Manhattan State Hospital, where during the first week she had auditory hallucinations; thought she was chloroformed; that she was operated on; was to be killed; was in great agitation, and for three days cataleptic in the left arm and analgesic. She made frequent threats of suicide and said she was out of her mind. Upon coming out of this state she had no recollection of its events.

This patient with an anxious depression probably largely on a hallucinatory basis; undoubtedly hysterical as shown by the above symptoms; and admitted at a time subsequently shown to be included in her hysterical amnesia; did not when admitted present hysterical stigmata, and the last mention of anesthesia in her case record is when two days after coming to herself she "Claims to feel pin-pricks less on the right hand than the left." This case and others that are to follow it will prepare us to be slow in positively stating that a depression of similar character is not hysterical because at the time of

examination hysterical anesthesias cannot be shown. Janet goes so far as to say that as a rule in hysterical deliria the stigmata disappear. This was not found to be the case with the next patient, who showed variable contraction of her visual fields usually worst while she was amnesic and distressed, and clearing up when clearer mentally. It may be noted, however, that with her the scant evidences of tactile anesthesia were found only at the onset.

Case No. 5.—A single nurse maid, aged 24, a native of Ireland, but who has lived for 12 years in the United States. Insanity and alcoholism in the family history are denied and she was considered more capable than the average. She was always nervous at her menstrual periods, and three years before, after an operation for varicose veins, she had to give up work for three months because of nervousness. It was subsequently learned that at this time she manifested ideas of violation very similar to those of the attack which we observed. She was chaste; cared little for masculine attention; was highly regarded by her employers, and had spent the summer at Bensonhurst in charge of a little girl. She was admitted to the Manhattan State Hospital West in October, 1904, and was observed on the ward assigned to the Pathological Institute. At this time she said that three weeks before her admission and the day before her return to New York her room was entered by a waiter who boarded in the same house. He was, she said, offensive in his endearments (she did not like him), and she resented his advances. The lock on her door was not strong, and the next morning she thought that it had been tampered with, and the thought came that perhaps she had been violated in the night. On returning home she could not get the occurrence out of her mind; slept poorly, and at the usual menstrual time the menses did not return. She says something about their return after the use of instruments. Her mistress called her own physician, who prescribed sleeping powders, but the patient took all at once with suicidal intent, and slept for many hours. She was distracted and dazed on coming out, rose twice in one night to play in the nursery with her charge's playthings, and was sent to Bellevue Hospital. There she was depressed; was sure she was to die; could not possibly get well; was suicidal and said she had brought all her trouble on herself by doing wrong. She was thought to be retarded.

When admitted to the Manhattan State Hospital she was oriented, coherent, and told part of her story and suppressed the rest. No distinct hysterical stigmata could be made out. Later very variable contraction of the visual fields and slight transient cutaneous anesthesia were observed.

October 5, one day after admission, she had an episode

of amnesia, beginning in sleep and lasting about an hour and a half. She did not know her own name, mistook the doctor, gave varying accounts of her age, fabricated freely when pressed to answer. She could not add, say the alphabet, or her prayers, or tell the time of day. She puzzled over the name of a key; decided it was a hairpin, and tried to use it as such. She continued to remember that she was crazy and in an asylum. She was drowsy, and on coming out had no recollection of what had been said and done.

After this amnesia was present in varying degree much of the time. As a rule the more drowsy and forgetful she was the more marked was the visual field contraction. Often she was able to recite poetry when she could not recall the names and uses of common objects.

Oct. 13 fever began. At first no cause for it could be found. October 17. She was averse in her attitude and would not speak. The next day she spoke of herself as killed; said the skin was burned off her body; she was in a prison cell. Said she expected to be burned again, but made no effort to escape. In explanation of being in prison she made hazy statements that they say she killed a woman. She did not know pieces of money, and knew her name only when it was called. Visual fields about 10 degrees. An hour later she was clear, and still later the same day in great agitation.

Another day, having heard, on the ward, talk about the birth of a child, was sure she had a baby and that they were hiding it from her. The next day she had insight into this idea, also another that she had not previously talked about, an accident on the street; a ride in an ambulance; and the suture of her wounded neck with big colored stitches. Oct. 21. She was in great agitation from fear of poison. Ascribed her past conduct to stubbornness, but is perplexed herself and says, "I didn't realize it either." She groaned over and over that she was to be killed and in this as in all other such beliefs which appeared at this time, arguments or assurances entirely failed to affect her agitation, restlessness and fear. Blood pressure was not increased. She remained in the above condition until Oct. 29, when the New York City board of health reported a Widal reaction. Active treatment for typhoid fever was instituted. A severe catarrhal pneumonia developed, and a second blood examination showed Widal reaction absent. She was very sick for several weeks, tuberculosis present on admission became active for a time. On coming out she had only fragmentary recollections, upon which she insisted that an effort to poison her had been made, and did not recall at all most of what she had said. She was subsequently discharged. Dr. G. H. Kirby, who has kept in touch with the patient, very kindly reports that at the last

she has lost all recollection of her fears of violation, poison and physical injury.

Summary.—An hysteric attack following prolonged worry over a possible shocking mental experience with at first distracted and inexplicable conduct, with suicidal tendencies, then by period of general and partial systematized amnesia, followed by extreme agitation and fear for her life, and complaints of torture, the subsequent course obscured by physical illness but ending with subsidence of the mental disturbances and loss of memory of their content.

Case 6 is another with periods of amnesia. She was admitted and discharged at a time when the writer had not learned of the conception of hysteria here presented and the observations are far from full.

Admitted to the Middletown State Hospital May 27, 1902. A domestic, aged 23 years. Single. A cousin is said to have been insane. Particulars are not given. Her father in the petition assigns religious excitement as the cause and the patient later said she had had no shocks or griefs. Her mother was very nervous while carrying her. She had infantile convulsions until three years of age. Then no convulsions until one and a half years before admission. She was always very religious and liked the society of elderly ladies best. She had been irrational, at intervals for 7 or 8 months, had made numerous suicidal attempts to choke herself and tried repeatedly to kill her parents.

When admitted her principal mental symptoms were: "Intense fright, hallucinations of sight, a frenzy of fear, resistive, apprehensive. Does not talk except incoherently as a rule, but occasionally answers rationally." She told the nurse her head felt in a whirl and spoke of seeing snakes and animals and wanted to go into the water and woods away from them. Two days later she came to herself in the bath. Said she had had these spells before but did not remember them, and so could not tell how long this one had lasted. Jesus, she says, talks to her and tells her to kill herself. May 30 she was bright and less depressed, and told of seeing seven old men with long white beards for a few minutes in the morning.

June 5 she had an attack. She was swaying and the nurse caught her. She did not froth at the mouth. After rousing from this she had the same frightened look as on admission; appeared to see things that frightened her. On the 6th she had a convulsive seizure, frothed at the mouth, rolled her eyeballs, and when she came out cried, called for knives and had to be held in bed. During the night she cried and had indescribable hallucinations of sight and screamed all the next morning, saying her father and mother were dead and buried.

During her screaming she referred often to having her

teeth filled, which had a few days previously been proposed. She said she has "a devil of a head," and that she "felt damned queer," and on remark about her language became more profane. Claimed the woman physician was her sister.

The next day she was quiet and well behaved, did not remember the incidents of the previous days and took up the thread of things from the time she "fainted away," as she called it.

July 21 it is noted that the night previous she began with visual hallucinations, was frightened, bit her hands and was put in restraint until noon, when she became quiet. This attack was less severe than its predecessors.

July 23 the next recorded attack occurred. She clutched her hands. Tried to throw herself out of bed, bit her tongue, her face was white, she resisted pressure on her ovaries. After her convulsions she talked a great deal about domestic cares and a baby—"You will have to get supper. I ain't able to get up and work. Just bring the baby. I will take care of it awhile, for I am afraid when she gets supper she will scald it. The others can walk. They are big enough to go out and walk. Take that stuff away. It stings and pains my throat. The others can go out in the shade and play if you will just bring the baby. If I wasn't so sore and sick I would get up and get it. Oh, Ella, you don't know how my head hurts and feels so hot inside. Bring the baby; bring him here while you get supper, or you will hurt him." These sentences she repeats with a whine. She sheds tears. She talks more and deranges her bed more when she sees herself observed. She also talks of killing the baby with carbolic acid. She jumps up suddenly and stares—"Oh, see them pretty gold stars shooting up before my eyes. Bring the baby so he can see them. It makes me think of heaven." On the 25th she was as well as usual and without recollection of her conduct.

On the 26th she had a new attack. Complained that empty cars on a railroad switch would run over her. She was in the ward. She tore her hair without evidence of pain and was insensitive to pin-pricks. She was much worse when the physicians were in sight and was relieved by a cold spray bath. On the 30th she remained analgesic except about the nose and mouth, but was quiet and realized her surroundings.

September 22, after three months' interval, she menstruated without incident. Except for complaints of headache she got on well, was intelligent and tractable until October 20, when she became excitable and confused in the night, was again fearful, mistook the identity of almost everyone about her, and said her head felt half as large as the room.

October 27 it is noted that she complained of her head feeling big and of a swimming sensation, and on November 25 while folding counterpanes, stared momentarily and seemed unconscious, and that morning was in a sort of a stupor for about half an hour. She stayed at the hospital without other marked incident until March 27, 1904, when she was paroled home. She was well and working as a nurse when last heard from September, 1904.

Summary.—A young woman whose hysteria apparently began in religious excitement; manifested itself in convulsive attacks with cutaneous anesthesia in periods of amnesia and in attacks of delirium accompanied by auditory and visual hallucinations; by fears for her own safety; by the reviving of what were perhaps old experiences, and at home by attacks on her parents. She recovered and when last heard from remained well.

The next case was recognized as hysterical only in her fourth attack.

Case No. 7 was on May 8, 1903, when last admitted, 23 years of age. She is of German birth. Her father and an aunt were said to be very nervous and excitable. The father died of cancer of the stomach.

Personal History.—She was always of excitable temperament. When four years of age fell, striking her forehead. She had severe bleeding at the nose, became unconscious and was sick for two months. She was in school from 6 to 15 and has a good intermediate education. When 16, after the sentence of her lover to 15 years imprisonment for insubordination to an officer, she had her first attack of insanity and was for nine months under treatment for depression.

In 1899 she came to the United States and worked as a domestic and later in a bakery. She became overworked, nervous and tried suicide by carbolic acid, but was saved. Soon after she had ischo-rectal abscess. The physician who attended her sent her to one of his relatives in the country. There she had periods of intense excitement. Ran up and down stairs, threw herself on the floor or in the arms of her attendants. She mingled sane remarks with random talk and laughter; at times was perfectly sane. She did not sleep even with hypnotics and attempted suicide by taking carbolic acid. On admission she gave an excellent account of herself and throughout her stay continued able to do so. There was a marked chorea on admission which subsided after three months' treatment. She was discharged as recovered from acute melancholia.

On leaving she went to a high class boarding house. The proprietress became ill and the management devolved upon the patient, and it is said that she did well in this capacity,

but under this strain and a well-grounded fear of pregnancy her depression returned, she walked the streets in an erratic manner, and tried suicide by sucking dilute carbolic acid through a straw. When readmitted September 9, 1901, she was extremely nervous, intensely anxious, depressed, wished to die and it was noted without elaboration that "hyperesthesia of the senses" was present. During her stay she had crying spells and frequent headaches and her chorea returned, especially when she was excited, but was less pronounced than in the previous attack. Six months after admission she was delivered of a male child. A place was found for her and her child and she remained there nearly one year, until May 8, 1904, when she was the third time admitted. Her place was a good one and she has since returned to it and gets on well, but at that time she had attacks when she became depressed, had much headache, her face was congested, she appeared dazed. She seemed well a few days and the same condition returned and she took her usual dilute carbolic acid. The attacks were described as follows: "Her eyes were open and glassy, pupils dilated. She seemed dazed and mumbled to herself and her hands twitched. The spells ended in profound sleep and apparent forgetfulness of what had happened.

Soon after admission she had one of these spells. It began with vertex pressure and severe pain in the left chest. She tossed about in bed. Face was congested, her hands and feet cold. She cried, talking to herself and to imaginary people. Talked of severe pain in the left side, and was so restless and excited the nurses sent for medical help. She made a grab at the doctor's pencil. Suddenly came out of the dazed state and professed no knowledge of what had just transpired.

As in previous attacks she was intelligent and adaptable, and she exhibited not a settled sadness but an emotional instability; easily moved to tears and to laughter through her tears. She was fond of her child and reluctant to be separated from him. She complained of little but frightful dreams, the content of which was not ascertained. July 4, while watching fireworks, she had another attack. She suddenly called the nurse, her face was red, pupils wide and staring. The nurses partially carried her to the ward and called a physician. Then she was throwing her arms about and grabbing at the nurses. She recognized persons, called them by name and intelligently answered questions. Except for these two attacks no positive mental disorders were found during her stay. She has never shown retardation, never flights of ideas, rhyming or sound associations and there is no trace of mental deterioration. Contracted visual fields were found during her stay. Since her last discharge she has

been able to make a trip to Germany to visit her relatives and to return without incident. She now remains well.

Summary.—A young German woman who has been four times confined as insane. Her first attack followed the sentence of her lover to prison, and two subsequent attacks have been induced by mental strain. In two attacks was choreic and in each relapse feebly attempts suicide, always in the same manner. Has an hysterical disposition, a few hysterical stigmata. She does not deteriorate.

The next is another patient whose psychosis might also be readily passed by under the heading of lactational insanity, especially as at no time during her stay in the hospital were anesthesias, or contractures present. No adequate search for amnesias were made. The symptoms, however, totally fail to fall within the categories we are accustomed to consider in this connection, dementia præcox, manic-depressive insanity, or exhaustion or infection delirium, and following her child's birth she had a convulsion, and again about two months later another series of convulsions which were said at the Post-Graduate Hospital, where she was treated, to have been hysterical.

Case No. 8 when admitted was 25 years of age. Nothing significant is learned concerning the family history except that her mother is miserable most of the time with gastric trouble and "growth in the throat and abdomen." She was born in New York of German parentage. At school she was an average scholar and after leaving school she worked in a factory four and one-half years. She always worried easily and was subject to headaches. She was a member of the Salvation Army and always religious. At 22 she was married. Her first child died of hydrocephalus at 6 months. Following her second labor she had post-partem hemorrhage and convulsions. She talked of religion, was emotional, restless and acted queerly, but grew better and assisted her husband in his business as florist, but convulsions again suddenly supervened and she enacted the death-bed scenes of a lost friend, called in her family to save her, and sang hymns. She was 12 days in the Post-Graduate Hospital, then was kept home until two months before her admission to Middletown.

When admitted she was a slightly built, poorly nourished woman who complained of dull headaches, a band-like sensation about her head, ringing in her ears and insomnia. She required urging to take food. She sat in bed wringing her hands or got up and walked about in agitation. She cried easily and could easily be made to smile; simply to let her tell of her troubles being often enough. She worried about her soul being lost because of things she had done, e.g., religious duties neglected but on telling about it, kept wandering on

to some other instances and back again. Questions about going home, seeing her mother, her husband and especially her pastor were reiterated by the hour, though already answered over and over. Yet her speech was coherent and relevant. She was always correctly oriented and had a good grasp of the situation. She had no hallucinations or illusions in contrast to Case No. 4, was not suspicious, but rather trusting and confiding. She was not retarded.

After three months she improved materially, became quiet and industrious, and in five months was paroled home. At the end of 30 days her husband wrote that she was as well as she had ever been.

The following case, No. 9, was observed in the wards of the Manhattan State Hospital through the courtesy of Dr. E. C. Dent and Dr. Geo. Campbell, and the following account is abstracted from the hospital records.

Case No. 9, a housewife, aged 34, was born in New York of Swedish parents. Her mother drank immoderately and separated from patient's father years ago. She had ten children, one at a time, in twelve years. Only three were alive at time of separation. One of these three, a brother of 26, is dissolute, irritable, feeble and childish. A sister has pulmonary trouble and hysterical attacks. A maternal aunt was confined as insane.

Personal History.—The patient, the oldest of ten children, was always excitable, walked in her sleep and had terrifying dreams. She obtained an ordinary education, then worked as a servant, was industrious, economical, efficient, well liked, made and retained friends. At 21 she married a good husband but had no children. She complained of uterine trouble. Her husband says she has been nervous and had irritable spells and hysterical attacks of shouting and screaming for eleven years.

The Psychosis.—Two years ago she heard a favorite niece had gone astray and suddenly fainted. For four weeks she was alternately in bed and about. Talked irrationally and brooded continually over the disgrace. When she took up her work again was irritable and quarrelsome, would fall into a faint and become rigid; she never hurt herself by falling. The attacks were preceded by headache and followed by passage of a large amount of urine.

About April she began to take care of a sick woman who died October 31, 1904. She fainted when her patient died and had to be carried to her bed. When she awoke she acted as if fearful; thought the house would be set on fire; thought she would be shot. At night she had attacks of screaming, cried and at times became rigid. Usually an attack was aborted if she had a drink of water. She became quiet and said the water relieved her heart.

The family of the dead woman had family trouble. She imagined she was to be subjected to the same, and in her ravings expressed those fears. She could not sleep, but ate well.

After persuasion she went to Bellevue November 10, 1904, and from there was sent to the Manhattan State Hospital West. Upon admission she was nervous, suspicious and troubled. At first was compliant, intreating the nurse to take good care of her. Soon after admission became tearful, showed considerable fear, begged God to have mercy upon her, insisted that she was going to be killed:—"Oh, doctor! Oh, I know that I am going to be killed, and I know you are going to kill me, and you are doing all my thinking for me, doctor—doctor (whining) Oh-Oh-Oh! (wrings hands) Oh, my God!" etc.

Orientation was correct, memory of the recent and remote past was good and her grasp of school and general knowledge corresponded with her education, but in calculation tests begged "I am trying to think—give me a little time." She read and remembered well. Nervous tremor interfered with her writing.

Her hysterical stigmata were sensitiveness beneath the breasts and over the abdomen (not constant); a sense of suffocation, as if chloroform and steam went through her body; temporary anesthesias of the cornæ and of the fauces and reduction of the visual fields to a point.

A few days later was doing fancy work very well. Nov. 15, slept only four hours last night; said she thought she had been given poison; that she was hypnotized and compelled to say certain things and do certain things; complained of a bad smell, iodoform, on her clothing; called peculiar motions which she made with her hands "resistances" in order to free herself from the coma brought on her by the odors. She seeks sympathy.

November 16, restless during the night. Tearful, feels nervous, accused the other patients of threatening her. Crying, cowering, shivering this morning, complaining of having to say foolish things. Fretful and peevish.

November 19, restless, tearful, and nervous, cornæ insensitive, also membranes of the throat. Breasts hyperesthetic and painful.

November 21, makes peculiar motions, insisting she was under an influence. Great emotional variability.

November 25 she was quiet and less emotional. Improvement continued and hysterical stigmata disappeared. On December 3 they had reappeared. On this date she explained the emotions she found herself going through when she awoke from her dream at night, as calisthenics, saying she

had been reading in the papers about physical culture. She had previously said that a Mrs. Meyer had killed her. She does not remember saying such a thing. "If I said that I must have been in a sort of a dream." She cried easily on questions, for instance about her mother, who was dissolute, and was easily consoled but not to the extent of being positively happy. Up to the end of the course at the Pathological Institute on December 23, 1904, when she was last seen, she remained fretful, easily discouraged, uneasy, working at fancy work and doing it well, but fitful in her application. In everything she disclosed a lack of the balance which comes with internal calm. She was discharged recovered in April, 1905, after her fitfulness had disappeared.

Summary.—A woman of 34 years who for eleven years exhibited nervousness and hysterical attacks of crying and screaming. Four years ago her condition was aggravated by worry over a niece's wayward conduct. About two weeks before admission when a woman she had cared for died, she fainted and awoke in a frightened delirium. After admission she was oriented, natural in her reactions to her environment in most particulars, and begged for good care. Later she feared that she would be killed, thought she was hypnotized, made to say things against her will. She said that she had been given poison, that there was a smell of iodoform in her clothing and made peculiar motions, "resistances," against the odors. If asked to calculate or do anything requiring thought there was a sense of insufficiency.

The last case of this series is one of the older cases and the account of its onset is fragmentary. Very distinct hysterical symptoms were noted during her stay.

Case 10 was admitted to the Middletown State Hospital June 13, 1901, aged 35 years.

She was married and the mother of two living children. Family trouble was assigned as the cause of her psychosis, which was said to have begun about May 1. The earliest symptoms mentioned were on June 8, when she was "taken violently insane." She tried to set fire to the house and tried to commit suicide, and was restrained by calling in the police. She was removed to a hospital where she required constant watching. To the medical examiner she said she had committed the unpardonable sin, could not get well and that suicide was the easiest relief. She was nervous and had a shrinking and frightened look. When admitted her speech and actions were just as when examined. Her face and body were covered with erosions where she had picked sores. At night she was very restless and wandered about the ward with a woebegone expression on her face and wringing her hands. She cried from fear that she was to be scalded to

death, moaned because she could not go home, and because of restlessness and self-mutilation was for a time kept in a waist. "Very restless" is a note that recurs continually with "depressing delusions and great apprehension." She would harp on one thing and ask the same question a hundred times a day. On one occasion she picked her neck with a hairpin until it bled, with suicidal intent. A year after admission she had nausea and vomiting, followed by fainting and convulsive attacks distinctly hysterical, more pronounced when observed, complained that all was black before her eyes, opisthotonos and after several hours convulsions, she came out and jested with one of the physicians. The attack was followed by the passage of much pale urine. In July there was another convulsion and in August it is noted that she "yells until she is hoarse." "Exposes her person shamelessly, soils the bed, sometimes purposely. Well nourished. Incorrigible." She became very untruthful, made many flimsy efforts at deception, was penitent when detected, but without stability in attempts to do better. Some days she sang and some days she cried so loudly as to disturb the ward. The next summer she was quieter and mutilated herself less.

October 31, 1903, after two and a quarter years, she was transferred to the Hudson River State Hospital. She was observed at a time when the present investigation of hysterical insanities had not been conceived and many hysterical traits probably present were not sought. Just before her transfer, however, when she was quiet and clean and much pleased with the prospect of a change her visual fields were examined and found contracted to less than 20 degrees, though by artifices the fields could be extended to 35 degrees. At the Hudson River State Hospital she improved for a time, then was not so well. Was transferred to a private sanitarium and finally returned home where her friends since report she is entirely recovered.

In reviewing these cases of the distinctly hysterical group, varying widely in their particulars, many points of fundamental similarity are found. Several of them belong to nervous families and where the details are followed up, especially families some of whose other members have shown hysterical features. They are persons who seem to have been fairly up to their family levels before the onset of the hysteria, or at least they were not markedly inferior individuals.

The prominence of emotional disturbances in precipitating the attacks is especially noteworthy; an abortion, an accident in which the patient might have been hurt; mental stress over a newly consummated marriage; an attempt at seduc-

tion; religious excitement; the disgrace and imprisonment of a lover; a childbirth, and a death figure as exciting causes; and bear a very distinct relation either to the original onset or the onset of particular exacerbations.

In all these cases at intervals or in some subjects the manner is quite natural and things are correctly apprehended. Many matters are viewed from the sane viewpoint and appreciated at their true worth. The psychosis is not fantastic, absurd or disconnected in the same sense as in dementia præcox. A patient may talk of suicide; destroy her clothing; tear her hair or throw herself on the floor, but these acts are the natural outcome of the emotions then present in consciousness. Her actions are controlled by ideas that have become dominant, but as soon as her self-control is regained, when her personality re-establishes its ascendency over the interrupting mental state, she looks upon such acts as does any other observer, or she tries to defend her conduct on rational grounds.

On the other hand the symptoms are of wide variety and may be of extreme violence. It is a mistake to think that because a patient is hysterical that all the mental symptoms are shallow and ephemeral and scarcely are real at all. A hysteric often acts under profound emotion and may do any rash or violent act that profound emotion suggests, as for example, Case No. 5, in the present series, took with suicidal intent all her sleeping powders at once, and recovered because the total dose was not lethal rather than because her suicidal act was consciously inadequate. More or less determined and entirely real efforts at suicide are decidedly common.

The hysterics forget what they did during their attacks completely in most instances, sometimes there is a partial remembrance left as in some attacks of cases 1, 5 and 7. This forgetfulness is not characteristic, however, for epileptics forget their attacks, and active manic states and febrile and exhaustion deliriums leave but a fragmentary recollection.

The symptoms in all these cases are of a decidedly episode character, much more so than in the other insanities commonly seen in a public insane hospital. The symptoms vary greatly from day to day, and while the patient seems most wrapped up in some delusional or hallucinatory experiences she yet, if her attention is distracted to something else, may

talk very well about the new topic. The mental symptoms themselves too vary greatly from day to day, perhaps with more or less complete alteration of personality, or as in most instances the patient remembers who and where she is but has varying unexplainable beliefs cropping up in her consciousness. It is noticed that as the cases become more chronic these seem to become more stable and in those under long observation the same old ideas insistently recur in the same form.

When the psychosis is most active the patients are sometimes dazed; do not know where they are or place themselves in scenes altogether foreign to their actual surroundings, but in the clearer phases they become oriented, have insight, realize that something has been wrong mentally and often insist then or at all times that they are crazy. Very uniformly they appreciate that there is some change in themselves and complain of it. One almost universal complaint which does not lend itself well to presentation in the abstracts of the cases, is a sense of inability to do mental or physical work. Janet has a chapter on abulia, which he believes makes one of the stigmata of hysteria, and he describes cases presenting to an extreme degree a feeling of difficulty in accomplishing anything. The patients herewith presented could calculate but complain that it is hard to do so, and do not like to set themselves to this or any other task. All these cases, however, present spontaneous movements that are free and active and usually lively. Uniform motor retardation is absent and there is, except in some cases to be quoted later, no hint of motor retardation at all. Dr. August Hoch teaches that in the mildest cases of manic-depressive insanity, "We may have only a slight retardation of voluntary efforts which manifests itself in a feeling of inadequacy and an inability to decide or accomplish anything for which decided effort is required; while emotional depression may be absent, or on the other hand so pronounced as to hide the other alterations." This exactly describes the state of many hysterics. They stand or sit about unemployed, vaguely depressed and unable to apply their minds to tasks, while at intervals this apparent retardation of thought and motion is swept aside and submerged by anxiety and fear.

There are cases of this character without hysterical stigmata on the one hand, and without manic-depressive alteration on the other, whose exact relationships cannot yet be satisfactorily determined.

No boisterous hysteric has been observed whose symptoms would be confused with the manic phase of manic-depressive insanity unless Case No. 22, to be added later, be so considered. Some scene is lived through; contortions are intense and from internal motives; not the busy, easily diverted manic activity governed by trivial events in the immediate vicinity of the patient.

Hallucinations are a prominent symptom; much more so than in mania, and hysterics seem especially prone to combined hallucinations involving two or more senses at once.

Most of these cases exhibited fear and anxiety, either continuous or episodic, and they are arranged progressively according to the prominence of this feature. This fear is very great, even fear for their lives; it is in several cases persistent and the most prominent feature of the psychosis. Anyone considering in detail the anxious symptoms of these hysterics must be struck by their close correspondence to those of the involutional depressions. In both conditions the patients sleep poorly, worry, have somatic complaints and allopsychic fears.

Involutional depressions do not constitute a homogeneous group. Kraepelin has separated from his melancholia only toxic depressions and those which are really equivalents of manic-depressive insanity. Certain of the remaining cases deteriorate in a way strongly suggesting a late dementia præcox and this suggestion is reinforced in some instances by finding a depression at the involutional period in one generation of a family and a dementia præcox in the next. Another group of involutional depressions is that associated with arteriosclerosis and high blood pressure, and usually (as in hysterics) precipitated by adequate moral causes. It is this group that figures especially as involutional melancholia.

It is quite a common experience, too, to find in depressions after 40 that do not present the features of manic-depressive insanity and in which the manic-depressive temperament is not in evidence; that is to say in anxious depressions with and without increased vascular tension that have been considered as examples of involutional melancholia; that there has been

a previous attack of depression in early life usually in connection with some mental shock. This has occurred 9 times among the 50 women admitted as melancholia, and once among the 17 men so classified between January 1, 1903, and April 1, 1906, in the Middletown State Homeopathic Hospital. Some of these cases are at least open to the suspicion of being hysterical. In considering the relation of hysteria to the melancholias of the declining period the relative frequency of the latter in women may be significant. As already noted the preceding cases have been arranged to show how the every day symptoms of hysteria lead over into this complex of agitated anxious depression and to indicate that we might expect at least some melancholias to be hysterias and the first case of what we may call the involutional group of hysterias shows that it is so.

THE ANXIOUS TYPE OF HYSTERICAL INSANITY

Case No. 11 was admitted July 29, 1904, aged 43. Her maternal grandmother was "weak like," "worried much and cried easily." Her mother, aged 68, was "quite sickly" in her younger days, but lately has been healthy. She has had much trouble, but "stood up under it all" and was not especially nervous.

The patient, the youngest of five children, was a healthy girl, received little education, married at 17, had 9 children, all of whom are healthy and rugged, and two miscarriages. She was of cheerful disposition, slow to anger, of good habits; always handled the family purse and paid the bills. For three years she had a hard struggle to get along, worked unusually hard and at admission weighed only 85 pounds.

Her trouble began with a fainting attack about April 15, 1904. Three days later she began to think she was to be arrested, put in the electric chair; seemed weak, tried to work, but could not. She walked the house at night, wanted to go out of doors; was in constant fear that the constable was coming for her; especially if a wagon passed the house looked worried; refused the food she was given, but ate food surreptitiously obtained. During April she stole out of bed at night and ran 400 yards to a mine shaft. In fifteen minutes she returned wet from head to foot and told her husband she intended to drown herself. From that time she required constant watching to keep her at home.

When examined after admission the same fears developed. Restlessness was not very pronounced and some of the staff thought they saw signs of retardation in her speech and activities, but this was slight and intermittent.

The high tension of an involutional melancholia was not found; she did not give very full accounts of her recent or remote past, yet had many incidents at her command. She slept little, repeated "Oh, dear" over and over, complained that her head hurt all over, that she was wicked and had committed bad sins, and made such spontaneous remarks as "I suppose you will leave me, won't you?" She was not entirely without insight, something often observed in hysterical cases, and said that she was a little off her base.

The diagnosis was not clear, manic-depressive insanity, involutional melancholia, and a condition allied to melancholia, were all considered and the last was as near as the case could be definitely placed. One symptom of the original examination failed to receive the consideration it should have received; that is, hypoesthesia of the calves and the infra-mammary region, which indicates that hysteria should even then have been considered.

During the ensuing months she changed little, was depressed and inactive, said she had killed seven people, wept a great deal periodically, thought her bowels filled up and that she could not eat; that she had poisoned her little children one by one with mine-hole water with a rattlesnake in it. She saw her daughter at night "just as natural as you and all covered with blood," and wept profusely as she told about it. One night she was much disturbed because she thought one of her children told her to be prepared for two men who were coming to take her away, and another night she saw one of her sons in brass buttons at the foot of her bed. He wanted her to put down eleven letters on a sheet of paper, evidently a combined hallucination.

This was the status of her case until Feb. 20, 1905, when one morning she complained that her left arm was numb and that she could use it but little. It was found that her knee-jerks were equally increased, that her pupils were equal and their reactions normal, and that there was no paraiysis of the face; but that the whole left side of the body was insensitive to pin-pricks. The anesthetic hand was livid and cold and she said that she could not move it, and made only very feeble efforts to do so, but was able to use considerably more power in resisting passive motions when their continuation for a short time became unpleasant to her. Two days later she spoke only in a whisper, was blind in the left eye and could not hear with the left ear. These symptoms had come on with a severe headache during the night. The hemianesthesia was complete, except about the ala nasæ, including the mucous membrane of the mouth and half of the tongue. Her visual field on the right was by rough tests contracted to about thirty degrees. The paresis of the hand was rather more pro-

nounced, she walked slowly but without limp. If, with her eyes covered, her left hand was displaced from its usual position at her side, she was unable to find it.

The next day sight had so far improved in the left eye that with it she was able to follow a moving object but not count fingers, as they seemed to have a "mist" over them. The anesthesia had changed to the stocking and glove type, reaching on the leg to just above the knee and on the arm to just above the elbow, with hypoalgesia over the remainder of the left side. Tactile, pain, temperature, and pressure sensations were lost over the anesthetic area. Feb. 25th, two days later, hemianesthesia had returned. After equal pressure on the backs of the two hands, the color returns to the right hand in six seconds, and to the left in twelve seconds.

Within the next two weeks there were times when the anesthesia disappeared, but after a sleepless night it returned. Now it is gone again, but she is depressed and rather worried. She thought her sons when they visited her did not look like her boys. Her coming here she says she does not remember, and she recalls no incidents of her stay here until Christmas when she was transferred to the ward where she has since been. She denies too ever having been in a mine-hole, or thinking that she had poisoned her family; such things could not be; but she says instead that she drank some whiskey as red as blood, and that then her memory was lost until last Christmas.

Summary—A woman of 43 with nine healthy children. Her trouble began with a fainting attack followed by allopsychic fears, refusal of food, sleeplessness, restlessness, and probably a suicidal attempt. The arterial tension was not high. At the hospital some hypochondriacal ideas developed, and at night, combined hallucinations, perhaps in connection with dreams; and six months after admission the classical syndrome of hysterical hemianesthesia developed.

The following case, a man, more nearly approximates the type of the worrying depression so commonly seen especially in women between 35 and 60 years of age. Definite stigmata of hysteria are not present, yet there are symptoms approximating them, and the individual symtoms as they develop seem to arise very largely on a mental basis, as if they were self-suggested; to rise as do the complaints of so many so-called nervous persons who suffer from what they think is the matter until something distracts their attention from themselves. The stress that he has laid on his dreams is noteworthy too in this connection. We do not have facts sufficient to let us definitely place such a case as this, but perhaps it may be usefully introduced as in the borderland of the group and at least as an indication of the further study required by such cases.

Case No. 12, a man aged 47, was admitted Feb. 18, 1901. A country laborer, temperate, married and with grown up children. His attack was of rapid onset and came on within the previous month. It was ascribed to la grippe. Insane heredity was denied. His father died of an epileptic fit or heart trouble. At home he talked of having done wrong, of killing himself and putting his family out of the way. At the hospital he thought it a criminal offence that three times in a year he had served on a jury and that on account of it the schools and churches throughout the country were closed and the ice harvest on the Hudson River stopped.

He did not think it worth while to eat, and the feeding tube was necessary for nearly two weeks. He made such statements as that he should be in State's prison, that his friends are against him and brought liquor here to "condemn" him and show people here that he is a drunkard; he did not see the liquor but "knows" it was brought.

The West Shore Railroad trains were all stopped on his account; he went about deeply dejected; wanted to die and asked the barber to cut his jugular vein. He talked of his wife and family being in the building. After a year he was better, worked out of doors, and in July went home, but no sooner reached home than he wanted to come back, to be sent to jail, talked of the wrong he had done, slept little, and would not leave the house. In five days he was back at the hospital, just as he was before he went away. He worked in the laundry, and when not at work sat in his room with an air of settled melancholy whining if he might not come home or if his folks were heard from.

He complained of sore feeling all over, headache and sparks before his eyes. Told often of waking up at night frightened, hearing his mother call him, then he would see her. "As plain as if she were living and right by me, dressed in black the same as she wore for a long time."

He gnawed his knuckles until they became calloused; bit his nails and picked his fingers sore.

In the summer of 1904 he swayed in walking and especially with his eyes closed. His gait was shuffling, the toes first striking the floor. Sometimes the left leg seemed weaker and sometimes the right; he gave the impression of not trying very hard and moved the legs well in bed. The legs felt numb, but no sensory tests are recorded, but in the fall he gradually walked normally.

In 1905 he complained that he was weak and that the left leg was weaker than the right. He had no grip, or at least would exert none, and his arms, which bear fair muscles, put forth no strength. His deep reflexes were all increased, and more active jerks followed striking the muscle bellies than on striking the tendons. One day he stated that

he felt no pain from pin-pricks even deep enough to draw blood, and was unable to tell the head from the point of the pin, at another time he told the head from the point and said he felt the pricks as such, or felt them over a given area, and a few minutes later did not feel them. Touch sensation was preserved and he could, with both eyes closed, place either hand in a position the other was passively made to assume. An examination of his visual fields showed an irregular contraction varying at different stages of the examination and coming, on the whole, to be narrower as the examination progressed. Since then the contraction of the visual fields has disappeared, to return again after a trial at home which did not succeed because of the prompt return of worry and sleeplessness at home.

Summary—A melancholy man with much self-accusation, and admitted in 1901 at 47. He was sleepless at night and troubled with vivid recurring dreams. He has presented hypoalgesia of the skin, and contraction of the visual fields of the neurasthenic type increasing with the length of the examination; and at an earlier stage what appeared to be a hysteric paralysis which prevented walking.

Regarding the possibility of certain obscure involutional depressions being hysteric, we may mention the case of a man, aged 62, who, without signs of local irritation, believed he had bugs in his skin; insisted that his physician had told him so and that it was a fact. He also believed his dandruff turned to microbes. In the course of a few weeks he thought the condition grew better and finally said they were gone, though even then he stoutly maintained they had been present. Since his discharge five years ago he has remained well. Hysteria did not come into consideration at the time, but has been suggested by the subsequent admission of his daughter with a hypochondriacal hysterical depression.

Case No. 13 was during her stay at the hospital perfectly oriented with good mental grasp and retention of her stock of knowledge, but not spontaneously very active mentally or physically. She complained of weakness, pain in the abdomen, the side, and the neck; of headache and backache, of a lump in the throat, and especially of photophobia. She stoutly maintained that she could not go out walking until two men were detailed to help her down the stairs, when she sprvly made the trip without this assistance. Long-standing inability to bear light was relieved for purposes of examination by a drop of water in each eye and then continuously by plane lenses of clear glass which improved her vision from 15—60 to 15—20. Her visual fields at the first examination normal; were subsequently invariably found contracted usually from 20 to 40 degrees. Skin anesthesia has not been found.

She was always willing to talk at any length about her pains, and smiled contentedly while so doing. She employed herself very little; complained that she could not, but occasionally had a day when she worked intelligently and well. Ordinarily she was content to spend her days if possible in bed.

She began in the spring of 1903 with worry over a dissolute husband who failed to provide for her and her two children. This passed away until the spring of 1904, when she began to worry about herself and her physician performed an operation on her for a lacerated cervix, but without relief. She went to the poorhouse; then to live with a sister, where though fat and rosy, she bored the family with her complaints, and finally threatened suicide, her friends think to frighten them. She was admitted to the Middletown State Hospital August 2, 1904, and discharged fourteen months later, improved.

Summary—A hypochondriacal depression in a hysterical young woman whose father four years before recovered from a short attack of mental disease, the principal symptom of which was a hypochondriacal delusion that vermin were boring under his skin.

Reference has already been made to cases symptomatically considered Involutional Melancholia, but which had in early life depressions of a similar character. Two of these cases follow in brief abstract. In both the friends say her previous attack was "like the present one, only not so bad." They say nothing of slowness, unwillingness to talk and quiet depression, but talk of restlessness, sleeplessness, lamentations and self-mutilation instead. Kraepelin describes such a psychosis only as an involutional symptom complex, but it does not, judging from our Middletown histories, seem very uncommon in youth. When attention was first directed to these cases we inclined to consider them as anomalous cases of manic-depressive insanity, thus tacitly assuming that manic-depressive insanity, perhaps with allied conditions, is the only recoverable insanity except those mentioned above. Longer acquaintance, however, seems now to make this view untenable, the cases present no tendency to manic alternations and we can instance no case with manic alternation presenting depression of the same character as these cases present. It appears they must be separated from the manic-depressive group, and because their symptoms remind us of those we have seen in hysterical cases, they are presented here.

Case No. 14, a single woman, aged 42, was admitted at the Middletown State Hospital May 9, 1904. She is of a good Norwegian family free from other insane members except a sister who at 20 had an attack of depression with cessation

of the menses when she was very nervous and excited, but recovered after ten weeks in a sanitarium. The sister remains well.

The patient was handsome, bright and clever. She had many admirers but never married because she had an attack of insanity shortly after her mother's death. She could not sleep, wanted to die, made several suicidal attempts, picked at her finger nails and cried a great deal. She was taken to the country and recovered in seven months without medicine, and afterward led a gay social life, was quite a coquette, came to this country, made good wages in a fashionable dressmaking establishment, was efficient, of sweet disposition, and unusually well liked by everyone, but considered the member of the family least fitted to work for a living.

In the summer of 1903, being in poor health and troubled with "nervous dyspepsia," she tried going away with a nervous patient, but it wore on her own nerves. In August a favorite brother died, and she grieved over his death, and during the fall became gradually depressed until she could think of nothing but her misery. The climacteric was approaching with irregular menstruation; she was unable to banish sexual thoughts from her mind and reproached herself accordingly. Six weeks sanitarium treatment made her no better and she was committed.

Upon admission she was restless and agitated, moaned much and walked the floor, said she would be chained to the wall and spanked until the blood came. Here the agitation and depression continued though blood pressure did not seem raised. She was oriented, conscientious, and the speech clear and coherent. She reproached herself; said that she had by lack of self-control brought on her own trouble and believed herself the cause of all the sickness in the sanitarium where she had been. Her memory was good except that she could not tell the time when things happened and she was twelve days wrong in the day of the month. She slept poorly, pricked her skin sore and bit her nails. The change in herself she was at pains to observe in the mirror and deplore, and she fingered taste solution bottles to ask if they contained anything to end her misery. Hysterical stigmata were not found. She sometimes spoke of being confused and was not able to do calculations, and these were seized upon as evidences of retardation, but in the general picture they are minor features. A continuous restless, apprehensive depression is in the foreground. She occupied herself whenever self-control was sufficient, in reading and in beautiful fancy work, but lamented that she could not do such work as she had formerly done. In April, 1905, after a sleepless night, there was an episode of fear and apprehension. She walked about

wringing her hands, cried out in distress, and begged the nurse not to allow anyone to injure her. She looked about the room with frightened, dazed expression, said there were snakes crawling over her bed and around her neck, and begged to have them taken away. She said she had "such a rapid sweep of thought" and that she could not control herself. For a month afterward her depression was aggravated but without further hallucinations and she was but little improved when paroled home in June, 1905, and a few weeks later sent to a sanitarium in the country.

Summary—A single woman of 42, of good mental capacity. At 24 she had an agitated suicidal depression treated at home, then remained well until the fall of 1903, when she began to worry over real trouble, lost her self-control, became restless, anxious and afraid, slept poorly and continued in this condition while under observation, more than 18 months after the onset.

Case 15 is a woman of 41. She has been married nine years. Her family history is good except that a paternal cousin is insane. She has always been considered one of the brightest and most ambitious members of the family, but of dissatisfied temperament. Active and nervous. Her fingers are disfigured from long biting her finger nails.

When about 21 she had an attack of mental trouble over a love affair. She thought she was pregnant, walked the floor all night long, could not sleep, at times would start in to sing and whistle, and in a few minutes from that time would cry. In about three months she recovered without treatment and has been mentally well since. For about one year before the present attack, following measles, her bodily health was poor, and she was dissatisfied with everything, became depressed, and talked of killing her child to save it from a worse fate. When admitted November 9, 1903, she was poorly nourished, constipated, pulse 100, tension rather high as estimated by the finger. She has a goitre. She was oriented with a good grasp of her surroundings and a good memory, but too preoccupied to try to recall unless urged. Dejection and despair were her prevailing moods. She bit her nails. She was not retarded but talked constantly, was restless and did not want to apply herself to thinking tests. She was in great mental distress, she said because one of her ovaries burst during copulation twelve years ago and that she is not right and her child is not right on this account. Some terrible fate hangs over them because of her sins and they will be burned to a crisp for her wickedness. She is not man and not a woman, her bowels are wrong side out and her eyes starting from her head. She must expiate not only her own wrong but the sins of the world, and then she will be as great

as Christ. There was also some feeble Nihilistic belief that she and her child are not real.

Under observation she is able to help and part of the time helps well with work about the hospital. She does it willingly, but in the intervals walks the floor despairingly, crying out the same self-accusations and that nothing can help her. At one time she had all her hair picked out, which seemed to give her no particular pain. Sleep throughout the illness has been poor.

Summary—A married woman of 41. At 21 after sexual indiscretions, had a restless, sleepless depression with emotional variability. Recovered at home in three months. The present attack came on during physical ill health. She was admitted in November, 1903, extremely agitated with delusions of abdominal somatic changes; of sin and that herself and child were to be destroyed. She has not materially improved and at one time picked all her hair from her scalp.

These two cases, No. 14 and No. 15, were treated at home and only a hearsay account of their early psychosis is available. Probably, as such patients remain oriented, coherent, and realize their own condition, they are not as a rule considered insane and are kept at home. One case, however, apparently of this character, was observed on the wards of the Manhattan State Hospital West, while in attendance at the New York State Pathological Institute.

Case No. 16 is an intelligent young woman of about 21, who has been employed as an assistant bookkeeper in a department store. She was a religious girl, punctual in everything which she thought her duty; with little taste for the society of young men, she said, because her young friends could scarcely support themselves. For many years she was troubled by an offensive breath due to nasal catarrh; a source of mortification to her, increased at times by the complaints of those who worked with her. No mental symptoms can be traced to this cause but in the summer of 1904 she began to have pain in her eyes; had glasses fitted with little relief, and in September one day heard a woman say that anyone with specks in his eyes would go blind. She had been regularly at work until this day. That night she came home, spent much time looking in the glass for specks in her eyes, cried all night, and for several days afterward, and has not since been able to go to work.

At Bellevue she was intensely depressed and apprehensive, said she could not talk or think, said that her throat was grown together. She had unspecified persecutory and self-condemnatory delusions, talked slowly and with apparent effort, and was quite emotional.

When admitted, November 26, 1904, she was found pale,

Hb. 70 to 80. Heart apex, 10 cm. from mid line, but no murmur. Chronic naso-pharyngeal catarrh, occipital headaches, possibly slight contraction of the visual fields; left Achilles jerk and plantar reflex diminished. She slept poorly. She was compliant in bathing and dressing and eager to talk about herself.

She says: "Can't I have an operation on the brain? You know I tried to choke myself and I don't remember the days any more or the years or nothing. . . . Every day I don't remember the day. . . . seems like eternity. . . . I ain't afraid of anybody—generally you know you are afraid of your superiors—don't you think the operation would be all right? . . . . I know I will be crazy all my life if I don't get an operation. . . . . You know you should have feelings for people. . . . you feel sorry for people when they have trouble—when there is a death you sympathize with them but I have no feeling now."

Again says: "I can't get my senses back, can I? I have to stay here all my life—I beat my breast" (illustrates). She choked herself and beat her head and wants to be operated on as the tubes have been forced up into her head, or killed because there is no hope for her. These are delivered quite calmly and with normal rapidity. Reiterates the same thing over and over. Her motions are free. With the progress of the examination she nervously works her feet up and down in bed, looks ready to cry but sheds no tears, and says in a whining voice: "Oh, mamma—mamma—oh, mamma, why did I do that—why wasn't I like other girls; work for you. . . oh, mamma—I broke my mamma's heart—I can't be of any use any more." She was correctly oriented as to place and persons. Soon after admission gave a good account of the admission, but two days later could not say when she came or give any recent dates. Her data of personal identification were unimpaired, and retention was excellent for things she was charged to remember. Her school knowledge corresponded to her education—100—7 to 0 test done in sixty seconds with one mistake, 47 for 37, and finished with "Do you know, my brain is all gone—do you know through choking myself I caused it?" Multiplication was done more rapidly than by the examiner. She says that she is crazy, she cannot recover, that her mind is a blank, and illustrates by saying that when she thinks of 26th Street she does not see 26th Street—"I see nothing." Since her admission there is no change in these symptoms, except that she is more calm and she sleeps well.

Diagnosis—No evidences of the lack of interest or the mental disorganization of dementia præcox are present. Her somatic delusions—she persistently refers to an actual cause—her attempts to choke herself or beat out her heart. She un-

derstands all that is said to her and is invariably coherent and correctly adapted to her surroundings, except as these adjustments are interfered with by anxiety and lamentations. Her depression in no way resembles manic-depressive insanity, except in her feeling that she cannot work and is of no further use. It is associated in her mind with a few specific casual relations, *i.e.*, her choking herself because of poor eyes and the alleged bad effect of her suicidal attempts. She spontaneously emits a continuous flow of recurrent lamentations when anyone will listen, complains because the nurses make her be quiet in bed, and shows no retardation in thinking tests.

Her anxiety has many of the characteristics of the involutional anxiety psychosis, but lacks high blood pressure, 115 Gaertner's Tonometer. It has, moreover, the characteristics we have seen in hysterical anxiety states: Clear consciousness with at best normal reaction to the surroundings; harping again and again on specific complaints, and utter failure of reason to influence the stream of thought; a feeling of an emotional change; the loss of memory of certain phases of recent events, which loss she appreciates and magnifies; and occupation with and talk about her own feelings, are entirely in harmony with an hysterical disturbance. Only slight evidence of stigmata have been found, possible contraction of visual fields and a very feeble pharyngeal reflex.

This patient was observed in the ward of the New York State Pathological Institute at the Manhattan State Hospital, and Dr. G. H. Kirby, who has followed the case, says she improved enough to go home, but is in feeble health and still inclines to think that there is something wrong with her throat and brain.

Summary—An anxious depression in a girl of 21 with ideas of bodily change. Slight and uncertain traces only of hysterical stigmata. She is without mental failure or definite indications of manic-depressive insanity.

We are uncertain as to just how these cases should be regarded and they are contributed less to advance any final theory as to their nature than to show that anxious depression is not by any means limited to what may be called involutional changes. One of these cases, and it happens not a recurrent one, after months of non-characteristic symptoms in a continuous mental disorder developed symptoms clearly due to dissociation of hysterical type. Other cases with attacks in early life following mental and physical distress show depression very similar to that seen in hysterical cases.

We have not been able to point out the dissociations here but the causes and symptoms are so uniform in all that we must suspect the mental mechanism for the production of all these anxieties to be closely similar. Similar causes and similar results arouse similar mental processes in both conditions, and we think that the facts warrant careful scrutiny of all anxiety psychoses for hysterical traits and that the recurrent cases, especially those with attacks in early life, may be tentatively thought of in the hysteria group.

(To be continued.)

AMERICAN NEUROLOGICAL ASSOCIATION.

Held in Washington, May 7, 8 and 9, 1907.

The President, Dr. Hugh T. Patrick, in the Chair.

(*Continued from page 776, 1907.*)

Traumatic Lesion of the Pons and Tegmentum with Direct and Retrograde Degeneration of the Median Fillet and Pyramid, and of the Homolateral Olive. By Dr. Adolf Meyer. (*See this journal*, 1907, p. 699.)

Dr. W. G. Spiller said he was exceedingly interested in Dr. Meyer's report, and while he has the greatest admiration for Dr. Meyer's work, he is somewhat in doubt as to the practical value of glass reconstruction, particularly as to whether it is of great benefit to the undergraduate student or not. Those who teach undergraduate students know how hard it is for students to obtain a knowledge of the finer anatomy of the central nervous system. Dr. Spiller believes that before any student can grasp what Dr. Meyer had presented, it is necessary for him to make microscopical sections of many different portions of the brain, and to study them carefully, as well as the region of the brain from which the sections are taken. When he has done that he will not need the glass reproductions.

As for the separate slides which Dr. Meyer showed, Dr. Spiller thinks that the actual sections showing the lesions are more valuable for demonstrations than drawings made from them. In his lectures on neuropathology Dr. Spiller uses the microscopical slides in the lantern. As for putting the glass slides together afterward, as Dr. Meyer does, this might be of assistance to a student who had done some work in neuropathology and was therefore able to enter into the details of Dr. Meyer's pathological findings.

Dr. Spiller said he had been especially interested in Dr. Meyer's description of the median fillet, and it happens that he has been examining recently a case in which the median fillet was destroyed by a lesion in the lower part of the medulla oblongata, and the degeneration extended from that point as high as the level of the red nuclei.

Dr. H. H. Donaldson said he was sorry to differ with Dr. Spiller on this point, but he thought one should emphasize the idea which Dr. Meyer had brought forward, that by use of these drawings, either on paper or upon glass, it is possible to differentiate before the student those portions of the section concerning which we may make moderately complete and accurate statements, and draw attention, by contrast, to the parts of the section which contain structures that are still in need of interpretation.

As regards the glass models, Dr. Donaldson said that his experience with classes had been that the most difficult thing for the student to get was the three dimensional relation of the parts, and these glass models were particularly well suited to give the three dimensional picture, which was better than the projection on the screen would indicate.

Dr. A. R. Allen said he considered the chief benefit to be gained by this glass reconstruction was that it gave the student a much better

geometrical conception—the third dimension concept—of the architecture of the brain than any other method of illustration.

Dr. B. Onuf said he could only voice Dr. Donaldson's and Dr. Allen's sentiments as to the value of this method. He thought that for plasticity, for showing the special relation of fiber tracts, it has an extreme merit. To give an illustration, he said he believes it happens to many of us to have only a hazy view of the spatial relation between the fillet and the superior cerebellar peduncle, and the relations of the three cerebellar pedunceles to each other, and he thought all these things cannot properly be brought out by wax models.

Dr. C. K. Mills said he could only join with the others in commending and approving of the work of Dr. Meyer, and would attach value to this method, although perhaps not fully capable of appreciating it. He said there was one thing he would like to call attention to. Dr. Meyer has spoken (and it seems to be a common feeling nowadays) of the little value, or relatively little value, of "schemes" in teaching. He said it is quite true that, if the schemes of to-day were like the schemes of localization of the sixteenth century, for instance, or even many of those of the last century, originating chiefly in the inner consciousness or in the fancy of those who invented the schemes, they would be of little value. Marie has delivered himself of opinions somewhat similar in regard to the schematic representation of aphasia.

Schemes and diagrams are worthless or valuable according as they are faithful representations of good or bad inductive or deductive methods. Dr. Meyer's glass reconstruction is one of the latest and best of these methods.

Dr. A. Meyer said that the trouble about the schematic method lies in the fact that so many things are apt to be introduced without sufficient proof that the points really represent insufficiently established facts. The method presented is as objective as possible and still has the simplicity of a scheme.

With regard to the question of the fillet concerning which Dr. Spiller remarked on marked downward and upward degeneration, Dr. Meyer said that we are dealing there with principles which hold for all central degenerations. It is the principle that he (Dr. Meyer) spoke of at the demonstration concerning the optic apparatus. In the central nervous system, degeneration involves the entire elements; especially when the lesion is near enough to the cells of origin there is always a complete degeneration of that mechanism, consequently the Wallerian law requires adjustment in this field.

The Symptomatology of the Lesions of the Lenticular Zone, with Some Discussion of the Pathology of Aphasia. By Dr. Charles K. Mills and Dr. William G. Spiller. (*See this journal*, p. 558, 1907.)

Lesions of the Corpus Striatum and Lenticular Zone. By Dr. Charles L. Dana. (*See this journal*, p. 65.)

A Case of Aphasia, Both "Motor" and "Sensory," with Integrity of the Left Third Frontal Convolution: Lesion in the Lenticular Zone and Inferior Longitudional Fasciculus. By Dr. F. X. Dercum. (*See this journal*, p. 681, 1907.)

Dr. Prince said there were one or two points which he would like to bring up; one is in regard to this motor defect. As Dr. Prince understands Drs. Mills and Spiller and also Dr. Dercum, they consider that

the motor defect is entirely an inability to co-ordinate or correlate the movements necessary for speech, and that the inability to do that is due to a lesion of the lenticular nucleus. It seemed to Dr. Prince that that is substantially the same view as Marie's. Marie maintains very definitely that the motor defect, the anarthria, as he chooses to call it, is not a total loss of motion for all purposes, of those parts which are used in speech, the tongue, throat, larynx, pharynx, but the inability to use them for purposes of speech alone, and he locates this function in the lenticular zone, which Dr. Prince takes it, includes the lenticular nucleus and the whole neighborhood. Aside from the question of location there may be this difference between Marie's anarthria and the motor aphasia of the readers; namely, patients with anarthria know what they want to say, while with motor aphasia they do not. A person who has inability to use his paralyzed muscles to co-ordinate them should know what he wants to say if it is a matter of speech, or know how he wants to move his paralyzed arm if it is a matter of the limb. Stammering from one point of view is anarthria, but a stammerer knows what he wants to say and could not be called aphasic. This is a point which wants clearing up. Dr. Prince understood from Dr. Mills and Dr. Spiller's report that their patients did not know what they wanted to say. If it can be shown that a lesion of the lenticular nucleus causes loss of speech with loss of internal language it would be a severe blow to the doctrine of Marie; but can it be said that that defect with a lesion of the lenticular nucleus is due to the latter alone and not to implication of the third frontal convolution, that the cells in the latter are not affected? (Remarks unfinished on acount of time limit.)

Dr. P. C. Knapp said, in answer to the point which Dr. Prince had just brought up with regard to the patients knowing what they want to say, that in some cases the patients evidently have at least some idea as to what they want to say, and yet there seems to be considerable mental defect. A patient recently under observation showed sufficient mental impairment to warrant commitment to an asylum. In this case there was some defect to be found in every manifestation of the motor and sensory sides of speech, yet she could understand some questions and begin a sentence reasonably well with fair intelligence, but would then lapse into a jargon aphasia. It seemed to Dr. Knapp that the cases of Dr. Mills, Dr. Spiller, and Dr. Dercum have shown pretty conclusively something which, if his memory does not fail him, they seemed disposed to deny at the meeting of the New York and the Philadelphia Neurological Society in Philadelphia; namely that there can be a destruction of Broca's convolution without motor aphasia, and destruction of Wernicke's convolution, without sensory aphasia.

Dr. B. Onuf said he was interested in what Dr. Mills and Dr. Spiller said regarding the lenticular nucleus and wished to give some of his personal experiences on this point. He and Dr. Fraenkel in three personal cases with autopsy and 102 cases collected from the literature, studied the subject of aphasia. They had had one case of dysarthria, that is a mechanical disturbance of speech in which there was practically total destruction of the lenticular nucleus and of the lenticular zone in general. Dr. Onuf said if he remembered aright Dr. Marie defines as the lenticular zone the white substance between the cortex of the insula and the lenticular nucleus plus the outer layers of the lenticular nucleus. In this case there was practical destruction of this zone, only a corner the size of a

bean in the lenticular nucleus being left. There was nevertheless no aphasia, but only a mechanical disturbance of speech. This would rather speak against considering the lenticular zone as connected with the function of speech in the manner in which Dr. Mills, Dr. Spiller and Dr. Dana have pronounced it. These disagreements may perhaps be reconciled and harmonized by taking the view which Dr. Fraenkel and he have put forth, that the centers of the lower end of the central convolutions form a so-called articulomotor center (Elder); that they are directly connected with the mechanical act of speech, and that this center is connected with Broca's convolution by an association tract, and that lesion of this association tract would make it impossible for Broca's convolution to continue its control of the articulomotor center and would then give the picture of so-called sub-cortical motor aphasia. The anatomical basis of this subcortical motor aphasia has so far found very few clear substrata; that is, there are very few cases reported in which the lesion was small enough to define with reasonable certainty the locality which gives rise to this form of aphasia. In one case reported by Edinger, however, in which there was a very small, very circumscribed lesion, the lesion would quite conform to the view they had put forth. It was a lesion between the medullary layers of the second and third frontal convolutions, really in the third frontal convolution, but it was situated in the centrum ovale where it joins the medullary layers of the third frontal convolution. This view may be reconciled with the finding of lesions of the lenticular zone in as far as a lesion of the lenticular zone is very apt to pass over towards the third frontal convolution.

Dr. Prince wished to conclude the last point he had begun to make; namely, that it seemed to him absolutely impossible to say that the third frontal convolution is not affected in such cases as those reported unless we use the same method which Campbell used in his work; that is to say, Campbell's work depended very largely upon the detection of changes in the motor cells brought about by the "*reaction à distance*," that is retroactive changes, secondary to lesion of the peripheral termination of the neurones. Dr. Spiller, who was the first to use that method in amyotrophic lateral sclerosis, had shown changes in the cortex in that disease. Similarly it may well be that the third frontal may be affected by a lesion of the lenticular nucleus in its neighborhood. A microscopic examination alone would reveal it.

Dr. Mills said in the first place regarding what Dr. Dana said about the lenticula being a vestigial organ, Dr. Dercum has said what he (Dr. Mills) would say in answer to this. With regard to the gas poisoning cases Dr. Mills said it must be remembered that the cases referred to were instances of acute poisoning, and coma being present simply means that all study of other symptoms of lenticular lesion was obscured.

Dr. Dercum, like Marie, seems to be obscuring issues by including in the lenticular zone regions like the insula which have functions entirely distinct from the lenticula. What part in speech, according to Dr. Dercum, does the insula play? He, Dr. Mills, believes it to be an integral part of Broca's area. Motor aphasia may result from lesion of Broca's convolution and the insula or from lesion of either of these areas, although the aphasia may be more partial in those cases in which both Broca's convolution and the insular are not involved together. This last remark would apply in part to what Dr. Dana has said as well as to Dr. Dercum's remarks.

Dr. Onuf seemed simply to be repeating his (Dr. Mills') opinion and that of Dr. Spiller with regard to the functions of the lenticula; namely, that it is concerned with the mechanical act of speech, not with the organization of language for expression any more than with sensorial images.

Dr. Mills said that he did not believe that he and Dr. Prince were far apart.

Dr. Spiller said that the functions of the lenticular zone is one of the most difficult subjects, and he has no doubt that after further investigation he may alter some of his views. He said that the lenticular zone is defined very accurately by Marie, and includes the island of Reil. In his use of the word anarthria Marie says he does not care to coin a word, and so uses one already in existence, but uses it in a somewhat different sense.

Dr. Spiller said that he had never denied that a destruction of the posterior part of the left first temporal convolution may exist without sensory aphasia, because at the time of the meeting to which Dr. Knapp refers he (Dr. Spiller) had reported a case in which that zone was destroyed without sensory aphasia existing.

Dr. Spiller said that Dr. Prince's remark on the possible changes occurring in the nerve cells of Broca's area as a result of injury of the fibers coming from these cells, was important, and such changes should be taken into consideration in judging of the integrity of this area.

Dr. C. L. Dana said that his own observations of the cases of lenticular lesions which he had reported are mainly negative, but they are positive in this respect; they seem to him to show conclusively that the lenticular nucleus and the corpus striatum in general are not a center for language, and that both can be destroyed without producing aphasia. He does not think that any facts have been presented that show that the lenticular nucleus has any control over speech except that dysarthria may occur. Dr. Browning reported a case of double lesion of the lenticular nucleus with pseudo-bulbar paralysis, and other cases have been reported. There have been a good many more cases of lesion of that region without any such symptoms.

Dr. Dana said he would like to mention one point with regard to the gas poisoning cases. He thought that we cannot make out anything regarding the function of the lenticular nucleus from a few cases of gas poisoning, but he was looking for the late effects of gas poisoning which were due probably to a softening of the lenticular nuclei.

Dr. F. X. Dercum said that it is not the first time in the history of medicine that we have been obliged to unlearn facts, or supposed facts, and he believes that this is the position we will be obliged to take with regard to the function of the left third frontal convolution. The historical facts with regard to Broca's first cases are of extreme interest. It has been shown by Marie that in Broca's first cases there was not only a lesion of the third frontal but also of Wernicke's region as well. His second case was one, the history of which reads not like a case of aphasia, but of senile dementia. We must remember that these cases were studied in the very early years when notions with regard to speech function were very vague. With regard to the question of anarthria, Dr. Dercum said that it is perfectly true, as Dr. Dana says, that you may have a lesion of the lenticular nucleus on one side and only anarthria. If you have a double lesion of the lenticular nucleus you have the same condition present as in pseudobulbar paralysis. When do you have the difficulty of enunciating words in connection with aphasia? Just as soon as you have

in addition to a lesion of the zone of Wernicke also a lesion of the lenticular zone or of the inferior longitudinal fasciculus. Dr. Dercum said he agreed with Dr. Prince that every case should be studied microscopically, but first we have to do our gross work. At the same time if the third frontal convolution has to do with speech, he thinks it is incumbent upon those who maintain that old view to prove it.

THE LOCALIZATION OF THE MOTOR AREA BASED UPON EXACT FARADIZATION.

By Dr. Alfred Gordon.

A case of focal epilepsy, in which the convulsions were confined to the left arm. A large osteoplastic flap was removed and the Rolandic area was exposed. Careful fradization with the unipolar method showed that irritation of the ascending frontal convolution and of the fissure itself gave distinct contractions on the opposite side. Irritation of the ascending parietal convolution gave also contractions on the opposite side, but far less marked.

A Stury in Tactual Localization in a Case Presenting Astereognosis and Asymbolia Due to Injury to the Cortex of the Brain. By Dr. Morton Prince. (*See this journal*, p. 1.)

Dr. M. Allen Starr said he felt that this paper of Dr. Prince's is a very interesting one. It is very evident that careful psychological analysis must go along with our analysis of the symptomatology in all these cases. He said he had been accustomed to teach that the symptom of astereognosis was due not so much to the loss of function of any special area of the cortex as to the interference with the association processes that are necessary to the building up of the concept, using the word concept in Charcot's sense, which is familiar in his illustration of the concept "bell." The point in the brain where the majority of these association fibers cross one another lies just beneath the cortex of the inferior parietal lobule just posterior to the posterior central convolution, and all the cases that Dr. Starr could get together that presented symptoms of astereognosis showed a lesion of that area of the brain, not merely cortical, but many of them (and he thought practically all of them), involving the subcortical association fibers that pass beneath the cortex at that point. It seemed to him, therefore, in speaking of astereognosis, as Dr. Prince has very well brought out, we are dealing with a complex association process rather than with the loss of a single sense, and that the reason why we have to locate the astereognosis lesion at that region is because that is the region where these association fibers particularly intersect.

Dr. P. C. Knapp said he had always felt much skepticism about this question of astereognosis, primarily from the fact that he had never yet encountered the report of a pure case of astereognosis; namely, a case of the inability to recognize an object when every form of tactile and muscular sensibility was normal. Even to-day our ideas of the visual and auditory agnoses have received what might be termed a jolt, as was shown in the discussions of aphasia at this meeting; but the idea that was formulated in regard to both soul blindness and soul deafness, or better visual agnosis and auditory agnosis, was that those conditions were the inability to recognize by sight or by hearing, although the visual sense

and the auditory sense were found to be perfectly normal. He said he had been able to find no case recorded in which there was a tactile agnosis, and yet the various forms of tactile and muscular sense were perfectly normal. In the second place, approaching the matter from the psychological side, we could hardly admit any very large center for the recognition of objects by the touch in the brain, because the ideas which we derive from the sense of touch are so extremely slight, and are limited, perhaps, to the ability to recognize the difference between a jack-knife and a latch-key in our pockets, or something like that, excepting, of course, in such cases as that of Laura Bridgeman or Helen Kellar. Theoretically, of course, we may assume that there is a group of neurones, as Dr. Starr has suggested, more of an association process, which are devoted to the recognition of tactile impressions. Dr. Knapp said he doubted if these are sufficiently extensive so that one can speak really of a stereognostic center. The distinction which Dr. Prince draws between pure astereognosis and asymbolia is rather subtle, yet perhaps hardly warranted, inasmuch as in the more highly developed senses, such as hearing and sight, we do not make quite that sharp distinction. Of course the perception of space relations in sight is as great or greater than it is with the touch, and that is a complex process, but it is simply the degree of complexity. In the particular case which Dr. Prince speaks of, Dr. Knapp said he had several times found a very slight loss of sensibility to touch, but he was not disposed to attribute that to fatigue. Dr. Starr has spoken of the difficulty being due to a deeper lesion involving the association fibers beneath the cortex. That may perhaps be an explanation, although with the loss of the different forms of sensibility there is, as Dr. Prince has very clearly shown, the failure of sufficient impressions reaching the brain for us to form any real judgment.

Dr. C. K. Mills said that he thought the main questions for discussion in Dr. Prince's paper (or among the main questions) are those concerned with the existence of separate regions for stereognostic perception, or conception, as Dr. Mills prefers to call it, and also the possibility of subdividing these centers of stereognostic representation. He said he thought that sometimes the views of those who believe in a very large and rather extreme division of functional regions in the brain, and indeed in the nervous system, are to some extent misunderstood. His own belief, as is well known, is that this subdivision is very great, that in the process of evolution centers have been added to centers in large number.

He has based his idea of the existence of a stereognostic area on the general biological assumption of the gradual development of association areas between the primordial projection areas. One of these areas, the posterior association area of Flechsig, he has designated as a concrete concept or concrete memory field. In this great field have developed in the course of racial and individual evolution, many cortico-subcortical centers of many sorts dependent upon the evolution of association processes. A certain portion of this region, the superior parietal lobule, is intercalated between those cortical areas which are concerned with the recognition of objects. It is next door on the one hand to the visual area. It is next door in another direction to what is probably the area of orientation; and it is next door in still another direction to the centers for cutaneous and muscular sensibility. And in this region, which is located between all these sensory areas, would naturally develop in cortex and sub-cortex, the highly evolved stereognostic area.

Dr. W. G. Spiller said that so far as he is familiar with the literature no reported case justifies the assumption that any cortical region except the parietal lobe is concerned with stereognosis. When we try to define the word center it is with more or less uncertainty. No one can separate clearly, in the use of such a term, the cortex from the subjacent white matter. Any center that we choose to refer to, the center of word hearing, for instance, certainly is dependent upon the integrity of the white matter immediately beneath the cortex of that part, as well as upon the integrity of the cortex itself, and therefore on the integrity of connecting fibers. When Dr. Spiller has used the word center in speaking of stereognosis he has used it in this sense. In all of the cases he knows of there has been some involvement in the sense of position or of some other form of sensation. He is inclined to think that the greater part of the parietal lobe is concerned in the function of stereognosis and not merely the lower part.

Dr. Morton Prince said he had ended his paper by saying that tactile stereognosis and asymbolia are abstract symbols. They are symbols of complex tactual defects, and if the terms are adopted they should be used only as symbols to describe such defects. He does not regard them as functions at all. They are judgments which are dependent upon the information given us by our senses, nothing more than that. Therefore to attempt to localize a symbol, an abstract symbol, is simply localizing a general intellectual process, and so far as our present knowledge goes we cannot localize an intellectual process.

As to what Dr. Knapp said as to the distinction between stereognosis and asymbolia, Dr. Prince said he would agree with him very largely, nevertheless we do find cases where the loss of tactual defect is sufficiently slight to allow one to recognize the object itself. Therefore these terms are convenient expressions to describe the amount of tactual loss.

With regard to what Dr. Mills said, Dr. Prince recognized, of course, the biological argument, but it does not seem to him to touch the problem. It is a matter of induction from facts. Now the facts are these. If we have extensive loss of certain tactual perceptions we cannot possibly recognize the test object.

In every case of asymbolia which has been reported and which has been sufficiently tested there has been loss of one or more of the tactual perceptions, such as localization, pressure, etc., needed for the recognition of objects.

As this loss will abundantly account for the asymbolia (stereognosis) it is bad reasoning and unjustifiable to assume a special function of recognition of objects and localize it on this evidence. No physiologist would dream of localizing such a function without demonstrating its loss with preservation of all forms of tactual sensations and perceptions concerned in it. The evidence justifies the localization of these tactual functions, nothing more.

A NEW TYPE OF ATAXIA.

By Dr. M. Allen Starr.

Patient in otherwise perfect health has slowly developed during eight years a progressive bilateral nerve deafness, and with it the symptoms of cerebellar ataxia. Is now totally deaf and walks with much difficulty.

Knee jerks normal. Pupils normal. No ataxia of hands or when seated. Diagnosis, primary atrophy of both divisions of the auditory nerve.

Dr. H. H. Donaldson said he wanted to ask just one question of Dr. Starr. Were any tests made to determine whether dizziness could be induced in this patient?

Dr. Starr replied that he twirled the patient round, holding him carefully, and made him throw his head back and forth, but could not elicit the symbol of dizziness at all.

Dr. Donaldson said that fitted perfectly with the results of William James's study made some years ago on the population of an institution for the deaf. In a large number of these cases the semicircular apparatus had been destroyed. Dr. James got the absence of dizziness, the absence of sea-sickness and the loss of direction under water.

Dr. P. C. Knapp said that Grasset in his book on equilibrium and orientation has pointed out the possibility of ataxia as arising from this lesion, although he has studied no case. Dr. Knapp said he did not catch, however, in Dr. Starr's description whether there was any sensory loss in the limbs Dr. Knapp also asked if the ataxia was influenced by closing the eyes.

Dr. Starr replied that there was none at all, no disturbance of sensation. The ataxia was increased by closing the eyes.

Dr. Schwab asked Dr. Starr whether this could have been one of the rare cases of tabes beginning with auditory symptoms.

Dr. Starr replied that he was not aware that tabes does begin with auditory symptoms, but he should not undertake to make a diagnosis of tabes when every other symptom of this disease is absent.

(*To be continued.*)

Periscope

Deutsche Zeitschrift für Nervenheilkunde.

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7. The Localization in the Spinal Cord for the Motor Nerves of the Anterior and Posterior Extremity, Particularly in Apes (Cercopithecus), in Comparison with Results of Investigations in the Dog, and Partly also in the Cat. BIKELES and FRANKE.
8. Localization in the Spinal Cord. Further Contribution. BIKELES.
9. Remarks Upon the Hemiplegic Contracture. LEWANDOWSKY.
10. Contribution to the Pathology of the So-Called Compound Tract Disease of the Spinal Cord. ED. MÜLLER.
11. Contribution to the Etiology and Symptomatology of Syringomyelia and Hysteria of Traumatic Origin. CURSCHMANN.
12. Paralysis of the Eye Muscles as a Result of Chronic Lead and Nicotine Poisoning. HAMMER.
13. Brief Communication. A Nervous Form of Disease with the Superficial Characteristics of Myotonia. v. BECHTEREW.

7. *Localization in the Spinal Cord.*—Bikeles and Franke cut various nerves in the extremities of dogs, cats and apes, and studied the degeneration in the cells of the spinal cord according to Nissl's method. They draw conclusions only from those cases in which the results are circumscribed and precise. When diffuse changes are found they ascribe them to complications. The results are partly tabulated, particularly for the segmental localization, and partly given in diagrams, especially for the groups of cells affected according to the section of the various nerves. The retro-medial group is always intact, no matter what nerves are cut, but the other nerves are affected differently for different nerves. For the detailed results reference must be made to the original.

8. *Localization in the Spinal Cord.*—(Continued.) Bikeles continues the preceding article, giving careful measurements of the lateral extent of the centers for the various sensory nerves and muscles in the spinal cord of the dog. He considers that these prove that the cells corresponding to the ventral portion of the myotom are situated medially; those corresponding to the dorsal portion, laterally. The long axis of the individual nuclei all exhibit a slight obliquity. The same groups of muscles in the more proximal sections of the body, considered in sagittal direction, are always anterior to those for the other muscles of the same myotom. The arrangement of the cells is unquestionably due to a morphological principle. It gives rise to the fact that sometimes, after section of the spinal nerves, or the spinal cord, the symptoms are peculiar, suggesting nerve localization. Among the minor points developed by this study may be noted, first, that in a group of cells apparently belonging to a single nucleus, nearly half the cells may show no changes by Nissl's method, although the others are distinctly degenerated. It also appears that, with few exceptions, the fibers entering the cord from an anterior root, are distributed to cells in the same segment of the spinal cord.

9. *Hemiplegic Contracture.*—Lewandowsky, after discussing the various theories regarding the contractures of hemiplegia, and reaching the conclusion that there is a general agreement that they are due to a nervous, and not to a direct muscular condition, combats energetically the idea that there is any inhibition, and suggests that there must be irritation of the central nervous system somewhere peripherally from the lesion. He does not believe that the apparent predominance of the flexor contraction over the extensor contraction is due to superiority in power of the flexor muscles. It is at least significant that in tabes dorsalis hemiplegic contractures usually fail. It seems to him that there is an active, or, as he expresses it, a voluntary element in these contractures, and, in addition, a certain element of inhibition of the antagonistic muscles.

10. *Compound Tract Disease of the Spinal Cord.*—Müller reports the case of a woman forty-three years of age, who nearly five years before death had weakness in the legs. The movements of the limbs were somewhat uncertain, although not markedly ataxic. The reflexes were increased. There was a moderate Babinski's sign, especially on the left side. The speech was monotonous, the movements stiff, and there was some hypertonia in the arms and legs. Station, without slight support, was impossible, sensation was normal. The patient ultimately died, and at the autopsy there was found diminution in the size of the whole central nervous system, involving particularly the dorsal region of the spinal cord and the cerebellum. Sections revealed only a combined system disease of the spinal cord. The case, therefore, represented a slowly-developing spinal spastic paresis. Müller suggests that in many of these cases not only the spinal cord, but also the whole nervous system is involved, although possibly this cannot always be demonstrated. In the present case, for instance, there is nystagmus. Of this he recognizes three forms: the true nystagmus, the nystagmus-like twitchings which are not rhythmical, and ataxic movements of the eyes. For the differential diagnosis between multiple sclerosis and combined visual disease he regards the behavior of the visual fields as most important. In multiple sclerosis scotomata frequently occur: in combined system disease, uniform contractions. He regards as the essential symptom of disease of the lateral pyramidal columns increase in the tendon reflexes, hypertonia of plantar spinal reflex, certain muscular changes, particularly the tibialis phenomenon, and paresis of the predilection type, which may indeed lead to paralysis. He discusses at considerable length the causation of these various symptoms and their relation to the pathological condition. In combined degeneration we have in addition symptoms produced by the involvement of the posterior columns and roots. Among these may be reckoned the disturbance of the deep sensation, the ataxia, and anomalies of the bladder and rectum. Müller is an ardent believer in the sensory origin of spinal ataxia. In cases of primary lateral sclerosis the diminution in the hypertonia may be regarded as an indication of the subsequent involvement of the posterior columns. In addition, in the course of the disease other portions of the nervous system may be involved without necessarily the development of a diffuse process. (The article covers seventy-four pages, and it has been impossible to make an exhaustive abstract of it.)

11. *Etiology and Symptomatology of Syringomyelia.*—Curschmann divides his cases into groups. First, those in which syringomyelia involves primarily the extremity subjected to injury. The first case, a man of thirty-five, received a severe injury to the right hand. During the third week after this injury the movements of the hand became clumsy, and

there was pain, and a diagnosis of post-traumatic ascending neuritis was made. Subsequently paresis, degenerative atrophy, vasomotor and trophic disturbances developed, giving rise to a typical picture of syringomyelia The second case, a man of fifty-four, received a severe injury to the right hand, followed by loss of a portion of the fourth finger. A little more than two months later he developed pain in the hand, and gradually typical symptoms of syringomyelia appeared in the arm. The third case, a man, received severe injury to the left hand. Six or eight weeks later paresis of this hand occurred, which increased until the left arm became useless. There was loss of the temperature sense, and the other characteristic symptoms of spinal gliosis. Fourth case, a man of thirty-four, received a splinter of iron in the right index finger. This was followed by infection, requiring amputation. Two or three months after the injury his movements became so clumsy that he was obliged to give up his work as a surveyor's assistant. There was gradual increase in the size of the hands due to thickening of the skin, atrophy of the muscles and the characteristic sensory disturbances. The clinical picture resembled Morvan's disease. Fifth case, a man of thirty-three, after an attack of rheumatism, with involvement of the heart and loss of compensation, was obliged to work from thirteen to sixteen hours a day for many weeks, using chiefly the right hand. Gradually this hand began to become fatigued more easily, trophic disturbances appeared, and when examined the condition was characteristic. The sixth case, a man of thirty-one, seven years previously had had a severe injury to the back of the head and upper portion of the spinal column. This was followed by symptoms of commotion of the central nervous system. Later there was apparently thrombosis of the veins of the left arm, and when finally examined he had the typical symptoms of syringomyelia. These cases can be divided into two groups: those in which the injury affected the periphery and those in which it affected the central nervous system. Two theories have been suggested, one, that the gliotic process existed and was in a sense activated by the injury; the other, that it was produced by an ascending neuritis. Curschmann, after discussing the evidences in favor of both views—particularly that resulting from the works of Schlesinger and Zappert—concludes that objectively the possibility of the traumatic origin or development of syringomyelia must be admitted. He then reports briefly a case of traumatic hysteria occurring in a man of thirty-three, in which there were some sensory disturbances in the left arm which might have suggested syringomyelia. He discusses very fully the differential diagnosis given by Laehr and Schlesinger, and concludes with his own views as follows. First, the character of the sensory disturbances is not a certain differential diagnostic criterion between hysteria and syringomyelia. This applies not only to the pain and temperature senses, but also such details as the behavior of the touch sensibility, the sharpness and character of the limitations of the sensory disturbances. However, the true dissociation of the sensory disturbances, either of the superficial sensory qualities to each other, or to the deep sensation, is a strong indication of syringomyelia. Disturbance of the muscular and joint sense, and of the stereognostic sense, while touch remains intact, is also evidence of an organic lesion. Particularly is this true of the distribution of the trigeminal nerve. Second, in regard to motility. Degenerative atrophies, progressively increasing paresis, and contractures are definite signs of syringomyelia. The nature of the occurrence of the paresis, chiefly because it is so difficult to determine from the history, cannot be considered of value. A quiet inten-

tion ataxia is rather in favor of gliosis and against hysteria. Third, the ordinary teaching of the skin and tendon reflexes holds good in these conditions. Fourth, vasomotor and trophic disturbances, if light in character, can only be used for differential diagnosis with great caution. Severe trophic disturbances which cannot be imitated by the hysterical patient, such as the arthropathies, are certain indications of syringomyelia.

12. *Paralysis of the Eye Muscles.*—A girl of sixteen had been working at the glazing of crockery. She developed symptoms of lead poisoning and difficulty with her sight. When examined there was found bilateral paralysis of the abducens nerves. Vision was reduced to five twentieths in each eye, and there was papillo-retinitis. The tendon reflexes were greatly reduced on the left side. The patient responded to treatment, and made a complete recovery. The number of cases in which visual disturbances occur as a result of lead poisoning is extremely small. Bilateral paralysis of the abducens has been recorded in only three cases. There is no case on record in which there was transient loss of the tendon reflexes unilaterally, particularly as in this case it seemed to involve the reflex arc. The second case, a man of fifty-nine, had had syphilis at the age of forty-seven. He had always smoked to excess. The abducens and oculomotor nerve on the left side were paralyzed. Vision was greatly reduced as a result of toxic retro-bulbar neuritis. There were no symptoms of tabes. The character of the retro-bulbar neuritis indicated nicotine poisoning. It was therefore assumed that the muscular pareses were due to the same cause, but no improvement occurred when the patient stopped smoking.

13. *Nervous Form of Disease with Superficial Characteristics of Myotonia.*—Von Bechterew reports the case of a man of twenty-one who had gradually developed a curious muscular condition in which it was difficult for him to make any spontaneous movement. If the eyes were closed they could only be opened by the aid of the hands. If the mouth was closed the same was true. If his fists were made he could only open them very slowly with the aid of the other hand. There were no myotonic reactions in any of the muscles, and no muscular atrophies. The tendon reflexes were normal; the cutaneous reflexes showed slight variation; sensation was normal. There was some fibrillary twitching of the tongue, and the patient, on account of the muscular condition, was unable to speak.

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Archiv für Psychiatrie und Nervenkrankheiten

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1. Pathological Anatomy of Paralysis Agitans. KINICHI NAKA.
2. Pathological-Anatomical Alterations of the Brain in Leprosy, Leprosy Bacilli in the Gasserian Ganglia, and the Anatomy and Pathology of Nerve Cells of the Brain in General (conclusion). HUGO STAHLBERG.
3. The Question of Amnesic Aphasia and its Distinction from Transcortical and Glossopsychical Aphasia. KURT GOLDSTEIN.
4. A Contribution to the Study of Dissociation of Temperature and Pain Sense in Injuries and Diseases of the Spinal Cord. J. PILTZ.
5. The Influence of Climate on Seven Epileptics. GEORG LOMER.
6. Investigations on the Widening of the Pupil as a Result of Psychical and Sensory Irritation, together with General Considerations on Pupillary Reactions. A. H. HÜBNER.

7. The Process of Healing in Softenings, "Lichtungsbezirke" (Granular Disintegration) and Cysts of the Brain. S. SALTYKOW.

1. *Paralysis Agitans.*—Naka speaks of the relative infrequency of paralysis agitans and the confusion which has surrounded the study of its pathological anatomy. The various findings of the different investigators are detailed and afford an excellent example of the wide diversity of opinion which has existed regarding the essential pathological alteration of the disease. Two cases are reported in detail. From a study of these two cases a rather broad generalization is made that a brain lesion probably lies at the basis of the affection and that certain slight cell changes of cerebral and cerebellar cortex are presumably to be regarded as the cause of the symptoms.

2. *Leprosy.*—An exhaustive study of the alterations of the brain in leprosy by Stahlberg results in the following conclusions: It is unusual to find leprosy bacilli in the cerebrum, cerebellum or medulla oblongata. Leprosy, both of the tuberous and nerve variety, leads to degenerative changes in the brain, appearing in nerve cells and nerve fiber. These changes are not to be regarded as characteristic; they are unrelated to disease of peripheral nerves; they do not cause alterations of sensibility at the periphery. The lesions are not pathognomonic, since in other severe, chronic infectious diseases similar alterations of the brain occur. Through the entrance of leprosy bacilli into the nerve cells of the Gasserian ganglion a variety of vacuole degeneration develops which leads to destruction of the cell.

3. *Amnesic Aphasia.*—On the basis of an exceedingly careful study of a case of aphasia, Goldstein discusses particularly the question of the amnesic form and attempts to draw distinctions which our later teaching incline one to regard as somewhat hair-splitting. The analysis into which the author enters does not permit of a statement in an abstract review.

4. *Dissociation in Cord Diseases.*—The interesting question of the dissociation of temperature and pain sense in lesions of the spinal cord is discussed by Piltz with, in part, the following conclusions: Alterations of temperature and pain sense may be of cerebral, spinal or peripheral origin. The topography of spinal thermoanesthesia which occurs in a large number of spinal cord diseases is radicular in its peripheral distribution. A special tract for temperature and pain exists in the spinal cord, which is anatomically separated from that of tactile sensibility and muscle sense. This tract presumably crosses in the anterior commissure of the spinal cord and runs upward in Gowers' bundle. An unilateral lesion of the gray substance of the cord gives thermoanalgesia when it is limited to the dorsal horn, and a crossed analgesia when the gray substance in the neighborhood of the anterior horns is destroyed at the point where the secondary neurone passes through. Following these general anatomical facts an accurate localization of the limits of thermoanalgesia in relation to various lesions is attempted. The paper is a valuable contribution to the difficult subject of the localization in the cord of a special type of sensation.

5. *Epilepsy and Climate.*—Lomer contributes a short article on the relation of weather conditions to the welfare of epileptics and the effect of various seasons. Investigation of 194 attacks during the months of May, June and July showed that the greatest number occurred in May, and the fewest in July. As regards time of attacks, it is found that the maximum number occurs from five to eight in the morning, or at the time of lightest sleep and waking. In the afternoon from four to five the minimum number of attacks occur. The study on the effect of weather

and air pressure led to certain results of possible significance, but the number of cases investigated does not permit of a statement of dogmatic results. It is proposed to study the matter further on the basis of a larger number of cases.

6. *Pupillary Widening and Psychical Stimuli.*—Hübner offers an interesting experimental study of dilatation of the pupil as the result of psychic and sensory stimuli, and brings his findings into accord with various investigations which have been made on nerve and mental disease from the diagnostic standpoint. In general, the method employed was an exact arrangement of a source of light in an otherwise darkened room in constant relation to a patient. After testing the light and convergence reaction, certain questions were asked the patient, and finally the subject was violently frightened through the dropping of a heavy object or through hand clapping. To the question whether normally every mental process is accompanied by a widening of the pupil an affirmative answer is given, although exceptions are recognized as possible. What the writer calls "Pupillenunruhe," or constant oscillation of the iris, is found to be a practically constant phenomenon in healthy persons. Its possible value in the determination of mental or physical defects is discussed. The psychoreaction, or widening of the pupil, as a result of a psychic stimulus, as, for example, the asking of a question, is likewise regarded as a normal phenomenon in mentally sound individuals. Starting with these ascertained facts, the article concerns itself with an interesting discussion of various pathological conditions, both mental and physical, in which this pupillary phenomenon does not occur, or occurs in abnormal fashion. The limits of this review do not permit us to enter into details regarding the somewhat involved results which the author deduces from his experiments, but he finds reason to think that the phenomenon of dilatation of the pupil is of much diagnostic significance.

7. *Healing of Brain Defects.*—The somewhat disputed question of healing of brain defects is discussed by Saltykow. He finds as the result of his study that the following facts appear to be established: In the scar following an area of softening neuroglia and connective tissue share in the process but in varying degree; the gliomatous and connective tissue overgrowth occur almost simultaneously and have the same significance. There is also a complete gliomatous healing of softenings and not necessarily in extremely small areas. The "Lichtungsbezirke" (granular disintegration, Clarke) are not characteristic of multiple sclerosis, as has been assumed. The typical "Lichtungsbezirke" heal exclusively by neuroglia. There are, disregarding the ordinary "Lichtungsbezirke," larger areas which are visible macroscopically as gliomatous scars, and others which have the appearance of small softenings. Perivascular cysts are essentially walled in by neuroglia. They may, however, be obliterated by glia or connective tissue following the growth of a preëxisting intralymphatic reticulum. There is the greatest variety of transitional forms of the three lesions in the healing of which scars of complicated and atypical form develop.

E. W. Taylor (Boston).

Book Reviews

STUDIES IN CLINICAL PSYCHIATRY. By Louis C. Bruce, M.D., F.R.C.P.E. The Macmillan Company, New York.

From the introductory foreword in this present volume, we have looked with considerable anticipation for something that would be new and stimulating, perhaps the recording of new facts out of which larger opportunities for psychiatric research might come. We very frankly state that we are disappointed and have put down the work with a sigh as being one more of the "faddy" outlooks in a field that is in so much need of cultivation.

Although the author very modestly calls his work "Studies in clinical psychiatry," in reality we have presented an attempt at a systematic textbook. As for the clinical studies we feel that they are misnamed, and that if the work were called "clinical studies of the blood in mental disorders" it would really represent the facts. There is not a trace of true clinical work in psychiatry in it, but there is a praiseworthy amount of attention paid to the general medical features of patients suffering from mental diseases. This point of view, however, is not new, as the author in his introduction would have us believe. It is a stage through which psychiatry has evolved and gone on to larger and more important problems. We venture to point out that the psychiatry of the eighteenth century is replete with observations naturally not made with modern instruments of precision—concerning the bodily functions, the neglect of whose study Bruce would stigmatize as contributing to our lack of advance in psychiatry.

The tendencies in the present volume we believe are retrograde rather than pointing to an advance, and it is quite consistent with this retrogression that the author should say that the less one studies the facts concerning mental life, of consciousness, and of mental experience the quicker psychiatry will advance. He says that when the study of the mental life is divorced from psychiatry advance will be made. This can be interpreted only by assuming that the author means by "psychology" "introspective metaphysics." If this be his interpretation of psychology it is apparent why he does not show evidences in his work of understanding either the primary principles of psychology or of psychiatry.

But to be more specific. The author's discussion of the mental functions is amusing if not pitiable; archaic is the only proper term to apply. In his chapter on classification he makes the totally gratuitous assumption that conditions which he names as acute melancholia (which is almost anything in which depression occurs), folie circulaire, acute mania, katatonia, hebephrenia, and a number of other things are all due to toxins. He offers no proof; indeed there is none, and much to show to the contrary, if the entire group be taken, and the evidence of the author's absolute ignorance of the immense amount of work done along these lines in the laboratories throughout the world, particularly those of Folin, make one stand aghast at his lack of knowledge or his presumption.

The author's general viewpoint may be further illustrated by the following quotation (p. 173). "Patients suffering from general paralysis are liable to attacks of mental disease which may be of any known form of insanity—excitement with or without confusion, melancholia, delusional states, katatonia, and folie circulaire." This is such rubbish that it is grotesque. It is the old one-symptom psychiatry familiar in the days of Pinel and Esquirol or even before. Depression equals melancholia; excitement is mania; depressed one day, excited another, folie circulaire; wax-like rigidity, forsooth, katatonia, etc.; the whole attitude on general paresis is so unscientific as to be amusing. We cannot refrain from offering the suggestion that the author study carefully the masterly work of Alzheimer and Nissl on general paresis in Nissl's first series of studies. That secondary toxemias may take place in the terminal stages of paresis is well recognized and belong to the large group of terminal infections so well studied a decade or so past.

The great pity is that so much good energy should be misdirected. A year's work on psychiatry proper in a German Klinik—Munich, or Giessen, or Würtzburg for instance—or Ward's Island, New York, or anywhere outside of England, would put the author in the frame of mind in which he could understand what is going on in psychiatry at the present time, and his splendid gifts of observation in clinical medicine might be made available in this special field.

A. W. S.

ATLAS DER PATHOLOGISCHEN HISTOLOGIE DES NERVENSYSTEMS. Redigirt von Dr. Victor Babes. III Lieferung. August Hirschwald, Berlin.

In this third contribution to the pathological histology of the nervous system Babes and Marinesco offer a study on the pathological histology of nerve cells, specially of the spinal ganglion. It is written in French.

They first outline the historical development of our staining methods as applied to the finer structures of the nerve cells, and naturally discuss the various hypotheses concerning cell structure as brought out by these various staining methods. The results of the researches which are here illustrated in a series of beautiful plates, nine in number, show that in the great majority of cases there exists a very close relation between the modifications of the chromophilic substances and the neurofibrils. The action of different traumatic agents applied to the peripheral nerves involves not only the chromophilic elements of the ganglion cells of origin of these nerves, but also the neurofibrils of the cytoplasmic reticulum. It is of such a nature that in the phase of reaction of the cell following nerve section a concomitant action within the two elements takes place. In different pathological states, such as myelitis, in the infectious diseases, one finds different modifications both in the chromatic and neurofibrillar substances. The authors confirm in part the observations of Cajal relative to the influence of heat and cold on the neurofibrils, but are not prepared to accord with his statements relative to the coalescence and association of neurofibrils in hyperthermia.

The various plates illustrate the authors' researches and should be seen in order to be appreciated.

JELLIFFE.

TRAITEMENT DES CHORÉES ET DES TICS DE L'ENFANCE. Par Dr. André Bruel. G. Steinheil, Paris.

It is usual, the author writes, to describe chorea and ties as two distinct nosographical groups. This distinction is justified, for there is no reason why one should fail to distinguish a true tiquer from a true choreic. There exist, however, between chorea and tics, especially in young subjects, pathogenic and clinical analogies which are frequent and undeniable, so much so that a differential diagnosis is impossible. Further the results of similar treatment for the two affections permits an inference as the underlying relation of the two.

After discussing the various meanings of the term chorea, the author limits his subject to the choreas of Sydenham and of Brissaud, and the infantile tics. His therapeutic procedures consist in feeding, isolation and psycho-motor training. Feeding, rest and isolation are desirable for the choreics, psycho-motor training for the tiquers. The various degrees of bed treatment consist in increase of night period; additional day time in bed; absolute bed treatment with isolation; absolute bed treatment with isolation in the dark. The psycho-motor training contains no new features.

The monograph is readable and sensible, but contains little new or novel.

JELLIFFE.

HISTOLOGICAL OBSERVATIONS ON SLEEPING SICKNESS AND OTHER TRYPANOSOME INFECTIONS. By F. W. Mott, M.D., F.R.S. William Wood and Company, New York.

Dr. Mott has here brought together in compact and convenient form a number of observations on the alterations which take place in the brain and spinal cord consequent on infections by the chief trypanosome *Trypanosoma Gambiense*, the reputed cause of sleeping sickness, and a number of less known forms.

His observations were made on material sent to him from Africa, and consisted of the nervous tissues from twenty-four cases of sleeping sickness occurring in natives, portions of the brains of eight monkeys experimentally inoculated with *T. Gambiense*, two oxen infected with other trypanosomes, tissues of a monkey inoculated with sleeping sickness organism, a rabbit's brain with surra infection, and the tissues of two Europeans who had sleeping sickness.

From the comparative point of view it is interesting to note that there is a marked similarity in all the trypanosome infections, but the main interest of the study centers about the changes in man, and the relation of the changes to other lesions of the nervous system. The usual noteworthy character of the sleeping sickness is the development of a chronic meningo-encephalitis which at first sight is very highly suggestive of the changes observed in general paresis, but which on close observation are seen to differ from these in some notable features. The general infiltration of lymphocytes and plasma cells around the blood vessels, as well as a marked neuroglia proliferation, are markedly similar to the picture of true paresis, but the absence of parenchymatous changes, and the limitation of the inflammation to the lymphatic tissues are in marked contrast with the lesions of paresis—the Stäbchenzellen of Nissl and Alz-

heimer are absent. Mott makes the note that he believes the Stäbchenzellen of Alzheimer are collapsed capillaries.

The small monograph is one of considerable merit, and is a solid addition to our knowledge of a type of chronic inflammatory change heretofore an isolated phenomenon of mental pathology.

K. V. HOWARD.

DIE MELANCHOLIE, EIN ZUSTANDSBILD DES MANISCH-DEPRESSIVEN IRRESEINS. Eine klinische Studie von Dr. Georges L. Dreyfus, Former Assistant in the Psychiatric Clinic of Heidelberg University. Gustav Fischer. Jena.

Melancholia, one of the oldest terms of psychiatry, has seen numberless variations in its meaning. In days when superficial resemblances set their stamp upon nosology, the picture of depression was sufficient to warrant a diagnosis of melancholia. The tendency, at the other extreme, now showing itself, particularly in the work of Kraepelin and those stimulated by his conceptions, is to do away with the term entirely, preserving it perhaps as a relic of the period when it possessed value, but rejecting it as a present guide, because of the necessary incrustations that time has deposited upon it, and which obscure rather than clarify the facts.

Kraepelin in making his synthesis of the manic-depressive states cut at one blow into the ancient conception of melancholia, but saw fit to reserve the term as valuable, under which could be arrayed a number of depressed states occurring in the years of involution, which further possessed certain characters which the single and recurring depressions of earlier years did not seem to possess, at least not in so marked a degree.

Many students have struggled with these distinctions, and with varying degrees of self-satisfaction, but increasing experience, especially taking longer periods of observation into consideration, has seemed to demonstrate a marked augmentation in the difficulties of finding constant differences in the picture of manic-depressive insanity and involution melancholia.

This is the standpoint of the present author, to which in the preface Kraepelin gives his assent. By making an exceedingly minute and exhaustive study of the after-lives of all the patients thought to have been suffering from involution melancholia in the Heidelberg Clinic since the year 1892 Dreyfus comes to the general conclusion that we are not warranted in separating involution melancholia from manic-depressive insanity. The very exhaustive analysis of the cases shows that none of the supposed differentials have absolute value, and that for those who have been able to grasp what Kraepelin means by manic-depressive insanity in its entirety, involution melancholia appears as one of its many variables. The main interest of Dreyfus's work apart from its being a model of clinical psychiatric research, is the great impetus that his study must impart to the depressive states of advanced years and which have been grouped under the terms senile and arteriosclerotic depressions, late katatonias, anxiety psychoses of the aged, etc. In this field a mass of material is at hand that is in need of careful clinical analysis.

If Dreyfus' conclusions are to be accepted, and his methods and arguments admit of little question, most of the involution depressions must be regarded in a better prognostic light than has hitherto been the

feeling, and we see once more the opening up of therapeutic possibilities which a premature pessimism has neglected.

The work shows an interesting sidelight on the open-mindedness and true scientific spirit of the leader of modern psychiatry. The gradual and steady evolution, the ability to modify one's opinions, the freedom from dogma are characteristic in the attitude here of master to pupil, even though the latter should tear down some of the edifice of the former. It is a refreshing sign.

WHITE.

UNCONSCIOUS THERAPEUTICS; OR THE PERSONALITY OF THE PHYSICIAN. Alfred T. Schofield, M.D. Second Edition. P. Blakiston's Son & Co., Philadelphia.

When a book has reached the second edition it usually indicates that the book is worth reading. The author's main argument: that the personality of the physician is a powerful factor both for the welfare of his patients and his worldly success must be conceded by anyone who gives it a thought. To those physicians who pay no attention to the appearance of themselves or of their surroundings or to those lesser details of manner and conduct which serve to create at least the outward personality this book would be an intellectual stimulant. Quackery owes most of its successful cures to the undoubted effect of mind on matter, and it is unfortunate that such a valuable therapeutic agent is not used by physicians generally, and taught in the medical schools. This book shows the necessity of such instruction, but is too diffuse to be used as a text-book.

C. D. CAMP (Philadelphia).

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Original Articles

A CASE OF RECURRENT AUTOHYPNOTIC SLEEP, HYSTERICAL MUTISM AND SIMULATED DEAFNESS; SYMPTOMATIC RECOVERY WITH DEVELOPMENT OF HYPOMANIA

By Bernard Oettinger, M.D.,

NEUROLOGIST TO THE HOSPITAL FOR THE CITY AND COUNTY OF DENVER, AND ST. ANTHONY'S HOSPITAL, DENVER, COL.

Ignorance as to any rational explanation for the inconsistent manifestations of hysteria, recognition of genuineness which some of these compel, not alone in the face of frequent voluntary exaggeration but even where actual symptoms are associated with other conditions entirely simulated, wring from the clinician a tantalizing interest. All too frequently for his peace of mind, the latter may observe typical anesthesias and paralyses less amenable to treatment than those which result from organic lesions. Rarer forms, however, presenting pathological sleep and mutism, are infrequently observed in English-speaking countries if we may judge from paucity of communications relating to these conditions in the medical literature of our tongue. For this reason the following report:

January 23, 1907, a male patient was brought to the Denver City and County Hospital who presented the following picture: a man who lies quiet and dorsally recumbent. He appears to be sleeping, his condition unlike stupor or coma, is suggestive of the hypnotic state. Facial innervation is not relaxed, the brows are wrinkled into a frown, there is a constant tremor of the closed eyelids. Attempts to open the latter are

resisted, at which time the eyeballs are also turned upward. Respirations 20 per minute, somewhat superficial. Pulse 66, small, compressible. Temperature 97 deg. F. (axilla). Face rather pale, no cyanosis. No paralysis of cranial nerves to be noted on inspection. Test of pupillary reaction impossible because of upturned bulbi when lids are forced apart. No external lesion whatever but examination of the skull shows that at some time previously this had been trephined on the left side over the anterior temporal region. The opening is 2 inches from above downward, about 1½ inches laterally, the central point of the aperture being 3½ inches upward and slightly forward from the center of the external auditory meatus. Heart and lungs negative except for slight muffling of heart sounds. The limbs are occasionally moved voluntarily and also in response to pin-pricks and when touched by a test tube containing very hot water. No muscular rigidity. Extremities when raised fall with normal tonus. Abdominal, cremaster, patellar, and Achilles reflexes present but somewhat sluggish. Babinski negative, plantar reflex positive. A catheterized specimen of urine shows a clear fluid of light yellow color, S. G. 1,030, acid reaction, albumin and sugar negative, some epithelial debris.

The patient could not be roused no matter how loudly called to. Pressure at the supraorbital foramina also failed to awaken him although the effect of pain was noted in a more pronounced wrinkling of the brows. In all, the state of unconsciousness presented inconsistent features, namely: on the one hand, a sleep apparently less profound than stupor from which the patient could not be roused by ordinary means; on the other, the irritable conjunctivæ, absence of stertor or any variation from normal respiratory rhythm, were findings not in accord with the clinical picture of coma. Most puzzling was the relation, if any, of the trephining to the presenting condition. Tendency to somnolence without paralysis suggested possible left frontal lobe lesion. Opposed to this supposition was the site of operation which lay posterior to the area of probable election, had direct access to the frontal lobe been intended.

The patient was given an enema and fed milk, and egg and milk by spoon. He swallowed without difficulty. It may be added that throughout this sleep, the bowels moved only in response to enemas and that the patient held his urine unless catheterized.

Several days later I had some of the following history from the wife. From time to time she added more, so that knowledge of its entirety was obtained long after observation alone precluded all possibility of an organic lesion in this case.

W. S., aged 34 years, occupation retail clerk, is of German parentage but born in the United States. He was hit by a baker's wooden paddle on the left side of the head in May, 1905. At the time of the accident the patient was standing on a freight elevator in the basement of the store where he was employed. The paddle fell from the third floor and although the man had on his hat, the blow "raised quite a lump." S. was not obliged to discontinue work, but he began to be forgetful. About three weeks after the accident, he awoke one night and complained of a queer feeling in his head, and a little later of terrible pain where he was struck. Thereafter he felt unable to work, became mentally depressed and in all ways apathetic of his environment. He undertook nothing upon his own initiative, and if sent to make small purchases, his memory often failed him. A trip to the mountains for several weeks resulted in no improvement. The wife, believing that a change of climate might benefit her husband, went East with the patient, returning to his former home. The headache grew steadily worse. He was placed in a hospital and in January, 1906, was trephined over the site of the original injury. Following the operation his memory improved and the headache abated for a time, but in February the pain returned. The patient now developed also, a spasmodic torticollis which drew his head to the left. One morning following a restless night, S. could not be awakened. He slept for four days in spite of all efforts to arouse him, including those of several physicians. The patient then awakened of his own accord, and remained awake, except for normal sleep for two days. He then fell asleep and so remained for 46 days. The first day of this—the second sleep—he was returned to the hospital. After fourteen days the trephine flap was turned back and immediately closed, nothing abnormal being found. The awakening this time was gradual, covering a period of several days. He now helped about the wards. Speech and all movements were very slow. S. had no memory of the events which occurred during the two days' interval between these two periods of sleep. He also had the belief that he would never leave the hospital alive, and that his people wished to put him out of the way. For this reason he refused to eat any of the delicacies his relatives brought him, although he gave these to other patients. He also cooked his own food. In August, 1906, the wife insisted that S. leave the hospital, and thereafter he no longer referred to the delusions mentioned. During the same month S. accompanied by his wife and child went to St. Louis. Immediately and of his own accord he looked for work which he found first in a lead pipe factory and later with a firm that manufactured terra cotta. He got along nicely, but wishing to again come to Denver, S. re-

turned to this city with his family in October, 1906. Again he immediately sought employment and within a few days took a position with a grocery firm with whom he remained until the second accident occurred. As an employee, the firm found S. satisfactory, yet thought him peculiar. In January, 1907, the patient was kicked in the left thigh by a horse and was disabled for about two weeks. During this time he became drowsy. For several days previous to January 16, 1907, S. came to meals when called but slept all the time between them. On that date he went to sleep and could not be roused. One week later he entered the City and County Hospital.

Even so much of the history as I obtained in the first interview with the wife together with the objective findings, were sufficient to make certain the diagnosis of hysteria, a term I use with never-failing repugnance because it comprehends so much which is inexplicable. Yet certainty of presenting functional disturbance did not entirely satisfy because of the trephining undertaken by careful observers and the knowledge that organic and functional disease are not infrequently concomitants. Inquiries of both physicians brought replies which substantiated the wife's statement, namely: that operation was done for relief of headache and not because of any distinct localizing sign.[1] With this information, probability of organic lesion could be eliminated. The patient's condition remained unchanged from day to day except for slight loss in weight and a rise of temperature on three days due to a mild cystitis which readily yielded to bladder, irrigation. Occasionally S. changed his position in bed, moaned or muttered "Helen," "home," and "water." I now endeavored to arouse the patient, using first apomorphine. A 1-10 grain under the skin induced copious emesis but the patient vomited in sleep and continued sleeping. Better results were attained by cool, full baths given three times a day at the bedside. After the first bath, S. resisted so strenuously by grasping the bedrail or blocking the porter's movements with a foot, that both men received bruises. About the fifth day the patient was awake, his return to consciousness taking place gradually. A photograph (Fig. 1) taken during this time shows the right eye partially open. The third prolonged sleep lasted 35 days.

After his return to a normal habit of sleep and waking hours, S. was mute. On the first day he was awake (Feb. 28, 1907), S. opened his eyes promptly upon my request; the next

[1]It is due the surgeon to state that he reports having found a cortical cyst which was evacuated, and adds that operation result was practically nil. According to the wife's recollection post-operative improvement of the headache and memory occurred, the former, however, returning with renewed severity the following month.

day and thereafter, he was apparently deaf to language and ordinary sounds until June 19, 1907. The patient told me that he regained his hearing simultaneously with return of speech. Strange to say, throughout the period of seeming deafness, S. heard the infant cries in the adjoining ward, to which he would respond by preparing a food bottle or by some other self-assumed service. At this time he consistently defeated every attempt on my part to surprise him into betrayal of audition. One reason for this lay in the difficulty of communicating with the patient in any way. The presenting con-

FIG. 1.

dition appeared to be a general retardation of receptivity. For instance, immediately after S. awakened, on being told to sit or stand, he appeared perplexed and unable to decide carrying out one or the other movement. Within two days the patient appeared completely deaf. All attempts to gain his attention by speech were futile. That S. retaining hearing for the cries of children, should not comprehend spoken words, was certainly inconsistent, but we were dealing with hysteria, and if the thought of mind deafness for ordinary sounds with retained ability to hear those of emotional content, presented itself, the idea was seemingly corroborated by an associated

partial mind blindness. At this time S. would pore over the simplest written question so long that an extended conversation was impossible. Here, too, defective cerebration seemed to manifest itself in retarded receptivity, for the question once grasped was answered with comparative promptness, in spite of very deliberate penmanship. It may be added that suddenly and for a few days only, S. merely copied the question put, instead of answering it, and also that stereotypy, too, had been noticeable. Immediately after awakening from his prolonged sleep, the patient again and again wrote, "Clean clothes," "Helen," "Go home," and as often as he repeated the last, he invariably wrote "Home" and prefixed "Go" (Fig. 2).

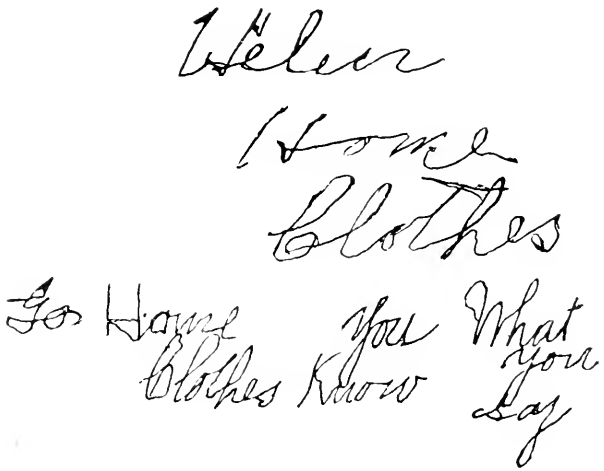

FIG. 2.

A strong induced faradic current was applied to the larynx externally with intent to bring about vocal protest, if possible. The result was nil. No greater success attended the attempt to hypnotize the patient. His facial expression denoted apprehensive excitement during the seance, but suggestion attempted by gesture and mental effort was not responded to. Later the patient spoke of his successful resistance to suggestive influence with complacency. This, then, was the condition of S. at the time my service ended, but I continued to see him twice or thrice a month. For about two months I could observe little change, then a gradually increasing celerity of movement was noticeable. Now and again a nurse would tell me she thought the patient could hear, not only because of his voluntary services in the children's ward, but for the reason that occasionally he would carry out spoken instructions. Once

he had closed a faucet as if the running water disturbed conversation. By the last of May he would converse fluently in writing. I therefore wrote: Q. "Do you hear anything that is going on about you?" A. "No, sir." Q. "Then how is it that you hear when the babies cry?" A. "I can tell on their features." Q. "But you hear them when you are in this room and they are outside?" A. "No, this is an impossibility." Q. "Try to use your voice; you can if you make a big effort." A. "That is past." Q. "Wouldn't you like to go out of the hospital and keep house again with your folks?" A. "I would not go anywhere else; I've been in so much. There are lots of useful things that can be done even if you can't talk or hear." Q. "Now I am going to talk to you with the lips. You answer on paper." A. "I can catch some of the words probably."

The result of these efforts was that not once when the nurse or I framed a question by lip movement did the patient comprehend. The same questions, for instance concerning the day of the month, etc., asked in an ordinary tone of voice always resulted in his writing an appropriate word or two, to which S. would point and look inquiringly. In spite of the palpable fraud and my accusation of deception on his part, he insisted that he could not hear. This attitude, inexplicable at the time, became less so as the psychosis became more pronounced. Two weeks later I saw the patient again. He wrote:

"Dr. Wood[2] says I can talk in a few weeks. I am well satisfied to get this chance" (*i.e.*, promotion to position of porter with pay, the same being dependent, however, upon his ability to speak). Q. (spoken) "When will you begin to talk?" A. (written) "That is as near as I can say." Q. "You can hear me now, can't you?" A. "No, sir, it is from the lips."

Nevertheless S. regained hearing and voice suddenly the next day. He spoke with fluency after a period of mutism lasting 4 months and 19 days. There was no dramatic prelude to the change. He was given porter work to do but it was at once apparent that whereas during the time he was mute, S. had evinced a willing and kindly disposition, had been industrious and had well planned his work, he now passed from one thing to another and failed to finish a single task. He was garrulous, boastful, irritable and in all ways exceedingly officious. He got for himself various little luxuries with the money he had, or could borrow and as much as he could obtain on credit. Discharged as porter, he refused to leave the hospital, and said he was "done with outside life."

[2]For ten days, Dr. Wood, one of the house doctors, had been making a show of treating the patient's throat.

Claiming his life was endangered by the hostile attitude of the hospital help, he purchased a revolver, and this in turn led to the patient being placed in the insane ward, as the other employees began to fear the man. During this time I re-examined S. and found him a man of powerful physique and weighing almost 200 pounds. Sensation was normal, but the plantar, pharyngeal, nasal, aural and conjunctival reflexes were diminished. There were no hallucinations. He explained his actions with plausible complacency and delusion referred only to incorrect judgment arising from an exaggerated sense of capacity and self-importance. After spending 10 days in the insane ward, S. willingly left the hospital and got for himself a position of clerk in a retail fruit store. But within a week he was arrested for disturbance. He had peddled his card to passers-by, to gain, as he explained, a personal following in his business, and this action led to a quarrel and to his arrest. An additional sentence was imposed for carrying a concealed weapon, which sentence he is still serving. No effort was made to obtain the man's release as he preferred to remain in jail to returning to the insane ward, but that he will soon bring about his detention in the hospital, by irrational conduct of some sort, there can be no doubt.

In all, the history of this patient is interesting and instructive. For one thing, the ease with which even careful diagnosticians may mistake hysterical pain for that due to organic lesion, is amply demonstrated. Likewise is shown, how in a given case, one may be required to differentiate function suppressed in response to abnormal egocentric ideas from actual simulation, both conditions presenting. Especially interesting to me has been the constant yet varied picture of mental dissociation. Thus the patient, to some extent depressed while dominated by the idea of mutism, exhibits approximately normal cerebral reflexes as regards his work, which he performs in an orderly and otherwise adequate manner. Stimulated to overcome speech inhibition and thus acquiring a normal reflex in this respect, depression is very soon replaced by hypomania, continuous futile exertion and material loss of judgment. The factor of dominating pathologic mentality will again be referred to. Consideration of individual symptoms suggests, as here in place, a line of thought concerning therapeutics in this class of cases.

Psychotherapy in the church or out of it and in the profession, has attained an established place in methods of treat-

ment. As thus recognized, cure is obtained through a persuasive idea of well-being, reception of which is voluntarily undertaken by the patient. It is obvious that in the present instance, because the avenues of sight and hearing were not available to conversational suggestion, such procedure was not feasible. For this reason physical means embodying some discomfort were employed in the hope that mental resistance to their effect would invoke sufficient effort of will to overcome inhibition of centers usually automatic; hence the battery, the use of apomorphine and finally cool immersions. The last were sufficiently discomforting to arouse the patient from abnormal sleep in five days. Had I been able to magnify the gripping impressions which were induced by nothing more formidable than rapid immersions by aid of a ducking stool, I feel sure the awakening of S. would have occurred within the same number of hours or perhaps minutes as were required in days. As the public now thinks, the attempt to restore certain central reflexes to the norm by harmless yet forceful mental impressions through physical aids, would no doubt be largely misinterpreted. The physician would need to not only manufacture his own paraphernalia, but reconstruct public opinion as well. Yet we know that when sudden and vivid mental impressions from natural causes have been brought about, the effect on hysterical loss of function has always been marked. In the train of the great San Franciscan earthquake and succeeding fire of 1906, the restoration of function in many paralytics has been related. Especially in point is the history of a case of prolonged sleep related by Herbst.[3] This person, an Austrian woman, had slept in bed for 17 years and during a period of six years before this time had often slept whole days and once continuously for 30 weeks. Nourishment was taken at regular intervals and delay in its offer produced great restlessness. When the woman wanted water, she smacked her lips until it was given. This person awakened during an alarm of bells rung because of fire near her dwelling. She was at once mentally clear and remembered all that had occurred previous to her sleep.

And again, if mental suggestion by aid of another person be employed, there is no reason why the stimulus should be

[3]Herbst, E. Wiener Med. Presse, No. 6, page 272.

confined to a single or a few human emotions. Religious healers use the idea of an all-pervading love, suggestion as employed to-day by regular practitioners, usually comprehends the certainly less potent thought of confidence in the return of one's own power to be or to act. However satisfying to contemplate the employment of such attractive sentiments, in the abstract, we know that there are other passions which play as great a part in human life and one may therefore ask, why not develop systematic use of these more common but less beautiful emotions, *i.e.*, to inspire return of function through fear, hate, jealousy, avarice, etc. I know of an instance where a patient long bedridden because of hysterical paraplegia, left his bed and the hospital to recover a wallet containing money, shortly before inherited, which had been stolen from under his pillow. The point I wish to make is this: that I believe the scope of psychotherapy may be enlarged by systematic use of disagreeable as well as pleasing emotions, and that suggestion through material means may at times be profitably substituted for that which is personally intimated.

Finally, however, with good effect which accrues from suggestion conceded, we need remember the limitation of benefit to be attained in hysterical conditions by the suppression of any one symptom, whatever the means employed. The abnormal mentality remains and even may be accentuated by a seeming clinical victory, as happened with my patient. And because the essence of the affection is a psychosis, no matter what the nature of its physical expression, lasting good from therapeutics can only come with capacity to treat morbid ideation from the viewpoint of its etiology. In respect to this momentous problem, we do well to look for little assistance from the present-day psychologist. Like his predecessor the metaphysician, his service to the study of insanity must continue to be confined to analyses of thought processes. His labor as regards cause of insanity will never be elementally constructive while the relation of brain cell and nascent idea remains unsolved. The prospect is brighter respecting the researches of physiological chemistry. Psychologist and chemist alike can only record the manifestations of vital phenomena and not explain them, yet the latter has the advantage in possessing as his sphere of action, the study of material

reactions which seem to be an integral part of such phenomena. The chemist has therefore to do with mere raw material and material byproduct of mental metabolism, a problem, intricate enough yet humbler and therefore, possessing better promise of solution than the work of the psychologist who analyzes mentality after its transmutation from physical elements. It is true that as yet little time has been devoted to the study of metabolism with direct reference to mental processes, which is explained by the fact that our profession does not yet recognize the urgent need of it. And yet since the discovered cause of cretinism, wherein absence of glandular function predicates with absolute accuracy the character of the associated psychosis, treatment of insanity may be viewed in a new light. In a former time the attempt to restore abnormal mentality solely by specific correction of body chemistry would have been deemed absurd. To-day the same measure forecasts the most hopeful and rational procedure for the cure of insanity. Our dependence upon the improvement of metabolism to benefit mental conditions is well reflected in the clinical pictures of such forms of insanity, acute and subacute, which are called curable, in that physical improvement goes hand in hand with mental betterment. More than this, however, it may be noted in the sole methods of treatment which we empirically employ in these conditions: we induce sleep, increase elimination, raise the per cent. of iron content of the blood, correct circulatory disturbances and in all ways try to improve body chemistry. On the other hand, the mental sluggishness of acromegaly and myxedema, the delirium of Graves' disease, the brain and cord affections in grave anemias and cachexias, the frequent brain and cord lesions associated with perverse metabolism resulting from, but not directly due to, the virus of the infection, as in syphilis—all these are links in a chain of analogies strongly presumptive of this fact, viz.: that not only is every psychosis the effect, direct or indirect, of disturbed metabolism, but that differences in character make-up within limits of normal deviation, are not less dependent upon varying totalities of body chemistry. In accordance with this idea, even as a mere working hypothesis, we should have laboratories at, or associated with, our State Hospitals for Insane where the study of

relation between glandular organic toxins, systemic ferments, etc., and clinical manifestations of insanity should be assiduously furthered. Such investigation concerning the effect of systemic toxins on mentality would be in line with active work of the same character, now being pursued to establish etiological factors of morbid conditions which belong to other departments of internal medicine. This study should furthermore be undertaken in each State by a commission with required full co-operation of governing boards of State Asylums, a condition which would permit the problem of prognosis and treatment of insanity to enter into a new and therefore more hopeful era.

ADDENDUM

The clinical history as it appears in the foregoing, describes the patient's condition up to about August 1, 1907. When seen six weeks later S. was no longer a hypomaniac but presented a delirium so expansive as to preclude, as I believe, possible recovery without a residual dementia in some degree. The patient was returned to the Insane Department of the City and County Hospital on a commitment sworn to by the wife, who feared violence on his part. As I entered the ward S. called to me and extended an invitation to witness his marriage to a beautiful lady of 19 years. On being reminded that he was already married, S. replied knowingly that he would get around that by joining the Mormon church. The head nurse, whom he gave the title of "Judge," was to perform the ceremony. Upon my expressing a doubt as to the correctness of the title, S. replied "At least he has always carried himself with magisterial dignity." He had been nominated for President by the Workingman's Party and his election a certainty. About his neck S. wore a long strip of elastic to which was pinned, by a penny brooch, a slimsy bit of red satin cut in the shape of a cross. Tied about the right and left upper arms were respectively white and black ribbons, while upon several fingers, worn ring fashion, were yellow strings from tobacco pouches. These were some but not all of the fantastic adornments. Nevertheless enunciation and gait were normal except for evidence of affectation which both disclosed. The patellar reflex was bilaterally somewhat exaggerated. Pupillary reflexes were prompt and normal in reaction.

A NEW DIAGNOSTIC SIGN IN RECURRENT LARYNGEAL PARALYSIS[1]

From the Department of Neurology of the University of Pennsylvania.

By Alfred Reginald Allen, M.D.

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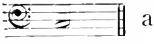
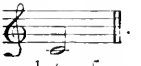
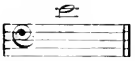
I make the report of this sign, not with the expectation and hope of its becoming one of the great diagnostic methods, but rather because it seems to me of interest in that it permits a quantitative estimation of the contractile ability remaining in a vocal cord in cases of recurrent laryngeal paralysis.

I have noticed in these cases where the lesion is monolateral that there is a very material difference in the upward excursion of pitch when the vocal apparatus is stimulated electrically during the singing of a tone.

To demonstrate this it is necessary to bare the neck completely and by careful palpation determine the angle on each side of the larynx formed by the cricoid and the thyroid cartilages. A small button electrode with a thumb circuit breaker is placed in this angle over the cricothyroid membrane, pressing back slightly the sterno-hyoid muscle, and then the patient is instructed to sing the note C. For a man this should be  and for a woman  . On the normal side there will be a rise in pitch equal to from seven to fourteen half-tones, or in other words the excursion will be an interval of from a perfect fifth to a major ninth or over. On the paralyzed side, however, the tone will only be raised from two to three half-tones—a major second or a minor third.

As the amplitude of this excursion varies in the normal individual according to the pitch of the note from which the test is

[1] Read at the thirty-third annual meeting of the American Neurological Association, May 7, 8 and 9, 1907.

made, it is desirable to employ in all cases the same note. For this reason I have suggested the C indicated, as it is found in every voice no matter of what register. To illustrate how different the findings might be if a different note were taken, suppose that a man were told to sing E,  , and let us suppose that the voluntary range of his vocal scale only extended to the G—a minor third above this—it would be manifestly impossible to get an excursion of more than three half-tones, although his vocal cords were in perfect condition. The C indicated gives a note in every voice which is free from muscular strain, and for this reason offers the best starting point for the test. The exact point at which to apply the electrode, although easily located from the superficial standpoint, is at times hard to find and frequently trials with gentle pressure at different angles are necessary to elicit the phenomenon. But when once found the reaction is certain and admitting of no doubt. The strength of current used has never to be such as to cause pain.

Unless the physician is able to place the tone C without aid—which is by no means common—he must have recourse to a tuning fork or some musical instrument. With a little practice one should become fairly familiar with the musical intervals if not cortically deficient in this respect. I should like to call attention to the possibility of confusing perfect fourths with perfect fifths, and vice versa. This is easily explained when we remember that the inversion of a perfect fourth gives a perfect fifth, and of a perfect fifth gives a perfect fourth. The ear hearing the interval of a fifth at times refers the upper note down one octave or the lower note up one octave, in either case producing mentally a fourth. By the same mental process a fifth can be perceived where a fourth is sounded. The intervals of the octave, the second, major or minor, or of the sevenths, major or minor, will cause no trouble in detection. The interval of the diminished fifth, or, what is the same thing, augmented fourth or tritone will cause confusion to the unmusical.

Thirds and sixths are of easy detection, but it is not always possible for the uninitiated to differentiate between the major and minor thirds and sixths. This is possibly explained by the fact that an inversion of a major interval produces a minor and vice

versa. I do not think it advisable to more than touch on this subject of intervals because any one so inclined can get a full description of them in any text-book on harmony or thorough bass. For this purpose I most highly recommend the first chapter in Hugh A. Clarke's "A System of Harmony," Theodore Presser, Philadelphia, publisher.

The points to which I have called attention are most necessary to observe if anything like a quantitative test of vocal cord power is to be made.

A CASE OF MATRICIDE AND ATTEMPTED SUICIDE WITH BRIEF PSYCHOLOGIC ANALYSIS[1]

By W. K. Walker, M.D.,

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The following case was brought to the Western Pennsylvania Hospital for the Insane after a tedious court trial into which was injected much irrelevant testimony looking toward the establishment of motive for the "crime." Widely advertised by the public press it was thus needlessly made one of the most notorious cases of local history. The data of clinical interest are as follows:

Family History.—Parents, born in Germany, were first cousins. One set of grandparents were cousins. Father, a prosperous butcher, was a drinker of the German type, taking wine or beer with meals and other drinks between; had suffered several attacks of rheumatism; died of apoplexy at sixty-four. Mother was sober, industrious, and always well; she had six children. A brother of the patient shot himself beside his mother's grave one month after her death. A butcher by trade, he was a poor business man—always borrowing, always in debt; he was regarded as a "sissy," since he preferred doing cooking and housework to attending to business; he had a morbid desire to attend funerals, wanting to see the decorations and how the corpse looked. The other members of the family are healthy and apparently of normal type.

Personal History.—Twenty-two years of age (on admission); is tall, slender, fair, rather good-looking but ordinary in appearance; dresses conspicuously, and is very self-conscious.

The youngest of six children, she was pampered and spoiled by her parents, particularly by her father whose pet she was. She had a common-school education; "was only a fair student." She was always wilful, dictatorial and high-tempered, vain and conceited. She has been a moderate drinker all her life, taking wine, beer, or whiskey every day since she was twelve years old. With her parents she has always believed in mediums and has consulted them many times. At sixteen she had a "love affair,"

[1] Read at the thirty-third annual meeting of the American Neurological Association, May 7, 8 and 9, 1907.

opposed by her parents because the man was a bad moral character. She went out a good deal to parties and dances with those of her own set. Never had many girl friends but was popular with men. She has been talked about for drinking and immorality, but patient says that she has never been drunk, that it was always her custom to take wine, beer, and whiskey; and her moral reputation has suffered because she went with a man of whom it was said that "he wouldn't go with a decent girl." Patient states that these remarks do not worry her "because people will talk and there are worse troubles in life to think about." At various times since she was sixteen she has wanted to die, but never really thought of killing herself until after her father's death. Her father died suddenly in December, 1897. Some months previous to this, the patient, with her mother, consulted a medium who told them that her father was going to lose a mortgage of large amount; they both worried greatly about this. The patient was very much attached to her father, and his sudden death, from apoplexy, in her presence, was a severe shock to her. Grieving constantly over his death she began to lose sleep and appetite; her health failed, and she became so depressed that her mother consulted a physician about her. About this time a friend, who was a medium, told the patient that she had a vision of her father who told her that he was not pleased at the way things were going.

The patient continued to run down in health. In August (nine months after her father's death) her family decided to send her away to visit relatives, where she had often gone before, hoping the visit would cheer her up. Patient was willing to go, but more, she says, for the purpose of seeing a medium whom she had once consulted and who knew of her father's death.

At a subsequent meeting with this medium she was told that "her father was standing behind her with his hand on her shoulder; that he stooped and kissed her on the forehead, saying that he was lonely without her and her mother." The patient did not see or hear her father but she believed he was there. The seance lasted an hour and a quarter. She remained in C—— a week or ten days longer to consult another medium who was said to be better than the first. This one told her that her father was there and wanted to see her; but again she failed to see him. She was also told that her father wore a discontented expression and that he was unhappy in the spirit-world—that if only she and her mother were there his happiness would be complete. After this the patient was so distressed that she at once returned home. After her return, which was six weeks before she killed her mother and attempted suicide, her father appeared before her in her dreams; soon he appeared almost nightly. He would stand or kneel at her bedside and plead with her to take her own and her mother's life and come to him. She told him she would

willingly come alone, but that her mother still enjoyed life and she didn't like to kill her; but he would stay and plead with her to bring her mother too; finally she promised him she would do it, but next day changed her mind. Then, she says, her father would get angry and not appear the following night; next night he would come again, however, and plead with her as before. Promising again and again that she would accede to his wishes but losing courage when day arrived, she continued in this way for five or six weeks. During all this time she slept badly and ate but little. She was in continual torment because she felt that she *must* perform the deed much as she disliked the idea of killing her mother.

On the day before the tragedy she went to the city and bought an ounce of laudanum, deciding that she would kill herself only. That night she went to her room at eight o'clock, took a bath, put on clean linen, and made all arrangements for death. She did not hear her mother come up to bed, although she slept in the adjoining room. Patient states that she walked the floor for a long time; finally—" sometime between midnight and morning "—she mixed the laudanum with whiskey and was about to drink it when a voice from the spirit-world (not her father's) said, " Do it now! You must do it now! " Her arm stiffened and the glass dropped from her hand. After this all appears hazy. She took the pistol from the dresser drawer and killed her mother; she doesn't remember this, but thinks she remembers having heard the first shot. After this everything is blank. She killed her mother and then fired five shots into her own body, two of them into her head.

The deed of violence herein described would, from its general character, lead us to look for evidence of abnormality in the perpetrator. But just as more than one striking and characteristic phenomenon is needed to make out a case of bodily disease so, in the mental field, we must search for other evidence than that exhibited by this one act to firmly establish a diagnosis of mental disorder. Our procedure here differs in no way from that we habitually follow in the investigation of bodily disorders presenting diagnostic difficulties. In both instances it is by putting together the minor symptoms and early signs—the slight deviations from the physiological—that we are enabled to produce the complete clinical picture; and by this method we here obtain one of such clearness as to occasion a feeling of surprise that there could have been any legal controversy such as actually occurred regarding it. As a matter of fact the controversy arose (as it only could have arisen) from the " attempt to follow the

rules of mere plausibility and explain all the actions of the perpetrator in terms of possible motives of everyday life."

"Mind" and "will," "motive," "conduct" and "responsibility," are terms habitually in the mouths of everyone. To assert that we commonly fail to fathom their full significance would invite indignant criticism. Courts of law, the pulpit, and the public generally but too easily arrive at finality in terms whose meanings are closely bound up with the profoundest problems of life—not only moral, ethical, and social, but biological as well.

Manifestations of mind and of will appear to us as motived. Motive is implied as the ruling force of conduct and when definite ends are consciously aimed at. But that which is revealed to us of mind and will in conduct is but a fractional part of all that is implied by these terms. Rooted in the physical substratum, through which they are also manifested, they become intimately associated not only with the body's normal functions—with its needs as represented in appetites and desires and their accompanying emotions—but with their perversions and abnormalities as well.

Deviations from the normal in manner of thinking and feeling commonly furnish the earliest clinical symptoms of mental disorder. As in the sphere of bodily function these earliest disturbances are in the direction of excess or defect in reaction to stimuli; and they further tend to final expression in conduct or action.

In the sense that it is the result of "stimuli" we can truthfully say that "no act is performed without motive; that is to say—stimulus: even irrational acts are caused by something"; but we here seek the cause or causes—not only in motive, or conscious design on the part of the one exhibiting such conduct, but we must further trace their origin to the hidden inner influences known to originate the effects which we observe.

The organism not only reacts to stimuli pouring in from the outer world, but in its reactions exhibits innumerable modifications depending upon its stored-up sensations and to its individual peculiarities in recollecting in memory and in recombining them through the imaging faculty before they are transformed into conduct or action. Some of its variations can be traced to limitation of brain capacity owing to the influence of heredity; some to gradually acquired but all-dominating habit of thinking and of

feeling; while others may be due to definite alterations in bodily fluids from self-engendered poisons, or those introduced from without.

In the analysis of this case let us take up the actual events in their proper sequence and natural setting. At one extreme we observe phenomena which are characteristically morbid; these must be regarded as the last links in a chain the first of which were forged by heredity. An alcoholic father, who exhibited, also, other signs of instability, explains the potentiality of the nervous organization of the patient. In childhood slight deviations from the physiological are to be noted in her manner of reacting to mental stimuli. She is described as a spoiled and petted child, impulsive and wilful, and subject to sudden outbursts of temper whenever her will was opposed. In this description we have the picture of one driven by sudden emotions, appetites, and desires and who more or less promptly translated them into action. Early incapable of restraint—of that subordination of the lower to the higher inhibiting impulses which is the chief characteristic of the stable and well-balanced nature, we find, as she develops into womanhood, an accentuation of these impulsive traits together with others pointing to an all-dominating ego. The early characteristic of selfishness is now exhibited in an exaggerated self-consciousness with a manifest desire to attract attention by a showiness in dress.

It must be said that until the death of her father, which occurred ten and a half months before the killing of her mother and attempted suicide, she presented no deviation from her customary attitude and conduct sufficiently marked to excite comment or to label her markedly morbid.

Her father's death occurred suddenly. The shock and emotional stress were manifested in the patient by reactions which must be termed excessive in that they were unduly prolonged and severe. Her sense of personal loss was great; she gave herself up to brooding upon it. Long pondering over her bereavement engendered a state of mental depression manifested in general bearing and conduct so marked as to lead her relatives to seek medical advice. Although there followed some amelioration in what was, for a time, a settled state of melancholy, there nevertheless was the frequent revival in memory and consciousness of ideas and images serving as pivots about which revolved the

whole of her mental life. Her sense of great loss was thus constantly renewed; with this there was associated the wish that her father might again be with them, or, since that was impossible, that they might go to him. The later expressed desire that she and her mother might die and thus forever be with him speaks for the growing intensity of her feeling.

Sent on a visit to relatives with the hope of diverting her from a train of thought and feeling so evidently morbid, she, at this critical juncture, had the interview with the spiritualistic medium whose statements were accepted as genuinely coming from her father in the spirit-world; these are of great significance as having a direct bearing upon events which subsequently transpired. Because of this interview expectation is added to desire; and however vague this was, it is now seen to become more and more clearly defined. Belief in the medium's revelations is made easy to her uncritical mind; thus reinforced all her images revolve with redoubled force around desire and, with attitude of expectant attention, she is ever on the alert for impressions which are made to fit in with the medium's suggestion.

It is reasonable to suppose that even at this stage her vague ideas and longings had not assumed the proportions of definite design to kill her mother and herself, for many considerations remained which would tend to prevent so abhorrent a measure; she is still responsive to the deterring influence of normally restraining ideas and emotions which thus far are capable of being called into operation.

But we have seen that her power of restraint—of inhibition—was never very great; the impulsiveness which characterized her every act continuing into maturer age. No less apparent is her inability now to exert control over lines of thought dominated by feeling. The sad ideas clustering around this central fact of loss of her father, with the concomitant desire to again be with him, and bolstered up by acceptance and unquestioning belief in the statement of the spiritualistic medium, hold her for longer or shorter periods of time in the power of their fascination.

We know that "the repetition of an emotional movement facilitates its automatic reproduction, and the more the reaction is established in the lower centers the more it escapes from the control of the psychic ego" (Dubois). Support is given to the assumption that these ever-recurring ideas, emotions, and

desires now entirely dominate the subconscious mental mechanism; for in the frequent abstractions and moods of the patient—states furnishing the soil in which morbid impulses grow rank and flower into action—we have outward manifestation of their compulsive power. Reverie is given up to vague imaginings based upon underlying desire—that is, ever-present longing now felt as a need to be with her father. Although these must be regarded as the beginnings of action—as the stimuli indeed, which inevitably tend to outward manifestation in conduct—we may infer that higher inhibiting forces would still have exerted their controlling power but for the more marked involvement of the faculties next evidenced.

Disgressing for a moment from the reconstruction of this clinical history by means of its psychical deviations, I would review in the briefest manner the perversions and defects of consciousness in which conduct that is grossly morbid so often takes its rise.

Clear consciousness is based upon the activity and integrity of the associative functions of the cerebral cortex. Anything which interferes with this functional integrity delays or prevents the marshalling of ideas before the "mind's eye," that is, limits, obscures, or abrogates consciousness.

From the total abolition of consciousness down through its less profound modifications due to narcotic poisons and intoxicants (opium, alcohol, etc.), and the toxins of acute and chronic diseases, or in those psychopathic conditions whose chief diagnostic feature is disturbance of this function (I refer to epilepsy and hysteria), on through dream, semi-waking, and hazy states, we observe all shades and degrees of disorder in this highest function upon which depends the welfare of the organism as a whole in its multiform vicissitudes of social and physical environment; and not only from the dangers which assail it from without, but also from the results of its own blind caprices, impulses, and emotions.

This function of consciousness, we have already seen, has suffered marked limitation under the stress of grief. There is lessened ability to transfer the attention to objects other than those of its own narrowed sensations, mental images, emotions, and desires, which, by reason of this restriction of the field of consciousness, become vivified and proportionately increased in

importance. It is to this point that marked divergence from the individual norm must be traced; and here it is that characteristic and unmistakable symptoms of mental disorder appear; these are manifested as hypnagogic hallucinations.

With the knowledge we now possess of antecedent conditions in the mind of the patient—of her constant preoccupation with sad ideas and desire to be with her father—we can understand how the mental activity of the day becomes reflected in the partial activity going on within the cerebral cortex during disturbed sleep and in dream states. Dreams are of nightly occurrence. Her father appears before her and asks that she and her mother join him in the spirit-world. But they soon pass beyond this stage of simple dream states representing a slumbering brain-activity, and assume a character which gives them a far more serious significance: the visions of her imagination—of waking as well as dream states—gradually become transformed into hypnagogic visions, that is, into pictures so vivid as to be accepted as real; they are now to be regarded as exteriorized images of the inner cerebral activity—as visual hallucinations—which, in general character and mechanism of production do not differ from any other falsification of sense perception.

It is interesting to note, in passing, that the patient's high refractive error of compound myopic astigmatism, O.D. — sph. 1.00 ⊃ — cyl. 8.00 ax. 180., O.S. — sph. 3.50 ⊃ — cyl. 6.00 ax. 15°, precluded the possibility of her obtaining sharply defined visual images at any time in her life; her unquestioning acceptance of the dream-vision as reality may have been facilitated by the previously engendered mental habit of readily accepting blurred images, for only in this manner had her visual perceptions and impressions of the outer world been acquired.

Under the high tension of expectancy developed through her experience with the spiritualistic medium, and the consequent fascination of the attention, these dream visions are given logical connection with all that has preceded them in consciousness. They now assume, to her, all the significance of reality—are, indeed, reality—since even in her waking moments she is unable to correct the faulty impression by any process of reasoning. That they are not at once translated into action—that there is further resistance against the compelling power of her father's

wishes in this manner revealed to her, is due to a feeling of horror at the idea of taking—not her own but her mother's life, the now necessary preliminary to joining him. But under the influence of regularly recurring visions all ideas dictated by this residue of her clearer consciousness are rendered less and less effectual as restraining forces. Indecision is now replaced by certitude: and under conditions of partially lapsed or clouded consciousness—in the semi-waking state in which the hallucinatory vision first appeared to her—it is again presented. With all the conviction of an insane person believing in her visions, the voice from the spirit-land is all that is needed to release the stored-up impressions from the higher cortical into the lower centers whence they are discharged through the appropriate channels and almost automatically transformed into the act which has been so frequently pictured in her imagination.

Conduct resulting in matricide and attempted suicide thus receives adequate explanation—not through search for possible motives of everyday life, calmly reasoned and carried into execution by the light of clear consciousness; but through tracing to their sources ideas, emotions, and sensations which undergo transformation through the hidden inner processes of an unstable mental mechanism, becoming, in accordance with known governing laws, recombined, perverted and distorted under influences which are brought to bear from without and from within, with resulting exhibition of symptom-complexes of disordered attention, emotion, consciousness and will, and, finally, the more marked manifestations of hallucination with delusion based upon them; the whole picture conforming in its principal features to a definite clinical type of mental disorder.

GENERAL CONSIDERATIONS AS TO THE NATURE AND RELATIONSHIPS OF HYSTERIA

By R. C. Woodman, M.D.,

of Middletown, N. Y.

(*Continued from p. 108.*)

THE RELATIONS OF HYSTERIA AND OF THE PARANOID AND DEMENTING INSANITIES

Our material is sufficient only to throw light on this topic but not to make it clear. On the one hand a number of traits resembling hysteria may be observed in individual cases of dementia præcox. Not only does general psycho-anesthesia, without interruption of the peripheral nerves occur in both, but sometimes in dementia præcox, as in the case of a young man observed in Middletown, definite belts of anesthesia are found. This particular anesthesia proved its functional character by variations in its limits and by subsequent disappearance. Perhaps the thought disturbance in the two conditions may be profitably compared. We regard hysteria as a disturbance of the thinking process in which the mental equivalents of some experiences become dissociated from the larger body of recollections. There is a cleavage in thought. In dementia præcox there seems rather to be an irregular breaking up of thought forms. Experiences do not arouse their appropriate associations and emotions; patients feel their mental incapacity and say that their thoughts vanish or that their minds are gone; a disorganization of thought occurs, reflected by inadequacy of speech and action. The history of such patients as a rule shows them to have been forgetful, dreamy, and lacking in vigorous mental grasp before the onset of severe symptoms. All this may be easily conceived as the result of a process similar in character to that found in hysteria, but differing in details and on the occurrence of such partial dissociations in persons undergoing deterioration, the hysterical stigmata found in dementia præcox

may be hypothetically explained. In passing, too, we may suggest that such a conception of the mental changes in the deterioration psychoses may throw light on their origin at times of special stress, on their tendency to improve under the influence of regular occupation and orderly life, and especially of the surprising mental improvement occasionally reported after acute illness or the administration of thyroid extract.

The next case, observed in the wards of the Manhattan State Hospital, shows a paranoid psychosis in connection with hysterical symptoms, and in her family history is found what appears to have been an hysterical mental disorder passing over into dementia. Three relatives of case No. 17 were insane and all were confined in the State Hospital at Morris Plains, N. J., and our information about them is derived from an abstract furnished through the courtesy of Dr. B. D. Evans. One, a cousin of 26, wrung her hands and cried. She said: "Oh, dear, I am wrong, I cannot collect my thoughts." In a few days she was "highly hysterical," and had convulsions in which she rested on her head and heels. At a later period she had a clear notion of where she was, but she stood all day, twisting and tearing her clothing. She dabbled in water and spoke in a whisper. Later she was said to decorate herself, to become careless of her clothing, considerably demented, untidy in her habits, and at last filthy. In these later stages her physical condition was poor from tuberculosis. She was nearly five years in the hospital and died shortly after her removal.

Another cousin, a younger sister of the above cited case, became melancholy and low-spirited after the removal of her sister from home. She had outbreaks of anger from the start and thought that her parents no longer cared for her. Two and a half years after admission she was noted as "a perfect dement with maniacal outbreaks. Cries a good deal. Filthy." Three weeks after this note she died of rapid phthisis pulmonalis.

An uncle of hers and of these cousins whose first attack was said to have followed an accident, was insane at 25, at 30, and at 49. The last attack kept him twelve years in the asylum. He was suspicious and had periods of mild excitement, but did not lose his interest in home affairs. Ultimately he was discharged improved.

Patient No. 17, aged 39, whose cousin and uncle are referred to above, was born of neurotic parents. At 30 she was married to a man older than herself, her only child died a few days after its birth and her husband is dead. Since the death of the latter she has lived with her parents. For fifteen

years (Her father says since she was eighteen) she has been depressed and nervous each month at the menstrual time. She often thought that she was followed or annoyed but these ideas each time passed away. For two years before admission she had thought herself hypnotized by a man across the street from her home and wanted the members of the family to keep away from the windows; and the idea persisted even when she moved to another place. Four weeks before admission she began to think that she was ridiculed in stores and elsewhere because of hypnotism exerted on her, and had many ideas of reference, and of mystic influence, causing her heart to beat very fast and causing sensations like electric shocks directed at her sexual organs. One of her ideas was that the tobacco advertisement of a man up a tree with a bull beneath, referred to her. The man in the tree meant herself and the bull on the ground represented the man whom she blamed for all her trouble through hypnotism.

On admission the physical examination elicited nothing important. She was quiet, compliant, and correctly oriented, but complained that she was brought to the hospital by deceit. Her tendon reflexes were greatly increased.

In the next few days she cried bitterly because she was deceived into coming to the hospital, was restless at night, insisted that her mother was outside, that she was hypnotized, that she had known her doctor elsewhere under another name. About a week after admission she wandered uneasily about the ward in a perplexed way, threw her clothing on the floor, then ran blindly about. That night she jabbed herself over the heart, saying that as others were going to kill her she might as well kill herself. In the morning she groaned, wrung her hands, but would not speak. Corneal and pharyngeal reflexes were lost and to pin-pricks she flinched only a little on the left side, and none at all on the right. In a month and a half she was apparently convalescent and was sent to the convalescent ward, but after a short residence there became fault-finding and complaining. She did not interest herself in anything; sat about all day in one position; refused food; and had to be spoon-fed. The hysterical stigmata returned and she would get out of bed, wander about the yard with her eyes wide open, staring vacantly, and apparently was in a dream-like state. It became necessary to tube-feed her, but she voluntarily regurgitated food and came to be but a mere skeleton. Often she would scream at the top of her voice and five months after admission began a monotonous reiteration of "No never; no never; no time never"; or "White, white, clean people." She lay with closed eyes and whenever interfered with, held herself perfectly rigid. Her malnutrition became extreme and her temperature 103 deg. At this

point the continuous bath was used with great improvement. She became cleanly, neat, took food, attended to the details of her toilet and evidenced some interest in her surroundings. For the later details of the case I am indebted to the paper on "Hydrotherapy," read by Dr. E. C. Dent at the conference of State Hospital Superintendents at Albany, N. Y., September 12, 1905.

Summary—A woman of 39 with intermittent paranoid symptoms since early life who shows also a profound hysteria. Other relatives have suffered atypical psychoses, especially one cousin who presented hysterical symptoms early in her attack, but whose insanity terminated in dementia.

Another case seen at the Manhattan State Hospital raises the question of dementia præcox. Her father was an alcoholic and her mother had been confined for seven months as insane in 1897-98, but since her discharge has been able to resume her humble place in society. The mother had thought that she was influenced, her mind read, had hallucinations of hearing, and thought that persons with flashlights were trying to take her picture for the World's Fair. She threatened suicide and sought the protection of the police.

Patient No. 18 was defective. She has been operated on for genu valgum; was brought up in a mission and was always headstrong and disobedient. She was admitted from a Magdalen home and told a story of a widower who wanted her to come and live with him. At the hospital she sat a great deal with placid averted face and sucked her thumb. Though on admission she gave a good account of herself, she quickly developed an averse attitude of mind and would rarely talk. As a rule she was contrary, and cross to other patients who got in her way, but occasionally she would chat quite pleasantly. No cutaneous anesthesias were found, and on only three occasions would she co-operate to test her visual fields, and at each of these times they were found contracted to as nearly as could be estimated without a perimeter, about five to ten degrees. She was regarded at first as a case of dementia præcox, but it could be noted that contrary to what is usually seen in that condition her resistance was not passive, and without emotion, but active, with outbursts of temper and related to real causes. If questioned or annoyed her seeming indifference quickly passed to a self-defensive attitude, and when she thought her rights were interfered with she became profane, struggled and screamed. This attitude seemed constitutional and perhaps had been fostered by her early life in an orphan asylum.

Summary—A girl of 21 of defective antecedents and personality who in peculiarities of manner suggests dementia

præcox, but the symptom picture is incomplete and atypical. She has contracted visual fields.

THE DELIRIOUS TYPE OF HYSTERICAL INSANITY

We have two examples of a confused delirium of an atypical character and without exhaustion features sufficient to explain them. One of these cases developed her delirium in the course of an undoubted hysteria and we think that the delirium must be considered a phase of the hysteria. This case is related besides to those just quoted, for a distinct paranoid persecutory trend has at last submerged all other symptoms of the case. The other, a young woman, developed her psychosis and as far as we can learn, on emotional grounds. During the period of confusion the cases closely resembled each other.

The first of these patients, No. 19, was admitted in March, 1905, aged 39 years. She is a native of New York State. No significant family history was elicited. She was married in 1895 and has had only one child, which was still-born after an instrumental delivery in 1900. Her husband has been told that when about twenty-four years of age after having fifteen teeth extracted under ether, she bled profusely and had sinking spells; and after five or six weeks went into a trance that lasted twenty-four hours. She lay so still that her doctor thought her dead. She remembers from this trance a vision of a beautiful stream, bordered by velvet grass with a beautiful city on the other side, from which came wonderful music, and to which a child beckoned her across. She could not go and finally came back to consciousness here. She was sick about three months in all.

Her insanity was preceded by indefinite worries in the summer of 1904 and by a baseless fear that her husband would lose his position, which led her to see his foreman about October of that year. A month later she had the first of a series of what were called nervous spells. She complained of a sensation like a log in her stomach, and that she could feel her muscles draw.

The attacks lasted half an hour and in them she walked the floor, clenched her fists and tried to tear her hair, so that it took two people to control her. She visited in Middletown in January, 1905, and the doctor who saw her in some of her attacks called them hysteria or hystero-epilepsy. At first she did not lose consciousness, later she did so, and lay for half an hour after an attack, and when regaining consciousness appeared dazed and wanted to know where she was.

On returning home she grew worse. She was given large doses of bromide and a severe acne ensued. Before committed she escaped from the house, ran half a block in her night clothes, and resisted return with an empty revolver. She talked of peculiar noises over her head, of the chairs being full of wires, that someone came and made holes in the floor and threw water over the bed. Upon admission she looked dazed and weak. Her eyes were partially closed and she would smile back in a feeble manner, then her lips would tremble and she would begin to cry. Most of the time she mumbled to herself and when she raised her voice, what she said was incoherent. She had no idea of time or place and spoke of herself by her maiden name. During the early days of her stay she was nervous, incoherent and confused, but able at times to give some account of herself. Later she refused food because she thought it was poison, until April 10, when she declined her breakfast as usual, but in the forenoon there was a sudden change in her manner. She described it as like coming out of a dark room into the light. Her attitude changed entirely. She was willing to eat, talked pleasantly and said that she had only a partial comprehension of her surroundings up to this time.

She continued pleasant, agreeable and appreciative until May 4, 1905, when she was paroled home, and a month later was discharged recovered. Three or four weeks later when it was proposed to return to a town where she had formerly lived she objected, saying that she had not been treated right there. She went, however, and soon came to think that the neighbors annoyed her and were trying to blackmail her. She sat by her window, which opened on the street, and thought that she heard passersby talk of arresting her. In July she suddenly rushed from the house because she thought the police were coming to arrest her, and she was found some hours later in a field two miles away. She was excited and confused, and objected to returning home with her husband, saying that he was conspiring with the others to put her in prison. When calm again she had no recollection of the episode nor of any of its events.

Some days she could do her work, but more often she lay about the house, inefficient. Sometimes she talked of suicide, and sometimes would brood and weep for half a day. She had three or four of her spasms, the last two months before admission. In the spasms it is said that she felt nervous and faint; would pull her hair, clench her fists, then get up and walk around. They lasted a quarter or half an hour and she did not remember, afterward, what she did while in them. They were worse at the menstrual period. While still at home she took sudden dislikes to help brought into the house, so

that they could not keep anyone to care for her, and finally she consented to return to the hospital and was readmitted on October 19, 1905.

On readmission she was rational, coherent and oriented, but a little depressed and quite without cause, apprehensive as to her husband's safety. A few times she had "nervous chills" and felt faint, and although she denied hallucinations she talked a little of this being a Catholic institution and of priests here and of people talking outside her window.

In January, 1906, she had a severe cold and lost 15 pounds, but her amiable mental attitude remained. Early in February she called attention to red spots on her left arm and said she felt very tired and weak, had shooting pains up the arm to the ear, and that the arm was numb. The arm and left breast were anesthetic to pin-pricks and on February 15, when the anesthesia was charted, it was found to include the whole of the left half of the body above the level of the crest of the ilium. This anesthesia remained unchanged until late in April, when it was twice dissipated by the electric spray, but each time returned in a few hours. She was at this time taken home on trial for ten days, but would not remain there with her family, and was brought back. Since her return the anesthesia has not been noted, but she has been very averse in her attitude. She has sulked most of the time in her room and will not speak pleasantly to anyone. "No hands to enemies," she said to a physician who extended his hand and tried to be friendly, and she demands of all that they shall let her alone. She will not read, sew, or occupy herself in any way. Her husband is blamed when he visits her, for all her trouble. In September, 1906, in a fit of rage, she declared that the patients here were drugged and deprived of their proper rights. That she and others here have large inheritances that those in charge of the institution were trying to get. The sole aim of the physicians was, she said, to get them out of the way. A vaccination a few weeks before was no other than an attempt to introduce poison into her system and, she asserts, she narrowly escaped death from hydrophobia.

Summary—A woman of 39, who, fifteen years before, after having some teeth pulled under ether, was for some days in a state of trance. After indefinite worries in the fall of 1904 she developed hysterical convulsions, then a confused delirium of more than a month in duration, which suddenly ceased and gave place to apparent recovery. On reaching home suspiciousness of others, present at the start, returned; and once under fear that the police were coming to arrest her, she rushed into the fields and was lost for several hours. She did not remember the episode. Since then characteristic hysterical anesthesias have been present, and a suspicious paranoid trend has gone on to delusional elaborations.

The case just considered has through a course marked by symptoms which distinctly show the hysteria, gradually undergone an entire change in the attitude of the patient toward the world. Out of a hysteria she had developed what a few years ago we would have unquestioningly have been content to call a paranoia. From a pleasant, efficient, home-loving woman, it is said a notable housekeeper, she has slowly changed. Now she is suspicious, surly and idle and has evolved a systematized persecutory trend. Although this change has not occurred suddenly with some emotional or physical shock, it is none the less a complete change in the personality of the patient. It is this type of change of personality upon which Bianchi lays emphasis and goes so far as to assert that it is hysterical, where he has shown no attacks or physical stigmata, so to speak, of hysteria. The present state of the patient may be compared to that of B I in Prince's case.

Case No. 20, an unmarried woman of 24, was admitted at Middletown on April 14, 1905. There is a family taint toward nervous disorders. Her grandmother at 87 was said to be in failing mind, both her parents were spoken of as nervous persons, and a paternal cousin was twice at the hospital with an indefinite psychosis from which he fully recovered. Not long after his second discharge he succumbed to tuberculosis.

As a child she was delicate and so excitable and nervous that she could not attend school regularly. All children's diseases were very severe. She was set in her ways and being an only child was humored and not disciplined in self-control, but she was social and fond of many friends. She had no love affair. Puberty was established at thirteen and menses were regular and profuse until the onset of her present attack.

In March she was present at the death of a near friend, and afterward was depressed and "all worked up." She could not sleep well, trembled at times, dropped things and had hysterics. The summer she spent in the country and in the fall, though not sick, was still inclined to cry at any trifle. At Christmas time she was visiting relatives, and when she heard that a niece of whom she was very fond, was sick, she cried excessively, though assured that the sickness was slight. Two days later she came home and finding the child improved she ran errands, visited the public library and helped her uncle balance some accounts and at about 10.30 o'clock retired, apparently as well as usual. At midnight she came into her mother's room and insisted that her mother was sick, talked rapidly and was so excited that they had to hold her. During succeeding days she could not sleep. Sometimes she sang and sometimes cried. She talked of scandal and accused herself of sins that she did not commit. Sometimes she thought that she was married and had children and at other times that her food was poisoned.

As she did not regain her right mind at all she was taken after a week at home to a sanitarium, where on admission it is stated that she was greatly excited and "in a state of alarm." Insomnia and constipation were obstinate there. She seemed to have visual hallucinations and "paroxysms of hysterical contractures of various kinds." She said that she did not know her name, repeated the same words constantly, and required the restraint of the attendants to keep her in bed and clothed. She remained in the sanitarium some three months without improvement until April 14, when she was brought to Middletown.

Here she was found in good physical condition. Sensibility to pin-pricks was present, but everywhere slight. Her expression was as if dazed and confused or sleepy. She gave the year as 1904 and her age as one year less than it really was, knew her name and her home address, but nothing of the sanitarium. She resisted what was done for her and a great deal; took off her clothing and rubbed her body. Occasionally she became noisy, but as a rule said little unless addressed. Her speech was usually a peculiar drawl and was often limited to a prolonged "w-h-a-t" to all questions as if she did not quite understand. Often she asked for a knife to kill herself or asked the nurses to kill her or cut her in pieces, or again she wept because she would never get out alive. Her motions were usually slow and constrained as if very drowsy. For weeks scarcely a stitch of clothing could be kept on her, and without being immodest, her modesty was entirely lost. When for any purpose the nurses took her from the bed she made herself rigid, as a rule, and in bed she held her knees drawn up and resisted straightening them. Sometimes her arms kept for a few seconds a position in which they were placed.

A week after admission while fearful and awake at night she told her nurse of seeing coffins and dreadful things, and said that her face was on her back and she was turned around. A few days later she got up suddenly and tried to break a looking-glass with a broom, then rushed around pulling out bureau drawers, struck the nurses who interfered and kept repeating, "Give me fire," "Burn me up."

April 27 she was brighter in the morning and entirely coherent in the afternoon. She had no recollection of how or when she came. Two days later, though again confused, she explained that she stiffened out because she saw "all sorts of horrid things, snakes and everything!" After this she was as bad as ever again and with much the same symptoms. In June she was dressed and taken out of doors daily, and gradually she came to know where she was. In July her memory for her peculiar actions was fading or denied. She gradually improved during the summer, but was listless and always self-

indulgent. Her interests became more and more acute and when paroled home August 31, she was in excellent mental and physical condition. She was discharged recovered. More than a year later she is said by the family to remain entirely well. All her interests are preserved and she is just as she was before the attack. She is also physically well.

Summary—A delirium with complete disorientation and confusion in a single woman of 24. It arose after retiring at night after a day with unusual but not great emotional stress. This delirium with one short remission continued for six months.

Case No. 20 had a delirium very similar to that in case 19, and she came out of it into a state of mind which seemed normal, as did also 19, though the latter has undergone further development. The circumstances of the onset and the marked remission of a few hours' duration, followed by a lapse again into a state of complete disorientation, we regard as additional reasons for thinking the disorder probably hysterical in its nature. It will be noted that the symptoms quite fail to fall into any of the ordinary symptomological groups.

Doubts, fears, impulsions and obsessions seem to be common enough in the ordinary symptomatology of hysteria, but among our hysterics committed as insane we have found but one who has presented this complex at all prominently, and in this case the complex of depressed symptoms was also in the foreground. The case next to be presented was when first admitted considered a compulsive insanity.

Case No. 21 is a woman of 21 years. Insanity in the family was denied. Her father was a man of ability and she is one of fifteen children, all of whom were said to be in good health. She was of cheerful disposition and learned well at school; became a stenographer and was employed in this capacity for two years, but gave it up about two years before her admission. She worked some the first of these two years, but was more or less deeply sunk in morbid introspection and gave up successive places from ennui. This made her inefficient and work was definitely abandoned in the spring of 1904.

After admission it was learned that even as a child she had obsessions. One given as an example was that she must smell the window shades. At 17 she had left the high school because she was nervous and tired of life, and morbid because she had cheated at an examination. No cause was known, but a possible cause for mental disturbance was to be found in a love affair with a young Jewish lawyer whom her friends opposed but with whom she kept up an acquaintance. Sexual relations and masturbation were denied.

When admitted in August, 1904, she complained bitterly of a gnawing feeling in her chest, of oppression and of a lump

in her throat. Concentric contraction of the visual fields to 40 degrees was found, but no anesthesia of the skin, though she picked herself sore. She was extremely restless, walked up and down in agitation and often wrung her hands. Her expression was perplexed and worried, and especially when questioned she bit and moistened her lips very often, corrugated her forehead, drew down her upper lip, dilated her nostrils and moved her ears. "I get so confused," she said when asked to tell about herself. "There is nothing to tell. I can't connect it all. I have been through so much. . . . I felt unhappy—a peculiar feeling there (motioning to her chest), I tried to reason things, then I stayed home a year but I wrapped myself all up in myself—you know what I mean." She was perfectly oriented, could calculate, took stenographic notes and transcribed them correctly, and, at the same time, was afraid that she would not make herself understood and was under the necessity of trying to explain again and again; she analyzed her symptoms and what she had already said. Her mood was very unstable. Sometimes she was fairly cheerful for a little while, but her usual feeling was one of despair. She spoke of a spiritual load on her chest and of her belief that it signified that her soul and her body were separated and could not be brought together again, until she "dies to self." All her talk was of regrets, repinings and despair of the future.

No hallucinations were present. Her grasp of her surroundings was excellent; she made fine psychological distinctions and she insisted on them. She complained of inefficiency while really doing very well. Her accounts of her past life were full and complete except for the two last years, of which her account was hazy.

She did not improve until the next spring. She was continually pondering one or another of her psychological problems, but always with the conclusion that she did not know. Thus in February, 1905, while changing sheets on a bed, she did not know the next instant if she had changed them or not. She said nothing was real and talked in a fragmentary way about the Holy Ghost going away, about convulsions, about a free agent, about the time past seeming like a dream, etc., etc. Breaks out crying, "Oh, you don't understand. It came to save me and it left! Oh, dear! Oh, dear! Man is a free agent. Oh, you can't understand! Oh, I know that the Holy Spirit came to save me! Oh, oh, it is all so funny, funny, funny! To me things are funny—to me (gesture) things aren't real. You don't understand. You can't understand. . . . Oh, I'm talking crazy now. When you are crazy you don't know it. . . . I talked about God and yet I really, really—I—I—oh, I know I mean all right. No, I don't know what I mean. . . . Nobody understands me—the doctors

are getting all mixed up." As she talked she rubbed her hands, and pounded on the windows and corrugated her scalp. She tried over and over to explain, stopped and started again but could not clear up anything.

She was often willful and obstinate and careless of her appearance, willing to wear any old dress and neglected to becomingly arrange her hair. Sometimes she accused herself of being the cause of the death of her father and talked of her own unworthiness. The contraction of the visual fields continued.

In May, 1905, she was noticed to be less preoccupied, and began to help with the spring house-cleaning. In two weeks she had excellent insight into her former state and was assiduous in helping other patients to overcome their bad ways and false beliefs. When questioned at this time she said that her trouble began with being depressed and things went on and got darker and darker. She did not know any real cause. Then she began to feel mixed up and to think that every one else was mixed up. From that she began "cutting up" at night so that the family could not sleep, she lost interest in her work and thought and talked of herself all the time.

On May 29 she was paroled home and 30 days later discharged recovered. We only know of her subsequent history that she has married.

Summary—A single girl of 21 of good ability but who as a child had some obsessions and who was nervous as a schoolgirl. For two years before admission she had been introspective and increasingly unable to employ herself. After admission for eight months she was in distress, continued introspective and under the constant necessity of debating problems and of trying to explain, and in doubt about herself and her actions. When she really began to improve, the symptoms disappeared in about two weeks.

By some this case was regarded as a case of compulsive insanity, but we think that it may be further differentiated. With our conception of hysteria it is easy to conceive how fears and compulsions may arise as part of an hysteria; that is just as subconscious ideas are projected into the field of consciousness by post hypnotic suggestions. As hysterical stigmata were for a long time present we look upon it not as a psychasthenia with hysterical symptoms, but rather as an hysteria with some symptoms such as are commonly found in psychasthenics. The compulsive features: obsessions in childhood, a feeling that she is not saying things right, and that she must explain are but a limited part of the mental disorder. Case No. 9 already quoted presented temporarily distinct imperative acts.

HYSTERICAL MANIA

Reference has already been made to a case perhaps hysterical in character but with symptoms such as are found with the manic phase of manic-depressive insanity; a case that is unique in our experience and which does not furnish ground for treating all hysterical insanity under the head of "Hysterical Mania," as we have found some authors do. This case terminated in recovery and thus far has not relapsed.

Case No. 22 is a woman who was twenty-three years of age when admitted at Middletown on July 14, 1905.

She was a foundling adopted when 5 years old and had lived with her foster mother and aunt until eleven months before admission when she married a man who was intemperate and unkind to her. Her child was born July 1, 1905; following it her husband was in the house drunk a good deal. It is said she would not keep to her bed as she should, but she says that she had to get up and care for herself. Her foster mother who was with her after the confinement says that the attack was precipitated by her husband, who came in with another drunken man, quarreled with him and so frightened the patient that she ran up stairs, and was only restrained by force from jumping out of the window. Her doctor says her confinement was easy and the puerperal period free from sepsis or fever.

The fourth day after confinement she complained of a sensation up her neck as if a stream of cold water were falling on it, and this sensation shot into her head. The seventh day she talked in a silly manner, disarranged the furniture, laughed and cried alternately, and had what her husband called laughing hysterics. The medical certificate of July 11 says that she is incoherent and does not keep still long enough to talk, that she laughs and cries, walks the floor, will not be dressed and does not sleep. On the way to the hospital she repeatedly started to undress herself. She complained of her back and head, begged to see the priest. Asked fellow passengers if they were Catholics; she swore, cried and screamed alternately, slapped the nurse and tore her waist.

On admission she was perfectly oriented, but slovenly and immodest, complained of the nurse, fell on her knees and prayed, and again laughed outright and said, "I am so overjoyed I am satisfied to stay." When the mental status was taken she was disorderly in conduct, she was elated in the main with sudden crying spells and it is said continually trifling and distractable, but examples are not given. The physical examination disclosed nothing significant.

At the hospital she was so disturbed that hot packs were

used and on July 19 she collapsed in a pack, but the next day was very active. She greeted the doctor's good morning with, "Oh, go to hell!" Then slapped him on the shoulder, held her hand over her forehead and said her head ached. Then unbraided her hair and said laughingly, "I guess I am busy." She continued to talk, swear and laugh a great deal. August 14 a sample of her stream of thought was set down as follows: "I have had so many names since I have been here I do not know one from the other." Asks the doctor what she is scribbling. "I had dreams last night. Who in hell are you anyway? I have got lice. I had them when I came here." Laughs. "That is all you want to know. Everything from tip top to top tip, from right shoulder to left; from your gut bone to your gut bone. If there is a hair on your toe they want to know it, so pump on." She cries and asks not to be hung to-day, "You have tormented me so."

She grew slowly quieter and the early part of September lay most of the time with closed eyes in bed, smiling and occasionally whispering a little. She had to be fed and soiled the bed. Once when the piano played she sang out loudly and clearly. Ordinarily the flies walked unregarded on her face. She never cried. Usually she resisted being taken from bed but occasionally she walked the heads of the beds and turned the pictures to the wall. September 22 she was anesthetic and extremely cataleptic. She gave her name "Tessie" and said that she was in "heaven." September 25 slight corneal and conjunctival reflexes had returned. The catalepsy and skin anesthesia grew gradually less over two weeks, and October 8 were gone. She would then answer brief questions for the nurse, and two days later she ran about and splashed the water while in her morning bath. This she did repeatedly and rubbed her milk in her hair, but said little until October 27, when she became excited while the nurse was working with another restless patient, called the nurse obscene names and swore at her in an amazing way. Both before and after this time she rarely spoke aloud, saying that a nurse had hurt her neck, but on this occasion and a few times when singing her voice was strong. A letter she wrote November 6 was very dilapidated, and disclosed poorly elaborated paranoid ideas of her illness and of fear of an operation. She was restless and mischievous at this time, smiled at the doctor but would not talk, and when visited November 6 by her husband, who was intoxicated, she scolded him well and saw to it that he got away in time to catch his train.

November 7 after breakfast she remarked that the bathroom needed cleaning and proceeded to do it well. From that time on she has worked well, tending to exceed her strength. A long letter of November 18 is coherent and contains sensi-

ble reflections on her past life with her unworthy husband, and plans for the future. November 25 she introduces a good range of topics into her talk and when questioned says she thought men were fighting and that war was going on here. "I used to imagine that I was in church and could see all the pretty things like pictures on the ceiling." She also remembers thinking that she was in heaven and walking on the head of the beds. She explains, "I had an idea I had to do it." "That I was on the stage like and had to practice."

She remained because of her unfavorable home surroundings, at the hospital until Dec. 30, 1905, but without further symptoms of mental disorder.

Summary—A young woman who was frightened a few days after the birth of her only child, and within a week began to be insane. On admission she presented the symptoms of active mania. Two months later catalepsy and anesthesia displaced the mania. Soon after she recovered.

The diagnosis in this case is not clear. Thus far we have no evidence of manic-depressive alternations, yet the symptoms in the early weeks after admission were so like those of an ordinary mania that they received no especial study until the development of such characteristically hysterical features as catalepsy and anesthesia. The early symptoms, the course and the termination all combine to remove dementia præcox from the field of consideration. Some features of the onset were just such as might well usher in an hysteria: the fright when in a weakened condition, the complaints of peculiar pains, then what her husband called laughing hysterics. The notions about her neck later and the loss of voice are additional hysterical traits, and she came out of the attack just as hysterical cases often do. On the whole we think that the whole psychosis was probably an hysteria.

HYSTERICAL STUPOR

Hysterical states of stupor seem to be very common, for, of short duration, we see them occasionally among our woman employees, and it is to this class that many of the cases of prolonged sleep reported in the newspapers are said to belong; and certainly the accounts of these cases frequently mention other hysterical attributes. It seems, however, that as this group of symptoms is usually manageable in the home, that cases of this character are very properly not regarded as insane and suitable for commitment by either the patient's family or her physicians. Therefore we have but the most limited experience with stuporous hysterics. We remember

to have seen one such woman at the Manhattan State Hospital. The only case in recent years at Middletown is that of a man admitted as long ago as October 31, 1898. The details are very incomplete.

Case No. 23, a French Canadian man aged 24 years, had been found wandering along a railroad track in a dazed condition, was noisy and excited when arrested, and called for a gun to shoot Spaniards. He had an untouched bottle of whiskey and it was supposed had been drinking, though he did not smell of liquor. When taken into custody his excitement disappeared, he would not talk, but sat in his cell regardless of everything and automatically picked his clothing to pieces, thread at a time. After his admission here he still refused to speak, would lay in any position placed, and sat looking at his food without eating, unless some was first placed in his mouth, and then he ate ravenously. This condition continued for ten days, when he developed the symptoms of the passage of urinary calculi. The severe pain gradually aroused him. On his arrival he did not know where he was, had no recollection of events since some time previous to his arrest when he was drinking in a city some two hundred miles away.

He gave the correct address of his friends and asked that they be communicated with at once.

After coming out he was found irritable, petulant and jealous in his disposition. He could not endure the thought of anyone having extras or privileges of any kind unless he had them also and more beside, and he found slights and causes for offense in many little innocent acts of others. On two different occasions he had convulsions of hysterical type. After eleven months he was allowed to join his family in Canada, where we learned in a few weeks that he had been admitted to a hospital for the insane with symptoms similar to those that he had when he came here. We have been unable to secure any further account of him.

Summary—A man of 24 who while drinking developed boisterous conduct, which suddenly subsided to a condition bordering on stupor in which he sat and automatically picked his garment to shreds, or did nothing. This passed away during a period of severe pain. He then presented an hysterical disposition and a few hysterical attacks.

HYSTERICAL DISPOSITIONS

One more group remains: the cases without well-marked attacks that are commonly spoken of as hysterical or sometimes as of hysterical disposition. It is from these cases that the hysterical manias of former years seem to have largely

been recruited, while some of those in whom we have been able to find diagnostic traits of hysteria have not been recognized as hysterical at all. In the cases we are now considering, the major stigmata are not found. They are ordinarily fully aware of their surroundings; and are able to talk in a plausible way, but they are self-centered; interested in their own symptoms and ailments; jealous of any attention shown to others; of uncertain disposition, often most affable, but subject to violent if transient anger on slight provocation; unable to apply themselves steadily to tasks and correspondingly inefficient and untrustworthy; anxious to be noticed, to be at the center of passing events, and capable of any foolish action, any untruthful story which will attract attention to themselves; and perhaps in many instances no longer able to fully differentiate their fictions from objective truth. All physicians are familiar with cases of this kind, and they are commonly regarded as hysterical even when physical signs of hysteria are not noticed, for such character is often found in hysterical persons, and persons with such traits are subject to hysterical attacks. Perhaps it would be better not to speak of persons of this character as hysterical in the absence of the usual signs, but on the other hand, these traits when well developed are probably mental stigmata, that is signs of a narrowing of consciousness and of defective association and synthesis of ideas.

There is no need to give such cases in great detail. They are too familiar. To illustrate the group we will mention briefly but one case.

Case No. 24, a girl of 17, was brought up in hardship, first in an orphan asylum and later by a step-father. Overwork, meager schooling, cruelty, and much wrangling at home were the surroundings of her childhood. Later while working out she became zealously religious and broke down while keeping Lent with morbid strictness. She worried over her course while at home and became overwrought and laughed and cried alternately. She said that she felt cranky, she wanted to jump out of the windows; she trembled all over and she talked of her employer looking like Jesus Christ, and of his little boy being too good to live. There is no history of distinct hysterical convulsions or of attacks in which stigmata were shown, though symptoms of this nature may have been overlooked. At the hospital she was usually happy and helpful to others, but she cried easily and was sometimes taciturn

and sad, and she was always unstable. During the 19 months she was at the hospital she gradually improved in poise and self-control, and was finally discharged, recovered.

In reviewing the cases presented they fall naturally into two main groups, first those hysterical cases not very closely resembling other forms of insanity, and second, cases with a more or less clear hysteria, but with features that might readily lead to their consideration under other heads in a purely symptomatological classification though their fundamental traits seem hysterical in most instances. The second group will be further subdivided, making with the hysterical dispositions, seven heads in all. These are not introduced as a classification of hysterical insanities in any sense other than a brief résumé of the cases actually found, with the idea of pointing out what diverse forms of symptomology hysteria may assume and some of the situations in which it may be found.

The cases are then:—

First: A group of ten hysterical women all with definite stigmata, whose psychoses present certain features in common; emotional stress is very prominent as a cause; the patients though sometimes violently insane, tend, either in intervals or at all times to be natural in manner and point of view about ordinary things; the symptoms are of varying intensity; and the events of the more intense periods tend to be forgotten. On the other hand, the symptoms vary from an active delirium to restless melancholia and to passing or more or less persistent delusional states.

A second group presents one case of so-called involutional melancholia of hysterical nature, and other cases of depression which do no more than raise the question of hysteria. These patients apparently have a melancholia of the anxious type often seen in the period of involution, but have had similar and milder attacks in earlier life and without any tendency to an alternating manic phase.

Third: Cases resembling dementia præcox and the paranoid psychoses. In our case and in other members of her family the symptom groups are intimately combined.

In the fourth group are two cases of confused hysterical delirium; in one case with physical stigmata, in the second with mental stigmata only. The first of these cases has developed at the last a systematized paranoid trend.

Fifth, a case of impulsions and obsessive ideas in an hysteric.

Sixth, a manic attack which during convalescence showed hysterical traits and which was perhaps hysterical also at the onset.

Seventh, the hysterical dispositions; cases with only mental signs of hysteria and without such attacks as permit one to make a positive diagnosis. The cases are common and well known, and if not really hysterical are closely allied to the hysterical states.

No cases approaching a complete alternation in personality have been met. The single possible exception, a man of 32, was at the hospital so short a time and his history was so meager that nothing of value can be reported.

Our efforts at attacking the underlying mental cause of hysteria by methods such those advocated by Janet, Jung or Freud, have been few, and there are no successes to report. Most of the cases have improved so rapidly when placed in as favorable surroundings as possible, with abundant nourishment and friendly counsel and advice as to make any such attempts seem not only needless but perhaps harmful. Other cases have proven too inaccessible. We think it important that persons who tend to be hysterical be trained in habits of regularity, order and self-control.

These cases show that a comprehensive view of the insanities must include certain hysterical disorders. In some instances it is evident that what we have to deal with is hysteria and not some form of insanity as modified by hysteria. For while excitements occur with insane hystericals, they do not, except in case 22, which may or may not have been hysterical in all its features, present psychomotor activity for its own sake, elation and flights of ideas such as we now regard as manic features, but rather an excitement accompanying one or another kind of unpleasant emotion. The hysterical depression is just as distinct from that of the manic-depressive insanity and the more typical cases of dementia præcox, and resembles more that found in the depressions of later life, and this probably because both, in part at least of their symptomatology, are the reaction of the personality to unhappy and distressing thoughts, and perhaps oftener than is suspected to submerged psychic irritation.

On the other hand is the possibility that there is more than hysteria present in some of these cases. Thus in Case 22 the history does not adequately tell if hysteria was present at the start, nor if the symptoms apparently maniac appeared primarily or as symptoms in the course of an already established hysteria. In fact little of the real nature of manic attacks is known, and nothing is known inconsistent with the conceivable possibility that a mania such as develops independently, may also develop during an hysteria. Likewise we have no reason to assert that an hysteria may not come on in a person already manic. This case suggests one or the other possibility, but the incompleteness of the record and of our knowledge of the problem does not permit a conclusion on this point. It is entirely safe to say, however, on the basis of the whole group of cases and on our larger experience with manic attacks, that such a combination of symptoms must be a rare exception. In dementia præcox there seems to be a more intimate commingling of symptoms in certain cases as already noted under its appropriate head, a fact which goes to support the conception of dementia præcox advanced by Meyer at the Toronto Meeting of the British Medical Association. In these cases it appears that there is besides the systematic dissociation present, some additional disorder, functional or organic.

The mechanism of the production of hysteria is better known than that of the other mental disorders. The origin of its salient features in divisions in consciousness has been repeatedly shown. The cause of its more characteristic symptoms is found in thought. Few workers seek its cause in this or that unknown toxin. We can see that it is functional in the same sense that normal thought is functional, and that we have no more reason to look for a poison in the system or a change in the structure of the cells upon some given day, when a patient has become hysterical, than we have to look for them after any other change in opinion or point of view. We believe, of course, that some change in the physiological chemistry of the brain occurs with every thought and emotion, just as some change accompanies every movement of a muscle, and the change in hysteria at the moment of its inception seems purely a mental change. This does not alter the fact that mental stress, whether it be hysterical or not, sets up

nutritive disorders and secondarily extensive changes in the tissues and fluids of the body.

With the functional nature of hysteria in mind it is possible to regard the mental disorders from a new point of view, and to inquire to what extent they may be thinking disorders as well, and functional in the same sense. To be sure our knowledge does not suffice to answer the question raised, but such a query deserves consideration along with the hunt for bacterial poisons and cellular degenerations. Perhaps in this may be found the explanation of the surprising amount of old knowledge and opinion to be had from almost any long standing dement, if we can by any means temporarily overcome his lethargy, and of the improvement which comes when the patient's interest can be aroused. It seems in part at least as if such patients had merely fallen into bad habits of thought.

It is enough if through the concept of hysteria the physician's mind may be freed from a too exclusive pathology of toxin and cell change, and he may have a rational basis upon which to try what can be done to put the insane patient mentally at ease; to seek for and to remove hidden or obvious causes of irritation; to restore his self-respect; and to show him outlets for useful and pleasurable activity.

The recognition of hysteria as a psychosis we think tends to bridge the gap which has been allowed to grow between diseases of the body and diseases of the mind, and to give a wider outlook and a new point of view from which to regard the other insanities. It makes it possible to correlate the insanities with what most physicians in their practice and thought regard as nervous diseases, and through them the physician and the patient's friends can come to some conception of what goes on in the insane person's mind, and of how such insane thoughts can arise and be entertained. It need not be supposed that the hysterical process is the only one through which mental aberrations occur. Rather in the past its rôle has been almost totally overlooked. The studies in hysteria should be applied to the problem of insanity, and the functional element sought in every mental disorder, and its mechanism be made clear if possible whether it be hysterical or some other as yet unnamed type of reaction. Thus we may hope to attack psychically in a rational way the psychic element of psychic disease.

Society Proceedings

AMERICAN NEUROLOGICAL ASSOCIATION.

Held in Washington, May 7, 8 and 9, 1907.

The president Dr. Hugh T. Patrick, in the Chair.

(Continued from p. 117.)

Loss of Comprehension of Proper Names. By Dr. Frank R. Fry. (See this Journal, 1907, p. 617.)

Dr. M. Allen Starr said this was a very interesting case. It is unfortunate that in the study of aphasia too little attention is paid by students to building up speech in the infant's mind. Now those of us who have watched the development of vocabulary in infancy will recollect that the first words spoken are nouns. A child at the age of two does not put together two or three words. He names individuals and single objects, and his whole vocabulary is these nouns. It is not until a child is two and a half to three years old that the connective parts of speech, as "mama is sick" will come out. Dr. Starr said it seemed to him therefore that in the study of aphasia we must regard the manner of the building up of our processes of language, and it would look as if this defect in the use of nouns was a defect in the most fundamental of the processes of language. Now we have the converse in other cases. Dr. Starr said he had had a patient who was educated or grew up on the borders of France and Germany, in Alsace, and as a little boy he spoke French. As he got to be a little older, eight or ten, a result of the German war was a change in his language and he had to learn German. Then he came to this country and finally learned English. He had a gradually advancing aphasia in which he lost his English. Then for a time Dr. Starr had to talk German or French to him. After that he lost German, and Dr. Starr had to talk French to him entirely. Dr. Starr said he watched the case for three years and observed progressive loss of speech until just before death the man was completely aphasic. The last acquisition of language was blotted out first. The fundamental seemed to be lost to a certain extent, but the later acquisition was lost first.

Dr. B. Onuf said he had been much interested in listening to Dr. Fry's paper, and he would try to classify the case from the observations which Dr. Fry had reported. It seemed to Dr. Onuf that one ought to think chiefly of two possibilities; one, that this was a case of optic aphasia as Freund has described it, and the second possibility that there was a small circumscribed lesion of the third frontal convolution. In the case of optic aphasia we are likely to find in addition to the aphasic disturbance also visual disturbances, such as hemianopsia, or soul-blindness, more or less pronounced. Also there is very likely to be in many forms some word-deafness. In cases of "frontal aphasia," as we might say, if the lesion

is small, there may be at first complete aphasia, and then very quickly a return of the vocabulary takes place, and the functional loss may then manifest itself almost in the manner that Dr. Fry had described; that is, the words that are acquired with most difficulty are those that will be lost chiefly and perhaps solely. In distinguishing the two forms it is very essential to make a careful study of accompanying symptoms. One is more likely in frontal aphasia to have symptoms on the part of the centres situated in the lower part of the central convolutions; that is, on the part of the facial, on the part of the tongue, and on the part of the pharynx; while in the forms of optic aphasia we are more likely to have hemianopsia, hemianesthesia and word-deafness. In a frontal aphasia there is also agraphia. From Dr. Fry's description it would seem that there were no visual disturbances, no soul-blindness, no word-deafness; and that would make it more probable that the lesion was situated anteriorly; *i. e.*, in the third frontal convolution.

Dr. G. M. Hammond said the case reminded him of one that he reported of a driver who was struck on the side of the head in the temporal region by another man who had a loaded whip-handle in his hand. The man did not become unconscious for some twelve hours. He then had a convulsion and became unconscious, but regained consciousness very quickly. He was taken into the hospital. He was somewhat stupid, and could not say any proper names. He could say all other words except proper names. In describing how he was hurt he said, "a (indicating a man), hit me on the (pointing to his head) with a (imitating as near as he could the whip-handle)." He was operated upon, and a fracture of the temporal bone and a sub-dural clot covering the superior temporal convolution were found. The latter was removed, and in the posterior third of the superior temporal there was a hole in the brain which extended inward about an inch, and was about as big around as a lead pencil. This man was under observation for about two years and a half, and he slowly regained power of saying proper names. At no time was there any loss of the idea of things. He would take up a cup and saucer and use it; he could not say cup and saucer to save his life. At the end of a year's time he could say these words, but it was still impossible for him to use words of three syllables. At the present time he has regained his entire power of speech. Whether the word center for proper names is exactly in the area involved in this case or not it is difficult to say. The probability is that it is not always in the same place in all individuals. As Dr. Starr has just said, the fundamental speech of a child is the learning of proper names, and probably the memorization of these names occurs in the cells of the superior temporal, which are in the highest state of development at that time. Now this may not always be exactly in the same place in the temporal lobe. The cells in the temporal lobe may be more developed in one child than in another, and hence may not always have the same topographical locality in all individuals, but it would seem in this case, and others, that the proper name center is in the superior temporal convolution.

Dr. H. H. Hoppe said that in connection with this subject, and especially in connection with what Dr. Hammond had just said, he (Dr. Hoppe) would like to briefly mention a case which was under his observation for a good many years. The man was injured by having a brick fall on his head, and it produced a compressed fracture of the temporal bone over the temporo-sphenoidal lobe. The interesting feature in this case

was that the fundamental language in this man was German, and his acquired language, which was very imperfectly spoken, was English, and he lost his naming sense in the German and not in English. For instance, if shown an object, in a very broken way he would give the English name for it, but had lost the German name. This might indicate that there is probably a separate area for naming in the different languages. He spoke rather glibly in broken sentences, and for two or three years, as long as Dr. Hoppe knew him, he never regained the power to speak nouns in German.

Dr. B. Onuf said that Dr. Hoppe's observation may find some psychological explanation; that is, uneducated people in learning a foreign language are very likely to unlearn their own; in fact they come to the point where they cannot speak any language.

Dr. Fry said that he had carefully examined his patient, and there were no visual defects that he could discover. He was disposed to accept Dr. Onuf's idea that this must have been a lesion in the anterior lobe; *i. e.*, if there were any process which might be called a single lesion; at any rate he could not conceive of the process as being other than psychic in a sense, or involving primarily the region of control or formation of language.

The Classification of Psycho-Neurotics, and the Obsessional Element in Their Symptoms. By Dr. G. L. Walton. (See this JOURNAL, 1907, p. 489.)

The Use of Social Intercourse as a Therapeutic Agent in the Psychoneuroses. A Contribution to the Art of Psychotherapy. By Dr. Sidney I. Schwab. (See this JOURNAL, 1907, p. 497.)

Dr. A. R. Allen said he did not know to what extent this manner of treatment is common or uncommon among neurologists here in America, but seven years' association with Dr. S. Weir Mitchell in his office has shown him many such cases; in fact, he could say that it is the usual modus operandi of treatment of these cases coming to the office. As to the question of a year and a half or two years or three years under observation, he said that whereas the case may stay in the town and be a regular office patient for three or four or five months, it is a frequent practice to send that case out into the world, making the patient feel that the office is a harbor in case of rough weather ahead, and this relationship lasts for two or three years, and in almost every case with the greatest benefit. It is very interesting to see these points put down upon paper, because it is too little done, and one reason we as neurologists do not keep these cases is because men do not realize the importance of this therapeutic treatment. After all, we get very many more cases of neurasthenia and hysteria or neurasthenia combined with hysteria than we do of cortical lesions.

Dr. S. E. Jelliffe said it seemed to him that Dr. Schwab had emphasized, as he so frequently has at previous meetings, the desirability of a little more definiteness in therapeutic work in general. It is well known that the methods as outlined are widely employed, but are employed by many in a haphazard manner. These patients are told to go to the theatre or to read books, without telling them what to see or what to read; to go about and see their friends, without ourselves having investigated the friends. The ideas that Dr. Schwab has formulated and put in such

definite, tangible form are ideas that all can apply with more definiteness than has been done. Each and every man knows that in his own city there are different conditions; at the same time with all the disadvantages if we will take the trouble to make it a systematic method it can be done with more consecutive purpose than as a rule we do employ.

A Case of Matricide and Attempted Suicide, with Brief Psychologic Analysis. By Dr. W. K. Walker. (See this JOURNAL, p. 144.)

Have the Forms of General Paresis Altered? By L. Pierce Clark and C. E. Atwood. (See this JOURNAL, 1907, p. 553.)

Dr. Diefendorf said he believed that the difference in the percentage depends in part upon the basis of distinction or the standards used by the different men. The fact that the speakers had not entered more fully into the description of the various types led Dr. Diefendorf to believe that this is true. In his own experience he believed that the expansive type should be confined to those cases where the symptoms of megalomania are continuously present for many months, together with a certain degree of psychomotor excitation and the emotional state of euphoria. On the other hand, the demented types he believed should be confined to those cases where deterioration predominates. In the demented cases there are undoubtedly short periods of megalomania, but they are insignificant compared with the progressive and rapid dementia which predominates in the picture. These cases seen for the first time and only during such periods would unquestionably be called cases of megalomania. The depressive type should be confined to those cases where the depressive symptoms predominate through many months. Judged by this standard he found in a series of his own cases, numbering 272, that the expansive cases represent 50 per cent., that the demented cases represent 40 per cent., and the depressive only 7½ per cent. He failed to find any true agitated cases except as they represent the terminal periods of the demented type, or short episodes in the expansive type. He did find, however, that there is a considerable group of cases that might be called manic-depressive type, because of the transitions from the megalomanic to the depressive stage and vice versa. One may question the desirability of grouping cases of dementia paralytica at all. The only advantage seems to rest in the fact that we get a clearer insight into the course of the disease; for instance, the megalomanic type in his cases averaged seven months longer duration. Again in the matter of the paralytic attacks he found that the expansive cases present far more paralytic attacks, and furthermore, also in reference to the remissions, he found that remissions are far more prevalent in the expansive type than in either of the other types. Dr. Diefendorf said that he himself did not believe that in recent years there had been such a marked alteration in the type of dementia paralytica. The apparent change is rather the result of observations by different men who have employed different standards of comparison.

Dr. P. C. Knapp said he thought Dr. Diefendorf had pointed out one possible explanation of the idea that the type of general paralysis has changed of late years. Of course our methods of diagnosis have improved and we recognize to-day many cases as being paretic which were not so recognized thirty or forty years ago, but at the same time it is distinctly rare, in Dr. Knapp's experience very rare, to encounter the typical case of general paralysis with the extreme delusions of grandeur, such as was

described by Falret and Calmeil in the original accounts of general paralysis, and which was regarded as the classical picture. Of course there can be no distinct line drawn between the different classes. There are a good many cases probably which Drs. Clark and Atwood included in their expansive type, who show after all comparatively little in the way of expansion. It is more a dementia with a mild euphoria and an easy-going way of looking at things, but without any of the real expansive conditions such as were formerly described, and that would explain the difference between such figures as they present and the opinion which Mendel has maintained, that there was a distinct increase of the simple dementing type in comparison with former years.

Dr. C. K. Mills remarked that personal experience must be looked at askance, even if it has extended over many years. His own experience has extended over thirty years, and from the first years of the period until to-day he has seen very many cases of general paresis in public and private practice. The general impression from that experience is that we have less cases exhibiting the grandiose delusions in their most typical form.

Dr. G. M. Hammond said he must agree with Dr. Mills. In his experience in the last twenty years or more the picture of paresis has certainly changed. Where the expansive delusions and grandiose ideas were plentiful years ago, now in private practice they are not so. He said he would not say that a majority of cases have not some degree of expansiveness, but they do not present it to the same extent, not nearly to the same extent that they did in former years. In many cases we see we cannot find any trace of grandiose ideas at all. In regard to the causation, Dr. Hammond believes there is only one cause and that is syphilis; that alcohol, excessive venery, debauchery of all kinds, are simply contributory causes, and there must be the syphilitic basis, whether we have found it or not, in order for these contributory causes to develop the disease.

An Experimental Study in the Etiology of Epilepsy.—By Dr. Smith Ely Jelliffe. (To be published in this JOURNAL.)

Dr. L. P. Clark said he had but one point of regret that Dr. Jelliffe should have carried out his experiments on rabbits. They are the most epileptogenous of all animals. If he had done his experiments on dogs or apes it would have been different. As far as the changes in the cortex are concerned it would seem to Dr. Clark as though there has been a distinct trend of late years in showing that these gliosis changes are entirely secondary to the cellular changes. It seems perfectly natural and rational, and it has been shown more or less conclusively and definitely by a host of workers that the primary changes take place in the parenchymatous elements and the secondary change was, in greater part at least, due to glial replacement.

Dr. Jelliffe said that if Dr. Clark would subsidize him he should be only too glad to carry on the experiments on the animals suggested. Apes, as is well known, cost four and five hundred dollars apiece.

So far as the remarks regarding secondary changes are concerned, it seems to Dr. Jelliffe that it is absolutely true that the glial changes are secondary to the destruction of the nerve cells, but the method by which the secondary process takes place is the point which Dr. Jelliffe tried to make in this preliminary contribution.

Multiple Neuritis, Accompanied by Degenerations in the Cord. Exhibition of Microscopical Specimens.—By Dr. Graeme M. Hammond.

A New Diagnostic Sign in Recurrent Laryngeal Paralysis. By Dr. Alfred Reginald Allen. (See this JOURNAL, p. 141.)

Hysteria in Children. By Dr. John Jenks Thomas. (To be published in this JOURNAL.)

Dr. Mills asked what age had been fixed by Dr. Thomas as the limit in considering hysteria in children? The etiology of hysteria in children is to be sought for in two things—a neuropathic heredity and an intense neuropathic environment. These cases in children usually occur in families in which at least one of the parents is a hysteric or a neuropathic, not infrequently one of them or some other near relative having had mental or hysterical manifestations. One of the most interesting cases he ever saw was a child ten years old, the daughter of a woman whom he had treated some years before for hystero-epilepsy of the most typical form. He was called to see a case of supposed meningitis with convulsions, and found the patient was the child of the woman he had formerly treated, and that the case was one of hystero-epilepsy. Dr. Thomas replied that the age of limitation he selected was fifteen years.

Dr. P. C. Knapp said that his experience in the hysteria of childhood is much more limited than Dr. Thomas's has been, but he wished to introduce one point which Dr. Thomas raised; namely, in regard to the care in diagnosis, to be sure that we are not dealing with organic disease. It is so constant an experience to be called to a case and informed that it is a case of hysteria, and to find on examination that it is undoubtedly one of organic disease, that Dr. Knapp thinks that point cannot be emphasized too strongly. Only the other day he was asked to see a case of alleged hysteria in an adult, presenting the Brown-Séquard complex with a Babinski reflex and various other symptoms of organic disease.

Dr. S. E. Jelliffe said that Dr. Knapp's remarks tempted him to express some experience he had had in traumatic hysteria in children. There the problem of diagnosis becomes often more acute, especially in litigation cases, and he recalled two instances only last year coming under his observation, of traumatic paraplegia in children of eleven and twelve years, following a trolley car accident, in which the diagnosis was of extreme interest. From his point of view they were both hysterical cases, although in the contest in court an attempt was made to show the organic nature of the paraplegia, but subsequent investigation (calling on these children and finding they had both gone off to a party) confirmed the diagnosis of hysterical paraplegia. Hysterical paraplegia in children of this age, it seems to Dr. Jelliffe, is of extreme interest, and Dr. Thomas's suggestion that he had found paraplegias more common among children than other forms of the graver hysterical accidents is suggestive.

Dr. Morton Prince said that his experience, so far as this class of cases with which Dr. Thomas deals is concerned, would coincide with Dr. Thomas's. Of course his experience with children has been smaller. There is one point which Dr. Prince would like to make, and that is regarding the classification of hysteria. The answer to this question of the frequency of hysteria depends very largely upon what syndrome we include in hysteria. There is a very marked tendency in the profession to limit hysteria in the adult to those cases which contain the classical stigmata of analgesia, anesthesia, paralysis, and physical stigmata of that kind. He thinks we are getting away from this notion and that, on the contrary, those cases represent a very small proportion of the hysteria in adults.

Unquestionably a very large number of cases occur without any of these stigmata, and express themselves in what used to be called neurasthenia. Neurasthenia as observed in a very large number of cases is hysteria. He does not say that all neurasthenia is hysteria, but that the symptom-complex of much hysteria is neurasthenia. Some people prefer the term psycho-neurosis, but we are dealing with patients who are essentially of the same class as those other cases of hysteria, which exhibit the stigmata of anesthesia, etc. Now in children we do see the neurasthenic type, though not so commonly as in adult life. This should not be overlooked. But as Dr. Prince understands it, that is not the type to which Dr. Thomas has referred here. He limits himself to hysteria with physical stigmata, but if the neurasthenic type is included it must increase somewhat the frequency of hysteria in childhood.

Dr. M. Allen Starr said that hysteria in children is a very rare condition in his hospital and private experience. He said he could recall a number of cases in which the so-called hysterical syndrome had been present for some little time in cases that have subsequently proved to be brain tumors, so that he is always on his guard when the family physician says there are hysterical symptoms in a child under fourteen years of age, and he (Dr. Starr) always wants an ophthalmoscopic examination.

Dr. J. J. Thomas said he would agree with most of the statements which had been made in the discussion of this paper. He thought that the statistics which he has of the cases in the Children's Hospital show that hysteria is a rare condition in children, and he should say it was much rarer here than in Germany. The tendency to call a thing hysteria where the symptoms do not form any snydrome of symptoms is a dangerous one, and he emphasized, of course, in his paper that one should always be on his guard against a mistake in diagnosis. The history of the cases where the child is supposed to have meningitis which disappears quickly, is not enough to warrant us in calling these cases hysteria. Dr. Thomas said he should be much more likely to class them among the serous meningitis cases. Moebius makes the statement that only symptoms which can be imitated are hysterical. Dr. Thomas thinks that is certainly going too far, when we consider the hysterical convulsion. The same thing is true of other symptoms. Then one should remember (a thing which he has brought out in the paper more fully than in the verbal abstract) that after organic disease one sometimes finds one little characteristic which may be defined as a hysterical remain. There again, as in adults, one cannot always make a positive diagnosis between an organic disease and a functional one, the hysterical condition, except perhaps by watching the case. That is true of many of these hysterical cases in children, where we have to deal with only one or two symptoms, and it is only by the sudden appearance and disappearance of these symptoms, their acting in a way we know an organic disease could not act, that we can call the cases hysterical.

NEW YORK NEUROLOGICAL SOCIETY.

October 8, 1907.

The President, Dr. Charles L. Dana, in the Chair.

EXPERIMENTS ON SUBCONSCIOUS IDEAS.

By Dr. E. W. Scripture.

In this paper the speaker stated that the associations of ideas might be put into two classes. The first was that of associations by meaning (internal associations); for example, "hat—head" and "marriage—funeral." The other class included associations by similarities of form or of sound, such as "goose—loose," or "hat—bat" or "marriage—carriage," which might be termed external associations. To these might be added still another class to include cases where the patient did not associate by external form and only half grasped the meaning, and which might be called indistinct associations. Such associations as "paper—to use," "book—to have" occurred constantly under certain circumstances. Finally, we could conveniently put the last two classes together as "superficial" in contrast to the association by meaning, which might be called "deep."

To get at the subconscious ideas, Dr. Jung, of Zurich, has devised the following method: With a printed list of words before him he explained to the subject or patient that he wished to test his mental activity, and that when a word was called out he was to say quickly what thing he first thought of. The printed list comprised such words as hat, street, good, poor, love, steal, dead, revenge, mother, laugh, cry, blood, money, enemy, hate, sad, tears, despise, etc. As soon as the patient felt at ease and answered readily, the examiner took a stop-watch with split seconds and proceeded to call off the words on the blank, and an accurate record was kept of the time it took for the patient to reply. Many hundreds of tests had shown that when a certain word brought up an idea that was associated with an emotion, it distracted the person so that he did not pay full attention to what he was doing. This often persisted for several associations. The application of the method in the detection of crime might be illustrated by an experience of Dr. Jung's. A man complained to him that a sum of money had been stolen from his office, but that he did not like to suspect his nephew. The boy was induced to undergo the association experiments on the plea that something was being done for his nerves. Words relating to theft, money, drawer, etc., were inserted in the list, with the result that the boy hesitated, stumbled and forgot whenever he came to them. When the experiment was over, the principle of discovering complexes was explained to him. His own record was then shown him, and it was such a convincing document that he could do nothing else but confess.

Dr. B. Onuf said that in one of the cases referred to by Dr. Scripture, where the result of the tests was not entirely satisfactory, it could perhaps be explained by the fact that a displacement of the affect of the

complex took place. A patient, for example, had a certain conception with which was connected a certain affect, and that affect was of such an unpleasant kind that he tried to disassociate it from the conception and carried it over to another conception which was closely associated with it, but which was not so repugnant to him.

Dr. L. Pierce Clark said he had applied the method described by Dr. Scripture in a number of cases, one of the most interesting of which was that of a neurasthenic in which he had been unable to get the emotional complex which precipitated the episodes. In applying the test to this patient, he noticed that the words "unhappy," "blood," "child" and "accident" gave a very long interval, and the result was the same in repeated trials. When the patient was asked whether he had ever had an accident to a child attended with the loss of blood he denied it, but his physician subsequently stated that about a year ago this man's wife had become pregnant for the first time, and that she had an abortion produced without the husband's knowledge and much against his wishes. Upon his arrival home one day he was shocked to find her in a condition of collapse, with severe flowing, which nearly cost her life.

ON SOME PECULIAR HOLLOW NUCLEAR-LIKE STRUCTURES IN THE NEURONE BODIES IN RABIES.

By Dr. Ira Van Gieson.

The description of the newly-found bodies in rabies was of very much less importance than the endeavor to interest neurologists in a seemingly forgotten subject within their field of investigation. The subject of the Negri bodies had received little or no attention from neurologists, chiefly, it would appear, for the reason that these bodies were regarded as protozoa, and consequently foreign to neurological research investigation. As a matter of fact, the Negri bodies were degenerated products of the neurone under the action of a peculiar virus, and scientific study was bound to throw light upon the process of rabies in particular, and upon the life history of the neurone in general.

In his original article (*Zeitschrift für Hygiene und Infect. Krankheit.*, 1903), Negri described rounded or ellipsoidal bodies measuring from one to twenty-five or more microns, lying in or near the substance of the nerve cells in rabies. These bodies contained particles of ordinary chromatin in their interior. These the Italian writer considered protozoa and the parasitic causal transmitting agents of rabies, although any approach to proof or confirmation of this surmise was conspicuously absent in his paper. Besides these typical bodies, which were probably at the present time regarded exclusively as the Negri bodies, there was another form quite distinct and different. This second form of corpuscle lying in or on the neurone bodies in rabies differed from the preceding in that they contained no internal chromatin particles and appeared quite homogeneous without exhibiting any internal differentiated structure. Thus there were two sets of these bodies appearing on or in the neurone bodies in rabies, and the speaker advised their differentiation by characterizing the first type as the nucleated and the second as the non-nucleated bodies.

Negri described the nucleated forms quite thoroughly, but the non-nucleated forms he barely recognized and evidently had a confused notion of their individuality. He failed to identify them signally, and to distin-

guish and separate them from the nucleated type which he regarded as the characteristic, typical and *sui generis* body in rabies. Furthermore, his distinction of the second or non-nucleated type of body was vitiated by his hasty surmise that the bodies were protozoa. In addition to these two forms of structure in rabies (it was to be understood that only street virus rabies was considered), there was still a third structure recently found by Dr. Van Gieson which was again quite distinct from the other two. The third type of structure differed radically from the other two in that it was not solid as they were, but hollow, and composed of a delicate skein or network of chromatin material. These bodies looked quite like the naked nuclei of a great variety of cells, with a delicate chromatin network inclosing apparently empty spaces. These bodies the speaker had so far found only in the bodies of Purkinje's cells in street rabies, and he suggested that provisionally they be distinguished from the other two types as the fenestrated or reticular bodies in rabies. The possibility that these fenestrated bodies were normal structures or artificial modifications of the other two types induced by technical methods, or the nuclei of neurophagi had been eliminated.

Dr. Van Gieson's paper was illustrated by charts depicting the structural characteristics and differences of all three types of the bodies.

Dr. James Ewing said that ever since the publication of Negri's articles he had been engaged in the study of these so-called Negri bodies. He thought that a fair estimate of the situation as it stood today was that while the adherents of the protozoan character of the Negri bodies had had a fair opportunity to substantiate their claims, they had not yet succeeded in convincing the majority as to the correctness of their theory.

Dr. Ewing said he was especially interested in Dr. Van Gieson's division of the two sets of bodies that he had found in or on the neurone in rabies as nucleated and non-nucleated. Personally, he had observed reticulated structures without any nuclei, but he had never considered that they deserved a separate classification, and he thought that he had at times observed certain transitional forms between the nucleated and the non-nucleated structures. In addition to the two bodies described by Dr. Van Gieson, Babes had recently found a very minute granular body in the ganglion cells in rabies, so that we now had three different structures that claimed recognition in connection with rabies.

Dr. Ewing said he had searched through many different types of diseased ganglion cells, and had never been able to find anything that he could recognize as a Negri body excepting in rabies. The speaker said he agreed entirely with Dr. Van Gieson that the problem was one for the general neurologist who was able to recognize minute structural changes in the nervous system. No one who was not properly trained in that field could properly cope with the subject.

Dr. Van Gieson, in closing, said he had found these bodies in quite a variety of diseases of the nervous system, such as cerebro-spinal meningitis and in various types of delirium. He regarded them as a new form of neurone degeneration, and he did not think that anyone could say how far a study of them might lead us. The particular point which he wished to emphasize in his paper was that rabies should again be regarded as a proper study for the neurologist.

DISCUSSION OF ACUTE ANTERIOR POLIOMYELITIS.

By Dr. B. Sachs.

In opening this discussion the speaker said that epidemics of poliomyelitis had been noted for a number of years past by various observers. The more important ones had occurred in Norway, Sweden, and in several smaller towns of this country. Its occurrence in New York during the past summer afforded us the first opportunity of studying the earliest stages of the disease, and of showing whether or not it should be included among the acute infectious diseases of childhood. Many had noted in years past the seasonal occurrence of the disease, and many writers on the subject, including the speaker, had stated more than twelve years ago that there could be very little doubt that infantile spinal paralysis must be considered an acute infectious disorder.

Our knowledge of poliomyelitis, largely through the investigations of Charcot and his school, had been based entirely upon the late conditions of the disease. The study of the earlier stages had naturally been neglected, as the vast majority of cases were seen in dispensaries and out-door departments, where the earlier stages had been either overlooked or were simply described in accordance with the statement made by relatives of the patients.

Dr. Sachs called attention to some of the clinical facts that had presented themselves in the children's service at Mt. Sinai Hospital, in his own ward, and in some private cases. Of the twenty or more cases observed in the children's wards of Mt. Sinai Hospital, autopsies had been obtained in three, and in a fourth doubtful case.

In the study of the clinical symptoms, the most striking fact was that the paralysis was preceded for one or many days by symptoms that pointed merely to a general infection of the whole body. It was only after the infection became more distinctly localized in the spinal cord that the symptoms of the disease became well established, and there were already sufficient facts at hand to show that the disease is at first not merely limited to the anterior gray matter, for in a number of cases painfulness of the extremities and some slight swellings have been noted, and the vesical and rectal reflexes have been impaired. The speaker also referred to the frequent involvement of the upper extremities in the recent epidemic, and the resemblance of some of the cases to multiple neuritis. The question as to the possible relationship between cerebrospinal meningitis and poliomyelitis had been raised by various observers, and it was one of the points on which the study of the present epidemic should throw considerable light. It was for that reason, Dr. Sachs said, that he had encouraged the performance of lumbar puncture in the acute stage, and although up to the present time the findings in the hospital cases had been entirely negative, the procedure should be persevered in, for if it was not the diplococcus, there might be and in fact there must be some organism, or, at least, some other infectious agent which is the cause of the disease. Careful blood cultures should always be made. Those made at the Mt. Sinai Hospital had in nearly all cases shown a considerable increase in polynuclears, and had revealed other evidences of an active inflammatory process.

Dr. William M. Leszynsky said that since June he had seen twenty-one recent cases of acute anterior poliomyelitis, the ages ranging from seven months to five years. Prodromal symptoms of general infection,

such as malaise, headache and fever were present for several days to one week before paralysis appeared. In one case pneumonia had occurred several weeks before. In two of the cases all four extremities and the lumbar muscles were affected. In a child eighteen months old there was at first paralysis of all extremities and of the spinal muscles of three weeks duration: the leg and neck muscles then recovered, but the child was unable to stand on account of the paralysis of the lumbar muscles. In others, both lower extremities, both upper extremities or a monoplegia or hemiplegia was present. The speaker said he had learned of many other cases in isolated groups in the practice of various physicians. In two cases the facial nerve was also paralyzed. In five cases complete recovery took place at the end of two weeks. In the cases he had observed there could be no doubt as to the correctness of the diagnosis, and the possibility of polyneuritis could not be considered.

Dr. Leszynsky said that through the courtesy of Dr. Russell Hibbs, of the New York Orthopedic Hospital, he was enabled to make the following report of cases of poliomyelitis admitted to that institution since July 15, 1907: Total number of cases, 48; of these 24 were males and 24 were females. The ages ranged from six months to four years. The duration of the disease at the time of admission ranged from one day to eight weeks, the average being about four days. In four cases all four extremities were involved, and in two of these the spinal muscles were affected also. In eleven cases the paralysis was limited to both lower extremities and in 26 cases to one lower extremity. In four cases it was limited to one upper extremity; in one case it was limited to one upper and both lower extremities; in one case to one upper and one lower, and in one case to both upper and one lower. Nearly all of these cases came from the east side of the Borough of Manhattan.

Dr. J. F. Terriberry said he had seen probably 150 cases of acute poliomyelitis the past summer, and in all had inquired particularly into the dietetic and the environmental conditions. Most of the cases were in children from the lower walks of life who had been fed on the general diet of that class. Milk did not appear to be the causal factor. His investigations had disclosed the fact that most of the epidemics reported in this country and abroad had occurred in low-lying districts, not from the standpoint of sea level, but with reference to the particular region in which the epidemic took place. During the past summer he visited a village in Western Pennsylvania, where an epidemic had been reported, and he found a small place of about 6,700 inhabitants on the western watershed of the Allegheny Mountains, located at the junction of two streams, the water of one of which was very clear and wholesome looking, while that of the other was quite the contrary. The epidemic in this locality was reported as a triple infection of typhoid, cerebro-spinal meningitis and infantile paralysis. About fifty cases of infantile paralysis were reported. This epidemic occurred during the latter part of July and lasted about six weeks. Four deaths had been reported. In only one case, a child of ten years, was there any possibility of spotted fever. The reported presence of cerebro-spinal meningitis was not confirmed. No true cases of this disease were found.

The speaker said that in his experience very few, if any, children made a *complete* recovery from infantile paralysis of a severe form. There was one other feature to which he wished to refer, namely, that these epidemics often occurred during an unusually dry season. This past summer, during our epidemic, was an evidence of that fact, and it

was dry and hot in Ridgway, Pennsylvania, the seat of the epidemic before mentioned.

Dr. Simon Flexner said he had been away from the city, and had only recently learned of this epidemic of acute poliomyelitis in New York, so he had no personal observations to report. The possible relationship between this disease and cerebro-spinal meningitis had already been alluded to by Dr. Sachs, and had been pointed out in the literature. With this in mind, the speaker said he had examined a dozen or more specimens of fluid obtained by spinal puncture from cases of poliomyelitis from a few days to several weeks after the onset of the disease; he had failed to find that any of these specimens departed essentially from normal spinal fluid, and he had come to the conclusion that if there were inflammatory changes in the meninges in these cases, they were of such a character that they did not lead to the accumulation of inflammatory products in the spinal fluid. In ordinary cultures upon media rich in blood, nothing had been developed excepting a few micro-organisms which could be regarded as contaminations. His results, therefore, had been purely negative. They were interested in this study at the Rockefeller Institute, but the great difficulty they had encountered was in getting early cases. The earliest they had seen was of three day's duration; others were five and six day's, and some longer. They were also paying some attention to the study of the stools, simply on an empiric basis, and with the idea that the reaction in the central nervous system was not due to a micro-organism, but possibly to the presence of products of a micro-organism which might develop elsewhere in the body. This was pure assumption. Dr. Flexner deplored the fact that he had thus far no access to autopsy material, and he made an appeal to the members present to assist him in obtaining, where possible, fresh organs from fatal cases of poliomyelitis.

Dr. L. Pierce Clark said the treatment of acute poliomyelitis must be along the lines of that of an acute infectious fever, and in the cases he had seen at the Roosevelt and the Babies' Hospital he had been struck by the apparent improvement that followed treatment directed toward the gastro-intestinal tract. The speaker referred to the fact that the symptoms in many of these cases pointed to a severe and diffuse involvement of the gray matter of the cord, which was the most favorable prognostic sign in the present epidemic. In some cases there was a return of power within three or four days after the onset of the palsy in all the muscles that had been involved.

Dr. Charles D. Cleghorn said that since about the middle of July they had seen twenty-five cases of acute poliomyelitis at the Vanderbilt Clinic. In any previous year the highest number of cases seen there in a corresponding time was ten, and the average number was about six. Of the twenty-five cases seen the past summer, thirteen were boys and twelve girls. Most of the cases occurred during the second year of life. All the cases had a sudden onset, with the exception of one. Two of them were of a non-febrile character. The initial palsy was perhaps more marked than usually in the upper than in the lower extremities, but still the leg type predominated in the ratio of about three to one. The meningeal symptoms were quite marked in eight of the cases. Two showed strabismus and eight had retraction of the head. Quite a number of the patients were still nursing and had had no other food but mother's milk, while the milk taken by the others came from good dairies. All were in the habit of drinking the city water, the purity of which was rather doubtful the past summer.

Dr. J. Ramsay Hunt said they had seen nine cases of acute poliomyelitis at the Cornell University Clinic since June 1st, 1907. In all of these there was a history of an acute onset, with fever and vomiting, the paralysis appearing in the course of from one to three days. It was quite probable that the mild prodromal symptoms might have been present and overlooked in these young children, just as they frequently were in cases of herpes zoster, to which affection the microscopic findings in anterior poliomyelitis bore a strong analogy. In none of the instances that came under the speaker's observation was there more than one case in a family, and none were of unusual severity. In seven the localization was in the lumbar region on one or both sides. In two there was a cervical as well as a lumbar localization.

Dr. Hunt said that there had been seven cases observed at the New York Hospital in the service of Dr. Lewis A. Conner since the first of June. Of these four had died, a very high mortality. Two died of respiratory failure, while the other two deaths were apparently due to the violence of the infection. Lumbar punctures were performed in these cases and some bacteriological experiments were carried out, with negative results. In one or two instances staphylococci were found which were attributed to contamination. Dr. Van Horne Norrie had informed him that in the Children's Ward at Bellevue they had had five cases since the first of June; two of them were from the same family, and in both the onset had occurred on the same night. In two cases from the same family observed by Dr. Norrie in private practice there had been an interval of ten days between the outbreak. In two cases there were pain and rigidity in the paralyzed areas, and in one instance the rigidity was so extreme that it suggested the possibility of hip disease. These persisted some time.

Dr. William H. Thomson said that several years ago he was called to Dutchess County, N. Y., where an epidemic of this disease was raging at the time. There were about thirty cases in a sparsely settled farming district. Upon investigation he learned that the first case had occurred in the village, and a distinct chain of connection might have been traced between that case and the subsequent cases, as it was an isolated community. The speaker said he believed that in epidemic acute poliomyelitis such a connection always existed, just as in cholera, but in crowded communities it of course could not be traced. One of the patients he was asked to see there was a boy ten years old who was taken ill rather suddenly with fever and who gave the history of having had a classmate who sat next to him at school and who had been taken suddenly ill five days before and died in 36 hours. The description of the fatal case tallied closely with that of cerebro-spinal meningitis, while the case Dr. Thomson saw was apparently one of true diffuse myelitis. The patient had retention of urine, constipation and intense constriction about the abdomen. Three months later, when the patient was brought to his office, the paralysis of the legs was typical of anterior poliomyelitis. Another case which he saw during that epidemic was a young woman who was related to the boy and who lived on a farm about three miles from his house. She was in a state of coma, with the terminal symptoms of acute cerebro-spinal meningitis, and died the following morning. Meantime her cousin, the boy seen by Dr. Thomson as just detailed, had passed a night at the house of the latter patient five days before he was himself taken ill.

Dr. Joseph Collins said that since the first of last July 129 cases of acute poliomyelitis had been seen in his clinic at the Hospital for the Ruptured and Crippled, and during the last two weeks alone he had seen upward of 40 cases. From his observation of these cases he had come to the conclusion that though there were some very severe cases, especially those with bulbar involvement, the epidemic was comparatively mild, and in at least three instances the patients had recovered. Moreover, in many of his cases the paralysis, which in the beginning was quite extensive, presaging serious consequences, had passed off, leaving a residue of paralysis in one extremity or part of one extremity, and judging from the rapid and progressive improvement in this it could be assumed that a fair degree of recovery would take place. Of course this is not unusual, but the point is that the paralysis disappeared sooner and more completely than it usually does. In many of the cases the type of the paralysis was unusual for this disease, and involved the ocular, neck and back muscles. Meningeal symptoms were almost entirely absent.

Dr. Collins said that while poliomyelitis was probably dependent upon an infection, from purely clinical grounds he concluded the infection was radically different from that causing cerebro-spinal meningitis. Clinically, there was no more similarity between the two diseases than there was between pneumonia and typhoid fever. One was short, with a not very sharp or brisk onset; the patient was comparatively severely ill for 48 hours or so, rarely longer, and then developed the paralysis. In cerebro-spinal meningitis there was an overwhelming infection with a long prodromal period, protracted symptoms often lasting weeks and very unlike poliomyelitis. As a matter of fact, from a clinical standpoint, poliomyelitis comports itself more as if it were the result of an intoxication rather than an infection. The speaker said he was also convinced from clinical reasons alone that the disease bore no analogy or similarity to multiple neuritis.

Dr. Joseph Fraenkel said that for some years the alternating sequence of epidemics of cerebro-spinal meningitis, poliomyelitis or polioencephalitis, peripheral facial palsies (so-called rheumatic) and finally chorea, made him think of a possible relationship between the infective agents of these infections of the motor system.

Dr. Charles E. Atwood said that Dr. Cleghorn, in his statistics, had omitted to mention that ten of the twenty-five cases had a history of retention of urine. The speaker said that in one case which he studied pathologically in Vienna some time ago, the gray matter of the entire cord was affected, though chiefly in the anterior horns. The lateral columns were also affected in that particular case. The process began in the artery of the anterior sulcus and extended to the anterior horns. It seemed to be an acute infectious process and showed diffuse inflammatory infiltrations of the entire cord.

Dr. Herman Schlesinger of Vienna, Austria, who was invited to take part in the discussion, said that epidemics of acute poliomyelitis, such as had been reported in New York and elsewhere, had never been observed in Vienna, so far as he knew, although a mild epidemic, confined to children, had occurred a few years ago. Exceedingly close relations apparently existed between the acute infectious diseases and polyneuritis and poliomyelitis, and the speaker said that after a severe infec-

tious disease he had repeatedly observed a symptom-complex which agreed at least clinically with acute anterior poliomyelitis. He recalled one case in a girl about twenty years old who immediately after the cessation of a severe pneumonia developed a sudden paralysis of the left upper extremity, with high fever, with subsequent severe muscular atrophy and contractions. In another case a similar symptom-complex developed after an attack of angina follicularis. Whether such cases could be regarded as genuine examples of poliomyelitis must be decided by subsequent observation.

Periscope

Review of Neurology and Psychiatry

(Vol. IV, No. 4.)

1. Epidemic Cerebro-Spinal Meningitis. J. S. Fowler.
2. A Plea for the Scientific Treatment of Stammering. H. G. Langwill.
3 Return of Paraplegia in a Case of Old Infantile Paralysis. O. Crouzon.

1. *Cerebro-Spinal Meningitis.*—Dr. Fowler reports 23 cases at the Edinburgh Sick Children's Hospital, 14 of which went to autopsy. Nineteen meningococcal cases died. Lumbar puncture was of paramount importance in diagnosis. Often the diagnosis in acute cases in children was in doubt until the cerebro-spinal fluid was seen. No case occurred in an exclusively breast-fed infant. Autopsy showed in most cases that the cord lesion was older than that of the brain. The abdominal reflex was abolished early in all cases. There is some reason to suppose that the natural channel of entry for the infection is from the abdominal cavity.

(Vol. IV, No. 5.)

1. A Clinical and Experimental Investigation into the Lymphogenous Origin of Toxic Infection of the Central Nervous System. Dr. Orr and Dr. Rows.
2. On the Nature of the "Faisceau en Écharpe" of Féré. G. E. Smith.
3. On Another Form of Anomaly in the Cerebro-Pontine Tract. G. E. Smith.
4. The Relative Order of Innervation of Certain Muscles of the Arm. A. W. Mackintosh.

1. *Toxic Infection.*—As a result of their research these two pathologists conclude that (1) "In peripheral nerves, spinal roots, and cranial nerves there is a constant stream of lymph ascending towards the central nervous system whose main current lies in the inner meshes or lymph spaces of the fibrous perineural sheath. Toxins reach the spinal cord and brain by this channel; and although they spread to some extent in the lymph spaces of the pia-arachnoid, and so affect structures at a distance from their point of entry, for the most part they pass, in the main current of the lymph, along the nerve-roots into the substance of the central nervous system. Here they apparently follow the nerve paths of the affected roots, and show little tendency to diffuse amongst the neighboring fibres. (2) In their extra medullary portion these nerves are protected from the influence of the toxins by the vital actions of the neurilemma sheath, but on losing this in their intramedullary part they at once undergo degeneration. (3) The first change is a primary degeneration of the myelin; the axis-cylinders and nerve cells are evidently affected later."

It was further stated as a conclusion that the vascular changes found in the cord in acute myelitic lesions should not be considered as being of primary hæmatogenous origin, but rather as being reactive changes due to a primary lymphogenous infection of the adventitia of the vessel walls.

(Vol. IV, No. 6.)

1. The Occasional Long Duration of Brain Tumour, with the Report of a Case of Jacksonian Epilepsy of Eight Years' Duration as the only Sign of a Small Cerebral Glioma. W. G. SPILLER and E. MARTIN.
2. The Amyotrophy of Chronic Lead Poisoning: Amyotrophic Lateral Sclerosis of Toxic Origin. S. A. K. WILSON.
3. Further Bacteriological and Experimental Investigations into the Pathology of General Paresis and Tabis Dorsalis. W. F. ROBERTSON and D. M'RAE.

1. *Brain Tumour.*—The duration of this case was 8 years. The tumour found on autopsy was a glioma, 1.5 cm. by 1.5 cm. by 0.5 cm., in a man of 60, situated in the posterior end of the right second frontal convolution, and did not extend into the precentral convolution. It was subcortical and had been localized for operation by left-sided Jacksonian attacks, but was not found and removed on opening the skull.

2. *Lead Amyotrophy.*—Wilson gives a detailed history of four cases of lead palsy with progressive atrophies; and suggests that the associated signs and symptoms—of cramps in the limbs, fibrillation in the diseased muscles, involuntary spasmodic movements, weakness, increased reflexes, ankle clonus, some spasticity, diminished electrical reactions, and lack of sensory impairment,—are essentially the clinical syndrome of the classical Charcot disease and indicate a probable toxic origin. Unfortunately he is unable to support his opinion by post mortem examinations.

3. *Pathology of Paresis.*—The authors adhere to their germ theory and claim histological evidence of the presence of special infective foci, in the alimentary tract and bronchi of general paretics and in the bladder of tabetics. Direct invading foci were also found, e. g., in the cervix uteri, in the conjunctiva, in the nasal mucosa, etc.

C. E. ATWOOD (New York).

Journal de Psychologie, normale et pathologique

Fourth Year, No. 5. September–October, 1907.)

1. Some Functional Disturbances of Audition among Certain Mentally Weak Individuals. A. MARIE.
2. Pantomimic Dissociation among the Insane. DR. DROMARD.
3. The Theory in regard to the Origin of the Emotions and the Actual Data of Physiology. H. PIERON.

1. *Functional Disturbances of Audition.*—This is so long an argumentative and psychophysiological analysis of the function of hearing that it is difficult to make a satisfactorily brief abstract of it. Marie starts from the teaching of Preyer that man, along with other animals, begins life without the sense of hearing; then he hears some sounds imperfectly; next, others are perceived more perfectly; until finally he is able to distinguish a great number of sounds in a mass, his powers increasing thus gradually from the highest to the lowest tones. An arrest of development may occur anywhere in this evolutional process. As noted among idiots, the cessation of development doubtless occurred before there was any power of audition whatsoever. In other instances it must have occurred shortly after the first appearance of the function, the most frequent operative cause being here birth asphyxia, such as plays so prominent a rôle in the production of certain forms of idiocy. The effect of this respiratory failure upon the development of the apparatus of hearing is

elaborately explained by the author. A loss of proper stimulation from this defective peripheral auditory apparatus naturally causes a retardation in the development of the central auditory mechanism. A loss of appropriate stimuli may come about in other ways, as, for example, through the failure of ideas and of the sensory motor cortical activity with which hearing is physiologically more or less in association. In all of these various ways the evolution of the function of hearing may be profoundly checked. When the deafness is associated with feeble-mindedness, the cause of it should be made the subject of a most searching psycho-physiological analysis. In the further elucidation of this Marie discusses in detail orientation, auditory accommodation, auditory judgment, and tone psychology generally. In this manner he gradually arrives at a consideration of the various subjective disturbances of hearing, taking up for special discussion hyperacusis, megalecia, which in a way is comparable to megalopsia, microacousis, micromegalecia, autophonia, auditory asymmetry and polyecia, and diplocousis. The article should be read in its entirety to be fully appreciated.

2. *Pantomimic Dissociation.*—Under normal conditions there is perfect accord between the facial expression and the emotion or feeling that gives rise to it. When the facial expression is not in harmony with the associated feeling a condition of pantomimic dissociation obtains. This is frequently observed among the insane. The varieties of this dissociation and the pathogenesis of each are elaborately discussed by Dromard.

Among degenerates it must be noted first that certain congenital anomalies, such as asymmetry, cranial and facial malformation, deformity of the ears and nose, may give rise to the dissociation. Furthermore, certain morbid manifestations, not necessarily psychic in origin, such as the ties, choreiform and athetoid movements of all sorts, tremors, may lead to it. The same is to be said in connection with the muscular atrophies, hemiatrophy, that occur in and about the face. Likewise spasmodic and paralytic conditions are sometimes responsible for it. As a happy means of studying objectively pantomimic dissociation, the author suggests the facial contortions of the average circus clown. The true pantomimic dissociations of the clinic, however, are not always easily recognized; yet as a symptom it has long been given great importance by the psychiatrists.

In the explanation of the pathogenesis of this dissociation, no one exclusive theory can be adopted, for the pantomimic play of the countenance has its origin in two sources. In a general way it may be affirmed that pantomimic dissociation occurs whenever the feelings are badly expressed or whenever there exist contending complex and contradictory feelings. That such a condition is not unknown even in the normal state, is evidenced in the phrases, sarcastic grin, obsequious smile, insulting laugh. The pantomimic dissociation therefore may be said to be exhibited by anyone who attempts to produce a facial expression not in perfect harmony with his underlying psychic state. This class of pantomimic manifestations make up what Duchenne of Boulogne denominated "the discordant expressive combined contractions." The veritable pantomimic dissociations, however, those which answer to Duchenne's "inexpressive combined contractions," involve a somewhat different pathogenetic conception. Here it is not so much the emotion itself which is uncertain as it is merely that the muscles do not serve the emotion appropriately and do not translate it correctly. A particular physiognomy is not established by the action of a simple muscle but by the action of certain muscular groups,

of which some are in a state of contraction while others are in relaxation. The harmony between these opposite states of activity may be ruptured by an *excess*, a *failure*, or a *substitution* of action. Each of these the author interestingly discusses and illustrates. The article ends with a consideration of the subject *lateralism*, or bilateral inequality of facial activity.

3. *Theory as to the Origin of the Emotions.*—This is a study of the old question as to whether the emotional feelings produce, or are themselves produced by, the physiological phenomena that are usually seen to accompany them. Pieron takes the view that the source of emotion is the caudate nucleus of the striate body. He says that this part of the brain is no longer to be considered as a center merely for the expression of the emotion; it is the very seat of the psychic origin of the emotion itself. This view, he believes, is warrantable upon our knowledge of physiology at the present time. The theory which sets itself in opposition to the peripheral theory (James-Lange) of the origin of the emotional feelings is not, as is so often affirmed, a mere spiritualistic conception. It is quite as physiological as the other but it is central in its conception. Instead of regarding emotion as the simple product of a coenæsthesia, it regards it as a mental phenomenon, keeping its own individuality just as all intellectual phenomena do and being able to engender certain organic reactions whose repercussion may in turn influence the emotion without the emotion being in any way engendered by them. In fine, the facts of physiology clearly indicate, in regard to the origin of the emotions, that this central theory is a much more coherent and simple one than is the peripheral hypothesis.

METTLER (Chicago).

Revue du Psychiatrie et du Psychologie Expérimentale

(November, 1906.)

1. Instruction in Psychiatry in France and other Countries. SERIUX.
2. Conjugal Insanity, with Hallucinations of Both Parties. DUPOUY.

1. *Psychiatry in France and Other Countries.*—This article is a review of the work that has been done in developing the teaching of psychiatry—particularly in France, and is of historical interest mainly.

2. *Conjugal Insanity.*—An interesting report of two cases of *foile a deux* with hallucinations.

(December, 1906.)

1 Insane in the Army and Military Penitentiaries. PACTET.
2 Care of the Insane in Portugal. LEMOS.

These two articles are of local interest only.

(January, 1907.)

1. Mental Disturbances in the Different Varieties of Supra-renal Syndrome. P. JUQUELIER.
2. Insanity of Doubt and Illusions of False Recognition. DROMARD and ALBÈS.
3 Hysteria and Suicide. PAUL COURBON.

1. *Mental Disturbances Associated with the Supra-renal Syndrome.*—The authors historically review this subject briefly and call attention to the

fact that symptoms, mental and physical, may result from episodic insufficiency of the adrenal. The symptoms in the mental field, which may come on rapidly, are delirium which may closely resemble delirium tremens. It is generally brief, hallucinatory, and oneiric. The characteristic asthenia of Addison's disease is described. This results in a practical annihilation of the intellectual functions. To these symptoms are sometimes added epileptiform convulsions, coma, and the pseudo-meningitic syndrome, corresponding to divers forms of acute or chronic insufficiency.

2. *Insanity of Doubt.*—This is the report of a case of a patient in confirmation of the views of Janet in a recent article, who held that the condition at the basis of the illusion of false recognition would appear to be as often the negation of the present as the affirmation of the past.

3. *Hysteria and Suicide.*—This article contains a short summary of the views of many authors on this question and presents a case about which the author says in conclusion that it confirms the opinion already expressed by many, that when suicide occurs in hysteria the hysteria is complicated by mental degeneration.

(February, 1907.)

1. The Ideas and Experiences of M. W. MacDougall on the Physiology of Attention. MAIGRE.
2. Some forms of Stereotypy. H. DAMAYE.
3. Note on Delirious Interpretations in General Paralysis. M. DUCOSTÉ.

1. *Physiology of Attention.*—This is a long article which discusses the views of MacDougall, from a critical standpoint and in relation to the views of many other psychologists on this subject. MacDougall's experimental work on attention was performed in connection with vision, and his conclusions do not seem to have been definitely formed as to whether fluctuations in vision due to movements of the eyeballs and of accommodation were due to fluctuations in attention or were peripherally conditioned by the musculature of the sense organs themselves. The author says, however, that it is demonstrated that even if the sense organs are continually moving themselves it does not necessarily follow that all the changes in the state of these movements, still less the changes in perception, have their cause at the periphery. Cannot both sorts of movements rather be due to central fatigue?

2. *Forms of Stereotypy.*—The author cites several cases and concludes from their detailed study that stereotypy is not confined to dementia nor to conditions of confusion. He thinks that the more we study this symptom the larger we will find its field and the more different sorts of conditions we will find it associated with.

3. *Delirious Interpretations in General Paralysis.*—The author calls attention to the fact that in paresis one occasionally sees paranoid states with well fixed though perhaps badly systematized persecutory ideas of considerable duration. Of 150 cases which have come under his observation, however, he has only seen two such. These he reviews in considerable detail.

WHITE.

Deutsche Zeitschrift für Nervenheilkunde

(Band 29, Heft 5-6.)

15. Disturbances of Motion in Infantile Cerebral Hemiplegia and Double Athetosis. LEWANDOWSKY.
16. Further Communications to the Knowledge of the Anatomical Foundation of Syphilitic Spinal Paralysis. NONNE.
17. Disturbances of Sensibility in Acute Local Ischemia. SCHLESINGER.
18. An Abscess in the Left Temporal Lobe (Contribution to the Knowledge of the Localization of Mind Blindness and Alexia). NIESSL V. MAYENDORF.
19. Hematemesis in Organic Nervous Disease (Tabes). NEUMANN.
20. Torticollis Mentalis (Hystericus). KOLLARITS.
21. The Variations of the Irritability of the Motor Centers of the Brain, and the Alterations of the Effects of Stimulation from the Cerebral Cortex, Under the Influence of Various Agents. SPANBOCK.
22. Brief Communications:
    1. Clinical Contribution to the Knowledge of the So-Called Tumors of the Akusticus. KRON.
    2. Clinical Contribution to the Differential Diagnosis of Cerebral Tumor in Chronic Hydrocephalus. GROSS.
    3. Contribution to the Symptomatology of the Intermittent Angiosclerotic Disturbances of Motion in Men (Dysbasia and Dyskinesia). ERB.

15. *Double Athetosis.*—Lewandowsky has studied two cases of infantile hemiplegia. First, a man of sixty had hemiplegia at the age of three months; the second, a man of forty-four, had his attack at the age of four months. The most characteristic feature is the absence of the Wernicke-Mann dissociation, that is to say, in infantile hemiplegia the flexor and extensor muscles, functionally belonging together, are simultaneously affected, or else both escape. He regards this as the rule in infantile cerebral palsy. Of the movements the most important is athetosis. Lewandowsky discusses the various characteristics of athetosis. He does not consider it necessary that the movements should be exaggerated nor limited to the muscles of the hands. He draws a sharp distinction between it and hemichorea. He speaks of the spasm which may occur in the hand in the course of athetosis, and which may even be produced by certain degrees of resistance. This spasm is to be distinguished, by various characteristics, from the true hemiplegic contracture. In addition to athetosis we have the so-called associated movements. Thus both sides of the body may carry out the same movements or the limbs of the hemiplegic side may only show these associated movements when there is severe effort made by the unparalyzed side. Sometimes the athetotic movements are increased by effort made by the healthy side. After discussion of the theories of these associated movements, Lewandowsky reports four cases of double athetosis that he has been able to study. This is not a condition of bilateral hemiathetosis; it is really a form of generalized, but not identically associated movements. In certain cases there are curious disturbances of the eye muscles, such as inability to look upward or to close the eyes voluntarily, although the various reflexes of the eyelids are preserved.

16. *Syphilitic Spinal Paralysis.*—Nonne reports the case of a man who, at the age of seventy, had had syphilitic infection, for which he had taken

a series of cures. At the age of fifty-seven he had spastic gait, increase of the tendon reflexes, with patellar and ankle clonus, myosis and sluggish light reaction of the pupils. These symptoms remained unchanged until his death thirteen years later. Examination of the spinal cord showed degeneration in Goll's column, diminishing downward and disappearing in the mid-dorsal region; and, in the lumbar portion, an indication of degeneration of the lateral pyramidal tracts, and there was also a slight diffuse degeneration in the whole section of the middle and lower dorsal regions as well as a slight chronic meningitis. The case, therefore, represents primary combined degeneration with diffuse myelitis. There was also distinct endarteritis of the anterior spinal artery.

17. *Local Ischemia.*—The symptoms of sudden occlusion of a blood vessel by embolus are, first, pain in the region of the lodgment of the embolus, increasing to extreme intensity, and at the same time pallor, coldness and paralysis of the affected extremity. Immediately after the complete closure of the blood vessel there is absolute sensory paralysis, often in the form of analgesia dolorosa. If the circulation is not restored the sensory disturbance remains the same and gangrene develops. In one case of ligation of the popliteal artery, Schlesinger was able to observe that the boundary line of the disturbance of sensation was at the upper border of the lower fourth of the leg. This corresponds to the distribution of the terminations of the nerves. The pain ceases suddenly with the restoration of the circulation, and sensation returns within a very few minutes. Severe, but not complete, ischemia produces slight or no sensory disturbances, as in the case of artificial anemia and Raynaud's disease. In gradual closure of the arteries sensation is affected very slowly and slightly.

18. *Abscess of Temporal Lobe.*—After a discussion of the functions of the temporal lobe, von Mayendorf reports the case of a man whose trouble began with an empyema of the antrum of Highmore. Several days after a radical operation he developed intense trigeminal neuralgia, and was transferred to the medical service, where it was noted that his replies to questions were vague or absurd, although his hearing was excellent. He was unable to read aloud, or to comprehend that which he read; indeed, he had complete word- and letter-blindness, with the exception of his own name. He was able to name objects held before him, or which he felt with his eyes closed; he could comprehend gestures; he spoke fluently without any defect in articulation, but there were indications of verbal paraphasia. He remained in this condition several months, when he died. At the autopsy there was ascending purulent meningitis along the sheaths of the trigeminal nerve, with an abscess at the tip of the left temporal lobe. Von Mayendorf discusses the anatomical basis for the clinical symptoms, particularly those which produce the word-blindness, and he concludes that these are due probably to the interruption of the left optic tract which interfered with the stimulation of the memory centers for macular vision on the left side. Ordinarily, however, vision would be preserved, because the right optic tract was intact.

19. *Hematemesis in Tabes.*—A man of thirty-seven, at the age of nineteen, had luetic infection, for which he was not treated. Later he developed gastric crises and distinct tabetic symptoms. He began to vomit every morning, and finally the vomitus was either pure blood or had blood mixed with it. The total acidity was greatly reduced and there was no free HCl. The stools contained occult blood. Neumann collects the cases hitherto recorded in the literature, and discusses the various

means by which the hemorrhage could be brought about, calling attention to the fact which had impressed Charcot, that there is a close relation between the hemorrhages and the gastric crises. Neumann observed further that during the crises the blood pressure was greatly increased, in his own case to 170 millimeters, whereas during the interval it sank sometimes as low as 110. This leads him to suspect that the hemorrhage is due to rupture of small blood vessels.

20. *Torticollis Mentalis.*—Kollarits reports three cases of torticollis on a hysterical basis. He includes in his statistics three cases reported by Jendrassik. The cause in these cases was either disease, or, more frequently, emotional shock. The cramps were not limited to the distribution of the accessory nerves, and they could be overcome by various manoeuvres on the part of the patient. The treatment of these cases is purely suggestive. Kollarits, as a result of several unfortunate experiences, opposes surgery with the object of influencing the patient.

21. *Motor Centers.*—Spanbock gives a careful and thorough critical review of the literature upon the variations in the irritability of the motor centers of the brain, and the effect of various agencies, physical, chemical or physiological and pathological, upon these centers. The article is not adapted to an abstract.

22. *Brief Communications.*—(1) A girl of eleven, at the age of five years, had a severe fall, followed, apparently, by no permanent injury. A few months before admission to the hospital she had an attack of vertigo and fell to the ground. Twice after that she had vertigo with vomiting. Later she began to complain of diminution of hearing and vision, and occasionally there were headaches with vomiting. In the left ear there was tinnitus. When examined there was a neuritic atrophy of both optic nerves. There was some paresis of the left sixth and seventh nerves, and of the left side of the body. The patient grew rapidly worse, became completely blind in the left eye, and there was diminution of hearing in the left ear. Kron discusses the diagnosis, which he concludes is the so-called "acusticus tumor," situated beneath the left pontine cerebellar peduncle.

(2) Gross reports the case of a man of twenty-seven years who, subsequent to a severe attack of influenza, developed severe headache with vertigo and vomiting. There was rapid loss of vision, and some transient lesion of the cranial nerves. When examined there was bilateral choked disc, increase in the tendon reflexes, no Babinski's sign, bradycardia, but no ataxia. There were some tumors of the skin and exophthalmus was present. The tumors of the skin were found to be lipomata. The patient grew rapidly worse and a puncture of the ventricle was made, ten cubic centimeters of fluid being withdrawn. The patient died, and at the autopsy a large tumor of the left temporal lobe was found. Gross discusses the differential diagnosis between tumors and acute hydrocephalus, which had been supposed to be present in this case.

(3) Erb reports the case of a man thirty-two years of age who had had syphilis three years previously. He complained of a sense of extreme fatigue in the left leg after long walks. The pulse in the arteries of the left foot could not be felt. There was some improvement at first, as a result of energetic specific treatment, but later all the arteries of the left side appeared to be involved in the process. The patient was a Jew.

J. SAILER (Philadelphia).

Miscellany

NEUROSES AND PSYCHONEUROSES. F. Raymond (L'Encephale, January, 1907).

The author wishes to distinguish, in this the first of a series he contemplates upon his conception of the neuroses, those things which are definite and those which are in dispute. He first excludes from the classification of Grosset certain conditions he considers organic; namely, Basedow's disease, epilepsy, paralysis agitans and chorea, on account of their tenacity, comparative variability, greater gravity, as well as our increasing knowledge of their somatic pathology and etiology. He also excludes such mental states as hypochondriasis and neurasthenia, the former on account of being a true mental alienation, and the latter on account of the variability of its etiology and the fact that the syndrome is a mere accompaniment of so many affections which have been grouped under this head through a want of care and clinical insight. He restricts the term therefore, with Charcot, to the simple acquired form due to fatigue. He points out that fatigue alone cannot engender systematic aboulia, feelings of insufficiency, long standing obsessive states, fixed ideas or systematic tics. There remain then only two neuroses, hysteria and the psychasthenia of Janet. These he thinks will one day be regarded as organic in the dynamic sense to which he extends the term.

He point out the difficulty of diagnosis, sometimes on account of the complexity and variety of the symptoms, and sometimes of their lack. The author attaches himself to the school of Dubois of Bern when he minimizes the importance of distinguishing the different neuroses. The therapeutic indications are general and particular. Of these latter the mental state is the most conspicuous. He insists that psychotherapy is an affair of individual tact and requires a special education, and that attention should not so much be directed towards individual symptoms as to the whole state of the patient, "for the spirit of system kills true psychotherapy." As to hypnosis, he thinks it advantageous in certain cases. He concludes by pointing out the importance of rectifying the digestive and genital systems, and the reticence of patients regarding the latter.

TOM. A. WILLIAMS (Washington, D. C.).

THE SIGNS OF PRE-DEMENTIA PRÆCOX: THEIR SIGNIFICANCE AND PEDAGOGIC PROPHYLAXIS. Smith Ely Jelliffe (American Journal of Medical Science, August 1, 1907).

This article is a complete, comprehensive and highly scholarly communication to mental pathology. A thorough survey of the literature of dementia præcox is made, Bleuler, Babinski, Hall, Jung, Janet, Kraepelin, Meyer, and others of equal repute, are freely quoted and allusions made to their important works. The author makes no attempt to define dementia præcox, saying: "Just what is here meant by the term dementia præcox I shall not attempt to define further than by saying that the word stands for a provisional group of patients who gradually develop certain mental and physical characteristics first brought into special prominence by Hecker and Kahlbaum, amplified by Pick, and later developed and expanded by Kraepelin and his students—a group very clear as to its nuclear features, but misty in its outlying edges and far from constituting an unassailable synthesis—a fact well recognized by Kraepelin himself." The various early mental manifestations of dementia præcox are briefly outlined and explained psychologically. "If I might express

it crudely," says the author, "in dementia præcox we find a stage of emotional impoverishment due to the breaking down, perhaps, as Freud terms it, a process of conversion, of rich plexuses of association of many years growth—the changes in the affective life should be interpreted from this standpoint as a disintegration or analysis of what had heretofore been a developing and fairly fixed personality." The relation of hysteria to dementia præcox is discussed, and "hysteria" differentiated from "hysterical," the latter is a phase common to many forms of mental alienations, the former a group by itself. The etiology of dementia præcox is exhaustively treated, the author favors the hereditary taint, and as he puts it: "My own observations on cases of well-developed dementia præcox which I have been able to follow for many years and whose parents have been well known to me, has shown that three elements have been most emphatic in the ancestry: Dementia præcox itself, alcohol, and abnormal personality or crankiness, if I may so express it." In regard to mental surmenage, the author says: "While some may fail to be impressed by some of the many carefully conducted psychological studies into the influence of fatigue, these studies nevertheless show in a graphic manner that which clinical experience has amply demonstrated. School work is not the only cause of fatigue by any means, but it plays a role in the genesis of the neurasthenoid background which is so prominent a feature in many of the pre-dementia præcox signs, and one cannot fail to be impressed by the unnatural fatigability of these individuals. Such good observers as Emminghaus, Ziehen, Eulenberg, Pick, L. Strumpell, Kahlbaum, Hecker, Christian, Wille, Uffelmann, Binswanger, Babinski, not to mention scores of others of equal prominence, all describe well-marked neurasthenic developments in the child and young adult, and ascribe them to the over-burdening of school life."

The prophylactic measures consist of proper pedagogic methods which "can be of more than casual service in the prevention of mental breakdown." The children of abnormal parents should receive proper care and attention during puberty and early adolescence. Nervousness in young children should be early recognized and subjected to prompt treatment. Outdoor exercise, moderate amount of outdoor sports, avoidance of intellectual competition, change of occupation for agricultural pursuit, are extremely advisable for all those who show predisposition for mental diseases. The author concludes by saying, "There is a large opportunity for the teaching profession of this country to evolve the mental healer in this sense, otherwise even the crudities of the quackish upstarts will be welcomed by a distressed and despairing public." Perhaps it will not be amiss to state that the time is not far remote when our schools will be represented by experienced, scientific, and up-to-date psychiatrists who will be able to assist our pedagogues in recognizing the early psychopathic stigmata in children and thus check the development of a mental malady.

MORRIS J. KARPAS (Ward's Island).

THE RELATION OF SEXUAL LIFE TO THE ORIGIN OF NERVOUS AND MENTAL DISEASE. Aschaffenburg (Münchener med. Woch., 53. 1906. W. 37).

The author asserts that neither onanism nor abstinence causes the disease, but the imaginations which are associated with the abnormal acts. Masturbation may exert a pernicious influence in the following manner: Either onanism is carried on in a senseless way or the indi-

vidual shows a marked hereditary taint. Onanism is not the cause but a manifestation of the mental malady. He bitterly assails Freud's sexual theory of hysteria. According to Freud hysterical symptoms are never manifested so long as children masturbate, but as soon as they abstain from it the hysterical phenomena become apparent. However, this is not the author's experience. He decries Freud's method of psychological analysis, and claims that Freud's patients know before-hand his questions and act accordingly. The paper is worthy of careful perusal, but Freud's valuable contributions to hysteria cannot be undermined, and are worthy of weighty consideration and credence.

Morris J. Karpas (Ward's Island).

Sleeping Sickness and General Paralysis. Spielmeyer (Muench. med. Woch., May 28, 1907).

The author finds that the clinical manifestations and pathological findings of the two diseases are similar. Clinically sleeping sickness differs from paralysis by the presence of fever, and somnolence, but resembles it closely in the progressive psychical weakness, physical and nervous symptoms, and constant presence of lymphocytosis in the cerebrospinal fluid. Mott first described a chronic meningo-encephalitis, a diffuse meningeal and perivascular infiltration, containing plasma cells. The anatomical pictures of both diseases are nearly the same. The essential difference of sleeping sickness is in the general distribution of plasma cells in all organs, and the tendency of the plasma cells to leave the lymph-spaces, and pass into the nervous parenchyma. Schaudinn has observed transitional form of trypanosom and spirochets. Some of the late stages of trypanosomiasis resemble tabes dorsalis. The pathological and clinical facts seem to confirm the results of the purely biological investigation, and vice versa the results of the biological investigation, bring new light for the understanding of the clinical and pathological findings. The relation which exists between the exciting causes shows itself in the anatomical changes and clinical manifestations of the diseases.

F. J. Conzelmann (U. S. Army).

Alypin. Dr. H. Gebele (Münch. med. Woch., 54, 1907, May 28).

The author states that alypin in a 1 per cent. solution injected subcuteneously acts very efficiently in the majority of cases. It has mostly been used in ophthalmology, otology, rhinology and dentistry and very little in minor surgery. Stolzer, Dold, Borszeky, Stutzin and Venus have reported the results of their experiences with the drug. Anæsthesia usually occurred five minutes after the injection and lasted 45 to 60 minutes. No local or general disturbances were observed. A solution of alypin may be sterilized by boiling without decomposing the drug. The addition of adrenalin is not necessary to obtain anæsthesia. The characteristic local anæsthesia of alypin was very well illustrated in a case of multiple atheroma of the scalp. The four large atheroma were removed in the one setting. Three of them were removed with novacain as the anæsthetic, without complete alleviation of pain; the fourth was extirpated with the use of a 1 per cent. alypin solution without any pain to the patient. Shleick recently recommended a combination of alypin and cocaine. This combination is adapted to enormously enlarge the indications for infiltration anæsthesia. He recommends the following mixture,

cocaine 0.1, alypin 0.1, sodium chloride 0.2, distilled water 100. The writer had no opportunity to try alypin as a spinal anæsthetic.

F. J. CONZELMANN (U. S. Army).

LUMBAR ANESTHESIA. Dr. Alvin Ach (Münch. med. Woch., 54, 1907, August 18).

Corning, an American, made experiments for lumbar anæsthesia with animals in 1885. Bier, independently of Corning's work, used the method on his patients and reported the results in 1,200 cases of lumbar anæsthesia at the Surgical Congress in 1901. So enthusiastic over the efficiency of the method was Bier that he had it done on himself and a colleague. The writer gives a detailed description of the technic as is carried out in the Surgical Clinic of Muenchen. Mostly all authors agree as to the technic, but various opinions still exist as to the selection of the place for puncture, the position of the patient and the kind and dose of the drug which is to be used. Some surgeons inject in the lying, others in the sitting posture. One selects the first or second, another the third or fourth interarticular space for injection. Novacain or alypin are the selected drugs of one and stovain or tropococaine that of another. One injects large, another small doses, one employs concentrated, another dilute solutions. The author uses a 5 per cent. of tropococaine solution of which he injects 1.2 c.c., which equals about .006 of the drug. In short operations he reduces the amount to .003. For laparotomies he prefers the first interarticular space for injection; in operations lower down the second interarticular space is his choice; for hæmorrhoids he has found the third interarticular space the best. In 200 cases of the 450 cases which were observed the outward symptoms and after-effects were marked. In the 250 remaining cases, and especially in the last 150 cases, only about 20 per cent. showed any bad effects. He has had no death or collapse. In cases of collapse he would apply the following treatment: (1) Puncture of the arachnoidal sack for a withdrawal of from 5 to 6 c.c. of fluid. This he believes would remove the anæsthesia at once. (2) Artificial respiration. (3) Subcutaneous injection of caffeine to raise the blood pressure. (4) A bandage around the neck to produce stasis. As remedies to prevent after-effects, phenacetin, asperin, caffeine, etc., have been recommended. Kroening employed profuse sweating with good results. As prophylactic measures he gives: (1) A good technic. (2) The use of tropococaine. (3) Small doses. (4) The solution should not be concentrated. (5) Rest, dorsal decubitus, raising the head, at least after the operation. (6) The pelvis should be raised only when absolutely necessary. (7) Bandage around the neck to produce stasis. (8) Subcutaneous injection of caffeine after every operation. In laparotomies the pain from tearing the mesentery or from pulling on the stomach and intestines is very well relieved with an hypodermic injection of a 2 per cent. solution of morphine about .015 to .002. In this way gastroenterostomies, resection of the stomach, enter-enterostomies and perityphlitis cases have been operated upon without any attacks of pain. Contraindications for lumbar anæsthesia are: (1) Recent and poorly treated syphilis. (2) Fever of unknown origin. (3) Septic conditions. (4) Diseases of the central nervous system. (5) Marked scoliosis. Ach sees a great future for spinal anæsthesia.

F. J. CONZELMANN (U. S. Army).

Book Reviews

Essai Critique et Theorique sur l'Association en Psychologie. Par le Dr. Paul Sollier. Felix Alcan, Paris.

This little volume is one of the series of the Library of Contemporaneous Philosophy and consists of a course of lectures given by Dr. Sollier in the new University of Brussels during the year 1904–1905. He gave a continuation of a set given four years earlier, on the general problem of memory.

Sollier elaborates this study of association, carrying out the integral relations of this process with that of memory, associations being the most important factors in the mechanism of memory.

In a purely monistic and schematic manner he presents the various hypotheses concerning the laws of association, but has very evidently neglected entirely the work of those students of psychiatry who have been assiduously gathering objective facts concerning the character of the types of association. Viewed from this viewpoint the work is superficial and academic, although a very simple and pleasing setting forth of the general problems of association.

Howard.

Klinische Beiträge zur Lehre von den Degenerationspsychosen. Saml. Zwang., Abh. VII. Von Professor Dr. K. Bonhoeffer, Breslau. Carl Marhold, Halle. 1.60.

On a basis of the Breslau material Bonhoeffer contributes a highly interesting short study of the type of individuals made classical by the work of Magnan and his students. The author considers more particularly three class of psychoses which are apt to occur in those types. The first includes the simple paranoid forms occurring in degenerates. The attacks are acute or subacute with comparative clearness of thought and orientation and manifest themselves mostly by ideas of reference and delusional ideas of interpretation. Auditory hallucinations may accompany the attacks which may persist for a few months or a couple of years. Insight is gradually established and the patient gradually rights himself with no modification of his personality. These "Erethristic debilities," as the author terms them, are allied with the the episodics in the fields of hysteria and epilepsy.

A second group is made in which there is a permanent character anlage of the paranoid coloring. These individuals have "the tendency to a dysharmony in the dynamics of their ideas," in the sense that certain ideational combinations are accompanied by a permanently increased affect influence so that opposite and regulatory influences do not have their accustomed influence. In these patients the "überwerthigen" ideas of Wernicke show to best advantage and are the progenitors, as it were, of the delusional interpretations. Anxious depressions, anxious dreams, hallucinations occur in the clinical picture. In some instances these patients approach very close to many of the litigious paranoiacs.

The third group is characterized by the great lability of the conscious-

ness of personality which has many relationships to hysteria. These individuals under the influence of prison life develop in a manner much more rapidly than do the true paranoiacs, a subacute, paranoiac modification of the personality in the direction of an elevation of the sense of self-importance. Delusional interpretations are absent from the picture. Ideas of reference are not usual but fabrication and pathological lying is very characteristic. A tendency to turbulence and fractiousness under discipline is present and simulation is very frequent. A sharp distinction between delusion and phantasy lying is often difficult to draw, but the latter is the common type here. Recovery may take place with insight even after the condition has persisted for several years .

The paper is one of much value to students of medico-legal science, especially in criminological fields.

JELLIFFE.

DEPARTMENT OF NEUROLOGY, HARVARD MEDICAL SCHOOL. Contributions from the Massachusetts General Hspital, the Boston City Hospital, the Long Island Hospital, and the Neurological Laboratory. Vol. II. Boston, Mass., U. S. A., 1907.

The original publications of the Department of Neurology of the Harvard Medical School, for the year 1906, are collected in this volume, in accordance with the plan outlined in the first issue of the series. Nine papers are included, all of which had been previously published, chiefly in the *Boston Medical and Surgical Journal*. The chief contributors are Putnam, Taylor and Waterman; others are Knapp, Walton, Brewster, Linenthal and Lindström. Interesting articles on psycho-therapeutics, with illustrative cases, and on brain tumors, multiple sclerosis, and neuro-surgical cases, are to be found; also suggestions as to treatment of exophthalmic goitre, tabes, etc. All are carefully written and form together a volume reflecting credit on the department. Most of the articles have been previously "abstracted" for this journal.

ATWOOD (New York).

IBSEN'S NORA VOR DEM STRAFRICHTER UND PSYCHIATER. Von Staatsanwalt Dr. Erich Wulffen in Dresden. Carl Marhold, Halle.

The literature bearing on the psychological side of Ibsen's work grows apace, and in Wulffen's analysis we have one of the most extended concerning a single character that has been offered. To be appreciated it must be read, and no review can hope to present its chief features nor to criticize to advantage the positions maintained.

The criminalistic side is well considered from the standpoint of German jurisprudence; it is perhaps a slight caption to ask that this standpoint would better have been that of the Skandinavian laws, but as there is little question that the law-breaking is solely a setting, the exact significance of the deed from a legal point of view is of secondary interest.

From the psychiatric side Wulffen considers Nora a true hysteria. She has the heredity, shows to him great emotional instability, is morally anesthetic concerning her crime, flirts and coquettes with Dr. Rank, does not really love her husband, and does not care for her children. This is a short résumé of the author's position.

It is significant that the very dense and stupid Helmar, for certainly Ibsen has made him a prototype of the unutterably stupid and unseeing type of man, should have practically the same idea of Nora that Wulffen adopts, and if one reads carefully the author's argumentation it is as cer-

tain that he is as stupid and unseeing as Ibsen meant Helmar to be. He deliberately misquotes in his pamphlet (p. 39) or else the German edition differs from the Archer translation, and throughout, his understanding of certain beautifully intricate and complicated passages is silly in the extreme. He misses, for instance, the significant contrast of physical and moral disease in the delicate little scene between Nora and Dr. Rank on the subject of Dr. Rank's spinal complaint, and calls it a flirtation. One would think that the author should have escaped the rendering that he gives to the friendship of Dr. Rank and Nora since Ibsen causes Mrs. Linden to draw somewhat similar inferences and then indicates very plainly how incorrect they are. They are the thoughts which would naturally arise in the mind of the superficial observer and are a part of the deeper art of the author's merit to a great genius. For a psychiater to interpret them in a manner as shallow as Mrs. Linden's is no credit to his acumen.

The reviewer feels that Wulffen, like Nordau, has given an absolutely superficial and futile interpretation of Nora's character, one which the casual observer would naturally give but not the rendering that Ibsen had in mind if the internal evidence be taken into consideration. It is of interest to read, for in few fields of thought can opposing opinions be better supported by their respective adherents.

JELLIFFE.

UNTERSUCHUNGEN ÜBER DIE AETOLOGIE DER MANIE, DER PERIODISCHEN MANIE UND DES ZIRKULÄREN IRRESEINS, NEBST BESPRECHUNG EINZELNER KRANKHEITSSYMPTOME. Von Dr. Giovanni Saiz, Volontär Arzt der Klinik. S. Karger, 1907. M. 5.60.

This is a contribution from Ziehen's clinic in Berlin in which the point of view of this clinician is maintained. The study is based on an analysis of all of the cases of simple, recurrent, and periodic manias together with the circulars which had come under observation in the Berlin Psychiatric Clinic. The author has paid particular attention to the subject of the periodicity of manic attacks and gathered evidence bearing on the etiology of periodic attacks. He calls attention to the fact fairly well recognized by all, save those who persist in speaking of simple mania, that simple attacks of this disorder are extremely rare, and further attempts a clearer definition of what constitutes recurrence and what periodicity, although realizing that the conditions merge insensibly the one into another. He reserves the term recurrent for those cases in which the free interval is definitely great, six years being arbitrarily assumed as a criterion.

The conditions at the Charité Clinic are such that a complete history of the lives of the patients is impossible, and while many of his cases are registered as having but one attack, Saitz adds that it is practically certain that this does not represent the true state of affairs and that a one-attack mania is an extremely rare condition, i. e., if a strict definition of mania be insisted on; mania not being "excitement" as it is for so many. The great majority of these cases Saitz believes belongs to the "recurrent" class.

Saitz's definition of mania is that of Ziehen. It comprises those mental disorders in which there is an elevation of the mood and an increase in the rapidity of associations. Motor unrest, hallucinations and delusions may occur, the last being secondary for the most part and largely of affective origin; the hallucinations are only accidental accom-

paniments of the primary affect disturbance. When the hallucinations and delusions come to the fore and are more prominent in the picture, the elevation in the mood and increase in idea association occupying a secondary rôle, the term acute hallucinatory paranoia is applicable. When in addition to many hallucinations and delusions there remains either a primary flight of ideas or a primary elevation in mood, Ziehen would term these cases the exalted variety of acute hallucinatory paranoia, thus showing a gradual approach on this question to the Kraepelian doctrine.

The author presents a very interesting and complete historical summary of the general question of mania and its relations to what is so widely accepted as the manic-depressive psychoses. The general clinic statistics were for 24,705 patients, of whom 16,123 were men and 8,582 women. Of this entire group 213, or 0.86 per cent. of the entire admissions, suffered from either acute, recurrent or circular manic states, 88 acute (simple?) attacks, 51 recurrent and 74 circulars. The author then follows out a series of minute statistical analyses which will not permit of further recapitulation here.

On the subject of etiology he rejects the infection doctrine entirely and believes that the two important factors are heredity and exhaustion. Evidently not enough weight would seem to be put on the clinical differentiation of infection and exhaustion deliria by the Berlin school, or else this statement would not be made.

The study is an extremely interesting one of a type that is much needed in clinical psychiatry. It is to be regretted that more care were not taken in tracing out more complete histories, as has been done by so many of the Heidelberg School psychiaters. This will have to be done in order to determine the limitations of Kraepelin's group of manic depressives.

JELLIFFE.

ZUR KENNTNISS DER VARIABILITÄT UND VERERBUNG AM ZENTRALNERVENSYSTEM DES MENSCHEN UND EINIGER SÄUGETIERE. Von Dr. J. P. Karplus. Privatdozent für Psychiatrie und Neurologie, Universitat Wien. Franz Deuticke, Leipzig und Wien.

By reason of the wealth of detail in this research we can outline only the chief features touched upon by the author. He has carefully studied and compared the external form of the brain, with reference to the development and complexity of its external surface in 26 groups of human brains, four groups of dogs' brains, four groups of cats' brains, and three groups of monkeys' brains. In all of these the brains of two or more descendants have been compared with those of the immediate ancestor and in some the comparisons are made in twins, in triplets, or even quadruplets, as in the case of dogs and cats.

The research is presented as a detailed descriptive analysis of the shape, size, angle, depth, etc., of the different fissures, sulci and convolutions of the different areas in the brains under observation. The medulla and spinal cords are also described. The rich results bearing on variability cannot be gained without the time-consuming labor of reading pages and pages of these descriptions.

In general the author shows that there exists, so far as brain structure is concerned, a distinct family likeness which shows both in the general nature of the brain structures and in many of its special peculiarities. He shows that among certain monkeys, *Macacus*, that variability is extreme however, so extreme as to necessitate more detailed study in

order to appreciate its significance. Only occasionally for instance is there a distinct resemblance in the form of the brain of mother and child, and in certain brain areas, notably the occipital lobes, there is a very great degree of variability.

The general results are utilized by Karplus to open up new vistas in the field of the evolution of the nervous system, but these are hardly more than hinted at.

As a thorough, painstaking piece of descriptive research the monograph is highly commendable. It can appeal practically only to the anatomist and student of the problems of heredity in their relations to the handing down of form and structure.

HOWARD.

LE LANGAGE MUSICAL ET SES TROUBLES HYSTÉRIQUES. Étude de Psychologie Clinique. Par le Dr. Joseph Ingegineros, Professor a l'Université de Buenos Aires; Directeur du Service d'Observation des Aliénés. Felix Alcan, Paris.

The study of the psychophysiology of musical language is a fascinating one, which has even at the present time a rich literature. Its interest to the psychologist is no less vital than to the clinician.

The loss of musical language is of no particular import or plays a very secondary part in the study of those individuals endowed with little or no musical ability. It is even of minor interest in the musical imbeciles, and attains its greatest value as a subject for psychological investigation only in those people of intelligence normally endowed with musical faculty. Its perturbations in professionals constitutes a veritable malady, as the case of Donizetti amply illustrates. The sense of beauty of language, its richness and harmony and musical quality rests at the basis of the beauty of the metaphors of Victor Hugo, the purity of the style of Flaubert, the compelling quality of the flow of Poe and the nobility of much that D'Annunzio has written, and in the works of these and others only those who have an understanding of the powerful emotional element in musical language are fully capable of the analysis of its variations and perturbations.

It is from this sympathetic standpoint that the author would interpret his observations on the psychophysiology of musical language and its applications in certain hysterical modifications. This in the work before us of some 200 pages is well carried out.

In Part I the problems of the psychology of musical language are stated, the biological origins discussed and their functions dealt with. Musical emotion and the forms and evolution of musical intelligence are taken up in two very fascinating chapters.

The second part of the work deals with the pathological applications. Here the author treats of the general subject of aphasia, of amusia, of musical deafness, motor and sensory amusia, and aphemias. Musical impulsions, incoercable melodisation, paramusias, phonophobias, melodic obsessions, colored audition, dissonophobias, etc., these are some of the subjects written upon.

In brief, the monograph is one of the most complete studies on the subject that we possess at the present time. It is written in a very pleasing style; is replete with the citations of other workers in the field, showing the author's wide reading, and is a practical and thoroughly praiseworthy contribution.

JELLIFFE.

Notes and News

Congress for Carers for Insane.—A third International Congress for the Care of the Insane will take place in Vienna, the 7–11 of October, 1908, under the presidency of Professor Obersteiner. Applications for membership, reports and demonstrations should be arranged with Dr. Alexander Pilcz, Lazarettgasse 14, Vienna IX, up to July, 1908. Details of the program will appear later.

American Neurological Association.—The council announces that the Thirty-fourth Annual Meeting will be held in Philadelphia, Pa., on Wednesday, Thursday and Friday, May, 20, 21 and 22, 1908. There will be two sessions daily, from 9.30 a. m. to 1 p. m. and from 2.30 to 5 p. m.

If it is the intention of the members to contribute a paper, attention is called to a clause of article VII of the constitution, which states: "The reader of a paper shall not exceed twenty minutes in the presentation of his paper, and no one shall speak longer than five minutes in the discussion of a paper."

It is the earnest request of the council that they will, if practicable, present a verbal or short written abstract of the paper instead of reading it in full.

Attention is also called to another clause of article VII, which states: "Members must send the titles and *abstracts* of their papers to the secretary at least six weeks before the annual meeting. Titles and abstracts must therefore be received by the secretary on or before April 8. The council orders that no title shall be printed in the program unless accompanied by an abstract.

The headquarters of the association will be at the Bellevue-Stratford Hotel. The sessions will be held at the College of Physicians. Members are advised to secure hotel accommodations at as early a date as possible.

The secretary announces the following amendments to the constitution: To amend article II so that it shall read as follows: "The number of active members shall be limited to one hundred and twenty-five" instead of one hundred and ten as it now stands.

To amend article IV by inserting the words "it may be forfeited" between the words "or" and "by" on the fourth line of that article as it is now printed, so that the sentence shall read "or it may be forfeited by failure to pay the annual dues for three years." The Annual Dinner will be held on Thursday evening, May 21, at the Bellevue-Stratford. The council announces that the dues for 1908 will be five dollars.

GRÆME M. HAMMOND, *Secretary.*

A New International Journal of Epilepsy.—This new journal, to be called *Epilepsia*, is to be an international quarterly, devoted to the study and treatment of this disease from the therapeutic, social and judicial points of view. It is announced as under the patronage of Drs. W. Bechterew, O. Binswanger, J. Hughlings Jackson, L. Luciani, H. Obersteiner and F. Raymond. The editorial staff includes the names of H. Claude, Paris; A. Turner, London; L. Bruns, Hannover; W. P. Spratling, Sonyea;

J. Donath, Buda Pesth, who is the active editor for the first year; L. J. J. Muskens, of Amsterdam, secretary. The collaborators and assistants announced are: Apelt, Grainger Stewart, Lejonne, Maes, Perusini, Southard, Agostini, Aschaffenburg, Alt, Alzheimer, Bastien, Batten, Brandos, Bruce, Ferrier, Gowers, Haskovec, Van Hamel, Hebold, Heilbronner, Henschen, Horsley, Jelgersma, Jelliffe, Kocher, Kowalewsky, Krause, Kure, Lewandowsky, Maxwell, Meyra, Nonne, Oppenheim, Probst, Cajal, Redlich, Stertz, Tükel, Voisin, Weber, Salomonson, Wiersma, Winkler, Ziehen, and others. French, German, and English will be the official languages. The price is not yet announced, but it will be reasonable. Scheltemer and Helkema, of Amsterdam, are to be the publishers. Subscriptions may be forwarded through the JOURNAL OF NERVOUS AND MENTAL DISEASE, New York.

Dr. T. A. Williams has taken up his residence in Washington, D. C., after spending two years in the study of nervous diseases in Paris and other European countries.

He has been requested to give a course, which will begin in March, embodying more particularly the recent researches of the French School in the diagnosis and treatment of the psycho-neuroses.

The number will be limited, as individual attention will be given and the whole day utilized.

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The Journal
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Nervous and Mental Disease

Original Articles

HYSTERIA IN CHILDREN.[1]

By John Jenks Thomas, A.M., M.D.,

OF BOSTON,

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The object of this paper is not to discuss the nature of hysteria, or even many of its various problems, but simply to embody the experience gained in the clinical observation of a considerable number of cases in children, and to emphasize the difference between the clinical phenomena of the disease as it occurs in adults and in children. The processes of thought in children being simpler than in the adult also makes the direct relation of the suggestion to the symptoms of the disease much clearer than is usually the case with adults, and thus is of significance in our concept of the nature and origin of the disease.

I have taken for a basis the cases of hysteria observed at the Children's Hospital from March, 1894, to May, 1907, and those at the Boston City Hospital from May 3, 1901, to April 17, 1907, the periods being determined by the availability of the records of cases at these two hospitals in the indexes of diseases. The upper limit of age has been placed at fifteen years since most writers have taken this limit, in particular those publishing tables of cases.

[1] Read at the thirty-third annual meeting of the American Neurological Association, May 7, 8 and 9, 1907.

None of the cases included, however, had reached puberty when first seen, and as at the Children's Hospital the age limit, though not always strictly enforced, is twelve years, most of the cases are below the limit set.

Almost all writers on hysteria recognize the fact that it occurs in children, and some of them devote considerable space to its characteristics in the young, as Binswanger in various parts of his treatise on the subject. There is, however, a considerable variation among writers, both of neurological and pediatric literature as to its frequency. Holt in his text-book on the diseases of children considers it rare before seven or eight, but apparently not so in later childhood. Heubner again considers it relatively frequent in children, though like most German writers he thinks the disease does not occur in babies. Koplik, on the other hand, speaks of it as not a true disorder of childhood. Lloyd in Starr's "Text-Book of the Diseases of Children," and Mills in Keating's "Cyclopedia of the Diseases of Children" have written at considerable length upon the subject, but while expressing in a general way their opinion that the disease is not especially rare in children, give no figures from their own experience. Taylor, in his book on the nervous diseases of children, gives but little space to hysteria, aside from what he says about functional paralysis, although other phenomena are very frequently found in children. Sachs, in his book on the nervous diseases of children, says what is undoubtedly true in this country, that hysteria is a rare disease, but adds that it is still rarer in children. Our figures show that this is so, especially in regard to the proportion of children's cases to those of adults. At the other extreme are some of the French writers. One might think after reading the monograph of Bezy and Bibent that most children, from the cradle on, were hysterics, if they either laughed or cried, so varied are the symptoms described. Oppenheim, in his book on nervous diseases, says that he has repeatedly seen hysterical symptoms in children between four and six years of age, and well-marked hysteria between eight and ten years. Gowers makes no detailed statement, but quotes older authors, as Briquet and Landouzy. We shall return to the various questions connected with the frequency of the disease in greater detail in the discussion of the symptomatology.

The following cases are taken from the records of the Chil-

dren's Hospital and the Boston City Hospital, from the departments for Nervous Diseases in each case, and for their kind permission to use the material I have to thank my colleagues, Drs. Bullard, Prince and Knapp.

The cases with convulsive attacks of greater or less severity vary considerably in character. The first one, except for the rigidity, might be taken for simple fainting attacks.

CASE 1. Ida Z. (B. C. H. 11323) was first seen on July 20, 1904, when twelve years of age. She was born in Boston, but of Russian parents. The mother died of kidney disease in convulsions. The child had had measles, mumps and pertussis when an infant, but otherwise had always been well. For two years she had had fainting spells in which she says, "papa, my hand," and then falls to the floor, with the left extremities rigid, but no convulsive movements. This lasts for five minutes. She has never injured herself in the attacks, and has not lost control of the sphincters in them, nor has there been any drowsiness afterwards. She has had occasional frontal headaches but no vomiting.

On examination there was found a left hemianesthesia, including the left side of the tongue and face, with normal fields of vision and no changes of the reflexes.

There were no attacks in the week after the first visit to the hospital, and the patient was lost sight of. This case I think to be an example of suggestion, as the child had seen her mother in the convulsions preceding her death, and her attacks began soon after this.

The next case, too, illustrates the suggestibility.

CASE 2. Patrick K. (B. C. H. 12230), born in Boston, and nine years of age, was seen first on November 27, 1905. His sister was also a patient at the hospital (12422) and in her case a diagnosis of simple nervousness was made. She drank considerable tea, cried easily, and was excitable, having an attack of excitement first in September when afraid of punishment, and these had occurred a number of times. Her physical examination was entirely negative. The family history was otherwise negative, and the boy's health had previously been good. The two children had been a great deal together. The boy had been well till ten days before being brought to the hospital, when he saw his sister have an excited attack, whereupon he became violent, cried loudly, and had to be restrained. He did not seem to lose consciousness and did not bite the tongue. Since this time he had had a similar attack almost every day, lasting fifteen minutes. In these attacks he sways as if drunk, "works" the hands and feet, knocks his head, and screams. There had been no loss of sphincteric control, no amnesia for the attack and no subsequent stupor.

Examination showed slight concentric contraction of the field of vision, more marked in the right eye, but no disturbance of sensation, no palatal anesthesia, and otherwise normal conditions. There was slight suggestibility to waking suggestion.

During the next three weeks the boy had only three or four attacks, which were milder. Then a history was obtained that the previous winter the boy had seen a man fall off a wagon, and though the man was not hurt the child was frightened, and that each attack he had began with staggering, but with no hallucination of a man falling, and the boy stated that he had no feeling of fright. He recalls everything that happens in the attacks. The mother says that he has something like a "shock" in the leg and cannot walk for a couple of minutes. The boy resisted hypnotism, but a suggestion of weakness of the right arm made in the waking state was reacted to. Three days later he was put under slight hypnosis and the suggestion of no further attacks made. He awakened easily, with amnesia for the suggestion, and on April 9, 1906, some four months later, he had had no further attacks.

The next case is one in which the diagnosis of hysteria may be somewhat doubtful, but is the most probable one.

Case 3. The child, Ethel O., colored, born in North Carolina, and fourteen years of age, came to the Out Patient Department (B. C. H. 10181) first on November 19, 1902. The preceding February she had been a ward patient, when it was noted that during the examination the arms were acutely flexed, and there was occasional stiffening of the body, and spasm of the orbicularis palpebrarum. There was a systolic murmur at the apex of the heart, the knee-jerks were lively, and there was no hemianesthesia. She had been studying hard, and one week before her entrance she began to have nervous fits and fainted several times, complained of headache and was very constipated. She had two attacks while in the hospital which were said to be hysterical in character, though no description was given. She remained in the hospital from February 12 to February 24.

When she was seen in November she complained of pain in the back, and left side, and of facial neuralgia on the right, but she had no more fainting attacks since leaving the hospital in February.

On examination there was found a well-marked scoliosis with rotation of the vertebrae and a slight myopia, but no other ocular trouble. In December, 1903, she complained of sensitiveness of the left chest to a light blow, and it was noted that there was slight hyperesthesia on the right chest and back. On January 4, 1904, it was noted that the whole thoracic and lumbar spine was sensitive to light pressure and that there were tender points in the left back below the scapula, in the left chest, and the left iliac region, while touch and pain stimuli were felt better on the right

side of the trunk than an the left. On July 13, 1904, she had an attack which began with quivering of the abdominal muscles on the left, and this extended to the left hand and left leg and then became general, and there was considerable tossing about on the bed. After this attack she was more irritable than she had been. This was the first attack in six months. During the next month she had no attack, and after that she failed to come to the hospital.

The next case also seems to be one of hysteria.

CASE 4. Martin J. L. (B. C. H. 11071), was first seen on March 9, 1904, when he was nine years old. He was born in Massachusetts, of Irish descent, and was an only child. The parents were living and well. When two years old he broke his forearm, and he had had various infectious diseases, but all before he was five years old.

He was brought to the hospital because of attacks of laughing and crying, followed by aimless walking. These began the preceding April, when one day he was put through a neighbor's window, and she attacked him with a broom, and threatened arrest. After this he seemed nervous and would not go to school, saying that the boys threw pieces of mortar at him as he was going home, and that the children accused him falsely of making noises. He had had several attacks, which began by twitching of the mouth, laughing and crying, and then he would walk up and down for about fifteen minutes. If restrained he became violent, trying to bite, mumbling, and saying, "Let me go." He had occasional vomiting. He improved under hygienic treatment until in May the mother thought he was well. In November, 1904, he was again brought to the hospital, because going to school had brought on a return of the trouble. In December the mother gave an account of an attack where he swept the floor with great vigor, then washed it, and then for an hour walked from room to room. He then appeared weak and pale, and the mother thought he was unconscious for about fifteen minutes. There was no cry, the head was drawn back, the eyes closed and she stated that he frothed at the mouth, but there were no convulsive movements, and he did not pass urine or bite the tongue. There seemed to be no memory of the attack. Examinations, which were repeated, showed no organic diseases, no changes in the field of vision, or of sensation, and no tremor.

This case seems to me to be one of those with attacks resembling convulsive ones, following a fright, and continued through emotional causes. The boy's teacher, as often happens with hysteria, thought he was shamming in order to avoid having to attend school.

The next case is one in which I think we have a true combination of epileptic attacks with hysterical ones.

CASE 5. The child, Grace J. T., aged twelve years, was first

seen at the Children's Hospital on September 19, 1894. (A-166.) She was an only child. The labor had been a severe one with forceps, but she showed no symptoms of any trouble afterwards. At the age of ten months she had "inflammation of the lungs," and at eighteen months pneumonia, which left the lungs "weak" for a number of years. Since she was four or five years old, about once a year, she had had attacks of vomiting which would last for a week. She had had measles, pertussis and varicella. In January, 1892, she told her mother that she had had two or three attacks in which she was unconscious for a moment, and the stomach felt badly [aura from epigastrium?]. The first attack seen was in February, and since then they had been frequent, sometimes as many as four or five a day, and again she had been as long as a week without any. The longest free interval had been two weeks. These attacks resembled the minor attacks of loss of consciousness of petit mal. In June, 1892, she was bitten by a dog, and knocked down, and since this the attacks had been more severe, being accompanied by frothing at the mouth. There were no clonic movements of the extremities, but sometimes one arm and sometimes the other would be "contracted," with the fingers flexed and the forearm extended. This "contraction" lasts five to ten minutes. She had not been going to school regularly since the attacks began, but had gone for a week or so at a time, and this always made the attacks more frequent. Before the attack she had a sensation of faintness for two or three days, and at times a sensation of dizziness, which seemed to rise from the stomach to the back of the head and neck.

On examination there was well-marked hyperalgesia in both hands and arms, and tenderness in the iliac region on both sides. The touch perception was normal, and the examination was otherwise negative. One attack at the clinic seemed to be caused by making pressure on the tender area in the right iliac region.

Under treatment the attacks became much milder again, and less frequent, but continued for the seven months that the child was under observation. They finally assumed the character they had before the dog bit her, being merely momentary losses of consciousness. Here our chief reason for calling any of the attacks hysterical was the effect of pressure upon the tender area in the abdomen, which seemed to show that it was a hysterogenetic zone, though the majority of the attacks were probably those of petit mal.

Case 6. The next case, that of Helen K., seven years of age, was first seen at the Children's Hospital on July 30, 1904. (W-148.) The family and previous history contained nothing worthy of note so far as could be learned.

About eighteen days before she was seen she had a tooth extracted, at which time she was much frightened. After this she had seemed like herself for two days, and then it was noticed that

she held her hand to the right side of the face, and complained of her tooth, though not of pain, and she was observed to laugh in a silly way. Two days later she began to have attacks in which the right arm was held rigid for several seconds and then relaxed, and she would put the hand to the face, and the eyes were rolled up. There was no loss of consciousness, and no drowsiness after the attack. She had only fallen once in the attack. The child was very irritable, but there were no tender areas found and no disturbance of sensation. During the two weeks that the child was under observation the attacks were numerous, but they were without any apparent effect upon the general condition. She was seen in one attack.

The starting of these attacks immediately after an emotional shock, and the character of them, seem to indicate that they were hysterical in nature.

CASE 7. The next case was another one in a colored girl. Alice H., thirteen and a half years old, was first seen at the Children's Hospital (P-2) on July 6, 1901. The patient was the second child of eight, four of whom had died in infancy of bronchitis and digestive troubles. The other children were well. The birth was normal and so was the history till she had scarlatina at the age of six, followed by acute articular rheumatism, since which she had never been well. She had had no other acute illness except chills and fever a year or so later.

About nine months before she was first seen the left arm began to feel numb, and she complained of a "beating pain" in the palm. Then the numbness spread to the left leg and later to the right leg also. In January she had a fit lasting two hours. Two weeks later she had another, and still another two weeks after that. The last one was four months before. The numbness continued. After the fit she shakes all over. Has bitten the tongue, and once wet the clothes and passed feces. The movements begin in the left arm, and finally become general. About twenty minutes before they begin there is a sensation of numbness in the left arm. She also complained of a great deal of pain over the eyes, and with this sometimes had vomiting. Of late she had attacks in which she would shake violently, without loss of consciousness. She had polyuria in the attacks. Lately it had been noticed that she had become very much absorbed and forgetful.

On examination there was no paresis of the face or extremities. The pupils and muscles of the eyes were normal in every way, and the fundi were normal on ophthalmoscopic examination. The tendon reflexes were lively but alike on the two sides of the body, and there was no ankle clonus. The plantar reflexes could not be obtained. Sensation for touch and pain seemed slightly diminished on the whole left side, in the face, trunk and extremities. Attempts to elicit the reflexes at times brought on a

twitching of all the extremities, and the child cried out, but when questioned said she was not hurt. She cried several times during the examination.

At a later visit the following account of the attacks was given, which is perhaps more accurate than the one first obtained, and agrees better with the character of the attacks seen at the hospital. They were said to consist of a violent shaking, which affected the whole body, including the arms, legs, trunk and "back" (opisthotonos?). The eyes would wink violently, and a spoon had to be placed in the mouth to prevent biting of the tongue. The mother also said that four months before she had had so much pain in the legs that she could not walk. On July 24 she had an attack at the hospital in which she screamed with pain in the knees and arms, with violent movements of the head, arms and legs, without loss of consciousness. In another attack she first complained of pain in the left side of the back, below the scapula, and there was twitching, beginning first in the left leg, followed by rapid flexion and extension of the leg on the thigh. These movements did not stop on pressure on the iliac region, but were controlled by the child holding the leg extended by pressing with the hands upon the knee. After the attack she said that the leg felt disagreeable, but not numb, and there was no pain.

Case 8. Walter E. F. was first seen at the Children's Hospital (X-36) on September 10, 1904. He was an only child, and eleven years old. The only illness he had had was pertussis at the age of four. There had been no fright. He had been in the country, away from home for a year, and about three weeks before he had had two peculiar attacks, and since his return home, five days before, he had had several more. In these the boy seemed cold and frightened, and the teeth chattered, but there was no loss of consciousness. Some of these attacks came during sleep, waking him up. One attack which we observed began without warning with a twitching of the lower jaw, which was apparently beyond the boy's control, but even during the height of the spasm he could open the mouth wide in response to request. He was perfectly conscious and the pupils were not changed, and reacted to light normally during the attack. There was no froth at the mouth and no succeeding drowsiness or feeling of weariness. The boy said that the jaws felt a little stiff during the attack. There was no amnesia for the attack. The attacks were continued during his attendance of four months at the hospital, at times two or three a day, but finally they lessened, and during the last three weeks there was only one slight attack, so it is probable that they ceased. The physical examination of the boy was entirely negative.

These attacks are somewhat difficult to classify and the etiology was not clear, but their sudden onset, the character of the muscular spasm, and their rather rapid subsidence seem to show them to have been of a hysterical nature.

Attacks of dyspnea in children are occasionally due to hysteria, of which the next three cases are fair examples.

CASE 9. George M., twelve years of age, was seen at the Children's Hospital first on January 7, 1899. (I-12.) The father was born in Germany, the mother in Rhode Island. The latter was supposed to have consumption till she was thirty years old, ten years ago. Eight years ago she had an attack of "nervous prostration" with convulsions. She is also subject to unilateral headaches, which come every two or three weeks. The boy's half sister, twenty years old, about a year before had a stroke of "apoplexy," it is said, without any known cause, which affected both legs. She is also said to have kidney trouble, probably some disturbance of the control of the bladder. This person was not seen, but the description seems like some spinal cord disease. The boy is the first child of the second marriage, and had one brother at the time, who was two years old and well. He had pertussis at three years and afterwards had a persistent cough with some fever and loss of flesh for some time, but for the preceeding two years he had been well.

About two years before he began to have attacks of gasping, after which he would struggle and the face would become blue, the eyes bloodshot, and the body rigid. There was no twitching of the limbs and no loss of consciousness, the duration of an attack was about fifteen minutes. At first the attacks came about once in two or three weeks, but they had grown more frequent, and in the previous week he had had ten. There was no sleepiness after the attack. They usually came during the afternoon. The parents thought he was going into consumption, and the mother had been in the habit of taking his temperature frequently in the afternoon, and often found it 99° to 100.° After the attack the boy has what the mother calls a nervous chill, and he has complained constantly of a terrible pain in the left side. He dreads being alone and cries much, and is restless and nervous, walking about between the attacks. He is said to be feverish every day, and at these times wishes to eat all the time.

The physical examination was entirely negative, including the lungs and the fundi, and there were no disturbances of sensation.

These attacks continued pretty frequently, but with intermissions, for four months, and then almost ceased, and for the six weeks before his last visit to the hospital he had had none.

CASE 10. Mary E. C., nine years of age, was seen at the Children's Hospital February 7, 1906. (BB-148.) The father died of phthisis, and the mother stated that four of her children had died of spinal meningitis at different times, seventeen, six and two years before. Three other children were living and well and were not nervous.

Four weeks before, the child had pain in the left ear for several days, but there had been no discharge. Soon after this she

began to have attacks of crying, and choking or smothering, as if she could not breathe. She had had four or five of these. The first one lasted an hour, and the last twenty minutes. The attacks began with sneezing and then she would shake. There was no convulsion, and no pain or headache. There was no cough, and the ear had not troubled her for a week. The examination was entirely negative, and during the next month she had no more attacks and was apparently cured.

Case 11. Loretta B., eight and a half years old, came to the Children's Hospital on October 26, 1904. (X-130.) There was one other child, a year and a half old, who was well. Nine months before the child had been ill with what was called "malaria," the illness lasting five weeks. She recovered but ever since she had had peculiar attacks in which she thinks she is going to die, and says she cannot get her breath. The attacks last about ten minutes, and during them she rubs her hands together and walks the floor. They have been as frequent as two a day of late. Her temper is good, and she plays, but is inclined to worry. She has complained of some pain in the left side of the abdomen, but not at the time of the attacks. The child was rather poorly nourished but the examination was otherwise entirely negative.

On closer questioning it was found that the attack of so-called malaria began with croup. The throat was not sore, but she was very hoarse and had high fever and delirium. She also had chills in which she shook, and she seemed to be better every other day. The child recovered perfectly in three weeks, and was entirely well for about fifteen months, when she had a recurrence of the attacks, which came about once a day. This time the trouble lasted about a month but was not as severe as before.

The next case is one in which we have only psychical symptoms which seem from their character and the course of the trouble to put it in the group of hysterical cases, though it may be that we should differentiate such cases.

Case 12. Regina S., five years of age, came to the Children's Hospital for the first time on October 27, 1906. (EE-110.) Her mother was somewhat nervous but had no nervous disease. The brothers and sisters were well. She had had, at the age of ten months, what was diagnosed as a slight sun-stroke, when she was dull and sleepy for three weeks. Two weeks before she was seen she fell on the steps, striking the lumbar region on the edge of a step. There was a slight bruise, but she did not complain of the back after the day of the fall. A few days later a change in her disposition was noticed. While she had always been easy to manage, a good child and cheerful, she became fretful and restless, moving the fingers constantly, and making grimaces. She also had spells of crying, and of waywardness, saying unclean words, and when reproved said she could not help it, and that

her head felt funny. She also talked to herself, and preferred to be alone.

The physical examination showed nothing abnormal. There were no choreiform movements, and the spine was perfectly normal except for a slight abrasion in the lumbar region, but no tenderness, and the urine was normal. She was isolated in the hospital and in three weeks was discharged well, the symptoms having disappeared in about four days after treatment was instituted.

CASE 13. This case is one which illustrates very well the form of hysterical astasia abasia in children. Abraham M., eleven years old, was seen at the Children's Hospital on January 5, 1895. (A-238.) The patient was the third child of six, the others being well. The previous history was of no importance, measles at two years and scarlet fever at five being the only illnesses. In December, 1893, about a year before he was seen, he had an illness in which he complained of feeling sick, and could not eat, and was feverish and chilly. He also complained of pain in the head, and the light seemed to trouble him, but there was no retraction of the head. He was ill at home for three weeks, and then was at a hospital for two weeks more. Before going to the hospital he was unable to stand or walk, but could move the legs, and when he returned home he could walk. After two weeks at home this difficulty returned, this time, however, with no other symptoms, there being simply the inability to walk, and some complaint of pain in the head, and the parents said that he seemed frightened at his own shadow. This attack lasted only a few days. The following May he had a similar attack, lasting four days, from which he recovered suddenly. He told his mother that he could walk and got up and did so. In October, 1894, he was knocked down and was unable to walk for twenty minutes. When he was seen at the hospital he was complaining of pain in the thighs, arms, back and occasionally in the head. The mother thought he could not walk as straight as other boys.

The examination was entirely negative, except for moderate tenderness on pressure at the sixth thoracic spine, and some astigmatism.

CASE 14. This case is an excellent illustration of the type of flaccid hysterical paralysis in children. Morris Q., ten years of age, was first seen at the Children's Hospital on January 23, 1907. (FF-30.) Two younger children and the parents were well. He had had measles four years before and this had been his only illness except that since the preceeding June he had had several attacks of sharp pain in the hips, knees and ankles, when they would be tender to touch. Three days before he was seen he burnt the back of the left hand very slightly, and since then the left arm had hung helpless and motionless from the shoulder, there being no movement of the fingers, hand, forearm, or at the shoulder. There had been no pain in the arm or joints.

The examination showed a total flaccid paralysis of the whole left upper extremity, with no atrophy, no tenderness, and normal sensation.

At the next visit on January 26, three days later, it was reported that on two occasions, about ten hours together, he had used the arm perfectly normally and then had suddenly relapsed. At the time of this visit there was slight power in all movements. Four days later it was reported that the boy would use the hand naturally on getting up in the morning, but after a time he would begin to complain of pain in the arm and of its being weak and in a short time the arm would drop limp to the side.

The boy recovered entirely in a short time and has remained well since.

CASE 15. Polly E., nine years old, was first seen at the Children's Hospital on December 18, 1903, when she was admitted to the hospital. (U-30.) Two weeks before she had fallen down two steps. She walked home and for a week did not complain, but about this time the mother noticed that she walked "crooked." On admission she walked on the toes of the right foot, not putting the heel to the ground. The X-ray photograph showed nothing abnormal. The foot was put up in plaster at a right angle for about ten days, when the plaster was removed, and immediately after this she walked with the heel on the ground, but after a few days it was noted that she only placed the toes on the ground as before, though she did not limp badly. This was on January 11. On January 27, 1904, when she was seen at the Out Patient Department, it was noted that when she walked the right foot was reached forward, the toes placed on the ground and then with a hop the left foot was brought up to the right one. The knee jerks were very lively but equal, and there was no ankle clonus or Babinski's sign. When lying down the foot could be flexed on the leg, and the contraction nearly completely disappeared. There was no change in sensation for touch or pain anywhere on the body. There was some tenderness in both iliac regions more on the left. There was no tenderness of the spine or on the chest. The latter part of February there had developed some clonic spasm in the calf muscles which was well marked when the attempt was made to dorsiflex the foot. A plaster bandage was again applied, but the gait remained the same, the foot with the bandage being slid forward first, and then the other foot advanced by hopping without throwing the weight on the right foot. On March 12 she was again admitted to the wards, when it was noted that the right gastrocnemius was held in constant contraction, which was increased by attracting her attention, but relaxing slightly with manipulation, and when relaxed there was free movement in the ankle joint, but considerable grating could be felt as it was moved. The right knee jerk was somewhat greater than the left at this examination, but there

was no ankle clonus, and the plantar reflex was normal and also the fields of vision, and there was no anesthesia or hyperesthesia. A systematic attempt at training was now made by analysis of motions and careful execution of each part of the step, but failed entirely to benefit. Apomorphia with suggestion of improvement and etherization with suggestion in the same way were also without benefit. One day, under the stimulus of singing by visitors in the ward, she took a few steps very naturally, but this was only temporary. She was discharged not improved, on April 21, 1904.

She was not seen again till September 14, when the condition was the same, with tonic spasm in the right gastrocnemius, the right knee jerk greater than the left one, but no ankle clonus or Babinski's sign, and sensation normal. On September 30 she was readmitted to the hospital, where under ether the spasm relaxed completely, and finally, on October 11, tenotomy of the tendo Achillis and the ham strings was done, and plaster applied. On November 10, when the plaster was removed, it was noted that it was difficult to put on because of the spasmodic contraction of the toes and foot. On November 19, with the plaster bandage on, she was still walking in the same way, sliding the right foot along, and hopping on the left one. On January 10, 1905, the plaster had been taken off two weeks before and she was still walking by hopping on the left foot, but with the heel of the right foot on the ground and without the marked plantar flexion of the foot that had been present before the operation. She could dorsiflex the right foot well, but not always, as at times spasm of the gastrocnemius prevented. On March 13 she was walking much the same as before the operation with contraction of the tendo Achillis and the knee held stiff, while passive motion at the ankle, knee and hip was good, except rotation, which brought on spasm.

She was next seen on April 18, 1905, when she had been unable to sleep, eat or sit down for four weeks. She gets up on her knees on a bench and bends the body back repeatedly. The right knee is contracted, and she cannot dorsiflex the foot. When she is put in a normal sitting position she gets in a sort of opisthotonic *arc de circle*, with jerky movements which lift the buttocks from the seat.

She then came under the care of the Massachusetts General Hospital, where she made some improvement in the severity of the symptoms, but was not cured, when she succumbed to some acute disease, and at the necropsy a normal nervous system was found.

The next case was somewhat similar but drifted off without treatment and the outcome is not known.

Case 16. Stanley W., eleven years old, was seen at the Children's Hospital on May 11, 1901 (O-124), and had been per-

fectly well till the age of seven. At that time he hurt the right heel on a stone and ever since, for four years, he had walked on the toes of the right foot. A year before a tenotomy of the tendo Achillis was done without any distinct gain.

On examination there was no paralysis, and no atrophy of muscles, but there was a contracture of the tendo Achillis on the right, which could be overcome on relaxation when distracted, while by forcible effort to dorsiflex the foot the calf muscles are made to contract very strongly. The knee jerks were increased and about equal, but there was no ankle clonus, and no Babinski's sign, and the muscles reacted normally to the faradic current.

Case 17. This case illustrates well the multiplication of symptoms such as is apt to be seen in older children. Frances L., fifteen years, was seen at the Children's Hospital June 9, 1897. (F-28.) The history of the onset of the trouble is defective, but on examination it was noted that she walked with the left knee bent and there was hyperalgesia of the left leg. Passively the left knee could not be fully extended. The knee jerk on the left was increased, and there was a front tap contracture on that side. There was tenderness over the temples and the lumbar spine as well as over the left scapula, above and below the left breast, and in the left iliac region. There was also well-marked contraction of the field of vision in the left eye.

Case 18. Gertrude F. was first seen on May 26, 1900, when eleven years old. (Children's Hospital, M-78.) The family and previous history showed nothing, the child having always been well, with the exception of having measles and pertussis, and she had never been nervous or excitable. Two weeks before she had been struck on the right hand by a cane, the blow leaving no bruise. That evening tremor of the right hand began, but it had not been constant. For three days she had complained of pain in the ring finger of the right hand. There had been no other symptoms. The tremor was fine, rapid, from eight to ten per second, regular, and affected the right arm. There was a general diminution in the strength of this arm, especially in the grasp, but no paralysis, no atrophy, and all the muscles reacted normally to faradism. There was slight hyperalgesia of the whole right upper extremity, while the perception of touch was normal. The triceps reflex was good and equal in the two arms. She was next seen at the Boston City Hospital (11507) on October 21, 1904, when fifteen years of age. The tremor she had had in 1900 had lasted two months. She had then been well till a month previously, when she was startled by a cat jumping over her as she lay in a hammock. The tremor returned a few days after this and had been persistent. She also cried often without reason and felt depressed. The menses had appeared six months before and had been irregular. She was treated by suggestion and improved, but was made worse for a time when her brother

accused her of faking and after crying spells. The tremor was made to stop at a definite hour named to her as a test of suggestion and in three weeks had entirely ceased.

CASE 19. Elizabeth McG., thirteen years of age, was seen at the Boston City Hospital on March 8, 1907 (12983). She had sprained her right hand three years before and it had been put up in splints at the hospital. Two days before she was seen she fell, striking the elbow and the hand. The pain was severe enough to keep her awake two nights. There were no bruises, but extension of the arm and other movements were painful. The hand had been very cold. On examination the right hand was found to be very much colder than the left one. The circumference of the right upper arm was 21.5 cm. and of the left 22.0 cm., while the right forearm measured 19.5 cm. and the left 20.0 cm. The pulse was equal in the two wrists, but seemed of less volume on the right, while the circulation in the hand was good, as judged by the rapidity of the return of color after pressure. There was no paralysis of any muscle, but all the movements of the hand were weak, while the upper arm muscles were strong. She could flex and extend the arm fully, as well as supinate and pronate. The flexion of the fingers was complete, but weak, the grasp registering 0 on the right and 40 on the left. Abduction and adduction of the fingers were good, and so was extension of the hand. The muscles all reacted normally to galvanism and faradism. Sensation for touch over the right hand and forearm was diminished and almost lost on the dorsal aspect of the three outer fingers. There was absolute loss of pain sense in the hand and arm up to a point just below the elbow. All forms of sensation were normal everywhere else on the body. The knee jerks were lively and equal, and there was a patellar twitch. The ankle jerks were lively and equal, but there was no ankle clonus, and the plantar reflex was normal and alike on the two sides. The abdominal and epigastric reflexes were lively and equal.

CASE 20. Agnes L., thirteen years of age, was seen at the Children's Hospital on March 6, 1907. (FF-130.) She had been admitted to the hospital on February 6, 1907. In the preceding September she began to complain of headaches, which were quite severe and lasted all day. She also complained of a dull constant pain in the left leg, which changed its position frequently, from the foot to the ankle, leg, thigh and hip. At first she could not put the foot to the ground. She fainted once at the first of the illness. She had had considerable nausea at irregular intervals about two months before, which was without relation to the ingestion of food and without vomiting except once, when she vomited a large amount of greenish material. Examination was entirely negative in every way, including the fundus, except for a limp with the left leg caused by a failure to get the heel upon the ground, though the foot could be dorsiflexed fully,

both actively and passively, when she was not walking. The headaches ceased almost at once when she was told that she could not return home till they were better. She was said to have had high fever every afternoon but there was none during her stay in the hospital. The pain in the leg at first shifted occasionally from one leg to the other, but later was in the left leg and below the knee. There was no paralysis or paresis and no contracture though the limp and the failure to get the heel to the ground continued. All the joints and the spine were normal. There were no tender areas, no disturbance of sensation anywhere and no palatal anesthesia.

The pain continued, at times disappearing for a day or two but returning, and the child is still under treatment, though there has been some improvement.

CASE 21. George G., eleven years old, was seen at the Children's Hospital on May 9, 1900. (M-52.) He had always been a nervous boy, his head shaking, and being uneasy, with some twitching. A year and a half before he fell on the floor, injuring his left elbow. It was badly swollen and splints were applied for three weeks. After this he complained a little of the arm, saying that it was numb, and occasionally dropping things. The mother thought this was imagination till lately, when he had complained more. He had cried in school from the pain in this arm. He was restless at night and seemed to have pain in the arm then. Some slight digestive symptoms, pain and gas, were the only other things noted. The physical examination, except for a lateral tremor of the head, showed nothing abnormal. In seven weeks the pain had entirely disappeared.

These cases illustrate the most common method of origin of pain as a hysterical symptom in children. That is the persistence or increase of a pain from some injury or painful inflammatory affection. Two of these cases followed an injury, while in the third case, because of the presence of fever, it is possible that the pain arose from some joint trouble.

The next case is one of aphonia, a fairly frequent hysterical phenomenon in children, though rare among our cases at the Neurological Department, probably because such cases go more frequently to the physicians for throat diseases.

CASE 22. Vina M., thirteen years of age, was seen at the Children's Hospital July 1, 1896. (D-72.) The family history and that of the child were unimportant, except that at the age of nine she had scarlet fever, and after that for a time had a twitching of the face, possibly a chorea. She had not been as well as usual for some months, being irritable, and five months before she had had quite severe twitching of the hands, and a little later of the face also, and some in the legs. This twitching, which from the description seemed to be an attack of chorea, stopped about two and a half months before she was seen. At that time

she complained of severe headaches and "numb spells." She has had these in school so that she has almost fallen to the floor. She also complained of the stomach and bowels which she thinks are bloated, and she was very constipated. Two months before she suddenly lost her speech and could make only unintelligible sounds. After a week she was able to talk again, but indistinctly for two weeks and then ceased speaking entirely. For five months she had had the numb attacks spoken of above, and these lasted for from five to ten minutes and affected the hands only. At first they came daily, but she had had none for a month till recently. For about the same length of time she had complained of attacks of dizziness lasting for about fifteen minutes in which she could not see. On examination the child could not protrude the tongue or speak, but there was no paralysis of the face or extremities, the sensation was normal for touch and pain, there were no areas sensitive to pressure and the reflexes were normal, the whole examination being negative, except for the loss of power in the tongue.

In our experience choreiform movements in children which are hysterical in character are rare, the following being the only cases where this diagnosis seemed justified by the character and course of the trouble.

CASE 23. John K., eleven years old, was seen at the Children's Hospital March 11, 1905. (Y-110.) His father was dying of tuberculosis. Two other children were normal. The boy had had diphtheria six years before and chorea four years before. For ten weeks he had not been able to use the right arm or the right leg. The arm twists about and he holds the knee bent, while at other times the leg is thrown about and he falls often. On examination he walked with the right knee partly flexed and held fixed by contraction of the hamstring muscles. He could, however, relax this contraction and stand with the knee straight, and in putting on his drawers he extended the leg fully. He fell several times, crying out that he had pain in the knee, but immediately quieted down and seemed entirely free from distress or peculiarity except for the peculiar position of the leg and inconstant, irregular, choreiform movements of the right hand and arm. The arm was held extended, pronated and every now and then was carried backward by a series of jerky twisting movements. The arm, however, could be readily flexed. When he tried to write he could not hold the pencil on account of the unsteadiness of the grasp, the pencil being frequently dropped and the incoördination also interfering. He could use the pencil perfectly well with the left hand. The boy was extremely emaciated, the heart was enlarged and showed a mitral systolic murmur which was transmitted into the axilla. The visible mucous membranes were slightly cyanotic. No tenderness or pain in the joints could be made out on passive motion, and there was no

muscular spasm. The left knee jerk was present, the right not obtained, probably because he did not relax the muscles.

CASE 24. Bessie R., fifteen years of age, was seen at the Children's Hospital on March 10, 1900. (L-172.) She was born in London but of Russian parents. There had been no other illness. When ten years old she began to have twitching of the muscles, pretty generally distributed, and since that time she had almost never been free from it, though the severity varied at different times. It had been very severe for the preceding two weeks. The menses had not appeared. She had had considerable headache but no other symptoms. The habits were good as regards the use of tea and coffee. On examination there was a severe choreiform twitching of all the muscles of the extremities and trunk, but those of the neck and face were very little affected. Occasionally a spasm of the diaphragm produced a gasp in the breathing. Nutrition and general development were poor. There was no paresis, spasticity or change in the reflexes. The heart showed no arrhythmia and the sounds were normal.

The child was seen several times between March and the middle of May, during which time there had been no change. She was next seen in November. Soon after the visit in May the twitching had ceased entirely, rather suddenly, and she had remained free from it till about the first of November when it began again. At this time the face was practically free but the muscles moving the shoulders and all of those of the extremities, both upper and lower, were moved violently. She could feed herself and do most things for herself. In December it was said that she had been vomiting every Friday night. The twitching continued during the remainder of her attendance at the hospital up to the first of January, 1901, at which time there had been a slight improvement.

This case may belong among the myoclonias, but the sudden change in the course of the trouble made us think it probably hysterical.

In regard to the frequency of hysteria in children it is difficult to draw conclusions. At the Boston City Hospital, where there is a large neurological clinic, it must be remembered that the attendance is limited to residents of Boston, and furthermore that in all probability a great many of the cases of nervous diseases in children which would naturally be seen there go to the special hospital for children, while at the Children's Hospital the cases are drawn from the whole of New England, and there are no adults treated from which to obtain relative figures. At the Neurological Department of the Boston City Hospital from May 3, 1901, to April 17, 1907, practically five years, 4,068 cases of

nervous or suspected nervous diseases were seen. Of this number the diagnosis of hysteria was made 105 times. That is, the hysterias constituted 2.6 per cent. of all the neurological cases. The cases in children at this hospital during this period were only 6 or 5.6 per cent. of the total number of hysterias, and a little over 0.1 per cent. of all the neurological cases. This is a much smaller proportion than Bruns gives as his experience. He saw 144 cases of hysteria in children, as against 550 cases in adults, and this in about 8,000 cases of nervous disease. That is, his cases of hysteria in children were 20 per cent. of all hysterias, as against 5.6 per cent.; and the hysterias in children constituted 2 per cent. of all cases as compared with 0.1 at the Boston City Hospital. His total hysterias made up 8.7 per cent. of his 8,000 cases and ours were 2.6 per cent. Duvoisin, in his statistics of 24 cases of hysteria in children, gives these cases as 0.41 per cent. of all cases. Steiner gives the hysterias of childhood as 16 per cent. of all cases of hysteria.

At the Children's Hospital from March, 1894, to May, 1907, 3,240 cases were seen in the Neurological Department, in 19 of which the diagnosis of hysteria was made, a little less than 0.6 per cent. of all neurological cases. Hysteria in children in this country, as well as in adults, judging from these figures, seems to be less frequent than in Germany, while the proportion of children with hysteria to the adult cases of the same disease would also seem to be much less, though this is more difficult to determine.

Of the twenty-four cases reported in this paper 15 were in girls and 9 in boys. The youngest was 5 years and the upper limit of age was placed at 15, at which age there were two cases. This upper limit was chosen for the sake of comparison with tables of other writers.

	Girls.	Boys.	Total.
5 years	1	0	1
6 years	0	0	0
7 years	1	0	1
8 years	1	0	1
9 years	2	2	4
10 years	0	1	1
11 years	1	5	6
12 years	2	1	3
13 years	4	0	4
14 years	1	0	1
15 years	2	0	2
	15	9	24

More than half of these cases were between the age of 11 and thirteen years. Of cases 6 years or less there was but one. Meyer in his 103 cases found very similar proportions.

	Girls.	Boys.	Total.
5 years	0	1	1
6 years	2	2	4
7 years	0	1	1
8 years	4	3	7
9 years	8	4	12
10 years	12	5	17
11 years	10	8	18
12 years	16	8	24
13 years	9	3	12
14 years	4	2	6
15 years	0	1	1
	65	38	103

Clopat and Goldspiegel's table, quoted by Philippe in Grancher, Comby and Marfan's text-book on the diseases of children comprises 272 cases.

	Girls.	Boys.	Total.
Infancy	19	1	20
3 years	0	1	1
4 years	1	1	2
5 years	4	2	6
6 years	3	2	5
7 years	15	4	19
8 years	16	6	22
9 years	15	7	22
10 years	18	15	33
11 years	24	17	41
12 years	22	13	35
13 years	27	16	43
14 years	12	8	20
15 years	0	3	3
	176	96	272

From these tables it is seen that in younger children the proportion of boys to girls is about 1 : 1, while in older children it is about 1 boy to 2 girls.

In regard to heredity in hysteria our cases are perhaps too few to throw much light upon the question. I have rejected a case in which a parent died of a shock, and one where a half sister was said to be paralyzed in both legs, as these were cases in which we should infer no hereditary taint. As regards similar heredity we find this in five cases. In one the mother had "nervous prostration" with severe convulsions, probably hysteria, or possibly epilepsy. The father was reported to be nervous once, the mother twice, and once a sister was markedly nervous and a patient at

the hospital for this. In the other cases there was no history of a functional nervous trouble to be obtained or it was distinctly denied, the cases being about equally divided.

In regard to the impairment of resistance of the child from alcoholism or constitutional disease in the parents, we obtained a history of this in four cases. In three one parent or the other had tuberculosis or had died of it, and in one of these cases several children had died of what was probably tuberculous meningitis. In one case the child's father had drunk to excess before the birth of the child.

As in Meyer's cases, trauma was perhaps the most frequent exciting cause, examples being burns, blows, falls and a dog bite. This was by far the most frequent cause if we include under this head the cases of fright, the psychical trauma of Binswanger. Direct suggestion played a part in the evolution of some of the cases as the one where the child developed convulsions shortly after seeing the mother in a convulsion from uremia, and in the case where the sister's excitement appeared to start the manifestations of the disease in the brother. Overstudy seemed a possible cause only once. Parental anxiety and the fear of tuberculosis seemed a cause once, and here, as so often is the case, the character of the symptoms developed bore a direct relation to the form of the suggestion, in this case appearing as a disturbance of the breathing. At other times the suggestion appears to have been received directly from some illness from which the child has suffered, as in the case of the child who developed the attacks of dyspnea after some throat trouble, and the case of astasia which evidently had its origin in an attack of some acute disease.

The psychical changes in these children were as a rule slight, aside from some inclination to crying, irritability and excitability.

The question of the so-called stigmata in children is interesting. On the whole, their presence is rare, and in the cases which have lasted some time they are not very common, their frequency varying more with the age. The contraction of the field of vision was only found twice in this series, once in a boy of nine, where it was slight, and once in a girl of fifteen. Since this series of cases was completed I have seen this symptom very well marked in a boy of about eight, with a hysterical paralysis of the arm. Again, the disturbances of sensation, as anesthesia

and hyperesthesia, were rather rare. In fourteen cases it was expressly stated that there was no disturbance of sensation, and in one of the severest cases (Case 15) there was no trouble of this sort all through our observation of it. Three times slight anesthesia was found and five times more or less hyperesthesia, almost always in the form of a hyperalgesia. These disturbances were rarely seen in the form of an affection of one half of the body, but more often of the affected limb, especially where there was pain in the limb, and less often when it was paralyzed. Sachs, Jolly, Binswanger, Henoch and others speak of anesthesia being frequent. Our experience is more that of Meyer, who found diminished sensation only 13 times in his 103 cases, and never a marked anesthesia. He found anesthesia of the mucous membranes (throat) only three times, while in our cases it occurred only once. He also found hyperesthesia more common than a diminution of sensation. Tender points in the abdomen, chest or spine were observed five times, in one of which the tender point could be regarded as a hysterogenetic zone. In the other cases no such areas were found. Meyer found tenderness of the spine in 9, and of the abdomen in 14 cases of his 103. Pain Meyer found to be a frequent symptom, and this was in general true in our cases, in three cases it being the most prominent symptom. Contractures were found in four of our cases, and flaccid paralysis once, astasia once, aphonia once only, tremor once, choreiform movements twice, while convulsions or minor spasmodic affections were seen in a third of all the cases, eight, four girls and four boys, and fainting attacks in one other girl. Meyer found paralysis or contractures in one fourth of his cases, aphonia seven times, flaccid paralysis twelve times, spastic paralysis seven times, and both flaccid and spastic paralysis once. Paresis only we found fairly often, especially in the cases with pain. It should be noted that the pain in these cases was never a joint pain. Chorea magna was absent from our cases unless some of the cases I have included under minor convulsive attacks be classed in this group. The tendon reflexes were not often disturbed, but at times were somewhat increased. Ankle clonus and Babinski's sign were never observed. Hysterical cough and vomiting were not seen among our cases, nor were any trophic disturbances observed. Neither did we see any of the attacks of colic with separation of the abdominal muscles, which Friedjung has recently reported as cured by suggestion.

The question of the existence of infantile hysteria must be at least touched upon. In the diagnosis of hysteria in children as in adults it is first necessary to rule out other diseases, though in some cases the possibility of the combination of organic disease and hysteria must be considered, as in a case recently seen at the Children's Hospital where a girl of about nine years who had an organic hemiplegia dating from infancy with moderate incoördination rather suddenly developed marked spasm in the affected limbs so that she could hardly walk without the shoes, as the spasm drew down the foot and toes to such a degree that the dorsal surface of the toes touched the ground first. This excessive spasm was also evident in twisting athetoid movements of the paralyzed arm associated with attempts to walk. The spasm on attempts at voluntary movement disappeared almost completely after a few weeks and all through there was considerable variability in the condition from day to day. This combination of organic and functional disease was also probably present in Case 5 of this series. Though the difficulty of diagnosis is much increased by such possibilities as these of which I have just spoken, if not at first clear it can usually be made out by watching the course of the symptoms, the hysterical cases often betraying their character by sudden cure or shifting of the symptoms in a way that is impossible in organic cases. Many cases have been reported as hysteria in children which are undoubtedly cases of organic disease, as one case recently reported by a French writer as hysteria in a young child, where there were convulsions, high continued fever, ankle clonus and Babinski's sign present, or the case of Chaffey, where a boy of ten, with persistent vomiting and optic neuritis, due to an endothelioma posterior to the right optic thalamus, had been shown by him in the earlier stages of the trouble as a case of anorexia nervosa, due to the mental shock of the loss of a favorite sister. The chief advocate of the frequent existence of hysteria in infants and nurslings is Chaumier, who in 1888 at the Congress at Grenoble, and at the French Academy of Medicine in 1891, maintained this position. He claimed that it exists in nurslings in three forms: (1) Emotional form, as in rages for slight or no cause; (2) faintings, where infants when agitated and crying hard become cyanotic, the limbs stiffen, and then suddenly become quiet, the face pale, muscles rigid, and there is loss of consciousness, fainting, and (3) convul-

sions, the characteristics being that they are short and in many cases thought to be due to meningitis, but the child gets well, and the only symptoms are the convulsions. This position has been questioned by most writers since, including many of the French authors, though most of these content themselves with quoting Chaumier as authority for the statement that many of the convulsions of infancy are hysterical, usually adducing as a reason for believing this the fact that many adult hysterics give the history of having had convulsions in infancy. Ollivier, who had been appointed by the Academy on a committee to investigate, in an article in 1892, in reviewing this stand of Chaumier, questions his conclusions, stating that it is impossible to accept them. Chaumier thinks adenoids a cause of this infantile hysteria, which Ollivier investigated and denies. Certainly in all of Chaumier's three classes of cases the evidence of the trouble being hysterical is very vague and unsatisfactory, amounting to little more than a personal belief. For his first two classes of cases proof one way or the other is very difficult, and for the convulsive attacks what proof we have seems to be against his view. Thiemich has shown that in many of the convulsions of infancy there is a tetanoid condition, with increased galvanic irritability of the nerves, and concludes that they are more dependent upon food and digestive conditions than anything else. Bezy and Bibent, in their monograph on hysteria in children, say that in the case of convulsions in infants we must first rule out intoxications and infections before we are justified in calling such attacks hysterical, and this cannot often be done. This is a point of view with which I fully coincide, as well as practically every German and American writer. Bezy and Bibent say that of 3,000 children examined all those with hysteria were at least five years of age. There have been a few cases reported occurring between the ages of 2 and 5, however, in which the diagnosis is correct. Clopat, aside from the infantile cases in his table, gives three cases in children under five. Bruns' youngest cases were two of three years, one of contracture of the foot and one of tremor of the hand, both cured by suggestive treatment. Thiemich gives his youngest case as in the second half of the second year. Bardol reports a case in a boy of three and a half years. Ollivier, however, rejects one supposed case in a child of twenty-two months who had a convulsion at eight months which left a permanent

hemiplegia. Sollier reports a case of typical hysterical convulsions in a girl of five and a half which were cured in five days. Steiner's first case of a boy five and a half years old I should reject, as there was nystagmus, inequality of the pupils, diplopia and right facial paresis with recovery. He states that there was some hydrocephalus and makes a diagnosis of pseudo-meningitis and hysteria, which seems unnecessary if hydrocephalus was present as he thought. His last case, a girl five and a half years old, is also questionable, where hallucinations developed after a slight injury, accompanied by slight unilateral tremor and paresis. Hecht has recently reported a case in a boy of four with muscular spasm and pain succeeded by flaccid paralysis of an arm after a slight injury which was cured by a single application of strong electricity, and another case in a girl of four with hysterical convulsions and astasia, while his fifth case, in a boy of five, was one with hysterical cough followed by aphonia, both quickly cured, the first by emetics and the second by strong faradism. These are the youngest cases I have been able to find in a fairly extensive search of the pediatric and neurological literature which did not seem to me extremely doubtful to put it mildly.

The earliest cases of hysteria in children are probably the imitative forms where the symptoms of some person with hysterical or other disease seen by the child are reproduced more or less exactly as the convulsions after seeing epileptic attacks in other children or hysterical attacks of the mother. With these come also the cases produced by autoimitation, where a symptom of some organic disease remains as an hysterical symptom, such as a persistent cought after the cure of some chest trouble, an aphonia after diphtheria, or recurrent attacks of astasia after some acute illness or an acute joint affection. Of course, one must remember in such cases that the organic causes of these symptoms are the more common, such as a multiple neuritis or recurrent joint disease.

Injuries, especially in the form of trifling traumata, are seen very frequently as the exciting cause of hysterical trouble in children, but in these cases the complicating effect of sympathy is well illustrated by several of Hecht's cases as well as by some of ours. The cases of paralysis produced by pure fright, the psychical trauma of Binswanger, though this includes other emotional disturbances than fright, as well as the very frequent dispropor-

tion of the physical injury to the severity of the affection, shows the large element of suggestion in the cases caused by trauma. As Thiemich well says, all hysterias are suggestions in form, but part are in content not suggested, but a pathological reaction to emotional disturbances. This is often seen with especial clearness in the cases of hysteria in children, where the simplicity of the mental life and processes shows us more clearly the relation of the mental processes to the disease than is possible in the often extremely complicated mental processes of evolution of the disease in the adult. Writers on hysteria give many causes for the disease, usually dividing them into predisposing and exciting causes. In the first group the most important is heredity, which Charcot especially insisted upon. If our concept of hysteria as a mental disease is correct we may be compelled to accept this view, though it begs the question somewhat, as cases of hysteria in which there is direct heredity can usually be explained as well by the theory of suggestion as by heredity, and in the cases of dissimilar heredity suggestion usually plays an important part. In any event the presence of a neurotic predisposition appears in such a small proportion of the cases in most recent collections as that of Meyer, where in 103 cases he found 11 with severe and 15 with slight nervous troubles in the family, that one is inclined to doubt to a large extent the tremendous importance of this factor as one is inclined to reject the similar assumption in certain forms of mental disease. Some writers give much higher figures, however, probably from less careful scrutiny of the facts in each case, as Duvoisin, who reports heredity in 58 per cent. of his cases, which number only 24, though this variation may be accidental, as he found tuberculosis in the family in 50 per cent. of his cases, while Meyer, with 103 cases, found it in only 10 cases, less than 10 per cent. Alcohol in the parents, as well as tuberculosis, has been given as a predisposing cause to hysteria. The figures of Meyer and ours both show a small number of cases where either influence was present, probably not a greater percentage than is present in the population at large.

Mills includes under predisposing causes neglect of physical health, hardship, climate and depleted conditions of the blood, and Lloyd, acute diseases. While these undoubtedly lessen the physical, and so the mental health and vigor of the child, and so are factors in lessening resistance, it seems hardly necessary to

assign them a more important place in the etiology of the disease, as Thiemich well shows when he remarks that most of this class of causes would act on all the children of a family, and yet it is rare to find more than one child in the family affected by hysteria.

Mills places among the predisposing causes improper education and this often plays a large part if we include under this term the lack of training in self-control that a large proportion of these children show. Parental example, of which he also speaks, seems to act rather directly as an exciting cause through suggestion, while Lloyd's inclusion among causes of severity and cruelty can be explained in most cases certainly as exciting causes through the emotional disturbances caused by them rather than as predisposing causes. Heubner, Meyer, Thiemich, Bruns, Mills, Lloyd and almost all other writers recognize the predominant role played among exciting causes by trauma, physical and psychical, as fear, anger, chagrin, grief, regret and especially fright. In fact, it may be that the emotional disturbance, the psychical shock of Binswanger, is the essential element in every case, and certainly is an important element in the cases after physical trauma. This is often shown very clearly by the disproportion between the physical effects locally of the trauma and the symptoms produced by it. These cases among children are especially unfortunate for the theory sometimes propounded which explains the hysterical symptoms following upon some trauma as being due to the effect of litigation upon the patient's mind, as almost none of the cases in children are the subject of suits, because the trauma is usually received from some carelessness of the child or others, or at home, in both cases preventing anyone from being made legally liable for damages, and even where there is litigation this is frequently unknown to the child. In our cases only once was there a law suit. In children, even more than in adults, both in the cases following trauma and in others we see the bad effect of parental sympathy and anxiety. Meyer instances this fact and it is particularly shown in some of Hecht's cases and in some of those reported here. Thiemich thinks this is particularly apt to be the case with late and only children.

Thiemich, Meyer and others call attention to the role of imitation, as after seeing convulsions, or polyuria and excessive thirst where a parent is suffering from diabetes. Onanism as a cause

we have failed to find, and Meyer makes the statement that he has never noticed masturbation in hysterical children. Jolly thinks while it may be a cause it is rare. Henoch reports a case of hysterical paraplegia in a boy of seven which was thought to be due to this cause. This fact, that masturbation is so rarely a cause, certainly is against the views of Freud of the frequent relation of the sexual sphere to hysteria.

Very important in the diagnosis of hysteria in children, because of the frequent paucity of objective symptoms, is the effect of suggestive treatment, the sudden cures by this means being especially frequent in children. Important in the same way, too, is the variability of the symptoms, or their complete disappearance temporarily, as was the case in several of our cases. Often the diagnosis is impossible without some such fact to aid us, or else long observation of the case, and because of insufficient observation a few cases included in our table where the diagnosis was open to doubt have not been reported in this paper. This need for continued observation is especially necessary in children, because of the fact upon which Bruns in particular has placed much stress that the so-called stigmata are rare in these cases, and are usually seen only in the older children. On this account he has called hysteria in children mono-symptomatic, a term which is useful, but may lead to confusion if we do not bear in mind that what is meant in particular is the absence of disturbances of sensation in the skin and retina, and of the tender areas. Thiemich argues from this mono-symptomatic character of the diease in children that at the time the hysterical symptoms appear there is no deep alteration of the personality as a whole necessarily present, so the prognosis is relatively more favorable than in adults.

Thiemich quotes Moebius to the effect that we should consider all pathological alterations of the body functions as hysterical which are caused by ideas, and not only intellectual ideas, but also emotional states with excitement. From this conception he rules out as possible hysterical symptoms such things as atrophy of the optic nerve, choked disc, the reaction of degeneration, loss of tendon reflexes, hemianopsia, ankle clonus, Babinski's sign, and paralysis of ocular muscles. In the main this point of view is correct, in spite of the fact that in certain rare cases of hysteria some of these symptoms have been observed, and recognizing that others may be simulated by hysterical symptoms, as an eye muscle palsy by a hysterical spasm of an eye muscle.

The number of cures in these cases in children, as given by Bruns from his 144 cases, after rejecting 49 because they were only seen once, from the remaining 95 cases 85 were cured quickly, while 7 were long cases, but were finally cured, and three cases were improved only. Of these three uncured cases one was seven years old and the other two between twelve and fifteen years. Of Duvoisin's 24 cases all but two were cured quickly, but of these 22 children 14 had a recurrence of the hysterical symptoms later. Of Meyer's 103 cases 15 had recurrence, a much better proportion, and probably nearer the average figures. This number of cases showing a return of the trouble should make us somewhat reserved in giving a good prognosis in these cases, and should make us more careful to keep the children under treatment until the mental health is sufficiently established to lessen this danger.

Eshner, Hun, J. W. Putnam, Hecht and others in America have published typical cases of hysteria in children, the article by Hecht, which appeared during the preparation of this paper, being especially full and valuable in its discussion of the subject. Lloyd, writing in 1894 in Starr's text-book on the "Disease of Children," gives an excellent account of hysteria, but thinks tremor and some other symptoms more frequent in adults than in children, while our experience and the published cases show this to be fairly frequent in the young. He also lays little emphasis in his description upon the absence of the stigmata which is so common, a fact to which Bruns and Thiemich seem to have been the first to call attention. Mills, in Keating's "Cyclopædia of the Diseases of Children," in 1890, states that convulsions are rare before puberty. This seems hardly to be borne out by the facts, especially if we include the minor attacks in which a part of a typical convulsive seizure is seen, though severe hysterical convulsions are rare in this country in children, as is also the case in adults. Chorea, as a hysterical manifestation, Mills regards as rare, in which opinion we should concur. Most of the cases of hysterical chorea are doubtful, even in the epidemics of chorea in schools, though a few cases in which the cure is obtained in a few days by suggestion, such as the case reported by Stadelman, show that it occurs. Recurrences, sometimes regarded as a proof that a case is hysterical, are as likely to be seen in chorea as in hysteria. Paralysis, we have seen, can hardly be called rare before the age of thirteen, and hemianesthesia is seen not so much in the severer forms of

the disease as in the older children. Like Mills, we have found sensory symptoms, especially hyperalgesia and pain, common. The occurrence of night terrors and somnambulism as hysterical symptoms seems somewhat questionable; certainly we have seen no instances of these troubles which seemed to us to require such an explanation. Both troubles are common in children, and of course may occur in children who are hysterical. The cases of which Mills speaks where there is elaborate fraud, where the wits are used perversely, as in ringing bells, rappings, destruction of furniture and such things may be hysterical, but they have not been seen at the Children's Hospital clinic. So also attacks of great mental excitement in children may be hysterical. The psychical forms of hysteria are somewhat rare in children, unless we include such minor phenomena as attacks of laughing and crying, which are frequent in the hysterical children, but not rare in other children also. Such attacks, when combined with twitching and trembling of the hands, or the more complicated movements known as clownism, which seem to occur independently, as well as taking the place of the passionate attitudes seen in the adult hysterical attacks, are more surely hysterical than when simple emotional disturbances alone are seen. Not infrequently the attacks of running after a slight unconscious attack, or such acts as climbing up the wall by furniture or pictures may belong in this class of phenomena, though one must not forget that they may be seen in other conditions, particularly in epileptic attacks.

In regard to treatment, all authors agree that as a rule in children the disease is more easily cured than in adults, but intractable cases are not rare such as Case 15 of this series. The plan of treatment must necessarily consider the factors in the etiology of each case, or perhaps more properly the factors which favor the further development of the disease. Here the most important thing is to remove such influences as far as possible, particularly such things as the undue solicitude and sympathy of parents or others in charge of the child. At times this is simple, as an explanation of the importance of this step may be all that is required. More often this is of no avail or makes things worse, from the fact that the unwise training of a child which has favored the development of the disease is due to defects of those in charge of it, so that the only method that is effectual is to withdraw the child from its surroundings, either temporarily

or permanently. This in itself may be made a form of psychical treatment and can be made to act as a powerful suggestion of cure by rousing the desire of the child to return to its home. This is naturally an element in the isolation treatment which is advised so frequently at the present time in the treatment of hysterical and other psychical cases. Suggestion in another form of the unimportance of symptoms is the essential of one of the methods of treatment so strongly advocated by Bruns, as he calls it, the method of purposeful neglect. The details of this method are capable of great variation to suit the circumstances of each case, and may vary from simple encouragement with disregard of complaints carried out at home, to complete isolation, with no visits, no messages from home, and no companionship of other children or patients in the hospital.

Bruns' second method which in his hands has been used with great success, as well as by many others, is what he calls the method of surprise. The object of this is to show the child that a thing it has been unable to do before can be done by it, and in this way to remove the loss of voluntary control. For certain symptoms this method is of great value, as for the removal of the various forms of paralysis, while for others, as the convulsive attacks, it seems of little use. The treatment may be used in a great variety of ways. Bruns sometimes uses simply a stern command, or again may suspend a patient with astasia and suddenly let it drop upon the feet, then, as it stands for a moment in surprise, gives the command to walk, perhaps given in the form of saying that as the child now sees that it can stand, it can also walk. The sudden restoration of the voice, or of the movement of a paralyzed limb after a single painful application of electricity belongs to this method of treatment, which is capable of a large amount of variation to meet different conditions. Here again the method is one of a single powerful suggestion. The objection has been made that this method simply removes a single hysterical symptom, and does not cure, and in a certain sense this objection is a valid one. However one should remember that this in itself is one of the most powerful suggestions of cure that can be given a patient, and where the psychical dissociation is not profound, as is probably the case with most of the children with hysteria, it may be practically a cure in itself. Jolly advises its use only to remove a single troublesome symptom, and while if

this symptom is the only one present of the disease we may call this a cure, it certainly should be followed up by such change in the surroundings and training of the child as seem to be required to confirm its mental health and remove factors which predispose to a new outbreak of the trouble. Others object that the method is brutal and may result in a fright to the child which may do much harm. This objection is a very valid one, and the manner of carrying out this treatment should be carefully considered in each case with this possibility held firmly in mind. The method of reëducation as used for adults seems more suitable for the cure of the paralyses of various forms in the case of very excitable children than this method of surprise, and is open to fewer objections. On the whole, however, this reëducation is more difficult of application to children than to adults, though this naturally is subject to great variation.

To these methods the physiological ones of electricity, hydrotherapy and other methods of this sort may be added, and in the application of these forms of treatment the suggestion cannot be neglected, whether they act only through suggestion or not. In certain rare cases the application of plaster of Paris bandages or even operation as for obstinate contractures may be required, though we have usually found that these methods failed where others had already failed, or were of only temporary benefit. General tonic treatment in debilitated children and favorable hygienic surroundings are of great importance, and while often but little can be done to change them, the mental habits of the child and its training are perhaps of more importance than all other things together. Thus, a wise physician, by timely advice in regard to the management of children and the training of them in self-control, may do more to save the development of hysteria than he or anyone else knows. Hypnotism we have used in certain obstinate cases and usually to remove some troublesome symptom, and even then with somewhat poor success, perhaps due to unfavorable conditions. The method is certainly dangerous, and especially so in children, as Binswanger says, when he warns against the danger in children of bringing about autohypnotism, or an automatic confusional state. Freud's so-called cathartic method from our experience is of very little use in hysteria in children, certainly much less applicable than in cases in adults.

With the use of all methods there is a residue of cases in which we remain unsuccessful, sometimes for one reason and sometimes for another, but certainly, with proper coöperation on the part of parents, or control of the surroundings, cure in children is much more often obtained than is the case with adults. In other cases the cure may be only temporary, and the patient relapse later, especially if the surroundings are unfavorable and remain so, in which case we are apt to see develop the severe chronic forms in adult life, though the greater number of these cases in my experience begin soon after puberty, rather than before.

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A CONTRIBUTION TO THE PATHOGENESIS OF SOME EPILEPSIES. A PRELIMINARY CONTRIBUTION.[1]

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It is practically conceded, I believe, that there is more than one epilepsy. Clinically we recognize the classical forms of grand mal, petit mal, psychical equivalents, etc.; forms that dement rapidly, which are in marked contrast with those that do not. Pathologically a multiplicity of alterations have been described, all tending to show that different processes are active. Etiologically a similar variability of factors is observable.

Notwithstanding the desire to invoke the law of parsimony in our interpretations of epilepsy it remains that a mental survey of the work done upon the epileptic group reveals the fact that a single mode of interpretation for these disorders is unattainable, and that we are in the presence of a large group of very closely allied diseases of varying etiology, pathology, and clinical course, and that the grosser phenomena of the epileptic seizure have by their very prominence served to obscure the many minor points of variation, and to foster a narrow interpretation of this group of affections.

That the time has come for a more accurate study of different forms within the groups now so widely recognized, and an abandonment of the so firmly rooted conception that all epilepsies are alike and due to a single cause, is recognized by most students of these phenomena. Without burdening this communication with a host of citations from ancient and modern authors it will be readily conceded from this standpoint that many epilepsies are due to well-marked organic changes. Certainly the masterly studies of Alzheimer[2] on gliosis of the cortex and of the Ammon's horn and many others leave no ground for doubt in this direction.

[1] Read at the thirty-third annual meeting of the American Neurological Association, May 7, 8 and 9, 1907.

[2] Monatschrift f. Neurologie u. Psychiatrie, Vol. IV.

We may naturally exclude at the outset the epilepsies due to tumors of the bones of the skull, meninges and brain, inclusive of echinococcus, porencephalic defects and cysts, traumatic lesions, scars, hypoplasias and microgyria of a hemisphere or a part of one, chronic leptomeningitis, encephalitis, and insular sclerotic atrophies. Other epilepsies have no such organic basis and an explanation must be sought on the basis of a yet unearthed disturbance of cellular metabolism. Munson's recent summary is illuminating in this direction. Further, other forms, and many of these offer fine problems in diagnosis, are interpretable on the hypothesis of a vaso-motor disorder with or without other factors. These three general factor groups, expressed in the broadest terms, do not by any means exhaust the field, but their statement is sufficient to emphasize the point of view here outlined.

Such a general point of view I believe will necessitate the closer analysis of the clinical findings and will afford valuable diagnostic, prognostic, and therapeutic outlooks. To illustrate in one direction alone. We all have been called upon to make a differential diagnosis between epilepsy and hysteria (both terms here being used as word diagnoses for symptom groups of different orders). The ablest of neurological students have confessed their inability to pin on a proper label in some of these patients, even after, at times, weeks of observation. Certainly such borderland conditions, if included within the epilepsy circle, are very different from the epileptic seizures in a diplegic idiot, and between these widely contrasting epilepsies there is a host of gradations. An epilepsy so closely resembling a hysteria or a psychasthenia has an entirely different pathological foundation, a widely variant prognostic outlook, and is in need of a quite dissimilar mode of therapeutic attack.

While not in any position to make a clinical differentiation of any group here, nevertheless it has been borne in upon me that there is a class of epilepsies which have a striking clinical resemblance, due as I believe to closely related etiological factors. There are the epilepsies following an acute poison of some nature; usually the toxin of an acute infectious disease. The epilepsies so prone to follow scarlet fever, measles, diphtheria, whooping cough, streptococcus infections, and influenza offer the most striking examples. It is unnecessary to quote the numerous studies that bear on these infectious diseases as factors in the

etiology of from 5 to 10 per cent. of the cases subjected to the usual statistical inquiries.

While it is yet uncertain what are the affinities or perhaps lack of affinities of the poisons of these differing infectious diseases, and their relation to other poisons, it has been amply demonstrated by a number of workers in pathology that alterations in the character of the blood and changes in the walls of the blood vessels throughout the body are very characteristic, and it is not a far cry to apply the knowledge of such study on vascular alterations to such changes in the brain. Minute hemorrhages, capillary thrombi from agglutination, endothelial alterations are the precursors of the more extensive and permanent glia alterations, made necessary by the general phagocytic activity of these connective tissue elements. This introduces us to the subject of neuronophagia and its implications.

Having had under observation a striking example of poisoning by santonin in a young child with the production of a permanent epileptic state, I was tempted while in Munich last summer (1906) to make some experimental studies with this substance, in order to gain some information concerning its action on the cortex of lower animals. As this particular type of substance is well known to have an action on the motor cortex which is very striking, if not characteristic, it seemed an excellent opportunity for the carrying out of a histo-pathological research which might offer an interpretation for the histogenesis of some of the epilepsies that are met with clinically. I desire at this place to record my indebtedness to Dr. A. Alzheimer for his generous assistance while I was in the Psychiatric Clinic of Professor Kraepelin.

As I have already reported[3] the general clinical history of my patient I will give here only a brief summary of the striking facts.

The patient is a girl, the only child of healthy parents. She had always had fair general health, although somewhat nervous, and had always been much constipated.

In the evening of November 5, 1902, after passing a lumbricoid worm, the girl was given one santonin troche, U. S. P. (These troches are supposed to contain about one half grain santonin.) On the morning of November 6 she was given another troche of santonin and a tablespoonful of castor oil. The castor oil failed to act, and the girl had no movement on November 6.

At seven o'clock on the morning of the next day, November

[3] Medical Record, March 31, 1906

7, the mother noticed that the girl was unconscious and was in convulsions. How long the little girl had been so the mother did not know, as the patient made no outcry. She was then given a hot mustard bath and the family physician sent for. When he arrived at 7.30 A. M., November 7, the child was unconscious, and was having a spasm every fifteen minutes. She was given enemas, both superficial and deep. From 7 A. M., November 7, until 7 A. M., November 8, general convulsions occurred at intervals of from fifteen minutes to half an hour.

Beginning at 11.30 A. M. on November 7, chloroform inhalations were resorted to, to abort or relieve the spasms. These inhalations were kept up for twenty-four hours, or until the cessation of respiration during the spasms was so prolonged that it seemed inadvisable to employ it further. After the first twenty-four hours, it was necessary to resort to artificial respiration by the Sylvester method, during the spasms.

The patient recovered consciousness and spoke for the first time about 7 A. M., November 9—forty-eight hours after the onset of the attack.

The treatment of the patient during the stage of unconsciousness need not be here summarized as it is already available.

From November 9 on the restlessness and irritability were less and the interval between the convulsive attacks was lengthened. The patient soon began to show signs of exhaustion. When attacks came on the spasm of the respiratory muscles was so long that the patient became very much cyanosed. After the first day the patient could swallow nothing and was fed by the rectum.

At this time, and for three weeks following, the girl was blind, having scarcely any perception of light. No fundus changes could be detected. From November 9, the time of regaining consciousness, until November 25, a slight temperature elevation was noted, the girl was exceedingly restless, picking at the bedclothes and at her nose and ears, and keeping up a most exasperating cry most of the time.

The temperature had remained normal two weeks when December 10 there began to be a rise of one or two degrees in the evening, possibly due to a nephritis, which had been developing for some time. December 12, temperature 101° F.; December 13, temperature 102° F.; December 14, temperature 103° F.; December 15, 104° F.; December 16, 105° F.

December 16 she passed but eight ounces of urine. She was totally blind, restless; the skin was dry; the urine was loaded with albumin, and the feet were slightly swollen, eyes puffy.

The patient gradually recovered from the effects of the acute nephritis and by December 30, the temperature was normal and the albumin had disappeared from the urine. The girl's sight improved and returned to normal with the disappearance of the fever and the albumin. From this time on till January 20, 1903, there was a steady improvement in health and strength.

On or about the early part of February, 1903, the mother noticed that the child would suddenly fall down, or rather *sit* down, and as quickly get up and go on with her play, as if nothing had happened. This "sitting down" was a sudden bending of the thighs on the trunk, the knees remaining unbent. There was no cry, no pain, simply a sudden drop and up again, as though the muscles were weak and gave way. These fallings were more frequent during or after excitement. Usually they occurred about six to ten times a day. In March the girl began falling forward instead of sitting down, so that she frequently fell against the furniture and cut her face. In the latter part of April, 1903, the attacks ceased, but recommenced late in May or early in June of the same year, when she had from two to ten daily. Sometimes they lasted from half a minute to two (?) minutes. July 8, the attacks ceased for three days. July 11, the attacks recommenced more severely than ever.

On August 12 the physician was holding the child on his lap when she had a severe tonic spasm, very closely resembling an epileptic seizure. This attack continued for one minute, by the clock. The girl was in a sitting posture. Suddenly her head bent forward, her muscles were in a state of strong contraction. She cried out "Mamma, Mamma!" in a weak voice, and seemed to be in great pain. There was no suffusion of the face, no frothing at the mouth, and seemingly no unconsciousness. In a similar attack in the morning, she passed water, and while doing so she cried, "Oh, Mamma, I am peing!" She yawned several times immediately after the attack, and was evidently weak, but she showed no inclination to sleep.

While in my office at the time of the first consultation, the patient had two of the attacks, which have been characterized as simple muscular weakness. They were typically of this character. The girl, simply and without any loss of consciousness, seemed to sit down, or in one instance seemed more to slip, the floor being of polished wood. In no particular respect could it at this time have been recognized as a *petit mal* attack, notwithstanding the extreme variability of this type of epileptic seizure.

As for the distinct epileptic seizures, which ultimately developed, I have seen but one, and this was atypical. But the family physician has seen a number, and he has had a large experience with epilepsy in children. Thus there is little doubt in my own mind, at least, as to the correct diagnosis.

The further history is of interest. In a letter sent to me by Dr. Marshall, the family physician, he reports improvement:

"August, 1903, had fits, one, two or three times daily. Severe fits two or three times a week. September 12, 1903, began giving bromipin ℥ii, three or four times daily. No appreciable effect. Continued until November 11. November 1 the mother reported that the 'falling spells' had ceased for a week (the dropping on

the floor and getting up immediately, as described above), but that the 'real hard spells' were as numerous as ever. November 9 she reported that the child had had no fits during the day, but had had them in bed just before rising in the morning. November 11, 1903, began giving a bromide preparation. November 17, the child's legs were so weak that she staggered and could scarcely walk alone. Stopped the medicine one day, and then began giving it again, and have continued it to date, December 16, 1903. The girl has had no fits of any kind since November 9. The only other treatment beside the sedatives mentioned, has been the continued use, three times daily, of an intestinal antiseptic tablet."

On January 16, 1906, the family physician writes: "In reference to our patient, I would say that on looking over my book I find that the girl has had an attack which from the description, I presume to be epilepsy, about once in two months, since December, 1903. You have her history previous to that. Most of these attacks are very mild, and of only momentary duration. She had quite a severe fit on January 1, 1905. I was sent for, and the next day she showed the typical rash of measles, from which she recovered in about a week. In the fall I advised her mother to send her to a kindergarten. I am sure that the diversion and the getting away from her nagging mother has done her good. I expect her to recover fully some day. She has had from five to ten grains of brometone daily for varying periods, whenever she seemed nervous, and along about the first of the month, when an attack might be expected.

"My last report, just previous to going to press, February, 1908, is that the patient has not had a major attack for about a year, but that now and again attacks of dizziness occur, and the eyes turn to one side. On being addressed the patient pays no attention to what is being said, then in a few moments is all right. At these times while in class-room will execute automatic movements. On one occasion she marched out of school and came to herself on the stairs.

"Last attack was in November, 1907. On election morning, 1907, she had a nervous spasm, all of the muscles shaking. This was while asleep in bed. Intellectually she is on a par with others of her age."

Here was a striking case of poisoning by a comparatively well known substance which was followed by symptoms of marked cerebral irritation, by retinal edema, by edema of the kidneys, and by evidences of profound alterations in the blood vessels of these organs. This was followed by a condition of chronic epileptic convulsions due I believe to the alterations set up in the cortex.

The poisoning is not an isolated occurrence and the symptoms are classical, but the after-coming epilepsy is unique.

An examination as to the mode of action of santonin is of moment in this connection and as having a bearing on the subsequent animal experimentation a brief summary of our present knowledge concerning this substance may be of interest.

The pharmacology of santonin has attracted considerable attention, and for some time it was thought that the convulsive seizures to which its administration gave rise were of spinal origin, but of recent years the work of Turtschaninow,[4] Kramer,[5] Berkholz[6] and others has led to the belief that the epileptiform convulsions are due in the larger part at least to stimulation of the cortex—although it is highly probable that the medulla is involved as well. In Turtschaninow's experiments on dogs the sigmoid gyrus being removed epileptiform convulsions were not elicited, yet Luchsinger's[7] work, into which we cannot go at this time, might show that the entire spinal cord is finally excited to react in somewhat the same manner as with strychnine.

The symptoms as shown in the lower animals (rabbits) are very characteristic. After a small dose—0.1 to 1.0 gram of santonin to 1,500 grams of rabbit—in my experiments sodium santoninate was chosen as being soluble and as determined by previous observers, notably in Binz's experimental work,[8] showing the identical picture of poisoning—the animal commences to show in from ten to fifteen minutes marked increase in respiration and the pulse is quickened. The ears become redder and hotter, due to the increased blood supply and heightened respiration. Following the smaller dose this may be all that is observed. Following larger doses one observes in about a half hour after the injections the beginning of fine fibrillary contractions—the animals shiver and tremble but doses less than 0.8 gm. do not cause convulsions. In doses over one gram to 1,500 grams of animal convulsions commence to appear, and the development of the poisoning is more rapid.

Thus in a rabbit weighing 1,100 grams an injection of 1.5 grams of sodium santoninate was given at 9.20 A. M. At 9.30 breathing was very rapid; at 9.48 cramps began to appear, first in the muscles of the facial region. The nasal folds were ele-

[4] Arch. f. exp. Path. u. Pharm., 34, p. 208.

[5] Zeitschrift f. Heilkunde, XIV, p. 367.

[6] Dorpat Dissertation, 1893.

[7] Pflüger's Arch., 34, 1884, p. 293.

[8] Arch. f. exp. Path. u. Ph., 6, p. 300.

vated, the mouth was opened and closed with great rapidity; the rabbit jerked his neck back in clonic spasmodic movements. Respirations were 100. The spasms persisted a few moments only. The rabbit lay down quietly until 10 o'clock, when he suddenly arose, his head drew back, the limbs strongly extended; there were strong convulsive movements of the facial muscles at first, somewhat tonic in their character, and quickly followed by short, rapid clonic movements. These lasted only a few moments. The rabbit then ran about his cage, reacted to noises—had no loss of sight and apparently no variation of smell.

10.30, convulsion reappeared; in this attack the right leg was affected soon after the convulsions in the nose, eyelids and face; then generalized stiffness alternating with clonic convulsions occurred; opisthotonus developed, succeeded by rapid clonic spasms. This attack lasted about two minutes and was succeeded by a quiet period. 11, another attack, shorter in duration. 11.03, another, few muscle twitchings, first of mouth, the ears then drawn back, the teeth chattering, grinding, followed by strong tonic extension of all extremities, neck thrown back, opisthotonus, rising of animal on hind legs, clown-like tumbling over backward, the animal pitching over as a result of the clonic convulsive leg movements. The mouth continued to twitch throughout attack. Respiration 90. A few moments later a similar severe attack. Foam-like saliva exuding from mouth, increased secretion, chattering and grinding of teeth. From this time on the animal passed from one convulsion to another in a few moments with marked opisthotonus. 11.05, a similar attack, persisting 1½ minutes; 11.07 a slight attack; 11.09, severe attack with marked flexor contractions, signs of beginning weakness in fore-limbs; 11.20 rabbit unable to get up—twitchings and jerkings continue. Respiration 100, irregular; lay prostrate, exhausted from successive convulsive status. 11.27, opisthotonus strong, eyes closed; when opened noted sharp symptoms like jerks of eyeball. Pupils dilated, responsive to light. Rabbit lay on side, making ineffectual attempts to right itself. Strong contractions in muscles of neck, pawing the ground sideways with front- and hind-limbs. Clonic and tonic convulsions alternating, lightning-like twitches throughout entire body. 11.48, exitus, heart continued to beat two minutes after respiratory paralysis.

This is the picture in the fatal cases. Doses of 1.2 grams of

sodium santoninate will cause several convulsions from which the rabbit recovers in from one to two hours, and next day is well and running about the cage, eating apparently in a normal manner. From the clinical evidence it would appear that the convulsions are of a mixed type, and it is certain that they are not all cortical.

Three rabbits were under observation. After obtaining a clear picture of the acute poisoning by gradually ascending doses, it was my object to study two things: first, the changes induced by acute poisoning, and, secondly, the alterations due to chronic poisoning. Rabbit 1, whose story has been given, served as material for the first purpose. It had been poisoned daily for a week in the effort to determine the lethal dose. Rabbits 2 and 3 were kept on smaller doses 0.2–0.3 grams for a week and the dose gradually raised to 1.0 gram of sodium santoninate daily. The giving of 1.0 gram caused for the first three weeks marked convulsive movements in Rabbit 2, weighing 1,400 grams, but not in Rabbit 3, of 1,525 grams; 1.2 grams was needed to influence the latter. A gradual toleration developed, but the two rabbits were kept on hypodermic doses varying from 1.2 to 1.5 grams of sodium santoninate daily for a period of about ten weeks. It was my intention to subject one rabbit to the action of santonin for a period of five months, but owing to technical difficulties one rabbit was lost and the other was utilized for a photographic analysis of the convulsive movements with the biograph apparatus. Excellent films were obtained.

A complete study of the lesions of the nervous system, confined for the most part to the study of the cortex was made of the case of acute poisoning. The two cases of chronic poisoning have not yet received complete study, only a few slides having been studied for the chronic changes. The further work in these must be left for a later period of quiet.

The lesions of acute santonin poisoning in the cortex are of interest, first as showing marked similarities to changes induced by other nerve poisons, and secondly as affording evidence that the mode of experimentation is valid and can lead to useful results if followed out more fully and completely than the time at my disposal would allow. It is essential that for the study of the changes of chronic poisoning, and through these the interpretation of the acute, that a period of over four months must be allowed.

Although complete detailed studied of the cytology of the rab-

bit's brain are still a desideratum, the work of F. Bailey forms an excellent standard of comparison for work of this nature.

The methods of examination employed were various. For the most part the Nissl and Cajal methods were used as routine procedures. Special fat stains were employed to ascertain the presence of fatty degeneration, and the Weigert glia method followed out for the study of these elements to determine their alterations in chronic poisoning.

I am unable to compare the results obtained with those of others, as no work known to me has been done on santonin since the advent of the finer cytological methods. Kobert remarks in the last edition of his monumental work on poisoning that such histological investigation of santonin poisoning is highly desirable. Bechholz,[9] working in Kobert's laboratory, confined his studies to dogs and investigated chiefly the mechanism of the movements. He compares the movements with those of epilepsy, in which he states he agrees with other authors before him. He maintains that the psychomotor cortex is the portion involved, but does not give any histological evidence.

Damme, in an interesting study largely directed to the investigation of the temperature variations, calls attention to the very widely dilated blood vessels of the periphery.

There is naturally a large literature bearing on the subject of yellow vision, but no clear understanding of the cause for this phenomenon is forthcoming.

The alterations, gross and cytological, following acute poisoning are very striking, although they have many parallels in other convulsion-producing poisons. Macroscopically an intense congestion of the meninges and of all of the cerebral vessels large and small is apparent. This is not due to the convulsive movements exclusively, for the dilatation of the blood vessels of the ears and the elevation in temperature of the ears were very striking in our experiments, and Damme has, as already cited, made a particular study of these phenomena. It is interesting to note that this marked peripheral dilatation is largely conservative, and is apparently a device to cool the blood as rapidly as possible, and thus avoid convulsive seizures, for among the many experiments

[9] Experimentelle Studien über die Wirkung des Physostigmins, Santonin, Codeine, Cocaine, Strychnine und der Carbolsäure auf die psychomotorische Zone der Grosshirnrinde. Dorpat Dissertation, 1893.

made by Damme we find that if the animal is kept in an oven and this cooling is prevented there is a marked increase in the force and frequency of the convulsions. The redness and heat in the ears are pre-convulsive phenomena.

Microscopically one finds the meninges and vessels tinged with blood, and minute capillary hemorrhages are common and widely diffused. The cortical vessels are more often involved. Whether increased pressure or vessel wall alteration or both are responsible, I have not yet been able to determine.

The changes in the ganglion cells, in brief, are those analogous to many acute poisons already made known by many investigators. They are not specific so far as I have been able to observe, yet the chromatolysis and nuclear alterations are not as marked as in many other poisonings.

Metachromatosis under Nissl treatment is a striking characteristic. The glia cells show no marked alterations. They take on a greenish tinge, however, and come out in strong contrast with the blue of the ganglion cell. In view of an interesting swelling that is observable in chronic poisoning the metachromatosis is worthy of recording.

The motor ganglion cells are uniformly altered. The large processes show the influences of being swollen; in fact, the entire ganglion cells show evidences by the vacuolation lines of having been swollen. The vacuolation is not a post-mortem alteration, nor do I believe an artefact. Its prominence in the main dendrites is of interest. Acute irregular swelling of the main dendrites is also observable. The Nissl bodies which are well ordered in the rabbit in the cells of the mid-psychomotor zone have lost their outlines, have become finely granular, or have suffered complete destruction. There is a marked chromatophilia of the cells. Remnants of intact Nissl bodies may be noted in places. Many cells are so widely changed that their death is undeniable. Marked changes in the nucleus beyond the marked chromophilia of the nucleolus were not observed.

The neuroglia was altered somewhat from normal neuroglia, yet in what direction it is difficult to say. The metachromatosis is peculiar and there is seen a marked tendency for a grouping or hugging of two or more glia cells about the base of the ganglion cells. What the significance of this is is uncertain.

One might go into considerable detail but the main facts

brought out show for the acute poisoning marked alterations in the structure of the psychomotor zone. The ganglion cells are much altered, some are doomed to destruction, and there is a fairly constant reaction on the part of the connective tissue elements.

From the picture of the acute poisoning in animals it is a fair inference then to conclude that a similar acute process took place in the cortex of our patient, which was accompanied by some of the cell changes here noted. There is every reason to argue that the destruction of some ganglion cells took place, and we must turn to the chronic poisoning specimens to determine what were the subsequent developments in the cortex of the patient.

I have already intimated that I have been unable to finish my work on the chronic poisoning cases. This was due to the fact that the time chosen for the continuance of the poisoning was too short—ten weeks, it should have been at least four months—and that sufficient leisure has not been found to make the exhaustive search that such histological studies demand. The specimens of ten-week poisoning, however, do show enough to point the way in the direction of what takes place in the cortex after the acute process has passed.

From the observations to hand, following the death of the ganglion cells, a process of neuronophagia takes place. The precise character of this mechanism in chronic poisoning I have not yet been able to follow in many of its details, nor is there, in view of the work of René Sand on neuronophagia, any need of going over the evidence cited in this interesting monograph.[10]

Hajos,[11] in his excellent work on the cell changes in epilepsy, shows the effect of the same process, and the studies of Alzheimer, Franci, Weber, Orloff and others accentuate the value of the findings in this present study. Neuronophagia, or, as Marinesco prefers to call it, necrophagia, is a definite process in these experimental studies, and there is little doubt that it is the precursor to that gliosis which gradually replaces the site of the dead cells and which is such a characteristic picture in the brains of many epileptics.

In this preliminary communication all that I can hope to show

[10] La Neuronophagie. Par René Sand, L'Académie Royale de Médecine de Belgique, Vol. XIX, 1906. Memoir Couronné.

[11] Hajos, Archiv f. Psychiatrie, Vol. 34, 1901.

is that following santonin poisoning there results a definite cellular alteration in the ganglionic areas of the psychomotor area, with death and destruction. This accounts for the profound functional disturbances resembling epileptiform convulsions. That as a result of chronic poisoning by the same substance a definite form of neuronophagia is demonstrated, which by inference from the many studies bearing on this subject, may be interpreted as a forerunner of a glial infiltration. Whether this latter will be found in my specimens I cannot yet say. Should such be the result, it would afford excellent evidence for a more definite understanding of the cellular mechanisms which lead to the gliosis of many chronic epilepsies. Should an opportunity offer, I hope to be able to complete the analysis of the specimens secured, and offer at greater length a number of interesting features of the research, which have not been included by reason of the preliminary character of this communication.

Society Proceedings.

THE PHILADELPHIA NEUROLOGICAL SOCIETY.

October 22, 1907.

The President, Dr. Alfred Gordon, in the Chair.

A CASE OF CEREBRAL MONOPLEGIA.

By Horace Carncross, M.D.

In April, 1906, this patient (a woman of 39 or 40 years), after a period of overwork and worry, became paralyzed on the left side while in bed at night. She could not be awakened in the morning and remained unconscious for three weeks. No history of convulsions was obtained. At the beginning of the attack the mouth was drawn and the eyes were turned to the right. For a week after gaining consciousness the patient states that she was irrational. Then her mind became clear, and she also became able to draw up the left leg, which, as well as the left arm, had been utterly powerless. About two months after the onset of the attack she began to walk. The arm improved little until the next winter.

At the time of examination (October, 1907) the power of the right side was good. The left arm could be raised slightly, but the forearm could not be flexed unless the arm was raised so as to be assisted by gravity. There was no power of pronation or supination, there was complete wrist drop and an extremely faint grip. The left lower limb showed power practically equal to that of the right side. The face, which on casual glance appeared symmetrical, showed slight traces of the former paralysis in the lower part of the left side, with an extremely slight deviation of the tongue to the left.

The gait, station, coordination of right arm, cranial nerves, electrical reactions, sensations for pain and touch, and superficial reflexes were all normal. But the deep reflexes were universally increased, and those of the left arm were markedly exaggerated. There had been a clonus of the fingers of the left hand, which could not at this time be developed. There was practically no wasting.

The heart, which was enlarged to the left, presented a systolic murmur at the apex, with a relatively accentuated second pulmonic sound, and the pulse was irregular in force and rhythm. There was a history of two attacks of rheumatism in years gone by. The urine was negative.

The cerebral monoplegia which followed the original complete left-sided hemiplegia, was diagnosed as due to an embolism of the ascending frontal branch of the middle cerebral artery of the right side.

Dr. C. K. Mills stated that the case though simple in its aspects, is interesting because cortical monoplegia is comparatively rare. In regard to the loss of sensation, he thought it possible that the patient might have had a temporary loss of sensation and yet the lesion have been

largely restricted to the pre-central convolution. It is well known that in embolism we sometimes have an effect beyond the locality of the obstructive lesion, so that the post- and pre-central convolutions may have both been involved in the acute condition of the lesion. The impairment of sensation in such cases from purely motor lesion, may be due to disruption of the neuronal associations between the sensory and the motor area.

Dr. A. A. Eshner said he wondered whether it was perfectly precise to designate this case as one of monoplegia. Although Dr. Carncross demonstrated that the paralysis predominates in one upper extremity, he also pointed out that the reflexes in the lower extremities are exaggerated, which must indicate some involvement of the motor fibers passing to these parts. With respect to the situation of the lesion, the question naturally arises whether in the presence of a cortical lesion one would not have a right to expect some convulsive tendency, either a convulsion at the inception of the disease, or epileptic seizures since them.

Dr. Spiller said there are a few cases of monoplegia from lesion of the internal capsule on record. The case presented by Dr. Carncross, he thought, must be regarded as cortical in origin. The middle cerebral supplies the whole of the motor cortex of the brain except the extreme upper part. Dr. Spiller has a brain in which there was complete destruction of the middle cerebral and of the whole Sylvian distribution, yet the uppermost part of the pre-central convolution and the whole of the para-central lobule were preserved, as that portion of the brain is nourished from the anterior cerebral artery. In that case the fibers coming from the para-central lobule were destroyed within the white matter of the brain by the sclerotic lesion and there was hemiplegia. Dr. Spiller had another brain in which there is destruction of the cortex in the central convolutions only in the upper part, in the center for the upper limb. In the case presented by Dr. Carncross, we may suppose there was an embolus of the terminal portion of the middle cerebral artery, and that the cortex of the upper limb center was destroyed; it is possible that the anterior cerebral artery extended further downward on the lateral surface of the brain than in most persons, and that the center for the lower limb has been chiefly supplied by this artery.

Dr. Spiller doubted whether anyone can find in the literature a case of cerebral monoplegia of the upper limb without some involvement of the face. Monoplegia is to this extent a misnomer. He did not observe any weakness of the orbicularis palpebrarum muscle or of the flexors in the lower limb in this case that Dr. Carncross had shown.

Dr. Carncross did not know whether convulsive seizures had ever occurred. The increased reflexes are in the right and left leg and right arm, and especially in the left arm.

A CASE OF UNILATERAL HEMORRHAGE IN THE MEDULLA FOLLOWED BY SYRINGOMYELIC SENSORY DISTURBANCES.

By Alfred Gordon, M.D.

A man of 48 was suddenly seized with an intense vertigo, objects turning to the right. He soon lost consciousness. An hour later there was inability to swallow and expectorate, also shortness of breath. Upon examination were found right facial palsy, narrowness of the right palpebral fissure, with marked tremor of the tongue, difficulty in moving the

tongue, complete loss of the three forms of sensation on the entire left side, cerebellar asynergy, holding the head on the right shoulder, and titubation with a tendency to walk to the right.

At present he has some difficulty in swallowing on the right side, some shortness of breath, some facial deviation to the left, narrowness of the right palpebral fissure, syringomyelic sensory dissociation on the left side including the face, tremor and atrophy of tongue with R.D., and stiffness of the jaws.

Dr. Gordon said, as to the possibility of polioencephalitis, he considered that at the beginning, but he was absolutely unable to explain the condition in that way. The cerebellar attitude of the head, and the cerebellar gait were most typical of a cerebellar or peduncular involvement. How polioencephalitis could affect at the same time fibers of the peduncle he could not explain, so he had diagnosed hemorrhage. The latter could explain all the symptoms. The cerebellar symptoms cleared up.

As far as he knows only a few fibres of Gowers' tract enter the white reticulated substance. The majority of the fibers of Gowers' tract go around the lateral lemniscus to the superior cerebellar peduncle. Assuming that the fibers of the white reticulated substance are involved on one side, the minority of Gowers' tract would also be affected. That the destruction of a few fibres of Gowers' tract would give such a complete picture of sensory disassociation is a question which has remained unsolved in Dr. Gordon's mind. When he examined the patient three days after the apoplectic insult he found while pain and temperature were entirely abolished on the left side, touch was also somewhat affected, but later touch returned. As far as the sensory disturbance of the face is concerned, of course Dr. Dercum's suggestion would perhaps explain the question of polioencephalitis. But on the other hand it does go hand in hand with the entire picture.

THREE CASES OF LESION OF THE EPICONUS.

By William G. Spiller, M.D.

Minor, of Moscow, has pointed out that a lesion of the gray matter of the fifth lumbar and first and second sacral segments of the spinal cord gives a definite clinical picture, characterized by the presence of certain symptoms and the absence of others seen in lesions of the conus. He has given the name of epiconus to the portion of the cord mentioned. The symptoms are atrophy and paralysis of motion and sensation in the innervation of the sacral plexus, especially in that of the peroneal nerves. The gait is of the steppage type because of foot-drop. The flexors on the back of the thighs and gluteal muscles may be weak because of the implication of the fifth lumbar segment. The Achilles tendon reflexes and plantar reflexes are lost. The sphincters of bladder and rectum and the sexual functions are not affected because the conus in which the centers for these functions and muscles are situated and the white columns above the conus are not implicated. The patellar reflexes are preserved, as the lesion does not extend into the fourth lumbar segment.

Dr. Spiller has had three cases of lesion of the epiconus. The first and third were traumatic in origin. The first has already been reported by Dr. Weisenburg.

The second case was seen with Dr. Alfred Stengel. The symptoms developed after pneumonia, and the diagnosis was poliomyelitis of the

epiconus. The patient, a man, had bilateral foot-drop, slight on right side, intense on the left side, but in other parts of the limbs the voluntary power was good. The muscles of the legs below the knees were wasted. The patellar tendon reflexes were exaggerated. Ankle clonus was obtained on each side but was soon exhausted. The ankle clonus with the pronounced foot-drop of the left side was very striking. Micturition and defecation were not disturbed. Sensations of touch and pain were normal in all parts of the lower limbs. Babinski reflex was obtained feebly on the right side.

The third case was a man who on November 6, 1905, fell and struck his back on the lumbar and sacral regions. At the present time the sensations of pain, heat and cold are diminished, but not lost, over the outside of each leg and dorsum and sole of each foot, especially on the right side, but are normal on the inside of each leg and on the back and front of each thigh. Tactile sensation is normal everywhere. The patellar reflex is present on each side but is much diminished. The Achilles tendon reflex is nearly normal on the right side but is weak on the left side. Complete foot-drop is present on each side. Babinski sign is not present on either side. The flexor muscles on the back of the thighs are a little weak. He has no disturbance of bowels or bladder.

Lesions of the epiconus have attracted little attention.

THE PHILADELPHIA NEUROLOGICAL SOCIETY.

November 26, 1907.

The President, Dr. Alfred Gordon, in the Chair.

SYMPOSIUM ON ACUTE ANTERIOR POLIOMYELITIS.

THE POLIOMYELITIS EPIDEMIC IN THE STATE.

By Spencer M. Free, M.D., Dubois, Pa.

Dr. Free presented three cases illustrating the various types of the disease which has been epidemic in the interior of the State of Pennsylvania. Dr. Free gave the histories of about one hundred cases of poliomyelitis which have occurred in his own observation and that of others in the immediate vicinity. He told of the onset, duration and course of the epidemic and the cases he had observed. Apparently the disease was not limited to any one part of any infected town. It appeared suddenly and was quite severe, but the prognosis in most cases was good in as much as there were only a few deaths. The disease seemed to involve both lower limbs and presented meningeal symptoms and considerable pain. Among other interesting facts, Dr. Free related how some lower animals such as pigs and chickens were also affected with the disease.

A CASE OF ACUTE ANTERIOR POLIOMYELITIS WITH INVOLVEMENT OF THE MUSCLES OF THE CHEST.

By C. S. Potts, M.D.

S. P. Aged 10 years. When three years old had a fall. Two days after had a convulsion followed by high fever. On the next day it was noticed that the arms and legs were paralyzed. Some improvement grad-

ually took place and at present there is marked atrophy of the shoulder girdle muscles on the right side, some atrophy of the muscles of the arm and forearm, and marked atrophy of the intrinsic hand muscles with "claw hand" deformity. On the left side there is a complete disappearance of the deltoid, more pronounced atrophy and weakening of the muscles of the arm and forearm than is present on the right side, and apparently complete paralysis of the intrinsic hand muscles with very marked "claw hand" deformity. There is also disappearance of the pectorals on both sides, and the latissimus dorsi and back muscles appear to be atrophied. The patient can walk but has atrophy and weakness of the muscles of the right leg and thigh with absent knee jerk. Electrical examination showed that the muscles of the arm and forearm of both sides responded to the faradic current that KClC was increased and modal change was absent. The shoulder girdle muscles did not respond to either current. The leg was not tested. The patient has some use of the right hand, can write, etc., but has no use of the left hand.

EPIDEMICS OF POLIOMYELITIS IN PHILADELPHIA.

By Wharton Sinkler, M.D.

The writer remarked that in the *American Journal of the Medical Sciences*, April, 1875, he had made the observation that the great majority of cases of poliomyelitis occur during the summer months. This fact was confirmed by Barlow in 1878, and subsequently by Gowers and other writers.

Dr. Sinkler has recently studied all the cases of this disease that have been treated at the Philadelphia Orthopedic Hospital and Infirmary for Nervous Diseases with reference to the season of year at which the onset of the attack occurred, and finds that of 509 cases, 294 occurred in June, July and August. In September and October there were 122 more. Therefore, about 82 per cent. of the total number of cases occurred during the warm months.

During the past summer an unusual number of cases of poliomyelitis have occurred in Philadelphia; but not enough, in the opinion of the writer, to constitute an epidemic—probably about 30 per cent. more than usual. There has been a very distinct epidemic of the disease in Ridgeway, Dubois and adjacent towns in Pennsylvania, which will be reported by Dr. Free. Gibney has seen at the Hospital for the Ruptured and Crippled, New York, from October 1, 1906, to October 1, 1907, 550 cases. Of these 387 were observed during the hot months. He does not say whether the onset of the disease was during these months, or whether the patients applied for treatment at that time.

There have been a number of epidemics of poliomyelitis recorded. The first occurred in 1841, and is spoken of as an epidemic of paralysis in teething children. All the epidemics in which Dr. Sinkler could find the season of the year noted occurred in the summer months. It is evident that poliomyelitis is due to an infection, and that this infection is developed in some way by hot weather. The fact that in a great number of cases of this disease there are intestinal troubles at the onset would suggest that the intestinal tract may be the avenue for the micro-organisms causing it.

EXAGGERATION OF THE PATELLAR TENDON REFLEXES IN ACUTE ANTERIOR POLIOMYELITIS.

By William G. Spiller, M.D.

It is taught that tendon reflexes in the affected limbs are lost in acute anterior poliomyelitis. This is usually the case, but occasionally exaggeration of these reflexes occurs. When the patellar tendon reflex is exaggerated, the wasting is chiefly in the leg below the knee, or in the leg of the opposite side. A case of poliomyelitis with exaggeration of the tendon reflexes was presented before this society by F. Savary Pearce some years ago. Another has recently been exhibited by C. E. Atwood before the New York Neurological Society (JOURNAL OF NERVOUS AND MENTAL DISEASE, 1907, p. 600). Two cases are recorded by Minor (*Deutsche Zeitschrift für Nervenheilkunde*, vol. 30, p. 398). All of these were clinical cases, and no explanation is afforded by them for the exaggeration of the reflexes.

In *Brain*, 1903, Dr. Spiller published the report of a case of acute anterior poliomyelitis with necropsy, following variola. The degeneration of the lumbar region was typical of this disease, and was confined to the anterior horns. In preparations made by the Weigert hematoxylin method the anterior horns contrasted strongly by their pale coloration with the white columns. A photograph of one of the sections from the lumbar region appears in the article mentioned. Implication of the lateral columns occurred in a limited area in the thoracic region. This case offers an explanation for the exaggeration of reflexes seen in certain cases of acute poliomyelitis. If the degeneration of the lower part of the cord does not implicate the cells innervating the thigh muscles, but is confined to those supplying the muscles of the leg below the knee, as is so often the case; and if the lateral column of one or both sides is implicated in the thoracic region, exaggeration of the patellar tendon reflex may occur. Poliomyelitis is not a process absolutely confined to the gray matter of the cord.

The following two cases are examples of acute anterior poliomyelitis with exaggeration of the patellar tendon reflex.

Case 1. R. G. J. Aged 29 years, male, white, was examined February 27, 1904. He had anterior poliomyelitis when about fourteen months old, and since that time the right lower limb, especially below the knee, was not developed properly. The right thigh is not much smaller than the left. Sensation in the right lower limb has not been impaired.

The right lower limb is much smaller than the left lower limb in all parts below the knee, where there is scarcely any muscular development. The right foot is in the position of marked talipes equinus, but the deformity can easily be overcome by passive movement. The man can flex and move the right toes inward, but has no power of extension of the toes. All movement of the right ankle is lost. The Achilles jerk is lost on the right side and is prompter than normal on the left side, but the patellar reflex is much exaggerated on the right side, and is also somewhat exaggerated on the left side.

The upper limbs appear to be normal.

Case 2. B. Colored, aged 14, male, came to the dispensary of the University Hospital, February 23, 1907. Three years previously he had some disease with fever and became paralyzed in the right upper and lower limbs simultaneously. He recovered the use of the right upper limb, so

that the grasp of the right hand is as good as that of the left, and the biceps tendon reflex is about the same on both sides, but not very prompt. The whole right upper limb is distinctly smaller than the left. The atrophy in the right lower limb below the knee is pronounced. The right foot is everted and contractured so that the patient walks on the inner side of the foot. He has steppage gait on the right side from foot-drop. The patellar reflex is exaggerated on the right side and about normal on the left side. Sensations of touch and pain are normal in the right lower limb. The electrical examination was made by Dr. C. S. Potts. In the right leg feeble faradic response is obtained in the soleus, gastrocnemius, flexor longus pollicis, flexor longus digitorum, and extensor longus digitorum muscles. The faradic response is lost in the tibialis anticus, extensor proprius pollicus and peronei. Galvanic reaction could not carefully be determined because of the patient's resistance, but there was evidently diminished galvanic contractility in the muscles of the distribution of the peroneal and internal popliteal nerves. The other muscles responded normally.

Dr. J. P. Crozier Griffith first of all had a few words to say about the etiology of poliomyelitis. The epidemic under consideration, like other epidemics, teaches us that the disease must be an infectious one. Nothing so much convinces us as personal experience, and the observation a number of years ago of the simultaneous development of the disease in two sisters led him at that time to accept readily the disease as infectious. He thought the whole symptom-complex indicated that it was an infectious disorder. He would mention in passing that he had the opportunity of seeing a few years ago a little child in the sixth day of the disease, who at the same time was suffering from pneumonia. Of course that is no proof of anything; it is merely suggestive. We know that all infectious diseases have a certain tendency to be accompanied by pneumonia, and that one may sometimes find in the lungs the specific germ of the primary disease, associated or not with the pneumococcus.

The matter of the method of infection in poliomyelitis is not at all understood. Dr. Griffith said he noticed that Harbitz and Scheel agree with the gentlemen who have already spoken that entrance is very likely by way of the intestinal tract. Though we have a germ reported by von Geirsvold this has not been corroborated, and the discoverer himself is not very certain that it was after all the cause of the affection. The matter is still *sub judice*.

Next, about the matter of mortality. Until the epidemics from Norway and Sweden were reported he had not realized that the death rate was as great as it is, varying from 10 per cent. to much more than that, one Norwegian observer placing it at 19 per cent. in the cases reported by him. We have been prone to view it as not immediately dangerous.

Last of all, about the clinical manifestations and the lesions. He has had occasion to observe one very interesting instance of involvement of the abdominal muscles, but the case which interested him most was one with involvement of the muscles of the face, originally combined with that of the muscles of the extremities, although recovery had been apparently complete in these latter. The case represented a combination of poliomyelitis with encephalitis. When Strümpell, many years ago, brought into prominence the occurrence of polioencephalitis considerable skepticism obtained regarding it. Now we no longer dispute its existence and acknowledge that it can be associated with poliomyelitis.

It is a mistake to view poliomyelitis as merely an atrophy of the anterior horns; it is more than that. Like other infectious diseases it spreads by way of the blood vessels. It is not simply in the horns alone, it is widespread, at first involving many parts of the cord and the large brain as well, and then, as a rule, disappearing from every place but the one region, but in some instances persisting in the brain also. This is another proof that we are dealing primarily with a blood infection.

Dr. McCombs gave statistics from the Children's Hospital of Philadelphia. His investigations embraced four and a quarter years, from July, 1903, to November, 1907. In 1903 (six months only) there were 6 cases; in 1904, 7 cases; in 1905, 6 cases; in 1906, 4 cases; in 1907, 26 cases. From July 23 to October 24, 1907, there were 24 cases. In addition there were during the last period 6 cases which applied for admission but were not admitted into this hospital. They were not, therefore, included in the following tables. Cases of less than one month's duration on admission: July, 1903, to July, 1907 (four years), 12 cases; July, 1907, to October, 1907 (three months), 18 cases.

Total cases treated: July, 1903, to July, 1907 (four years), 25 cases; July, 1907, to October, 1907 (three months), 24 cases.

The cases were carefully selected, all those that were doubtful being excluded. On comparing eighteen cases of less than a month's duration on admission with twenty-five cases in which the histories were complete enough to make valuable records, it was found that the cases occurring during the epidemic did not deviate from those occurring before. The discovery of paralysis as the first symptom was slightly in excess of an onset with initial fever and vomiting, which were most prominent in the twenty-five cases. The paralysis appeared most often during the first twenty-four hours, as in the twenty-five cases; and likewise involvement of a single extremity was the most frequent form of paralysis, closely followed by failure of both legs. Pain was noted in about the same proportion (one fourth) of the cases. Meningeal symptoms were lacking, and improvement seemed about the same as in the previous twenty-five cases.

Dr. Fox stated that the Department of Health received word in the early fall that poliomyelitis was prevalent in Butler, Venango and Elk counties. The Commissioner of Health asked him to go there to make bacteriological examination of the disease, to find some cause, bacterial or parasitic, for this disease, and to make some effort to trace the course of the infection. The facts of hygiene and the epidemology Dr. Free had given better than he could give at the present time, and the work on cerebro-spinal fluids, nose and throat cultures from animals, particularly chickens, and a few bed bugs, are not as yet completed. Dr. Fox said he feared they would be in vain. Everything seems to point in that direction. The work is being carried on at the laboratory of the Department of Health, and it is hoped that a publication will be made shortly, but not too shortly to be able to say something definitely. At present there is nothing in the research from the etiological standpoint, from the hygienic standpoint, from the bacteriological standpoint which would point to the cause, transmission and course of the disease.

Dr. W. B. Cadwalader said he would like to speak of a case he had the opportunity of examining which was under the care of Dr. Eshner at the Orthopedic Hospital during August of this past summer. The history which he could only speak of from memory at the present time was that the child originally developed pain in the leg, and on the second or third

day following he had paraplegia, and later on the fourth or fifth day when he was admitted to the hospital he developed some weakness of both arms, some stiffness of the neck muscles, was very drowsy, had rapid respiration, and finally the sixth day he died suddenly with what appeared to be acute bulbar symptoms. At the time there was some question whether it was a case of epidemic cerebro-spinal meningitis. The necropsy was performed by Dr. Hill, the resident, and the abdominal and thoracic organs were found to be negative, except for some congestion of the lungs; the cord and brain were preserved and Dr. Cadwalader had examined it. So far he has examined only the cord. The gross appearance of the cord was not very definite. There were a few areas of ecchymoses about some of the vessels of the anterior surface of the cord which seemed to extend practically the whole length to the pons, and on cross section there was in the lumbar and sacral cord, particularly, a very definite hemorrhagic area in the gray matter on both sides; the left side, perhaps, more than the right, and this extended in varying degrees all the way to the pons through the cord and medulla oblongata, but was more marked in both the cervical and lumbar regions. The dura was possibly a little discolored about the cervical region. Dr. Hill told him that when the cord was in the fresh state there was a definite hemorrhage in the dura at that point, but after a time when the tissue was in formalin it was difficult to make it out. In the stained sections of the cord, the central canal appeared normal, excepting in one or two sections, the cells lining the canal in some places appearing to be torn off. The dura was normal throughout the length of the cord microscopically, but the pia appeared to be separated and was infiltrated by round cells with a deep staining nucleus, some of them definitely polymorphonuclear leucocytes. The gray matter was very markedly affected in the cervical, thoracic and lumbar swellings, and there was an intense round cell infiltration about the vessels. This was most marked perhaps in the posterior part of the anterior horns about the columns of Clarke. The vessels were dilated and the whole picture was intense in the lumbar and cervical regions. The white matter was affected but not to such great extent as the gray. In a number of sections in the dorsal region the septa running through the white matter of the cord suggest the possibility of their carrying the round cells from the pia into the substance of the cord. That he did not feel at all certain about, but it possibly may be so. Dr. Cadwalader said he had not studied the sections carefully enough to be able to give information on this theory.

Dr. J. W. McConnell said in regard to polio-encephalitis and poliomyelitis, it was his fortune or misfortune to fall in with this epidemic in Corsica in this state. He was called to see a child who died a few hours before he got there, having been sick five days. It was first paralyzed in the upper extremities, secondly in the lower, and the day before death was unable to swallow and died apparently of respiration paralysis. The general history was that of acute poliomyelitis. A second case was seen in Philadelphia, similar in every respect to the advance of the disease, and as to the difficulty in swallowing, and as to death from paralysis of respiration. Another feature in the first case was that the patient was taken sick two weeks after the death of a child from the same disease in the family of the milkman supplying them with milk. It seemed significant in that particular connection. Furthermore, one of the points that struck Dr. McConnell in the study of the cases in Dubois and Corsica was the very marked, almost absolute constipation, that all these children showed, and vomiting and fever were present in all these cases.

Dr. Weisenburg thought that the participation of the lower animals such as pigs and chickens in the disease is very suggestive. In only one other disease that he recalled was the infection carried by means of the soil. This was in the disease known as beri-beri which is prevalent in tropical countries. He told of an instance in which patients who were quartered on one side of a military prison had the disease, while those on the other side were free from it. An exchange was made of these prisoners only to have the healthy prisoners infected with the disease when they were removed to the other side. In beri-beri it is supposed that the infection is carried by means of the soil.

Dr. Alfred R. Allen said his attention was called to the rarity of this affection in the colored race by Dr. S. Weir Mitchell some years ago. He himself had never seen the disease in a colored person.

Dr. Gordon said he wished to say in reference to Dr. Spiller's statement relating to exaggerated reflexes that he examined a child for Dr. H. A. Wilson in whom there was a typical onset of the acute infantile paralysis, fever, vomiting and diarrhea, paralysis of the lower extremities. The child was brought ten days after the onset of the disease. The patellar reflexes were markedly exaggerated, curiously enough the arms also presented the exaggerated reflexes, still the flaccidity of the paralysis and the onset of the disease spoke in favor of anterior poliomyelitis. Dr. Gordon thinks there are two great points which should be studied and which are extremely important: First, the question of the bacteriological basis; second, the manner of involvement of the anterior cornua. It is conceded that in a number of cases the pneumococcus and the meningococcus were found, and still in a number of other cases of the same form and same severity no microörganism was found. The bacteriology of the disease is entirely incomplete and absolutely uncertain. The experimental observations are also negative.

In regard to the transmission of the disease whatever the cause may be, whether it is a microorganism or a toxin, it is not at all certain that the blood stream is the carrier of the poison. In Landry's paralysis or rabies in which the lesions are almost analogous to the lesions of the anterior poliomyelitis, it is almost certain that the poison does not follow the blood stream. These two points: What is the real cause of the disease, and how the infection reaches the anterior cornua are the two great problems to be studied.

In regard to the epidemicity of the disease: In the Jefferson Hospital since July there were 21 cases, 7 of these 21 cases came from a single locality of the city. They did not occur about the same time, but pretty nearly about three or four weeks apart. Every year about the same time a certain rush of these cases comes to the Jefferson Hospital.

As far as negro cases are concerned: Dr. Gordon has seen two cases at the Douglass Memorial Hospital.

Dr. Free, in closing, said only one case had paralysis on the first day. He said that doctors, including himself, were wrong in giving the affection the name of poliomyelitis. The necropsies could not be obtained. A great effort to do so was made in all four of the fatal cases. In addition to the case in which lumbar puncture was performed before death, he wished to state that a lumbar puncture was done two hours after death in another case. In neither of these cases were meningococci found.

Dr. C. S. Potts said he could not add anything new in regard to the

treatment. He has seen very few cases in the acute stage. In the chronic the patients are treated by massage, electricity, exercises, and when indicated, by either the use of braces, nerve anastomosis or tendon transplantation. Nerve anastomosis may possibly be of some service in the case Dr. Potts exhibited. Treatment is usually symptomatic in the acute stage, with cold to the spine, mild counter-irritation and of course absolute rest in bed.

Dr. McCombs stated that Dr. Ludwig obtained some wonderful results from the Bier-Clapp method.

Dr. Gordon thought it significant that the child exhibited by Dr. Free which showed the most complete recovery, had been given antitoxin injections.

A CASE OF OLD POLIOMYELITIS (ACUTE) FOLLOWED BY MUSCULAR ATROPHY IN FACE AND HANDS.

By S. D. Ingham, M.D.

C. L. Age 42 years. Rug weaver. Born in Denmark.

Family History.—Both parents lived to be old. Patient knows of no similar disease among his relatives.

Does not remember ever having had any serious illness. Uses alcohol and tobacco moderately, but regularly. No venereal history. When eight or ten years old both hands were burnt by molten tin (principally on dorsal aspect). He states that the left leg has always been crippled, that "he was born that way," but examination shows that the condition was probably an acute poliomyelitis in infancy, the left leg from the knee down being shorter than the right and much smaller. The left foot is in cavus position, the only voluntary power in the leg being dorsal flexion of ankle and slight extension of toes. (Peroneal and anterior tibial muscles intact.)

Right Leg is normal as to power, development, reflexes, etc. The *face* shows muscular atrophy and weakness, as in attempts to show teeth, smile or whistle. He cannot close the eyes, attempts to do so leaving palpebral fissures nearly one fourth inch wide, in which condition they remain during sleep. Tongue and palate negative. Pupils and extra-ocular movements normal.

Hands, both right and left show atrophy of the intrinsic muscles. Thenar and hypothenar eminences are almost entirely absent. Grip is fair. No atrophy is present in forearm or arm.

Reflexes.—Biceps jerks and triceps jerks are present.

No atrophy is found in any other part of the body. No fibrillary tremors occur anywhere.

Electrical Reaction present in all muscles showing voluntary power to both faradic and galvanic currents. No R.D. is obtained.

The patient does not know when the condition in the face and hands developed. Inability to close eyelids developed about ten years ago during examination by ophthalmologist. He has noticed no progress in the condition, except slight weakness in voice (articulation) and occasional slight choking attacks, especially when swallowing liquids.

In discussing Dr. Ingham's patient, Dr. W. G. Spiller said that the case appeared to be one of muscular dystrophy developing after a long-standing anterior poliomyelitis. The patient was 32 years old when he first noticed the weakness of the orbicularis palpebrarum muscles, but there may have been some defect previously undetected. He has more

of the Landouzy-Dejerine type of muscular dystrophy. Dr. Spiller said that he reported a case of muscular dystrophy which began in the peripheral parts of the limbs. A few other cases are on record in which the atrophy commenced in that way.

Dr. J. W. McConnell said that the patient under discussion reminded him of a case at present in Blockley. The man had an acute poliomyelitis in early youth and at present time exhibits symptoms of chronic myelitis, both upper and lower extremities being paralyzed.

Dr. Alfred Gordon said he had exhibited two or three years ago, a case before the Neurological Society, a boy seventeen or eighteen years old, who had acute poliomyelitis in childhood. At the time Dr. Gordon examined the patient he presented about the same symptoms as this man, except the eyelids were not involved to the same extent as in this case. The reflexes were exaggerated. It was evidently a case of amyotrophic lateral sclerosis. Possibly this is too.

A CASE OF BULBAR PALSY WITH COMPLETE MUTISM.

By Alfred Gordon, M.D.

A woman of 40 had an attack during which the tongue protruded suddenly and was caught between the teeth. The jaws were closed so tightly that the protruded portion of the tongue bled. For 24 hours the jaws could not be separated. Gradual relaxation took place. Since then the following symptoms developed: Inability to swallow, to move the tongue, to masticate; difficulty of breathing, loss of speech and aphonia. Some improvement followed. At present there is absolute mutism, right facial palsy, atrophy of the tongue, also R.D. and fibrillary twitchings of the latter, palsy of the uvula, some difficulty of swallowing and of mastication, atrophy of the lips and R.D. of the muscles of the right side of the face; partial paralysis of the epiglottis, engorgement of the vocal cords.

After discussing the differential diagnosis, Dr. Gordon arrived at the conclusion that it was a case of acute bulbar palsy with unusual symptoms. Complete mutism cannot be explained.

Dr. Wharton Sinkler said he thought the case one of bulbar palsy. The condition of the lips, tongue, fibrillary tremor, atrophy, all point to that condition. The sudden onset is sometimes seen and is then due to hemorrhage in the bulb. Probably that was the origin in this case referred to.

Dr. Gordon asked Dr. Sinkler whether there could be complete absence of speech.

Dr. Sinkler replied that he had seen one case, a fatal case, in which there was complete absence of speech. The patient was not aphasic, because he made signs for a pencil and wrote what he wished to say, but he could not be induced to say or repeat a word.

Dr. Spiller agreed in the diagnosis of acute bulbar palsy. He referred to a case recently published, in which pseudo-bulbar palsy occurred without any implication of the limbs, but he did not regard Dr. Gordon's case as one of that type. Reactions of degeneration in the face and tongue were contrary to such a diagnosis.

HEREDITARY EXTERNAL OPHTHALMOPLEGIA.

By T. H. Weisenburg, M.D., and Wm. M. Sweet, M.D.

Congenital ophthalmoplegia is a rare condition in itself and not many cases have been recorded, but to have ophthalmoplegia in a number of generations of the same family is an exceedingly rare occurrence, and is important also in showing the influence of heredity. The case shown was that of a girl of fourteen who had bilateral external ophthalmoplegia. She was unable to move the eyes in any direction and there was considerable drooping of both upper lids. The child in the effort to see brought constantly to her aid the orbicularis muscles. It is interesting to note that the pupils were of normal size, and when the patient made an effort to look at a distance or at a near point there was a feeble attempt at movement of the eyeballs, and the pupils were dilated or contracted accordingly. There were no other symptoms of maldevelopment present.

The mother of the child had exactly the same condition. So far as the mother's history was concerned there were no facts obtainable. She had two children, the patient who was exhibited and another child who had exactly the same condition but who died some months after birth.

NEW YORK NEUROLOGICAL SOCIETY.

November 12, 1907.

The President, Dr. Charles L. Dana, in the Chair.

A CASE OF INFLAMMATION OF THE GENICULATE GANGLION.

By I. Abrahamson, M.D.

The patient was a man 67 years old, a native of Russia and a teacher by profession. His family history was neuropathic. He denied venereal diseases and used alcohol and tobacco in moderation. On October 18, 1907, he first complained of pain above, behind, and below the right ear. It was of a dull, aching character and persisted throughout the night. On the following morning, while washing, he noticed that he could not suck water into his mouth, owing to lack of mobility of the right side of the lips, and that his chewing power was impaired. He thereupon consulted his physician, who informed him that he had a facial palsy. On October 21, when he appeared at the Mt. Sinai Hospital Dispensary, there were slight swelling, redness and tenderness over the right auricle. Two days later an examination of the ear showed herpetic vesicles over the lower auricle, the external auditory meatus and the tympanum. Hearing was diminished over the right ear; there was tinnitus aurium; no vertigo, nausea nor ataxia; no epiphora; no salivary disturbance nor difficulty in swallowing; taste was slightly impaired. There was a complete typical right facial palsy, involving all three branches of the nerve. The pains behind the ear disappeared in the course of a few days, but the herpetic scabs were still present over lower half of auricle, auditory meatus, and one of them, which showed the site of a very large vesicle over the tympanum, was particularly noticeable.

At present there is right facial palsy, complete, involving all three branches of the nerve; left pharyngeal innervation was stronger than the

right, uvula to the left. Trigeminal nerve normal in every particular. Taste diminished somewhat over anterior of the tongue.

Hearing diminished during the activity of the zoster, later became augmented in the right ear. Electrical examinations in the early days showed increased faradic and galvanic irritability. Later the faradic became gradually diminished, muscle and nerve, and at present very strong currents elicit faint responses. Galvanic shows no polar change, though the contraction is slow and vermicular.

The case was especially of interest, on account of the moderate amount of pain, which in geniculate lesions is usually severe and persistent.

Dr. J. Ramsay Hunt said that the case reported by Dr. Abrahamson was quite typical, presenting the characteristic syndrome of the herpetic inflammation (posterior poliomyelitis) of the geniculate ganglion, *i. e.*, an eruption of the herpes zoster within the zoster zone for the geniculate ganglion (herpes oticus), a peripheral facial palsy, and hypoacusis. He had personally examined the case, and thought that the auditory disturbance was due to the inflammatory swelling of the tissues of the external auditory canal and tympanic membrane, and was not dependent upon a central involvement of the acoustic nerve or its terminations. Symptoms of Ménière's disease, not infrequently observed in this group of cases, were entirely absent. He emphasized the red and swollen appearance of the auricle, which was displaced laterally from the side of the head, and the narrowing of the external meatus, which no doubt, in not a few cases, gives rise to errors in diagnosis, such as perichondritis and diffuse inflammation of the auricle. The single large bleb referred to on the tympanum had been observed in other cases; within the canal and tympanum the vesicles often show a distinct tendency to coalesce.

Dr. Hunt desired to place on record a case that he had seen a few days previously which represented a different type of the same group of cases. This was a case of herpes occipito-collaris with seventh nerve palsy occurring in a man 71 years of age who developed a typical herpes zoster in the left side of the neck and occipital region. Eight days later there appeared a typical facial paralysis on the same side which had been preceded by tinnitus the day before. Taste perception was diminished on the anterior two thirds of the tongue on the affected side, and there was a slight but distinct diminution of hearing of neural origin on the same side. The facial palsy involved all branches, but very slight movement was still present in the upper and middle branches. There was no evidence of any eruption within the zoster zone for the geniculate ganglion. The speaker explains a case of this type on the basis of an inflammatory reaction in the geniculate ganglion.

MYOTONIA ATROPHICA.

By D. J. Ramsay Hunt, M.D.

Myotonia atrophica is an extremely rare affection; and so far as Dr. Hunt was aware these are the first cases of this affection recorded in this country. In a recent study of this subject by Pelz, the percentage of myotonia cases showing atrophy is given as 11 per cent. In the following two cases, myotonia and muscular atrophy are present in two brothers. Another brother, now dead, is said to have presented somewhat similar symptoms:

Case 1. The first patient was a man, 44 years old, a native of Ger-

many. His occupation was that of cleaner in a barroom. He stated that he first noticed a weakness in his hands about ten years ago, and six years later in the lower extremities. Three years ago the subjective symptoms of myotonia first manifested themselves, *i. e.*, while the first movements of the arms and legs were somewhat stiff, this would diminish and finally disappear after repeating the movements several times. The weakness and associated atrophy had gradually grown worse. He had had no pains nor paresthesia.

At examination the patient was well built and of good muscular development. His face was dull and lacked expression. His speech was slow and somewhat labored and monotonous. He walked with a steppage gait due to bilateral foot-drop, and on lifting the arms a wrist-drop became apparent. The muscles of the trunk, the shoulder and hip girdles and the thighs were unusually well developed. In the upper arms, the forearms and the calf muscles a well marked tapering atrophy was present, which was especially marked in the triceps, the ulnar side of the forearm and the extensor surfaces of the calves. The muscles supplied by the cranial nerves showed no defect, save the dulness of expression and the articulation disturbance. There were no pupillary changes. The tendon reflexes of the arms and lower extremities were abolished. The skin reflexes were present. The myotonia on voluntary movements was only distinctly demonstrable in the hands. On making a fist, the hands and fingers slowly relaxed in the typical manner, the stiffness growing much less after a series of the same movements. While the subjective disturbances were present to a slight degree in the lower extremities, they were not objectively demonstrable. No myotonic disturbance on voluntary movement was present in the eyelids, the jaws, neck, trunk, upper arms or lower extremities. The mechanical irritability of the muscles was increased and showed typical myotonic characteristics (persistence of the contraction) in the muscles of the face, tongue, arms, shoulders, thighs and calf muscles. It was exquisitely present in the intrinsic muscles of the hands. Gross motor power was well preserved in all the muscles of the body excepting those of the arms and legs. Flexion and extension of the elbow were very much diminished in force. There was almost complete paralysis of the extensors of the wrist and fingers; the flexors were also very weak, as were the intrinsic muscles of the hands. Flexion and extension of the knee were well preserved on both sides. There was, however, almost complete paralysis of the peroneal and extensor muscles of the foot. Plantar flexion of the ankle and toes, while present, was considerably diminished. There were no objective disturbances of sensation. The extensors of the wrist and fingers and the peroneal and extensor group of the anterior tibial region failed to respond to strong faradic or galvanic currents. The posterior muscles of the calves, the intrinsic muscles of the hands, the flexors of the wrist and fingers and the deltoids showed the myotonic reactions.

Case 2. This patient was a brother to the first case shown by Dr. Hunt. He was a man, 41 years old, a baker, whose symptoms dated back eighteen years and began with a weakness in the hands and arms. He found that he could not lift heavy objects, that his hands would not close tightly, and he stated that a boy could open his fist. He was positive that the weakness preceded the stiffness by many years, the myotonic rigidity first manifesting itself ten years ago. The trouble in the lower extremities dated back about ten years, and consisted of weakness and stiffness. Before that the patient could run and jump and was

a good dancer. At that time he found that upon arising from a sitting posture he had to wait a brief period before a certain stiffness wore off. He had also had some difficulty with speech; his tongue seemed heavy and he had difficulty in getting started in talking, but after talking a while, this slowness would wear off. A few years ago this disturbance of articulation was worse than it was now. He ate rather slowly and chewed his food for a long time; otherwise he had no difficulty in mastication or deglutition.

Status: The patient was well built and of strong muscular development. He bore a marked resemblance to his brother (Case 1), not only in physique, but also in face and coloring. His gait was slow and shuffling, with distinct steppage character (foot-drop). The facial expression was dull and heavy; articulation was slow, monotonous and indistinct. The muscles of the trunk, neck, shoulder girdle, hip girdle and thighs were unusually well developed and showed no signs of weakness or of atrophy. The upper arms, forearms and lower legs were distinctly tapering and atrophic, especially the lower portion of the triceps, the ulnar side of the forearm and the anterior muscle group of the lower leg. Flexion and extension of the elbow were weak, the supinator longus muscle was practically absent on both sides. Extension of the wrist and fingers was so very much diminished that it could be easily overcome by the pressure of a single finger. The flexor movements of the hand and fingers were also very feeble. In the lower extremities the dorsal flexion of the feet and toes was practically abolished. Plantar flexion, while present, was very much reduced. The pupils and cranial nerves were negative; the arm jerks and the Achilles jerks were absent; the knee jerks were present but very much diminished, reinforcement being necessary. All the skin reflexes were present (plantar flexion of the toes). The mechanical irritability of the muscles was increased and showed the characteristic response in the intrinsic muscles of the hands, the forearms and upper arms, deltoids, calf muscles, feet, face and tongue. The only demonstrable myotonic disturbance on voluntary movement was in the hands; here the typical rigidity persisted after a contraction, and gradually relaxed as the movement was repeated. Subjectively, the symptom of myotonia was also present in the face muscles of articulation, and in the lower extremities. The myotonic electrical reactions were demonstrable in the muscles of the lower face, the tongue, the deltoid, the upper arm and hands, and in the posterior muscles of the calves. The muscles of the forearm, especially the extensor group, showed a very markedly diminished response to both currents. The electrical reactions were practically absent in the anterior tibial and peroneal group of the lower extremities.

Dr. B. Sachs said the first case shown by Dr. Hunt bore a close resemblance to Thomsen's disease, but on account of the associated atrophies and the appearance of the arms it reminded him of the Charcot-Marie type or leg type of progressive muscular atrophy. The condition of the forearms, and of the legs, suggested that type.

Dr. L. Pierce Clark said that in a case reported by Kaiser, in 1897, there were three conditions present, namely, hypertrophy, atrophy and myotonia; the conditions apparently followed a cerebral palsy. The hypertrophy occurred in those muscles that were paralyzed.

Dr. Dana said he had examined these patients at his clinic, and the idea had occurred to him that they might more properly be called cases

of muscular dystrophy with myotonia. The dystrophy came on before the myotonia. They were perhaps examples of one of the forms of progressive dystrophy of the Charcot-Marie type upon which the myotonia had been implanted.

Dr. B. Onuf also agreed with Dr. Dana that the cases probably represented a myotonia implanted on a primary muscular dystrophy. The flaccidity of the muscles in one of the cases shown was extremely marked. In the other case the facial expression was so typically mask-like that it presented a very striking feature, and rather spoke against its belonging to the Charcot-Marie type.

Dr. Dana, in reply to Dr. Onuf, said that Gowers had reported one case of progressive dystrophy of the Charcot-Marie type in which the mask-like expression of the face was present.

Dr. Hunt, in closing, said that those who had written most extensively on this subject had tried to determine if this represented a distinct clinical type of atrophy, and whether it was a spinal or myopathic type, and the consensus of opinion was that it could not be brought under one of the well-known groups of atrophies. The atrophies in the cases he had shown seemed to be confined chiefly to the arms and forearms, with preservation of the muscles of the hands.

RESEARCHES ON THE EPILEPTIC VOICE.

By L. Pierce Clark, M.D., and E. W. Scripture, M.D.

Dr. Clark stated that for several years he had held that there was a characteristic voice in epilepsy which, if accurately analyzed, would possibly be an aid in the differential diagnosis, and with the help of Dr. Scripture's methods of voice study, which had been explained and illustrated at the April, 1907, meeting of the Society, he was now able to give a demonstration and analysis of the epileptic voice type. It was unnecessary to say that there was no past literature to refer to in such studies, as both the problem and results were entirely novel. After explaining and illustrating in detail the method of obtaining these records, Dr. Clark showed that in the voice curves from epileptics there was a peculiarity which struck the eye at once. Instead of the continual rise and fall in melody of the normal voice, vowels and phrases ran along on even tones. The speech of an epileptic was, in fact, "plateau speech": his melody proceeded by even tones. There was also a tendency to monotony, and the successive sounds did not vary much from each other as normal ones did. This defect of speech of the epileptic could be easily removed by training, but relapses occurred quickly. The degree of permanency of the removal was still to be proven. The speaker said he had seen the defect remain for several years after the epileptic attacks had ceased. He had no adequate explanation to offer for this voice defect in the epileptic. There was no parallel; no similar defect in any patient suffering from any other motor disorder of the nervous system, insanity or imbecility. Two of the record cases he had shown were entirely free from the bromides. They had universally found this epileptic type of voice in all well-established cases of epilepsy of whatever type. As a tentative explanation, they offered the theory that the voice was due to the chronic exhaustion of the nerve centers peculiar to epilepsy. The diagnostic value of the voice sign was obvious.

Dr. Charles E. Atwood said he had had the opportunity of following

a good deal of the work of Drs. Clark and Scripture in connection with the epileptic voice, and he could corroborate what had been said regarding its peculiarities. The character of the voice in epilepsy was so uniform that the diagnosis of the disease could frequently be made on that basis alone.

Dr. B. Sachs asked whether these voice peculiarities described by Dr. Clark were observed in recent cases of epilepsy or only in the chronic or very old cases. Also, whether the voice was confined to those cases that had been more or less thoroughly bromilized. The monotony of the epileptic voice must surely in the end be due to insufficient vibration of the vocal cords, or perhaps a slowness of vibration, and that we would expect to find in patients who were under the influence of the bromides.

Dr. E. W. Scripture said that while it was rather early to speak of the voice changes in paralysis agitans, they were distinctly different from those observed in epilepsy. In both affections the speech was monotonous in character, but otherwise they were different. The spaker thought it was quite possible to walk through the wards of the institution on Ward's Island and distinguish the various cases of epilepsy by the voice alone. The voice of imbeciles was also quite distinctive.

Dr. Edward D. Fisher said that in the three cases of epilepsy shown by Dr. Clark, the patients were of a low type of mentality. The speaker said that in epileptics of a higher type, those who were able to be about and conduct their business affairs in a satisfactory manner, he had never noticed any changes in the voice, and he was inclined to believe that such changes were an evidence of mental deterioration.

THE GALVANOMETER IN PSYCHOLOGY.

By Frederick Peterson, M.D.

The author said the discovery by Féré in 1888 and Tarchanoff in 1890, and the rediscovery by an electrical engineer in Zurich very recently of the influence of emotions on the galvanometer was at present arousing some attention here and abroad among those interested in normal and morbid psychology. The bibliography of the subject and the results of the investigations last winter by Jung and Peterson were published in the July (1907) number of *Brain*. The speaker showed an instrument which thus far had given the most satisfaction in these researches, and he hoped to demonstrate its utility as a measurer of the emotions so far as that was possible with a test person in the same room with a large number of people. Anticipation, apprehension and embarrassment might interfere with the experiment. The instrument was the mirror galvanometer of Duprez-d'Arsonval, made by Carpentier of Paris. (Model A, suspension thread 0.08 mm., duration of simple oscillation 1.69 second; resistance 6.2 ohms.) By means of an arc light the reflection from the mirror was thrown on a screen 12 to 14 feet away, so that every one in the room might note the deviations of the mirror. This was only for purposes of demonstration. For scientific use the light reflected upon a millimeter scale about a meter from the galvanometer, and a sliding index connected with a pen made a permanent record on a kymograph. An effort was being made to secure an automatic recorder such as was used in the cable offices, and if that could be perfected it would prove a most valuable adjunct to the apparatus. The test person was put in the circuit with the galvanometer and one dry cell, and a shunt was also employed to

regulate the amount of current. The electrodes were nickel-plated copper upon which the hands or feet might rest. The speaker said he had used one electrode of carbon and another of zinc, in which case a cell in the circuit was unnecessary. The fluctuations observed in the light were due to varying resistance in the skin. Moving the fingers or lifting the hand caused the light to recede because of increased resistance. Very strong pressure might diminish the resistance and cause the light to advance. Such muscular movements were readily detected after some experience with the instrument, and did not complicate or interfere with the movements due to the emotions. He had tried water contact, which excluded changes due to varying pressure. If the hands were blanched with an Esmarch bandage, the deflections were not affected. If they were completely dried by immersion in formalin, the resistance was so great that fluctuations did not occur. A deep inspiration or a deep expiration or coughing would cause a deflection, whereas ordinary respiratory movements did not affect the light. All of these influences were readily distinguishable by practice. If a test-person was placed in the circuit and every possible stimulus avoided, we obtained a rest curve, which was characterized by first a considerable deflection and then a slow recession of the curve to almost nothing, which was quite contrary to our ordinary experience with cutaneous resistance. When any stimulus was applied, sensory or psychic, we had after a latent period of from two to five seconds a rise in the curve, and then a gradual fall to the previous level. It was noted in Dr. Peterson's experiments that expectation or anticipation, that embarrassment and apprehension caused fluctuations. A sudden sensory stimulus aroused attention, so that all of these stimuli had a psychic element. As to the actual practical value of this instrument in psychology, the speaker said we had at least in it a new instrument of precision which was more accurate and certain than any other employed in psycho-physical investigation, such as the pneumograph, the plethysmograph, ergograph, etc. It promised to have a field of usefulness in all conditions in which study of the emotions or emotional complexes was of importance, as for instance in neurasthenia, hysteria, certain mental disorders, suspected criminality and malingering. As yet it was too new and untried, however, to permit of claiming so much as established. The psychometer might ultimately do more than we expected, or it might do less than we at present surmised. The explanation of its action was not yet clear. So far as we knew now, the deviations of the galvanometer were due to variations in resistance of the skin through an intimate connection of the sweat-secretory system with the nervous system, perhaps through the medium of the sympathetic nervous system. Yet it was also quite possible that some extra electro-motive force either from the tissues or from the cutaneous surface was added to the electromotive force of the single cell employed in demonstration.

PRELIMINARY REPORT ON THE WORK OF THE POLIOMYELITIS COMMITTEE.

By B. Sachs, M.D.

Dr. Sachs premised his remarks by stating that the committee was working in conjunction and entire harmony with a similar committee appointed by the Pediatric Section of the New York Academy of Medicine and a representative of the New York Health Department. The work

of the committee thus far had been restricted very largely to determining what methods should be pursued in order to elicit general interest in the work, and to gather as complete statistics as possible from New York and vicinity. Although numerous communications, asking for information, etc., had been received from distant parts of the country, especially from Boston, the committee had deemed it wise to restrict the field of their investigation to New York and its immediate vicinity. In order to get an approximate idea of how many cases of poliomyelitis had occurred in New York during the past summer, 4,000 return postal cards were to be sent out to physicians. For the purpose of gathering accurate histories of these cases, an elaborate history blank had been compiled through the combined efforts of members of the committee, and upon the receipt of the return postal cards from those physicians who had treated cases during the recent epidemic, copies of these blanks would be forwarded to them, and the information thus obtained would then be analyzed by the members of the joint committee. In their work, Dr. Sachs said, the members of the committee had received the cordial coöperation of the New York Health Department. The speaker invoked the aid of the New York Neurological Society, from whose members they naturally expected to receive the most complete histories of cases. The chairman also stated that Dr. Simon Flexner, of the Rockefeller Institute, would be pleased to examine any suitable specimens from cases of poliomyelitis.

Periscope

Deutsche Zeitschrift für Nervenheilkunde

(Band 30. Heft 1–2.)

1. Lesions of the Conus Terminalis and of the Cauda Equina. BALINT and BENEDICT.
2. Syphilitic Disease of the Basilar Arteries of the Brain. FALINYI.
3. Contribution to the Knowledge of the Intraventricular Brain Tumors. HUNZIKER.
4. Contribution to the Question of the Combination of Organic Nervous Diseases with Hysteria. HÜTTENBACH.
5. Pronounced Contractures and Atrophies of the Skeleton in Progressive Muscular Dystrophy. SCHLIPPE.

1. *Lesions of Conus Terminalis and Cauda Equina.*—Balint and Benedict report six cases of disease of the lower portion of the spinal cord. The first, a woman of 33, had fallen at the age of 14 years, striking the lower portion of the back. There was severe pain in the sacral region and she was obliged to remain in bed for a year with paralysis of both feet, and inability to pass urine. She had complete anesthesia of the rectum and vagina and bladder, and there were also some other evidences of disease of the lower part of the spinal cord. At the autopsy the fourth and fifth sacral segments were completely converted into neuroglia tissue, and contained neither cells nor fibers. The second and third sacral segments contained a few fibers, and above the fourth lumbar segment the cord was practically normal. The second case, a woman of 40, at the age of 19 developed severe pain in the sacral region. There was anesthesia of the skin of the perineal and gluteal regions, and of the dorsal surface of both thighs. There was also difficulty in urination. The third case, a man of 23, had a severe fall at the age of 5 years. At the age of 12 he had pain in both legs when he attempted to lift a weight. He gradually developed talipes varus in the left foot, which was not permanently cured by tenotomy, and later developed an ulcer. Upon examination there was found to be a defect resembling spina bifida in the sacrum. There was anesthesia in the perineal and gluteal regions. The fourth case, a man of 57, at 54 years developed pain in the sacrum. There was difficulty in urination, and the characteristic disturbance of sensation. Examination also revealed an aneurism, probably of the hypogastric artery, which had eroded the sacrum, and pressed against the lower spinal cord. The fifth case, a man of 27 was exposed to severe chilling of the lower part of the back. He then developed severe pains in the lumbar region, and difficulty in urination. He had the characteristic areas of anesthesia, and in addition anesthesia of the outer side of both feet. The sixth case, a man of 35, had a severe fall one year previously. Three weeks later he had pain in the sacral and gluteal regions. There was difficulty in micturition. There was anesthesia in the gluteal and perineal regions, over the lower portion of the left chest, and both legs. A diagnosis of probable central fibro-

matosis was made. In discussing the symptoms of these cases Balint and Benedict call attention to the fact that in all of them micturition was automatic rather than controlled in any way voluntarily. They show—especially the first case—that the automatic centers of the bladder are not in the sacral regions of the cord, but probably in the sympathetic cells. In regard to defecation they could state that the external sphincter was preserved. There was anesthesia of the rectal mucous membrane, and absence of the anal reflex. Nevertheless, the contraction of the rectum occurred, and therefore the center for this act must also lie external to the sacral cord. In regard to the sexual functions, the data are, to a certain extent, untrustworthy, but it appears that the center must be located in the ganglion cells of the hypogastric plexus, but there is also evidence which tends to show that the center for ejaculation is localized in the conus. The first patient, however, after an injury which destroyed the lower portion of the spinal sacral cord, had a perfectly normal child, the only remarkable feature being that the labor was entirely painless. It follows from this that the centers for uterine contraction cannot possibly be located in the lower sacral segment.

2. *Syphilitic Disease of Basilar Arteries of Brain.*—Falinyi reports three cases of syphilitic disease of the arteries. The first, a man of 22, had been infected two years previously. He had a series of apoplectic attacks, following the last of which he remained unconscious, with paralysis of all the extremities, and died. At the autopsy the chief changes were, thickening of the arteries at the base of the brain, particularly those going to the cerebral portion, some of them being completely obliterated, and softening of the external and lower wall of the left lateral ventricle, including the corpus striatum of the optic thalamus. The second case, a man of 32, had been infected in his youth. He was married, and became the father of five living children. He developed intense headache, diplopia, and impairment of vision. Gradually he emaciated, was persistently somnolent, and died with a diagnosis of cerebral tumor. At the autopsy there was found marked chronic adhesive lepto meningitis of syphilitic nature, syphilitic endarteritis of the right vertebral artery, and softening of the right temporo-sphenoidal lobe. There was also some hemorrhagic meningitis. The third case was a woman of 21, from whom no history could be obtained. There was evidence of an old syphilitic disease, and chronic gonorrhea. In mentality the patient was confused and demented. There were tonic contractions of various muscles, and finally the patient died. The Sylvian arteries on both sides were thickened and obstructed; there was softening of both corpora striati; and syphilitic hyperostosis of the skull. After a careful discussion of these three cases, Falinyi reaches the following conclusion. Syphilis can involve all three layers of the arterial wall. In the intima it produces principally extensive proliferation, which may lead to obliteration. In the least involved portion of the media it causes degeneration with circumscribed miliary foci, with the presence of giant cells. In the later stages of the process the tissue in all three of the layers is replaced by granulated tissue, only the fenestrated membrane remaining intact. The changes in the brain substance appear to be directly secondary to the vascular lesions.

3. *Intraventricular Brain Tumor.*—A girl of 11 years fell from a ladder, striking the left parietal region, and remaining unconscious for ten minutes. At the age of 24, after her first child was born, she had bilateral mastitis, and after this there was almost persistent headache. A year later she had an attack of influenza, after which the headache was

more severe, and often localized to the left parietal region. She frequently had attacks of vomiting without any gastric disturbance, and occasionally, involuntary micturition and defecation. She began to emaciate very rapidly, had attacks of fainting, and finally developed paresis of the facial nerve, the right hand, and right leg, with diminution of sensation in the whole right side of the body. There was also ataxia of motion in the right hand; tenderness to percussion over the left parietal bone, and choked discs. Von Monakow made a diagnosis of tumor of the centrum ovale of the left parietal lobe. Her condition grew steadily worse, and she finally became comatose and died. At the autopsy the left hemisphere was enlarged, and a tumor was found in the left lateral ventricle, adherent to the corpus callosum, and the septum lucidum, but hanging freely in the cavity of the ventricle. The greatest diameters were, transversely, 6.5 cm., and vertically, 5.5 cm. The tumor consisted of small round cells, and was very rich in blood vessels. The glia staining in the tumor substance was negative. Hunziker gives a summary of the cases of intra-ventricular tumors hitherto described. He assumes that his own case is probably gliomatous in nature, although the absence of neuroglia fibers makes it impossible to exclude an outgrowth from the connective tissue of the blood vessels. He notes that many of the tumors reported in the literature showed various intermediary stages between glioma and sarcoma.

4. *Organic Nervous Diseases Combined with Hysteria.*—Hüttenbach reports three cases of organic nervous disease, associated with hysteria, with the following diagnoses. The first case had organic paralysis of the right axillary nerve, association with traumatic hysteria, both conditions apparently following the same injury. The second case, a man of 33, had sciatica on the right side, in addition to hysteria, probably developed on a neuropathic basis. The third case, a man of 51, had a severe injury followed by hematemesis. There was also a fine tremor of the left side of the body, immediately after the fall. This disappeared, but a month later recurred when the patient yawned violently. The arm then became paretic; there was weakness of the left leg; the speech became monotonous, and festination occurred. The right side was not involved. The patient had also propulsion and retropulsion; both hands were in the position of writing, and the expression was mask-like. There was also muscular rigidity, and unilateral disturbance of sensation, involving the mucous membranes.

5. *Progressive Muscular Dystrophy.*—Schlippe reports two cases of progressive muscular dystrophy, in brothers, who, in addition to the very considerable atrophy of the muscles, showed marked atrophic changes in the skeleton, particularly in the shoulder-girdle and arms. In the first case there were marked contractures of the feet.

J. Sailer (Philadelphia).

Allgemeine Zeitschrift für Psychiatrie

(Vol. LXIV. Heft 1. 1907.)

1. Studies of Heredity in Mental Disease. Tigges.
2. Classification of Psychoses, particularly the Periodic Forms. F. Geist.
3. Contribution to Knowledge of Pseudologia Phantastica. Anna Stemmermann.
4. Depressive Psychical States due to Political Events. J. S. Hermann
5. Discharge of Insane Criminals from Asylums. Von Kunowsky.

1. *Heredity in Mental Disease.*—A statistical study, based upon the reports of a number of German and Swiss asylums, which does not readily lend itself to abstraction.

2. *Classification of Periodic Psychoses.*—The difficulties which still prevent a scientific classification of mental diseases, upon either a clinical or a pathological basis are considered, but the author can suggest no way of surmounting them at present. The one thing which forces itself upon us is the polymorphism of psychoses in point of view of symptoms. This stands in the way of a purely symptomatic classification, while in the domain of pathology we are still too much in the dark to use this as a basis. The author can suggest no better course than to utilize all the information which we can gather as to the mode of development, course and outcome of psychoses as criteria, a course which has long been urged by the Kraepelin school. He gives some histories which illustrate the difficulties of classifying certain psychoses showing a periodical course and suggests that we might do well to recognize a paranoid and a katatonic form of periodical insanity. Since so many cases of acute insanity show a complex picture in which symptoms of mania, of melancholia and of amentia are combined along with various hallucinations and illusions, he suggests that it might be well to group all these acute and presumably curable cases under the head of "acute simple psychoses." To regard a conception of secondary dementia as entirely out of date is he thinks to go rather too far, since there are undoubtedly cases which, while showing many symptoms in common with the acute simple psychoses, nevertheless run a chronic course for years without proceeding to dementia. For these he would suggest the name "chronic simple psychoses."

3. *Pseudologia phantastica.*—The histories of five cases which the authoress thinks typical examples of the pathological liar. In most of them there was apparently hereditary defect, if not so marked in the mental, at least in the moral sphere, she lays great stress upon the occurrence in these people of attacks characterized by headache and disturbed mental condition, at the end of which the patients seem confused as if in a "hypnoid" state. It is during such periods she thinks that persons already predisposed to lying bring out the phantastic tales which are characteristic of "pseudologia phantastica."

4. *Depressive Psychical Disturbances due to Political Events.*—Several Russian authors have put on record cases of insanity which seem to have arisen both in soldiers and civilians in connection with the Japanese war, and the unsettled political conditions in Russia. Soukhanow has divided the cases which he has observed in the soldiers into the following forms: (1) Depressive hypochondriac, (2) depressive amentic, (3) depressive stuporous, and (4) depressive paranoiac. The author—director of the Orel Asylum—gives the histories of seven cases, all from the working class, which he has observed at his institution. All were characterized by sudden outbreak, depressive anxious affect, hallucinations and illusions. In all cases sudden fright, in the course of one of the prevailing disturbances immediately preceded the outbreak of the mental symptoms. The author summarizes as follows: (1) The mental disturbance followed immediately upon the psychical trauma; (2) In all the patients there was observed extreme anxiety accompanied by ideas of threatening danger and of unavoidable destruction; (3) The delusional ideas were unsystematized and were directed toward no special person, the patient having rather a dread of danger from every side; (4) In all the patients there was mental anxiety and in the majority also optic and auditory

hallucinations of terrifying character; (5) The outcome of the disease is rather favorable, four out of the seven patients recovering; (6) In all except one of the patients, there were stigmata of degeneration.

5. *Discharge of Insane Criminals.*—A discussion of the working of the Prussian laws with regard to the setting at liberty of insane criminals who have sufficiently improved to make it necessary to come to a decision as to whether it is consistent with public safety to release them. The author relates some cases showing where a conflict of opinion between the asylum physicians and the police generally occurs, and ends with some remarks upon special asylums for insane criminals.

C. L. ALLEN (Los Angeles).

Psychiatrische-Neurologische Wochenschrift

(August 11, 1906.)

1. Psychopathic Tendencies among Physicians to the Insane. LOMER.
2. An International Course in Legal Psychology and Psychiatry. SOMMER.

1. *Psychopathic Tendencies of Alienists.*—Attention is called to the danger run by physicians in asylums because of their abnormal environment. While many only suffer from a neurasthenia, examples of much more serious moment are noted and "Anstaltsdemenz" seems to be characteristic. The author advises more rest by taking longer vacations.

2. *Legal Psychiatry.*—Professor Sommer announces a course in legal psychology and psychiatry at Giessen. It will include among other things a study of the forms of criminality, determinism and punishment, the psychology of evidence, heredity and environment, etc.

(August 18, 1906.)

1. Contribution to the Symptomatology of Petit Mal. BRESLER.

1. *Petit Mal.*—The author shows by a series of experiments in a case that although mental processes started before an attack they are completed after the attack is over. The effect of the attack is shown in some way, i. e., in beginning all over again. The attacks exercise at least a simple, mechanical, though not necessarily completely interrupting effect on perception.

(August 25, September 1, 1906.)

1. The Psychology of the Strange. ERNST JEMTSCH.

1. *Psychology of the Strange.*—The author starts with the thesis that a defect of orientation is associated with the impression of strangeness and develops it along the lines of the formula "old—known—trustworthy," "new—strange—untrustworthy." He illustrates this by the painful feeling of apprehension and strangeness produced by wax figures when they are life size and especially when they perform certain movements mechanically, while a doll that is perfectly familiar may be made to open and close its eyes without exciting any such feelings. This general idea is carried out by many illustrations, especially in the realm of art where the strange is often used to produce the emotions of horror and the like. Man is always reaching out for the intellectual government of the outer world. Intellectual certainty assures psychical refuge from the conflicts of the world.

(September 8, 1906.)

1. Weakness of Judgment. DR. H. SCHÄFER.
2. Care of the Insane and Insane Asylums of Italy. (Concluded.)

1. *Weakness of Judgment.*—A short account of a case of moral defect leading to crime and punishment. Of little interest.

2. *Asylums of Italy.*—A very good short directory of the insane asylums of Italy, giving their population, date of foundation, name of director, etc.

(September 22, 1906.)

1. Chiarugi. JOHANNES BRESLER.
2. Account of the First Year's Work at the State Sanitarium for Nervous Diseases at Chemnitz.

1. *Chiarugi.*—A review of the work of the Italian psychiatrist, Vincenzo Chiarugi (1759–1820); "Della Pazzia in genere e in spezia, tratto medico-analitico, con una Centiria di osservazioni" (1793–1794).

2. *Sanitarium at Chemnitz.*—The usual hospital report showing admissions, discharges, etc.

(September 29, 1906.)

1. Contribution to the Forensic Significance of Paramnesia. DR. H. ZINGERLE (Graz).

1. *Paramnesia.*—This is the detailed account of the case of a man whose daughter disappeared and who subsequently, under the insistent questioning of the authorities, confessed to having killed her. As the body had not been found he confessed further to having burned it, and on still more insistent questioning implicated his wife. The family history was very bad and the patient himself, it was shown, had frequent attacks of disturbance, when he was very irritable, incoherent, red in the face and apparently confused. The case came to a fortunate termination by the reappearance of the daughter.

(October 6, 1906.)

1. Contribution to the Forensic Significance of Paramnesia. ZINGERLE. (Continued.)
2. Official Statement of the Forensic Activity of Prussian Asylums.

1. *Paramnesia.*—Will be abstracted when completed.

2. *Prussian Asylums.*—A tabulated statement of the number of persons sent to the Prussian Asylums by the Courts for observation as to their mental state in accordance with the law.

(October 13, 20, 27, 1906.)

1. The Education and Organization of the Attendants on the Insane. DR. ERWIN STRANSKY. (Continued.)
2. Forensic Significance of Paramnesia. (Continued.)
3. The State Supervision of the Insane. MARCUS WYLER. (Continued.)

1. *Education of Attendants on Insane.*—A general discussion of questions relating to the attendants on the insane. The importance of the subject is considered very great for it is the nurses with whom the patient is left the greater part of the time and who should therefore be persons of character and ability.

(November 3, 1906.)

1. Contribution to the Psychiatry of Cataleptic States in Catatonia. F. RIKLIN. (Continued.)
2. Levulosuria and Paresis. E. JACH.
3. Forensic Significance of Paramnesia. (Continued.)

2. *Levulosuria and Paresis.*—The progress in the knowledge of certain general diseases of the body such as myxedema, diabetes, etc., has

exercised a certain influence on the conception of the nature of general paralysis of the insane. While formerly it was considered as purely a brain disease we have come more and more to consider the disturbances of general metabolism in the picture of the disease. The comparison of these disturbances with the similar appearances in the above mentioned disease has more and more led to consideration of paresis as a severe general disturbance of nutrition in which the brain to be sure plays the most important part, but a part only nevertheless.

The author examined 40 paretics, 40 epilepties, 30 other psychoses and 20 normal individuals. Positive reactions were obtained as follows: paretics, 72.5 per cent.; epileptics, 52.5 per cent.; other psychoses, 23.33 per cent.; normal persons, 20 per cent. This shows the highest degree of intolerance to levulose among the paretics. The value of the results are their indications as to the presence of liver disturbances and the relation of the body to carbo-hydrates.

WHITE.

Book Reviews.

MORRIS' HUMAN ANATOMY. Edited by Henry Morris, M.A. and M.B. Lond., F.R.C.S. Eng., and J. Playfair McMurrich, A.M., Ph.D. Fourth Edition. Part III. P. Blakiston's Son & Co., Philadelphia, 1907.

Morris' "Anatomy" is so well known that it is unnecessary to enter into a detailed description of it. The division of the work into five parts is advantageous, inasmuch as any one interested in a special branch of anatomy, the nervous system for example, may have in a volume of small size the portion of the book most useful to him. Part III contains the anatomy of the nervous system and of the organs of special sense. The names of those engaged in rewriting and editing the anatomy are a guarantee of the thoroughness with which the work has been done. The Basle nomenclature has been employed. Some of the terms are not very familiar at present; lateral cerebrospinal fasciculus, for example, requires a moment's thought before we realize that the crossed pyramidal tract is meant. A little use will make the new names acceptable. A large portion of Part III has been rewritten by Irving Hardesty, the section on the ear, tongue and nose has been rewritten by Abram T. Kerr, and a section on the eye is from the pen of R. Marcus Gunn.

The reviewer has not attempted to read Part III through, but he has examined the text carefully and studied the numerous and excellent illustrations. He has found little or nothing to condemn and much to praise.

SPILLER.

THE FRUIT OF THE TREE. By Edith L. Wharton. Charles Scribner's Sons, New York.

Mrs. Wharton's recent novel, "The Fruit of the Tree," has incited so much medical discussion that the laity has turned apprehensively to the profession for its verdict, much as it did after the newspapers burlesqued Dr. Osler's famous age-limit speech, by emphasizing his allusion to Bellamy's chloroform proposition at the expense of his own plea for a doubled salary for retired professors. The question which seems to affright the general public, on reading Mrs. Wharton's pages, is practically the same. Is it really true that the physicians take upon themselves the right to end human suffering when the end is inevitably near; and would a friend or nurse be ethically justified in shortening the term of suffering in an apparently hopeless case?

Because these questions have been threshed out in every medical journal of the country, it is hardly necessary to answer them first, especially as we do not feel that they are the only questions of this keenly analytical book.

The universal helplessness of human suffering, even in the presence of skilled medical attention, must have at some time deeply impressed the

author, just as it both impresses and oppresses the physician who reads his patients' lives as well as their symptoms. The mental anguish of the mill operator whose hand was crushed in the over-crowded factory, mingled with the physical pain that was the prelude to starvation for his wife and children, stands for the type of suffering that might have been prevented, if those who have surplus fortunes would be content to spend part of it on making their laborers' conditions healthful and safe. The hopeless agony of the beautiful society woman, the owner of the mills, as she lies after being thrown from her horse, with what is inferred to be a dislocation and a fracture of the fourth cervical vertebra and lesion of the cord, stands for the type of suffering that neither wealth nor medical skill can help.

But nearly every reader who has in imagination suffered with Bessie Amherst and Justine has at some time suffered or watched suffering of an agony that, whether fatal or not, is no less exquisite and intense. Patient women, heroic men, unknowing little children remember nights following operations or days of fever when, during every second of every minute of every hour, they were wrapt in an eternity of pain, which they accepted as inevitable, with no further expression than the moan or animal whimper that involuntarily accompanies the pain, and which the nurse accepts as a natural sequence, and records on her chart monosyllabically as shock, exhaustion, restlessness or delirium.

The nurse may herself be fully sensible of the degree of suffering, but it would be unprofessional for her to dwell upon it in the pleading terms that relative or friends might use, clutching at the doctor's presence and begging for relief for the patient; and unless the physician himself has had a personal experience of what an eternity of torture might be summed up in the charted words "restless night," he will accept the fact as a matter of course. Of the mental anguish of a patient the physician, or nurse, seldom takes further note than to say "Don't worry." "Depression," as charted in the "remarks," is also something to be expected. Unless he himself has once been in the depths, the busy physician has no conception of the hopelessness of John Dillon, at the thought of providing for his wife and babies without his right hand, and the terror of Bessie Amherst of "living like this," paralyzed and in torment from her neck to her feet.

The questions of bodily functioning and strength of the heart are rightly the all important ones to the physician and nurse. Like the captain and mate of a vessel, they must guide her safely through the storm, regardless of the seasick, terror-stricken souls on board; and yet the triumph of modern navigation lies in the fact that passengers may now cross the perils of the sea in perfect comfort without in any way endangering their safety.

It is not that medicine lacks the means, nor physicians the will to alleviate pain; but there is a hitch in the perfect adjustment of the mechanism, which lies in the "professional" attitude forced upon the nurse by her training.

The nurse is engaged by the physician, but employed and paid by the patient; but if the question of loyalty to one or the other arises, she must be loyal to the physician or resign her post. Mrs. Wharton has drawn the picture of a trained nurse who is more interested in her patients than in her professional relations. She frankly discusses the

future of John Dillon without his arm, even though the physician at the hospital, who is the brother-in-law of the manager of the over-crowded mills, officially states that the injury is slight, and that he will only lose a finger or two.

Technically a nurse must believe and act upon the physician's statement. Technically she is not supposed to help her patient by interesting herself in the family that is weighing on his mind, but in this strong rapid sketch of the relations of patient, physician, and nurse in the charitably endowed, modernly equipped hospital, and later in the boudoir stripped of its feminine luxuries and accoutered with all the grim paraphernalia of the operating room, we get a glimpse of the perfectly constructed system of medical attendance and professional etiquette that bridges the deep abyss of human suffering, without ever sounding its depths.

That the nurse, or physician, should go down, as Justine did, into these depths with her patient, should feel and see suffering from the point of the sufferer as well as from the point of the scientist, is we feel the plea that Mrs. Wharton makes. Justine, this woman of scientific perception and honest thought dares to question the sacredness of human life, either from the lawyer's and clergyman's and physician's standpoint, when confronted by hopeless and scientifically prolonged agony, and she gives an overdose of morphine to shorten the hours or days of inevitable suffering.

Mrs. Wharton chose her dilemma with the utmost nicety; the inevitably fatal termination of the accident was skilfully left open to question by her presentation of the case. Mrs. Amherst had been kept alive by powerful stimulants, alternating with morphine, for nineteen days. The morphine must be diminished, and the stimulants increased in the bare hope of life. Of health, of the power to walk there was absolutely no chance. A slight tremor once noticed in the hand, would indicate that if the patient lived, she might perchance recover some use of her arm, was the only faint reason for making life other than a hideous imprisonment of pain. Out of this prison, Justine let her friend.

That she afterwards married her friend's husband, and was blackmailed by the ambitious young doctor, who was trying to keep Mrs. Amherst alive, till her husband came home, for the sake of making a name for himself—all this is but a skilful complication of the normal question—Dare a man or woman take another life in mercy?

Is it merely a question of sentiment? One may take human life in battle, in the interests of the law, or in self defence, Is there justification in the present instance? This we leave open to the reader.

H. L. JELLIFFE.

JAHRESBERICHT ÜBER DIE LEISTUNGEN UND FORTSCHRITTE AUF DEM GEBIETE DER NEUROLOGIE UND PSYCHIATRIE. Herausgegeben von Dr. Ed. Flatau in Warshau und Dr. S. Bendix in Berlin. Redigiert von Prof. Dr. E. Mendel in Berlin und Privat dozent Dr. L. Jacobsohn in Berlin. X Jahrgang, Bericht über das Jahr 1906. Verlag von S. Karger, Berlin.

The tenth annual volume of the Jahresbericht consists of 1,300 pages and appears in the same form as its predecessors. It reflects the work of the specialties as does no other publication and is absolutely invaluable to all workers in neurology and psychiatry.

The JOURNAL has made the request of its readers from time to time to support such a valuable publication and we can only repeat it in this place.

The work done by the Jahresbericht is well done and no paper of value on any subject in the specialties represented is overlooked either in citation or by full analysis.

JELLIFFE.

ARBEITEN AUS DEM NEUROLOGISCHEN INSTITUT AN DER WIENER UNIVERSITÄT. Herausgegeben von Prof. Dr. Heinrich Obersteiner. Bd. XV. Festschrift zur Feier des 25 Jahrigen Bestandes des Neurologischen Institutes an der Wiener Universität. Herausgegeben von Dr. Otto Marburg. Franz Deuticke, Wien. 25 marks.

This Festschrift edited by Marburg, appears in two volumes, both of which are filled with excellent material gathered from his students now in different parts of the world. One American, Spiller, and one English neurologist, Beevor, contribute to the volumes.

Marburg gives a short introductory historical résumé of the Instiute, it being preceded by an excellent photogravure of Obersteiner. The papers are so numerous that to attempt to analyze them would be useless in this place. Among them may be noted, Oculomotor Paralysis, by E. Fuchs; Contributions to the Study of the Relationship of the Labyrinth and the Eye, by C. Biehl; The Comparative Anatomy of the Red Nucleus, by R. Hatschek; Contribution to the Study of Heredity, by A. Pilcz; On Diffuse Changes in the Cortex due to Brain Tumors, by E. Redlich; Paralysis of the Upward Associated Ocular Movements, by W. G. Spiller; Korsakow's Disease, by W. Serbsky; Changes in the Peripheral Nerves in General Paresis and other Psychoses, by E. Stransky; The Bromine Content of the Epileptic Brain, by H. F. Grünwald; Pseudo-Bulbar Paralysis with Complete Loss of Voluntary Respiration, by Ch. E. Beevor; Differential Diagnosis of Juvenile Bladder Disturbances, by L. v. Frankl-Hochwart; Spondylitis Infectiosa, by H. Schlesinger; Graphic Study of Foot Clonus, by E. Levi; Homicidal Melancholics, by E. Raimann; Congenital Heart Defects and Organic Brain Disease, by R. Neurath; Germinal Glands and the Nervous System, by A. Schüller; Peripheral Facial Paralysis, by A. Fuchs; Medical Report of Japanese Insane Asylum of Tokio, by S. Kure; Juvenile Insanity, by K. Miyake; Experimental Lesions at the Base of the Brain, by A. Spitzer and J. P. Karplus; The Treatment of Tetany by Epithelium Body Preparations, by F. Pineles; Comparative Anatomy of the Cerebral Trigeminal Roots, by E. Huller; The Spinal Cord of Ungulates, by P. Biach; The Pathological Anatomy of Chorea Minor, by K. v. Orzechowski, and Contribution to the Knowledge of the Cortex of Monkeys, by O. Marburg.

All of these studies reflect the high standards which have so many years made these Arbeiten so well and favorably known. This Festchrift is an honor to the editor and a fitting memorial to Professor Obersteiner.

Notes and News

To the Editor of the Journal of Nervous and Mental Disease:—The Sixteenth International Medical Congress will be held at Budapest, from the twenty-ninth of August to the fourth of September, 1909, under the high patronage of His Imperial and Apostolic Majesty. The Secretary General is Professor Emil Grosz, whose address is Budapest, Hungary.

Section II, the Section of Neurology, has been organized with Professor Jendrassik as president.

Professor Jendrassik has asked me to organize an American Committee for the purpose of encouraging the representation of American Neurologists at the Congress. The following gentlemen have kindly consented to act as an American Committee; their function being to promote an interest in the work of the Neurological Section. Professor Jendrassik is working energetically for the success of his Section, and expresses the earnest hope that America will be well represented. Dr. J. J. Putnam, Boston; Dr. Hugh T. Patrick, Chicago; Dr. J. W. McConnell, Philadelphia; Dr. Frank R. Fry, St. Louis; Dr. H. T. Pershing, Denver; Dr. H. M. Thomas, Baltimore; Dr. C. Eugene Riggs, St. Paul; Dr. Leo Newmark, San Francisco; Dr. C. L. Dana, New York, *Chairman.*

The statutes of the congress have been printed, and I have asked that copies of these, and other literature concerning the congress be sent to the members of the committee, for purposes of distribution to those who desire to take part.

CHARLES L. DANA.

American Medico-Psychological Association.—The sixty-fourth annual meeting of the American Medico-Psychological Association will be held in Cincinnati, Ohio, Tuesday, Wednesday, Thursday and Friday, May 12, 13, 14 and 15, 1908. The headquarters of the association at Cincinnati will be at the Hotel Sinton. Dr. F. W. Harmon, superintendent Longview Hospital, Carthage, Ohio, and Dr. F. W. Langdon, medical director The Cincinnati Sanitarium, 5 Garfield Place, Cincinnati, constitute the local Committee of Arrangements.

The following material for the programme has been arranged for: Psychiatry as a Part of Preventive Medicine, by Henry M. Hurd, M.D., Baltimore, Md.; Insanity Increases, by Carlos F. MacDonald, M.D., New York, N. Y.; Etiology of Paresis, by H. C. Eyman, M.D., John D O'Brien, M.D., Massillon, Ohio; The Bacteriology of One Hundred Autopsied Cases of Mental Disease together with brief Clinical, Anatomical and Histological Correlations, by F. P. Gay, M.D., E. T. F. Richards, M.D., and E. E. Southard, M.D., Hathorne, Mass.; The Need of Reform in Expert Testimony, by Walter Channing, M.D., Brookline, Mass.; The Imbecile with Criminal Instincts, by Walter E. Fernald, M.D., Waverly, Mass.; Traumatic General Paresis, especially in its Medico-Legal Bearings, by E. Phillippe Chagnon, M.D., Montreal, Que.; The Diagnosis of Psychic Epilepsy and Allied Hysterical States in their Medico-Legal Re-

lations, with illustrative Cases, by George Villeneuve, M.D., Longue Pointe, Que.; Epilepsy, by Everett Flood, M.D., Palmer, Mass.; Italian Immigration and Insanity, by Albert Warren Ferris, M.D., New York, N. Y.; Past History of Some Insane Patients Deported, by Sidney D. Wilgus, M.D., New York, N. Y.; Immigration—Legislative Aspects, by Thomas W. Salmon, M.D., Boston, Mass.; Some Data in Reference to Insanity in the Rural Districts, by Bigelow T. Sanborn, M.D., Augusta, Me.; The Relation of Urban Life to Insanity, by Michael Campbell, M.D., Bearden, Tenn.; Teaching Insanity, by N. Emmons Paine, M.D., West Newton, Mass.; Neuropathic Wards in the General Hospitals, by Donald Campbell Meyers, M.D., Toronto, Ont.; Heredity, by J. T. Searcy, M.D., Tuscaloosa, Ala.; A Study of Some Phases of Family Psychoses, by John Gerald Fitzgerald, M.D., Toronto, Ont.; A Method of Craniometry, by H. A. Tomlinson, M.D., St. Peter, Minn.; Concerning Thyreodectomy and the Thyreo-Lecithin Treatment of Catatonia—Ten Consecutive Cases, by Henry J. Berkley, M.D., Baltimore, Md.; Hydrotheraphy in the Treatment of the Insane, by George Stockton, M.D., Columbus, Ohio.; Electricity in the Treatment of Mental Disease, by W. M. Knowlton, M.D., Brookline, Mass.; Alcoholic Psychoses in Hospitals for the Insane, by J. M. Keniston, M.D., Middletown, Conn.; Insanities Arising in the Fifth and Sixth Decades, by E. E. Southard, M.D., and H. W. Mitchell, M.D., Hathorne, Mass.; Impressions of Some Asylums in Scotland, by Chas. A. Drew, M.D., State Farm, Mass.; Anxiety Psychoses, by I. G. Harris, M.D., Poughkeepsie, N. Y.; New Statistical Methods in New York State, by William L. Russell, M.D., Poughkeepsie, N. Y. Application forms for membership may be obtained from the Secretary.

CHARLES W. PILGRIM, M.D., *Secretary.*

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Original Articles

A CASE OF MENINGEAL TUMOR COMPRESSING THE CEREBELLUM

By Helen Baldwin, M.D.,
PHYSICIAN TO THE NEW YORK INFIRMARY FOR WOMEN AND CHILDREN

The symptoms of brain tumors, especially of rapidly growing neoplasms, are often so definite, so obvious and so distressing that they demand recognition and make a diagnosis relatively easy. With slowly growing tumors, however, the case is different and these may attain a large size without causing any one of the classical symptoms of new growths in the brain—headache, vomiting or optic neuritis.

The patient whose history is here given developed the first symptoms of her disease ten years before her death, the tumor grew to such a size that it would seem that it must have given characteristic symptoms, yet optic neuritis never developed, there were only a few attacks of vomiting throughout the illness, and these were evidently due to acute indigestion, and while headaches occurred for short periods daily in the early years of the disease, during the three years before the patient's death they did not recur. During the course of the illness the symptoms so closely simulated those of Parkinson's disease, of arteriosclerosis of the vessels in the cerebrum and cerebellum, and of cyst of the cerebellum, that while the question of brain tumor was always considered, the symptoms at no time seemed to justify an exploratory operation.

The first symptoms directly referable to the tumor were noticed ten years before the fatal termination of the disease. The

patient on waking one morning was seized with a very severe pain over the suboccipital region of the brain. This lasted only a few moments, but recurred each succeeding morning on waking. At about this time, when walking one day, she suddenly fell with no apparent cause, with no vertigo, and with no loss of consciousness.

The patient was at this time 54 years of age, a married woman who had borne three children. Her father died suddenly of

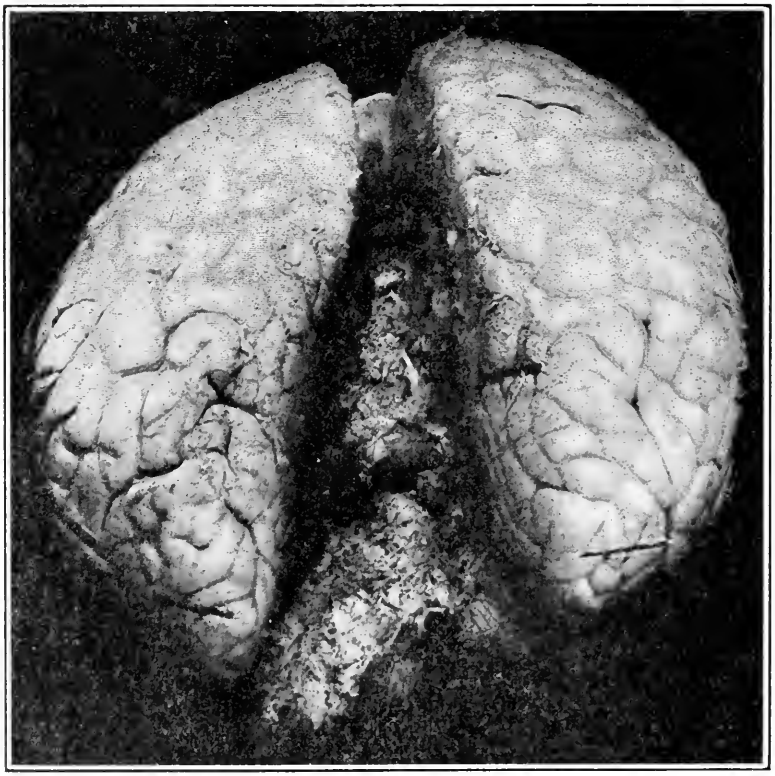

FIG. 1. Showing the Position of the Tumor.

heart disease. Her mother died of uremia, following an operation for pelvic tumor. The patient had ten brothers and sisters, all but two of whom survived her.

The early history of her sickness can best be described in her own words. This record was written in the fall of 1903.

. . . I do not believe that any one ever had such good health, certainly not any better, than I enjoyed until the age of 54 years. During this period, I sustained many trials, some of a nature peculiarly calculated to break down the nervous system of women, but I did not break down. Indeed I often reflected with pleasure that not only my muscles, digestive apparatus, etc., were always in perfect running order, but my brain was always singularly clear and buoyant. It seemed to me often as if I lived in a glass house on the summit of a lofty mountain where I could see in every direction an almost illimitable distance looking through an atmosphere of blue and gold. The delight I experienced in the clearness of this view was immense. On account of it I was never conscious of depression or of irritation for more than a few moments at a time. I lived in an equable golden calm as in a sunrise or sunset cloud. I emphasize this habitual condition because it was on account of it that the first symptoms of the present illness became so conspicuous from contrast and attracted my attention, as otherwise they might not have done.

In the winter of 1896, . . . on waking one morning I experienced a very sharp pain running transversely just below the occiput. It lasted between three and five minutes, then disappeared, and was heard no more of throughout the day. But the next morning at precisely the same time the suboccipital pain returned with precisely the same characters, and lasting precisely the same length of time. From this date, the same thing happened every morning for four years, and the pain never occurred at any other time of day. But in 1900 it did begin to come on occasionally at other times, always, however, lasting such a short time, three to five minutes, that it did not seem to me deserving of much attention, however severe it was while it lasted. Finally, in the year 1900, the morning pain instead of disappearing persisted and increased in severity, in extent, and territory, became complicated with nausea, then vomiting—assumed in fact all the characters of an ordinary sick headache.

I think I had never had a sick headache before. It lasted from early morning until early in the afternoon, then died away under the influence of phenalgine. This sick headache recurred every six months during the next year and a half. In the interval the head was as clear as usual, only the sharp attack of suboccipital pain continued to occur for five minutes every morning.

In June of 1901, I joined a party in an expedition to the Yellowstone Park, where I spent a week. On the first day, and after the thirty-six hours' railroad ride, I had a sick headache with moderate pain, but much nausea. The next day, and for a week, I was perfectly well. But the day before leaving I indulged in a hot bath from the geyser water and was seized in the night with an extremely violent pain in the head, not limited

as usual to its posterior third, but extending all over, and soon accompanied by retching and vomiting. These symptoms were so severe in the morning that I was quite unable to rise and accompany the party home. I remained in bed all day, took phenalgine, and gradually recovered. The following winter, however, I noted a gradual increase in the head symptoms occurring on waking in the morning, also a great difficulty in arising from a recumbent to a sitting position. Frequently there was an attack of nausea, and even vomiting, after getting out of bed. Great irritability of the bladder at this time, and that also frequently continued during the day, but altogether the first few hours in the morning were always pretty miserable. Nevertheless, I managed to do my work, usually after 10 A. M., and felt pretty well, but during the winter of 1901–1902, the attacks of sickness became more frequent, and towards spring about once every two or three weeks I was laid up in bed all day. At the end of May, I had an especially severe attack, which was arrested this time by nitroglycerine. I was then ordered to bed for a fortnight, the first such experience in my life, during which time I felt quite ill. I continued to take a few tablets of nitroglycerine daily.

On the seventh of June, I went into the country with my family, and stayed there until the seventh of October. Three days before I went to bed I suddenly lost in great measure my power of walking. I was walking home, . . . when it suddenly seemed to have become almost impossible to drag one foot after another. It was with great difficulty that I climbed up the steps to the house. This was the first of June, and from that date to the present my walking power has been greatly reduced. During the summer I at first only attempted to walk a few steps off the piazza. After a month I could sometimes walk a quarter of a mile, and in three months I could occasionally walk half a mile. There was no pain or stiffness in the legs at the time, but since the last week I have noticed, with some concern, that a great feeling of heaviness and dragging is liable to come on in the end of the back, and especially after walking. I cannot now walk more than from three to six blocks. At the time of this limitation in extent of walking capacity, I began to totter somewhat in walking. On going downstairs there was a tendency to pitch forwards. This tottering has not increased, but it has persisted. Since the last two months, I cannot walk more than three or four blocks, and that with the aid of a cane and an assistant's arm. From time to time I have fallen suddenly—not when out of doors, most frequently upon arising after sitting for a long time, perhaps especially in the evening. I would fall to the floor, and experience considerable difficulty in getting upon a chair. The fall was unaccompanied by either vertigo, giddiness or pain. Indeed no different sensation in any part of the body; the legs

simply gave way as if I had been on skates. After a moment or two, I could climb to my feet again and felt none the worse for the adventure. These attacks of falling have occurred about once in three or four weeks during the winter.

I do not find any symptoms of paralysis in any of the four limbs, either of motility or sensibility. Neither are there cramps, contracture of muscles, or stiffness in them. I can climb in and out of a bath tub usually with ease, though occasionally I need the maid to assist me, as also in arising from the bed in the morning. There is no steadiness or regular progression in this nor in any of the other symptoms. There is a constant sense of general fatigue and inability for exertion.

A slight tremor has occurred in my right hand during the last winter. It comes during repose and is scarcely rhythmical; its excursion is very small; it is not developed during motion or exertion. During the last two or three weeks, I think this tremor has been much less, and often for many hours it would be entirely absent. There is no fibrillary tremor.

A change in mental condition began in a subtle manner about six years ago. I began to lose the initiative, which had formerly been so active with me. I was not at all depressed or melancholy, but became relatively indifferent. It seemed as if a fine gauze veil were thrown over all the objects in which I had formerly been so intensely interested. It was like the life after death as the Greeks understood it when they described Hades. My interest in contemplation persisted, and even increased, but I acquired an increasing reluctance to effort and voluntary mental exertion. I appreciated Schopenhauer's "Condemnation of the Will," and felt confirmed in my view that his entire theory sprang from a deep inner consciousness of personal weakness of volition. This impressed me the more from its sharp contrast with the vivacity and strength of volition which had been a leading characteristic with me all my life. There was a facility of fatigue after mental exertion, quite comparable to that after walking. This became marked at the same time with the latter, that is after June, 1903, although the sense of loss of initiative had begun, as I have said, six years before. In the last week I have had for the first time a dragging heaviness in my left arm, and some stiffness when I move it backward.

In addition to the above symptoms recorded by the patient, there were others occurring at this time which were noted by her friends. These were mainly symptoms of ataxia. When walking about the house she would place her hands upon objects to support her or more often to regain her balance. When on the street she was inclined to walk with a broad base and with short

steps as though to steady her gait. When walking quickly she had a propulsive gait. If she wished to turn in her course she would stop abruptly and bend her body backward to regain her poise before turning. This propulsion was noted even five years before her death. In the latter part of this period it would frequently happen that if she turned out the light and the room became dark she would fall to the floor and be unable to rise

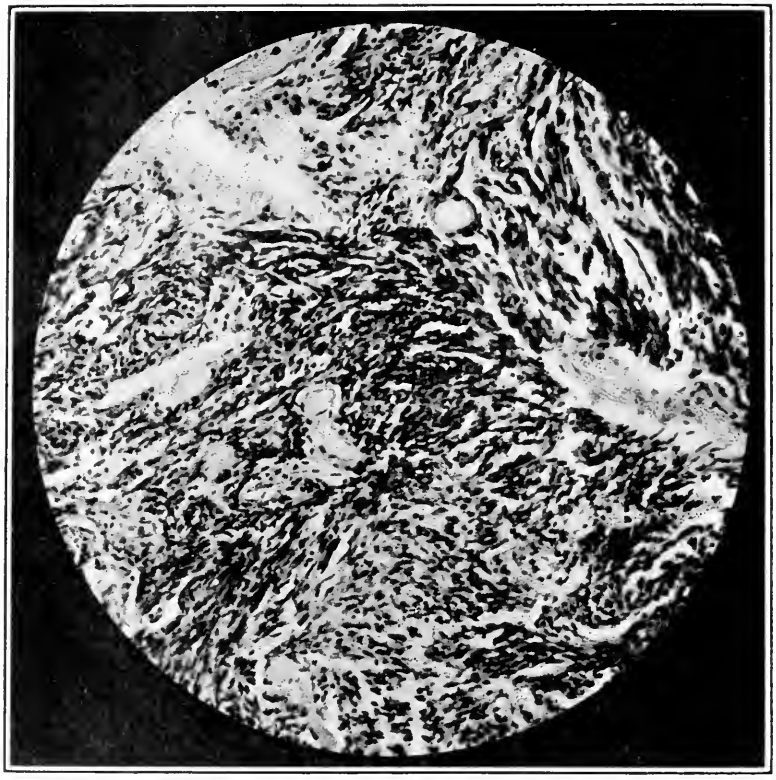

FIG. 2. Section of the Tumor.

until she had crept on her hands and knees to some object which she could use as a support. At this time also a tendency to drowsiness was becoming marked, and yawning with or without apparent sleepiness was a conspicuous symptom. After talking or laughing she would sometimes put her hand to the back of her neck as if in pain.

Three years before her death the patient came under the care of the writer. At this time the symptoms closely simulated those of Parkinson's disease, the gait being especially characteristic. She would start to walk with a resolute erect poise, but after a few steps she would begin to bend forward, and bending more and more, would hurry to reach some support. She had at this time a tremor of the right hand which with effort she could control for a time. She had also a slight tremor in her tongue. There was an immobility of countenance and an increasing slowness of mental processes. The fingers were held in a flexed position, so that at one time slight sores developed on the palmar surface of the right hand. The reflexes were normal. There was a general muscular weakness, more marked on the right side, but with no rigidity. When walking she had a tendency to fall to the right side. Sensation to touch, pressure and temperature over the skin seemed to be unimpaired. But she early lost the sense of taste and of touch within the mouth, so that she could not tell whether she was holding food in her mouth or not. There was at no time any paralysis of the palate. There was a weakness in the action of the epiglottis, so that frequently, while eating, particles of food or drink would pass into the larynx and choke her. Throughout the later years of the patient's illness the symptoms of intestinal indigestion were very marked. The feces contained masses of undigested meat and starchy food, while the urine gave marked reactions for the products of intestinal putrefaction. The following urinary analysis is typical of all that were made. The volume voided in twenty-four hours could not be measured as incontinence was a symptom throughout the later years of the patient's life.

Urinary Analysis.—November 28, 1905. Color deep amber. Reaction very strongly acid. Sediment, heavy deposit of uric acid and urates, with many epithelial cells and a few leucocytes. Specific gravity, 1029. Urea, 2.25 per cent. Uric acid, 0.16 per cent. Ratio, $\frac{\text{urea}}{\text{uric acid}}$, 14.2. Nitrogen of ammonia, 2.9 per cent. of total nitrogen. Indican, marked reaction. Phenol, very marked reaction. Acetone, considerable amount. At no time was there a trace of albumin or any casts found in the urine.

The blood pressure was repeatedly taken with an old type of Riva-Rocci instrument. The blood pressure in the left arm was

constantly about 118. That in the right arm was higher, being about 170.

The examination of the heart and lungs was negative until the development of the terminal pneumonia.

The eyes were examined at intervals of about six months and there was at no time any evidence of optic neuritis. Two years before her death she began to develop deafness which was more marked in the right ear, but the hearing on the left side was also affected. The deafness began in September, 1904, and was described by the patient as occurring in rather definite cycles which commenced as a feeling of fulness in the ear, confusion of sounds and increasing deafness which by the end of fifteen minutes became marked. This condition remained at its height for an hour or more and then gradually subsided until the hearing became nearly normal. There was no tinnitus, no vertigo and no nausea. These symptoms would be repeated from three to five times daily, and at the end of about two weeks she noticed that the hearing did not fully return after the attacks. On October 18, 1904, she was examined by Dr. Alice E. Wakefield, whose report is as follows: "I found the left ear practically normal. The right showed slightly dull drum membrane. Tone limits were from 16 to 512; bone conduction diminished; Politzer acumeter heard six feet (perception low)." On November 25, Dr. Wakefield reported: "Right ear—Deafness markedly increased; acumeter 18 inches; no fork heard above 256; bone conduction could not be elicited; no nausea and no vertigo; tinnitus intermittent—not marked. The cycles which characterized the beginning of deafness have given way to permanent and rapidly increasing deafness. The left ear is still practically normal, although answers to tests are slowly and hesitatingly given. Eye grounds—Optic nerves are pale and arteries are small, but not exceeding physiological limits. The lesion involving the auditory nerve may lie anywhere along its course. It seems to me from the general symptoms that the lesion may be along that part of the nerve which lies in contact with the right lobe of the cerebellum at its junction with the anterior end of the worm, and could result from degenerative processes involving the right side of the cerebellum."

At Dr. Wakefield's request, an examination was also made

by Dr. E. Gruening, on December 2, 1904. He made the following report: "There was nothing positive in the ocular examination. I found both eyes perfectly sound. The affection of the right ear seems to have its seat in the labyrinth, inasmuch as no laterization to the affected ear could be obtained in the Weber test and Rinné was positive."

As the disease advanced there was a steadily increasing mental lethargy. The mentality became slow, but was always accurate. The patient's mind was very quickly tired. There were no perversions of the mental processes, apart from the dulling and slowing of thought and memory.

The following record was made on February 21, 1905, eighteen months before her death: She can stand, if balanced, and can walk if she is commanded to, and her attention is kept fixed. The legs are strong and show no atrophy. The hands and arms seem fairly normal, and a tremor which was once present in the right arm is rarely seen. The dynamometer is, for the right 40, for the left 35. There is no tremor of the face or legs, except at times; then it is coarse and intentional. She writes rather slowly and poorly; speech is slow. She cannot move in bed without help. The eye reflexes are normal, the kneejerks the same. Ocular excursions normal, except that there is a paralysis of convergence only. No forced laughter. Deglutition normal. There is deafness in the right ear and slight hyperesthesia of the right side of the face. There is no ataxia of the arms, and finger nose test is normal. There is no astereognosis. The patient had had sudden attacks in which she fell to the ground, without any distinct loss of consciousness or any convulsive movement. The attacks begin with pain in the occiput, which runs over to the front of the head, then she suddenly sinks to the ground.

On January 12, 1906, Dr. Wakefield again examined the eyes and ears and reported that the retinæ were pale, and the retinal arteries were small. There was a conjunctivitis probably trophic. The condition of the ears was the same as the year before.

The progress of the disease during the last six months of the patient's life was marked by a steady loss in muscular power and a gradually increasing mental hebetude. She could not stand or walk unsupported. Her head when she was resting in

her chair had a tendency to turn toward the left shoulder. In the later months she could not maintain an erect position in a chair or support her head. At this time, there were no cerebral palsies, except the deafness in the left ear. There was slight dulness to sensation on the right side of the cheek and tongue at times. There was very great slowness of deglutition and speech. No nystagmus or oculomotor palsy. No ataxia of the arms. The kneejerks were fairly good. There was transient rigidity of the arms and legs. There was a gradual failure of the memory—extraordinary slowness and retardation of thought so that a reply would sometimes come out half a minute or one or two minutes after the question. She would start to express herself, then would lose the train of thought, which sometimes would come back to her later. There was no emotional disturbance, no headache or pain anywhere. She had no distinct paralysis of the limbs, but for months she was entirely helpless.

Two weeks before her death she had an attack of left hemiplegia, with clonus of both feet, and a very quick cerebral reflex to the plantar surface—like that of a baby, but no Babinski. There was a slight return of power following this attack, but an aspiration pneumonia developed which terminated fatally.

The autopsy was made by Dr. Libman and Dr. Israel Strauss. Their notes on the case are as follows:

Brain and Dura.—Dura, normal. Sinuses, normal. Brain, convolutions markedly flattened and fissures narrowed because of the condition of marked internal hydrocephalus. This was due to the pressure of a neoplasm lying on the upper surface of the left half of the cerebellum. The neoplasm was dark red in color, very vascular and in places showing psammomatous degeneration. It covered the entire left half of the cerebellum and encroached upon the worm. Posteriorly it was visible by its projection beyond the overhanging occipital lobe of the cerebrum. Anteriorly it reached as far forward as the corpora quadrigemina upon the left lobe of which it exerted considerable pressure. There was likewise a marked depression in the left crura cerebri and to a lesser degree in the cortex in the region of the hippocampal fissure. The left lobe of the cerebellum was reduced to almost half its thickness by the growth, but its substance was not infiltrated. The neoplasm was about 2 cm. in thickness, triangu-

lar in shape with its base posterior. It arose from the pia mater and nowhere showed any evidence of connection with the brain. Microscopical examination shows it to have been an endothelioma such as is characteristic of neoplasms arising from the pia.

Lungs: congestion at bases; in right lower lobe an area of purulent broncho-pneumonia, about 2 cm. in diameter.

Heart: brown atrophy with fatty degeneration; moderate atheroma of aorta; no congenital lesions.

Spleen: not enlarged, pulp soft, congested.

Liver: congested, fatty; gall-bladder and ducts negative.

Pancreas and adrenals: negative.

Kidneys: parenchymatous change, congestion; changes resulting from arteriosclerosis of the vessels.

Thyroid: normal.

In reviewing the history of this case, it will be seen that while the symptoms of brain tumor were not distinctive, those due to cerebellar involvement were marked throughout the course of the disease. These include the attacks of suboccipital headaches, the cerebellar ataxia, the cerebellar seizures—sudden relaxation and falling with no vertigo or unconsciousness—and the tendency when sitting to drop the head toward the left shoulder. The symptoms of involvement of cranial nerves also pointed to a localization of the disease at the base of the brain—the nerve deafness in the right ear, dysphagia and anesthesia of the tongue. But while the involvement of the cerebellum was evident, the lesion seemed to be one of degeneration rather than of compression from an extra-cerebellar growth.

MELANCHOLIA WITH DELUSIONS OF NEGATION: THREE CASES WITH AUTOPSY[1]

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While engaged upon a more general analysis of involution-melancholia, presenile insanity, and allied conditions, our attention was arrested by three cases of melancholia showing feelings of unreality. Recent interest has been so great in patients with feelings of unreality and nihilistic delusions that we have been tempted to anticipate our more general report upon the great subsenile group with a particular account of these three cases. The cases present a certain uniformity both from a clinical point of view and from the histopathological side. Anatomically, the cases belong in the catalogue of insane subjects with "normal" brains or brains with "unimportant" or "trifling" lesions. It is these cases which now deserve the closest histopathological attention, especially from the point of view of the intracellular pigments and fats and their distribution. The problem in these cases of "normal" brains might shift from considering foci of destruction to determining the degrees and loci of intracellular clogging with pigments and fats.

The paper deals with three cases of melancholia which showed feelings of unreality and nihilistic delusions, but is not to be taken as a brief for Cotard's syndrome as an entity among mental diseases. Nevertheless, feelings of unreality and nihilistic delusions, together with various other ideas of a metaphysical cast, are so striking an element in certain conditions of melan-

[1] From the wards and laboratory of the Danvers Insane Hospital. The term melancholia is here used in the older Kraepelinian sense.

cholia that we can hardly escape throwing such cases into a group for convenience at least. Moreover, although delusions of this type undeniably dwell in the minds of many persons in and about the climacteric era and although the climacteric state appears to have many features which are met again and again and afford some color to the claim of melancholia as an entity, we do not wish at present to concede that either Cotard's syndrome or melancholia at large is an ultimate group for a finished psychiatry.[2]

Histopathology can have little to say on the cerebral side so long as the metabolism of involution presents such meager data. The present cases, however, so far as their examination was carried, offer interesting data which support the claim of a central rather than a peripheral disorder as the basis for the mental states shown. Moreover, the central disorder appears to involve, not so much the great cells which are related to the periphery, as the smaller cells which must be concerned with the elaboration and transforming of peripheral data.

Should our hypothesis hold, the cases of so-called melancholia (including many of those which fall into Cotard's syndrome) might prove to be founded on a central disease. The importance cannot be denied of proving that these patients are not simply explaining and rationalizing their own perverted metabolism, but are actually suffering from a fault in the very mechanism of explanation and rationalizing. Naturally the cerebral fault may itself turn out to be metabolic. We can have nothing to say on this line and offer our cases as preliminary to a more extensive study of the group called melancholia.

Case I.—G. E. H. Admitted to Danvers Insane Hospital July 10, 1903. Age, 49. Occupation, clerk.

Family History.—His father was an eccentric, incapable man, and there was a history of occurrence of insanity in distant relatives on paternal side. The relationship and character of mental disturbance could not be ascertained.

Personal History.—Naturally, he was a man of cheerful, pleasant temperament, with average business capacity. His habits in regard to alcohol were good. There was no history or evidence of syphilis. Previous to present illness he had had lum-

[2] Since this paper was first presented, Dreyfus has published "Die Melancholie ein Zustandsbild des manisch-depressiven Irreseins," 1907, representing a reversal of Kraepelinian opinions concerning melancholia as an entity.

bago on several occasions, but no well-defined attacks of rheumatism or any other disorder which could account for the cardiac hypertrophy found on admission. The occurrence of previous mental disturbance was denied by relatives. During the winter of 1902–1903, he lost money, became depressed, slept poorly, lost weight and had to give up work in February, 1903. His depression deepened and he became restless and agitated, fancied that he was ruined and in a hopeless condition.

He was admitted to the McLean Hospital, April 28, 1903. We are indebted to Dr. Tuttle for clinical notes which have been used in the case description. Upon admission at Danvers examination was made by Dr. H. M. Swift.

Physical Condition.—July 11, 1903. His muscular development was good, but he was poorly nourished. Area of cardiac dullness was enlarged to the left, and apex beat was in sixth space. There was a systolic murmur heard most plainly at the apex and transmitted to the axilla. Pulse was 90–100, increased tension, and radials were somewhat thickened. Urinary examination was negative. Cranial nerves were normal. Pupils equal and reacted to light. Tendon reflexes were lively and equal. No disturbance of sensation was found.

The notes on the mental condition from April 28 to July, 1903, are as follows: During the month of May there were short periods when orientation for time and place was imperfect, and at these times he had imperfect memory and displayed great agitation. He was distinctly apprehensive, thinking the register was a machine to kill him; that he had signed away the whole United States; that he had opened all the asylums in the United States; that he had killed his wife and could see her blood on his hands. At times, he appreciated the absurdity of these ideas and would converse clearly. Gradually his apprehensiveness and agitation increased, though at intervals he would demonstrate a good grasp upon surroundings. He became resistive, talked in anxious whispered tones between moans and sighs, and developed the feeling of unreality, seated, rocking body to and fro, wringing his hands; he would whisper between his groans and sighs: "I am all mixed up. It's all a mistake. I'm not Mr. H——. I am all gone, I am dead to the world." Again he would say: "I'm not G. E. H., I ought to be him, but I've taken these names along when I have seen them in the yard. I've made myself as Gibbs, I've been here as G. E. H., and have a home in Haverhill; not I, but G. E. H., has a home in Haverhill. I've made myself a different one."

His agitation increased, and he became untidy and very resistive. His productions became desultory and disconnected. He denied identity, had no stomach, could not take food, and at the last had no idea of time or place. He died of pneumonia July 13, 1903.

Summary of Case I.—Father was eccentric and there were instances of insanity on paternal side of family. Age, 49. Habits exemplary. Loss of money only recognizable cause for gradually increasing worry and depression. No serious illness previously. Cardiac hypertrophy, arterio-sclerosis, only recognized physical ailments.

Mental symptoms appeared in following order: worry, insomnia, restlessness, depression, delusions, possibly visual hallucinations, agitation, ideas of negation and stuporous condition preceding death. Orientation fairly well preserved until the last few days.

Autopsy Findings.—The autopsy was performed by Dr. A. M. Barrett and showed death due to pneumonia of the right lower lobe, lower portion of upper lobe, and posterior portion of the middle lobe of left lung. The heart muscle was pale and flabby. The mitral valve was moderately thickened. There were a few patches of atheroma in thoracic portion of the aorta. The liver showed slight surplus of fat. The heart weighed 445 grams. There was a chronic fibrous peritonitis in the region of the appendix and upon the lower surface of the liver and around the gall-bladder. Examination of the skull showed the sutures preserved and the diploë in large amount. The dura mater firmly adherent to the calvarium. The brain with pia mater weighed 1445 grams. The pia mater contained considerable fluid and was hazy along the edge of vessels. Adhesions were found at the bottom of the fissures of Sylvius. The large blood vessels at the base showed some yellow atheromatous patches of irregular distribution. The lumina of the arteries were not diminished. The choroid plexuses showed a few minute cysts. There were no focal areas of softening in the brain. The ependyma of the ventricles was smooth.

Microscopical Examination.—Several regions of the cerebral cortex in both hemispheres showed an essentially normal layering (Nissl's method). There are nowhere focal destructive lesions. One lesion is common to all the sections examined, viz., a great quantity of pigmented cells in the adventitiæ of the vessels in all parts of the gray and white matter (best demonstrated in Heidenhain iron hematoxylin preparations). The pigment around the larger vessels is enclosed in cells of the usual meningeal phagocyte type. But about the capillaries of the gray matter the pigment is as a rule enclosed in neuroglia cells.

The source of the perivascular pigmentation cannot be made out from the vessels affected, since all the vessels with but few exceptions are equally invested with pigmented cells. The distribution of pigment in neuroglia cells is a little more characteristic. The pigmentation of neuroglia cells is not, as in some conditions, particularly found in the outer or subpial layer or in the inner or fusiform cell layer. The neuroglia cell pigmentation

tends to affect the intermediate layers of the cortex. This is best shown in the layers of large and small stellate cells of the calcarine areas. The frontal areas, however, show a fairly general distribution of pigmented neuroglia cells.

The nerve cell pigmentations are more characteristic in point of distribution. The large pyramidal cells of the frontal cortex are practically all pigmented and, in most cases, somewhat characteristically along the lateral borders and at the inferior angles, leaving the perinuclear region and the apical portion free of pigment. The pigment is not collected in a single sac, but appears to occupy interstices of the cells with perfect maintenance of their proper contours. The characteristic unifocal collections of pigment in the Betz cells of the paracentral lobules are absent in this case. The Betz cells have little or no pigment of any sort. The larger nerve cells (including the solitary cells of Meynert) of the calcarine areas fail to show much pigment.

The examination indicates a steady outward stream of pigment materials from the nerve tissues to the perivascular region. So far as the examination of a limited number of areas is conclusive, the autochthonous pigmentation of the nerve cells is maximal in cell types whose function is either unknown or receptive. Large elements like the Betz cells and the solitary cells of Meynert fail to show marked pigmentation. These larger elements are doubtless more directly related with the projection system than the others.

Case II.—T. H. Admitted to Danvers Insane Hospital June 13, 1904. Age, 75. Occupation, mason.

Family History.—Both maternal and paternal relatives were unusually healthy, long-lived people. His mother died at the age of 63, and had exhibited a continuous, melancholy depression for the last few years of life. One brother died of apoplexy at the age of 52. Family history was otherwise negative.

Personal History.—He had a normal development and became a capable and modestly successful contractor, retiring from business at the age of 65, with a small competency. His temperament was cheerful and sociable. Family relations were pleasant. At no period of life had he ever used distilled liquors, but occasionally he drank a little beer with meals.

At the age of 25, he had a venereal lesion, probably nonspecific. Aside from an attack of pneumonia seven years prior to commitment, he had always been healthy. His relatives had noted some memory failure for recent events during a few years before admission, and he had been troubled with insomnia for one year, but no symptoms of insanity were recognized until the week before his commitment. The loss of an only son eight years before, who left a widow and four children dependent upon the patient for support, had been the only known cause for worry. A sudden depression developed. He thought he had lost all his

property and that he was responsible for the consequent misery of his family. He became sleepless for a week and finally cut his throat, wounding the larynx. He struggled to prevent dressing of the wound, declaring that he did not want attention, that he must die; and threatened to accomplish suicide by some other means. He was soon admitted to the hospital.

Physical Condition.—He had a powerful muscular development, was well nourished, tissues showing very little senile atrophy. There was an infected wound involving larynx. Temperature on admission 99.4°. There was harsh respiration and occasional coarse rales were heard over anterior surface of chest. Area of cardiac dulness slightly enlarged. Second aortic sound was sharp and accented. First sound at apex was roughened. Pulse was 126, fair tension, irregular rhythm and occasionally intermittent. Superficial arteries were thickened and tortuous, and there was well-defined arcus senilis. Urine showed trace of albumin. No sugar. Sediment contained hyaline casts, pus cells, uric acid and calcium oxalate crystals. Strength was little impaired and he had perfect control of muscular movements.

Cranial nerves were normal. Pupils were responsive to light. Tendon and skin reflexes were normal. No sensory disturbance detected.

Mental Condition.—At time of admission, he was oriented for time, place and persons, and appreciated the character of his surroundings, but answers had to be obtained between his groans and protestations. His mental distress and agitation rapidly became more pronounced, and he would pace about his room, wringing his hands, his features contorted with emotion, muttering about his "most miserable state," his family and his deplorable acts. With great and steadily increasing difficulty his attention could be gained momentarily, but most of the time he took little notice of requests and struggled blindly against efforts to care for him. His spontaneous talk showed great depression, nihilistic delusions with the feeling of unreality; *e. g.*, "Oh! I am in a hopeless state. It is awful and nobody ever suffered this way. This is not a reality. This is eternity, this is eternity. Why such a thing as I the world never knew, the world never saw. Why! Why! Why! I can't move, I can't die." As nourishment was offered he said: "No, no, I can't take it. It won't do me any good. I can't take it, I am not a reality." At another time, he declared he was in a vault, and that the sunlight, streaming into the room was not real; that the sky was not blue; that the view from his window was not a reality; that the people about him were not real human beings, and that no person had ever been in such a miserable state as himself. His agitation was practically continuous. He would appear exhausted and lie down and secure short, fitful sleep. The suppurating laryngeal wound

eventually, prevented audible speech, and death resulted from pneumonia on the eighth day in hospital.

Summary of Case II.—Mother had mental depression during last four years of life. Brother apoplectic. Age, 75. Moderate beer drinker. Old age and worry over loss of son and responsibility of caring for son's family only known causes for a comparatively sudden outbreak. No known illness, excepting pneumonia, seven years previously. Arteriosclerosis, nephritis, myocarditis and bronchitis from wound in larynx were the only physical ailments noted. Slight memory failure and some insomnia preceded for some months a sudden depression, painful delusions concerning self and family, suicidal attempt, great motor agitation, feelings of unreality largely confined to external objects, and nihilistic ideas. Orientation fairly preserved to the last few days of life.

Autopsy Findings.—The autopsy was performed by Dr. A. M. Barrett and showed death probably due to hypostatic pneumonia. Suicidal wound of neck. The heart was fatty. Arteriosclerosis was general, involving the aorta and in particular the splenic vessels and the arteries of the base of the brain. There was well-marked chronic diffuse nephritis. Examination of the head showed the following: dura mater completely and firmly adherent to the calvarium. The pia mater free from thickening. Basal arteries beset with scattered foci of yellow thickening. Internal carotid arteries notably stiff and yellow. Small old cyst of softening just outside tip of posterior horn of right lateral ventricle. The brain with pia mater weighed 1,300 grams.

Microscopical Examination.—The frontal, paracentral and calcarine regions of the cerebral cortex show essentially normal layering. The most prominent feature is a universal pigmentation of perivascular cells, neuroglia cells, and nerve cells of about the same degree in all regions examined. The pigmentation about the vessels is by no means maximal, and is of a degree often shown in younger subjects under different conditions. The neuroglia cell pigmentation affects practically all the cells and, since it occurs also in the subpial layer, is probably due to fiber degeneration as well as to nerve cell changes. Satellite cell pigmentation is, however, also frequent and characteristic, so that a nerve cell origin for some of the pigment is highly probable. The examination of the nerve cell pigmentation is facilitated by parallel study of adjacent sections stained by Nissl's methylene blue method and by Heidenhain's iron hematoxylin method. By Nissl's method it is easy to make out the universality and extent of the senile yellow pigmentation in the layer elments, *e. g.*, large pyramidal cells, Betz cells, solitary cells of Meynert. Besides the yellow pigmentation of the banal type shown in old age, the Heidenhain preparations show an extensive development of pigment staining black by this method. This latter black-stained

pigment lodges in the nerve cells of the intermediate layers, but characteristically affects the smaller elements, *e. g.*, small pyramidal cells, as well as also the large pyramidal cells (in which the black-stained pigment accompanies the yellow pigment which remains unstained). The Betz cells and the solitary cells of Meynert, despite their accumulations of or distention with yellow pigment, fail to show more than a very few black globules in the Heidenhain preparations. The black-staining pigment seems never to alter the cell form, but to lie in crevices which may represent lost tissue. The Nissl stain shows that the Nissl bodies are throughout not well stainable; but this circumstance cannot yet be unequivocally related to the cell pigmentation.

The arteriosclerotic element in this case is not prominent, despite the old cyst of softening in the left occipital region. Nor is cerebral atrophy a prominent feature. The most striking features are extensive pigmentations, which are partly of senile origin and partly reside in elements which are of unknown or receptive function. These two types of pigmentation are easily separable in the Heidenhain preparations.

Case III.—J. H. A. Admitted to Danvers Insane Hospital, November 21, 1903. Age, 65. Occupation, shoemaker.

Family History.—Patient's father died of some cardiac disorder at age of 77. His mother died at the age of 67, after suffering from senile dementia for over three years. One brother died of tuberculosis. The history concerning grandparents and collateral branches was unobtainable.

Personal History.—Nothing was known of early life. He enlisted in the army, serving three years during the Civil War, and was afterwards employed as a shoe-factory operative. He was married in 1873, and has two healthy adult children. His wife stated that he had been industrious, kind to family, pleasant and agreeable in disposition. Since early life he had been a moderate user of tobacco and alcohol. He was not a daily drinker and was never known to have been intoxicated.

He had had a purulent discharge from the right ear following scarlet fever at the age of three. This was the only severe illness he had had until the age of 61, when he had typhoid fever and la grippe during the same year and was mildly delirious during both illnesses. He made good recoveries and worked steadily until nine weeks before his commitment, when he was discharged for failure to perform work properly. For two or three months previous to this event he had slept poorly, lost weight, worried over trifles, and had shown evidences of mild hypochondriacal depression. Following the loss of his position, mental symptoms had been progressive. He began to think that he and his family would starve and freeze, and was greatly distressed by fancied physical ailments; *e. g.*, he had the idea at one time that his head, eyes and limbs were growing large and later

thought that he had become much smaller than others who were correspondingly increased in size. Occasionally he would seem to have some insight, and he lost weight steadily. He never showed any suicidal tendencies. A sudden threat of violence to members of his family and refusal of food were the immediate causes of his hospital commitment.

Physical Condition.—He had slender muscular development, pallid complexion and was poorly nourished. His skin was dry and wrinkled. He had sordes on lips and teeth, tongue was coated and breath foul. Temperature ranged from 99° to 100°. Area of cardiac dulness was not increased. Second aortic sound was sharp and accented, and first sound at apex was roughened. Pulse was 96, feeble volume and irregular rhythm. Arteries were soft. There was a faint arcus senilis.

Abdomen was shrunken. Urine was turbid and had a foul odor. Acid. Sp. gr. 1013. No sugar or albumin present. Sediment showed squamous epithelial cells, few red corpuscles, bacteria and moulds. He had accurate control of muscular movements, and walked with a firm gait. He could hear faint watch tick with left ear and could read coarse print. The right pupil was slightly larger than left, and both reacted promptly to light. Tendon reflexes were generally hyperactive and equal. Sensory tests were of little value, as he declared that he had no feeling, although wincing at pin pricks.

Mental Condition.—Soon after admission, he objected to every effort to care for him. He refused to walk, said that he had no legs and could not stand; when an attempt was made to raise him from a sitting position he doubled up his limbs and shouted protests against the cruelty of trying to make a man with no legs walk. His enunciation was not impaired and he talked freely, excepting that he would refuse to answer some questions and remain mute for a short time. In answering questions to test orientation, grasp on surroundings and retention of school knowledge, he would give correct replies after protests, refusals, and nihilistic statements; *e. g.*, Q. Where are you? A. "I don't know, I've got no brains. Yes I do too, Danvers. I've been here. Down here for insane and you are going to report I am all right." Q. Are you sick? A. "Course I am, and you know it. I've got no brains and I can't talk and you are going to make out the wrong report. I've got no heart and no stomach, and I can't swallow. I haven't any feelings." His tones were sharp and manner petulant. He was apprehensive and showed some agitation. There was no evidence of retardation. After much urging he would swallow liquid nourishment in great quantities, but saying it did him no good, because he had no stomach, nothing but a hole and he would drink to fill that up. Every question would suggest some new idea. When asked if he knew any people about him, he answered: "Yes, you are all great big men

and so strong, you must weigh over 400 pounds, and I'm so small you can't see me. I'm freezing to death and you cover me up with great, heavy, wet blankets. I wouldn't burn if you threw me in the hottest furnace, but I can't suffer because I've got no brains."

He would say that he could remember nothing of events from day to day because he had "no brains" and in the same sentence make remarks that showed partial recollection. Depression was constant and at times was very marked. During the first week in hospital he was restless, walking to and fro in his room, groaning and picking at his clothing and fumbling with his bedding. He was occasionally untidy in habits. With increasing weakness, his ideas of negation and unreality became more absurd, and were more brokenly expressed. Death occurred December 19, 1903.

Summary of Case III.—Mother died of senile dementia at 67. Moderate use of alcohol. Age, 65. No assignable cause for insanity. Typhoid and la grippe with delirium accompanying both disorders, four years previously.

Emaciation, arteriosclerosis of mild degree, nephritis, cystitis and lively reflexes were the only important physical ailments. Mental symptoms gradual in onset. Slowly increasing hypochondriacal depression, with incapacity for work, was noted for four months before evolution of ideas of unreality and nihilistic delusions. Mildly agitated, petulant and complaining during the last. Orientation and memory quite well preserved.

Autopsy Findings.—The autopsy was performed by Dr. A. M. Barrett, and showed an extensive suppurative process in the retroperitoneal tissue around the bladder with abscesses anterior to the bladder and numerous narrow sinuous pus channels running backward on either side of the bladder. The process extended upward beyond the limits of the pelvis into the perinephritic fat tissue as far as the upper poles of the kidney.

The right auricle of the heart showed numerous small thrombi. There were numerous recent emboli in the primary branches of the left pulmonary artery. The aorta showed, 30 cm. below the arch, a large thrombus with puriform softening. The spleen had failed to react to septicemia. The lungs, pancreas, renal pelves, ureters and bladder showed various signs of acute inflammation. Moderate aortic sclerosis. The brain showed no gross signs of alteration at the autopsy and was hardened for four weeks in ten per cent. formaldehyde solution. More careful examination later also proved negative. The ependyma of ventricles was smooth.

Microscopical Examination.—Cultures from the heart's blood and retroperitoneal pus showed pneumococcus and a bacillus, probably bacillus coli communis. Examination of the organs of the trunk added little to the gross findings. The kidneys showed

a mild chronic interstitial change with slight fatty changes in the epithelial lining of the tubules.

Microscopical examination of the various regions of the cerebral cortex by Nissl's method, by L. Ehrlich's pyronin method for plasma cells, and by Heidenhain's iron hematoxylin method demonstrated a moderate and in places quite trivial accumulation of cells of the lymphocyte group in the sheaths of small vessels in the lower layers of the gray matter and the adjacent white matter. This accumulation of lymphocytes (and plasma cells) is strikingly even in degree in the parts examined (left and right superior frontal, left and right paracentral, left and right calcarine and occipital regions) and in most sections has to be sought with some care. There is no noteworthy alteration of the nerve-cells, if we except pigmentation of the larger elements (large pyramidal cells of frontal area, Betz cells, large pyramidal cells of occipital area). The other nerve elements (in particular the solitary cells of Meynert) fail to show pigmentation. Numerous neuroglia cells, as well as cells applied to the walls of capillaries, are well pigmented (seen especially in the Heidenhain preparations). The cell layers of the cortex and the cell arrangements of the white matter show no notable alteration.

The question arises whether the data admit the diagnosis *general paresis*. A strict histological diagnosis might be chronic degenerative and exudative encephalitis of a diffuse but mild nature with the exudative element slightly predominant.

Summary of Post-Mortem Findings.

The causes of death were: Case I, pneumonia; Case II, pneumonia following suicidal wound of neck; Case III, general infection from pelvic suppuration.

All three cases presented more or less sclerosis of the aorta. The senile case (II) and case I showed considerable sclerosis of the large arteries of the base of the brain.

Aside from a small old cyst of softening in case II, no brain showed focal destructive lesions. No brain gave sign of general or focal atrophy upon macroscopic examination. Case I was the only case to show pia mater changes, and these were confined to haziness along vessels and adhesions at the bottoms of the Sylvian fissures. Case III, despite the chronic exudate found upon microscopic examination, showed no macroscopic alterations of the pia mater. The ependyma was in all cases smooth.

Microscopical examination of three or more areas in each hemisphere was carried out by several methods, notably by Nissl's

original methylene blue method and by Heidenhain's iron hematoxylin method.

Common to all the cases is an accumulation of meningeal *phagocytes containing pigment.* This perivascular pigmentation is of about the same degree and quality as that found in most adults beyond the age of fifty years. Whether perivascular cell pigmentation signifies increased wear and tear on the part of the nervous tissues, or whether it rather signifies a reduction in the serum's power to take up these substances cannot yet be told with assurance.

A second common feature of the cases is *neuroglia cell pigmentation.* Whether the collections of pigment (or of fat-like bodies) here shown indicate phagocytosis on the part of these cells cannot be established. The accumulations of fat-like substances might be due to some neuroglia cell activity affecting materials brought to the cells in solution. The senile case showed well-marked satellite-cell pigmentation. The neuroglia-cell pigmentation which is found common to all three cases is pigmentation of the scattered neuroglia cells of the middle layers of the cortex.

The nerve cells also show deposits of pigment or fat-like substances. The Heidenhain preparations bring out the presence of two kinds of pigment, which can be distinguished according to capacity or lack of capacity to take up the hematoxylin. The type of pigment which fails to take up hematoxylin is collected in the familiar yellow sacks which form unifocal deposits in the cytoplasm of the larger cells. This yellow pigmentation is found in case II, as it is in numerous senile and subsenile conditions. It is this pigment which is so common that histologists attach little significance thereto. The other kind of pigmentation, so well demonstrable by the Heidenhain method, is developed in all of our three cases. It is in this pigmentation, if in any feature now demonstrable, that we must look for somewhat differential characters in diseases of the group under discussion. The most significant feature of this type of pigmentation is its plurifocal distribution in the affected cells. Whereas the yellow sacks of the more banal type of pigmentation seem to enter into no intimate relation with the cell and may not interrupt its paths, the plurifocal pigmentation, on the contrary, is so distributed that it may well modify or influence the progress of impulses through the cells. It may be noted that this kind of pigment is apt to

be deposited at the sides and inferior angles of the cells (*e. g.*, of the large pyramidal cells of the frontal cortex) and to neglect the perinuclear and apical portions of the cells.

If we take into account the iron-hematoxylin-staining pigment and leave out of account the banal yellow pigment, it appears that the larger elements (*e. g.*, Betz cells, solitary cells of Meynert) are not likely to be affected. The iron-hematoxylin-staining pigmentation proves to be maximal rather in those elements whose function we do not understand or term receptive. If this pigmentation can be taken as a sign of central disease, the disease appears to involve not so much the larger elements which come into immediate relation with the projection system as the smaller elements having to do with transference of impulses or having unknown functions.

General Summary and Remarks.

The disease group melancholia has a somewhat precarious footing in present-day psychiatry. The purest cases appear to occur in or about the climacteric era. The practical alienist is always reluctant to place a case in the group melancholia, lest it shortly transpire that the case should rather have been counted senile, or arteriosclerotic, or, in some other way, frankly organic. Moreover, it frequently appears that error may creep in the opposite direction and the patient, alleged to have melancholia, turn out to be actually a victim of some constitutional defect or of some acute psychosis.

We have found difficulty in resolving our ideas about the disease group melancholia and were for a time inclined to believe that any given case could perhaps be transmuted, as a matter of diagnosis, into some other group.

As a beginning in this group, we have here introduced three cases which, whether they fit any acknowledged grouping or not, appear to have certain features in common as well as certain instructive differences. The psychological color and, to some extent, the course of these cases recall some features of Cotard's syndrome.

In every case there were a few established instances of insanity in the direct line on one or other side. The hereditary features, interesting in each case, appear to have little in com-

mon, except the terminal continuous depressions in the mothers of two patients (cases II and III).

The lives of the three patients showed little which can be regarded as underlying their ultimate conditions. Case I used no alcohol, Case II drank beer with meals, Case III used alcohol moderately. Venereal history practically negative in all cases.

Occupations: clerk, I; mason, II; shoemaker, III.

Previous diseases: lumbago, I; pneumonia seven years before commitment, II; scarlet fever and purulent otitis media, æt. 3, and typhoid fever and la grippe with delirium, æt. 61, III.

Ages at onset: 48, I; (69) 75, II; 65, III.

Durations: 8 months, I; (8 years) 2 weeks, II; 24 ca. weeks, III.

Onset: gradual, I, III; sudden, II.

Assignable causes: financial worry, I; senility and domestic worry; nothing, æt. 65, III.

Physical conditions: arteriosclerosis in all cases; case III showed emaciation, nephritis, cystitis, increase of reflexes.

Mental states: ideas of negation in all cases, developing in case I after slowly increasing depression and agitation with delusions about self and family and questionable hallucinations; in case II, after gradual senile failure, as sudden deep depression with agitation, delusions of ruin of self and family, and suicidal attempt; and in case III, after slowly developing hypochondriacal depression.

The feelings of unreality and ideas of negation presented a certain variety in the three cases. Case I showed an alteration of personality, uttered frequently in such phrases as "I'm all gone, I'm dead to the world. I'm not G. E. H. I've made myself as Gibbs; I've been here as G. E. H." Case II showed a feeling of unreality, both as regards the outer world ("sunlight not real, sky not blue, people not real human beings") and as regards himself ("I can't move; I can't die"). Case III showed an alteration of point of view as to the outer world ("you're all great big men, and so strong; you must weigh over 400 pounds") and nihilistic ideas ("I am so small you can't see me; I've got no brains; and I can't talk; I've got no heart, and no stomach, and I can't swallow; I wouldn't burn if you throw me in the burning furnace").

The anatomical side of these cases presents several common

aspects, but little which promises to explain the disease. Arteriosclerosis, when confined to the large branches of the circle of Willis, can scarcely be invoked as underlying symptoms of such specialized character as those under consideration.

Just as the patients showed strikingly little in alterations of reflexes (increase in case III), so the brains showed strikingly little in the shape of gross or focal alterations (small old cyst of softening in case II and mild chronic exudative process in case III).

Moreover, the brains gave little evidence of general or focal atrophy. No striking alterations in cortical topography and arrangement of layers could be detected on microscopic examination. Pigment-bearing cells in perivascular spaces were constantly found; and, in default of any suspicious localization of these, we must attribute them rather to the results of advancing years than to a special factor.

Neuroglia cell pigmentation was also quite constantly found; but this was not so universal in distribution as was the case with the perivascular cell pigmentation. Common to all three cases was a neuroglia cell pigmentation in the intermediate layers of the areas of cortex examined. The relation of the neuroglia cell pigmentation to cortical activity could not be made out. Satellite-cell pigmentation was not constant.

Nerve cell pigmentation was constantly found in the elements of moderate size in all parts of the cortex examined. This pigmentation was strikingly brought out by the use of iron hematoxylin. The pigmentation in question has a somewhat characteristic locus in the affected cells, fails to destroy their contours, and lies apparently in interstices in the cells. This interstitial nerve cell pigmentation, as brought out by iron hematoxylin, is to be sharply distinguished from the familiar yellow sack pigmentation of the major elements.

Pending the increase of knowledge concerning the pigments and fat-like deposits in general, we can at least investigate their occurrence topographically. It seems to us that, however fragmentary the present findings are and however far we may be from bringing such findings into relation with disorders of apperception, we have at least a promising field for investigating the conditions of what seems to be a truly cortical disorder. We are at present at work upon accessible cases of melancholia.

SPINAL CORD TUMOR AND TRAUMA; A REPORT OF TWO CASES[1]

By Pearce Bailey, M.D.,

of New York.

In the two cases which I have to report, a severe and local injury to the back was followed by the development of symptoms of intraspinal tumor—a diagnosis demonstrated at operation. The injury did not, in either case, directly damage the spinal cord, its roots or its membranes—at least, there were no symptoms of such damage; and in neither case did the tumor involve the bone. The pain of the original injury merged gradually into the pain characteristic of nerve irritation—and this within a reasonable length of time. So the general relationship of cause and effect seemed close. To determine the details of such relationship is a far more difficult matter. When, as in these two cases, the cord and its envelopes escape all initial injury and the bones at no time share in the new growth, the modus nascendi of the tumor is largely speculative. For we do not know how the trauma acted or on what tissues. When a blow to a bone is followed in a short time by a sarcoma of the bone, or when a laceration or contusion of a membrane is followed by disordered new growth, we can readily explain or at least understand how the new growth came about; and with equal readiness whether we hold to the classic Cohnheim doctrine or the one that lays tumors at the door of parasites. But when the injury consists of a general contusion or sprain and the tumor occurs in parts supposed to be protected from such violence and which, moreover, gave no symptoms at the time of the injury, we are thrown back on guesswork explanations. We can assume some microscopic lesions of membrane, too slight to cause a loss of spinal cord function; or the existence of congenital tumor masses, which possibly might be impelled to growth by a very slight general commotion or concussion. But we remain in the land of speculation.

[1] Being part of the introduction to the discussion as to the relation of injury to diseases of the spinal cord, held at a meeting of the New York Neurological Society on December 3, 1907.

Both the cases I have to report were in the lumbo-sacral region, a region, I need hardly remind you, in which congenital tumors are most frequently found.

Case I. A woman, M. B., 32, married, no children, was always healthy until 12 years ago: then she was injured in a bicycle collision. While riding very fast, she collided with a man coming also very fast from a side street. She was thrown and both machines were thrown on top of her, the frame of one of them striking her in the lower part of the back. She was not bruised and the skin was not lacerated, but the pain, which was immediate, was terrific and increased by every movement. She nevertheless rode home on her wheel (a distance of two miles), though she had some difficulty in getting there. She had to be helped upstairs. She was confined to the house for three weeks, although she did not stay in bed all of the time. There were no symptoms pointing to spinal cord injury. Paralysis, sphincter trouble, anesthesia, were all absent. But the back was very sore to the touch and was particularly painful on movement.

After three weeks the pain began to let up a bit. Then she soon became able to ride her wheel again, some days as far as forty to fifty miles. From now on she was free from pain in the daytime, but, on lying down at night, the pain began and interfered seriously with her sleep. This went on for four years or so. Then she was married. The pain continued severe at night, and she says she has never had any really good nights since the accident. She took sleeping medicines but formed no habit, although she narrowly escaped it. After an operation (ovariotomy) five years ago, she began to have pain in the daytime as well as at night.

From the first the pain has always been in the same place, namely, in the region of the third lumbar vertebra, to the left of the median line. It was dull, aching, heavy. She had the feeling as though, if she could get her hand on the pain, she could pull it out. In May, 1907, she slipped and fell. This seemed to give the pain a fresh start. It took on all the frightful characteristics of the pain of spinal cord tumors. At the same time there began difficulties in walking, which became more and more pronounced through the summer.

I saw her first in early October, in the service of Dr. Hollis at St. Luke's Hospital. Aside from the spinal symptoms, physical examination was negative. The back was held stiff, any movements of it increasing the pain. There was marked pain on pressure over the third lumbar spine, and to the left of the median line for about two inches. In this latter situation, also, there was an increased resistance of the muscles in palpation. There was no pronounced anesthesia—certainly not enough to chart—although there was some vague blunting of sensibility in

the legs. The legs were weak, but all movements could be carried out. But there was marked ataxia of the legs and the Romberg symptom was pronounced. The left knee-jerk was absent, and the right much reduced. There was no involvement of bladder or rectum.

In the next few weeks the symptoms grew worse. The patient complained so bitterly and sincerely of the pain that that symptom alone would have justified the diagnosis of tumor. But, in addition, the ataxia of the lower limbs changed into a paraparesis, so that the patient could neither walk nor get in or out of bed unassisted.

Without any further symptoms, operation was advised and was performed by Dr. B. Farquhar Curtis on October 25. A soft, friable, non-encapsulated tumor, about the size of the little finger, was found beneath the first, second and third lumbar vertebræ, on the left side of the median line. The tumor was lying in the canal, having pushed the cauda equina to one side and had no firm attachments. Its exact nature caused considerable discussion among the histologists, though it was roughly classified as a fibro-sarcoma. It had all the characteristics of a slow-growing tumor.

Case II. A boy of thirteen was injured while coasting, by some one jumping on the small of his back. After this accident, he was ill in bed for a time. He suffered from pain in the back, which was greatly increased on movement. After two or three weeks, he returned to school. But he continued to have stiffness and some pain in the back. All muscular movements involving the back, and especially all jars, made the pain worse, so that the boy was shut off from active exercise. When quiet, however, he was free from pain. But, two years after the accident, a new pain set in. This consisted of a sharp, lancinating pain, which occurred independently of movement, and which, starting in the lower part of the back, shot down into the lower extremities. The area of this pain was the posterior surface of the right leg as far as the knee, and on the left side, throughout the distribution of the sciatic nerve. This pain became so severe as to be disabling, and the patient was acquiring the morphine habit.

Dr. C. N. Dowd, before referring the patient to me, had operated on the coccyx, which was found broken and tilted forward at a right angle with the sacrum. Dr. Dowd reported the case in the *Annals of Surgery*, Vol. 42, p. 279.

My examination was made in June, 1904. The young man had evidently sustained a considerable injury of the lower lumbar vertebræ and of the sacrum. The whole lower end of the spine was held stiff and, in addition, there was a lumbar lordosis and dorsal kyphosis. The patient could not bend the back to touch the floor. In carrying out this movement, the back was held

rigid, the knees only bending. This deformity was considered as being entirely due to bone injury.

The muscles of the whole lower extremities, but especially of the gluteal region, were atrophied—and the buttocks and thighs were constantly agitated by fibrillary twitchings. The legs were weak throughout, although the patient could execute all movements with them. When seated, however, he was unable to extend the legs, as at the beginning of the effort of extension, the flexors of the legs would be thrown into contraction, preventing extension. There was no anesthesia and no sphincter trouble. The knee-jerks were lively. These were the only symptoms. But the characteristic pain, coupled with the atrophy and fibrillation, was considered sufficient justification for operation, which I advised and which Dr. Dowd performed in two stages.

The first operation was on June 4, 1904. The opening was made at the site of the first sacral vertebra, a portion of the lamina of the last lumbar being removed. A tumor was found within the dura. Fearing that this was a glioma, and that the fibers of the cauda were too closely incorporated with it to permit safe removal, only a portion of the tumor was cut away for examination. The patient recovered promptly. His symptoms were very much relieved, and he passed several fairly comfortable months. The pathological report was given with reserve, fibroma being the most probable diagnosis.

In February, 1905, the patient again began to complain of the night pains, and on March 1, Dr. Dowd removed the tumor of the spine. The laminæ and spinous process of the fifth lumbar vertebra, most of those of the fourth, and part of the spinous process of the third, were removed. The posterior portion of the canal, corresponding to the first, and part of the second sacral, was also removed. The tumor was about two inches long, and one inch in diameter, and was adherent to the dura. The pathological examination showed that the tumor was an endothelioma, arising from the lymph spaces of the pia mater.

Reëxamined November 20, 1907. Now, the patient has no spontaneous pain whatsoever and has had none since recovering from the operation. He never has pain in the legs, but feels pain in the back when he receives jars. There is still some deformity of the spine and great stiffness of the lower part of the spine. The muscles of the legs have resumed practically a normal volume, although there are still fibrillary twitchings in the glutei and large muscles of the thighs. Sensory anomalies are absent. Never any bladder disturbances. Knee-jerks are now normal.

The chief results to-day are those due to the injuries of the bones of the spine. The patient gets about easily, is free from pain and is earning his living.

In closing, I want to emphasize the necessity of painstaking surgical examination of the back in all cases of injury. We should use the X-ray freely, especially in the cervical and lumbar regions. X-ray plates, in these parts, show structure with remarkable fidelity. At the same time it must not be forgotten that hysterical palsies may co-exist with broken bones.

The whole subject of trauma and spinal cord disease requires great conservatism and moderation in its discussion. Any one familiar with the confusion cast over these injuries by the writings of Erichsen, which exists even to-day, must feel the necessity of caution in giving expression to ill-founded views. For the subject has more than a medical importance. I hesitated a long time before making public these two tumor cases, for, in every case of litigated backache, I can already hear the question, launched at the witness with a smile of triumph toward the expectant twelve—"Doctor, may not this back-sprain turn into a tumor of the spinal cord?"

Society Proceedings

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The President, Dr. Chares L. Dana in the Chair

SIGNIFICANCE OF THE OCULAR SIGNS AND SYMPTOMS OF DEMENTIA PRÆCOX

By H. H. Tyson, M.D., and L. Pierce Clark, M.D.

This preliminary report covered a period of study of over 100 cases from private practice, clinics, and the metropolitan asylums. Definite changes were said to be invariably found. The fundus changes, as seen clinically, were divided into three stages in the order of their occurrence: First stage, congestion of the discs, hyperemia and edema, dilated veins, contracted arteries and blurring of the edges of the discs, all of varying degrees, constituting a low grade perineuritis of the optic nerve. Second stage, congestion of the nasal side with temporal pallor of discs, dilated veins and contracted arteries. Third stage, pallor of discs, dilated veins and contracted arteries, constituting anemia and partial or complete atrophy of the optic nerve.

All forms of demenetia præcox were under study. The more rapidly deteriorating forms showed the most marked changes. The cases embraced both users of alcohol and tobacco, and abstainers. Theoretically, the changes were probably due to a vascular toxin from liver or intestinal auto-infective origin. A vascular alteration with edema took place, resulting in disturbances of nutrition and slow degeneration of the nerve fibers. Cases in the first stage had shown fundus improvement under the usual treatment for intestinal auto-infective toxemias. Other eye symptoms universally found were enlarged pupils, negative sensory reflex, negative psychic reflex, negative Piltz reflex, diminished corneal sensibility and concentrically contracted visual fields. No other psychosis presented similar conditions. The findings should be found useful in differential diagnosis, to a certain extent in prognosis, and possibly in the study of the pathogenesis and pathology of dementia præcox.

Dr. Carl Koller said that years ago he had seen many cases of dementia præcox but had not examined them ophthalmoscopically. Lately he had examined such a case which presented a very striking ophthalmoscope picture, which however was very different from what Dr. Clark had described. The patient was a man of 31 years of age, had shown the symptoms of dementia for four or five years, and had been subjected to the usual treatment. About six weeks ago he contracted malaria and three weeks later began to complain of failing vision. Both optic discs were slightly edematous, the remainder of the retina being entirely clear. There were numerous hemorrhages in the background, involving both

artries and veins, big and small alike. In the macula of the right eye there was a geometrically regular diamond-shaped patch, slightly raised, yellow in color, with very sharp outlines; it was hemorrhagic. But for this latter character it resembled the choroidal patches which are so common in the secondary stage of syphilis and whose seat is the anterior (mostly lower) segment of the eye. The anatomical locality concerned was evidently the choriocapillaris, and the shape of the patch bore most likely some relation to the distribution of the smaller arteries in that layer. The appearance of the disc also mostly resembled the low degree of neuritis found so very frequently (almost he might say constantly) in the secondary stage of syphilis. In the left eye the conditions were identical; only the diamond-shaped patch was very much smaller. In the right eye it was at least of the size of the disc, in the left its diameters were one quarter the size. The speaker said he was mostly inclined to consider this a syphilitic lesion.

Postscript: After three weeks of treatment of which injections with salicylate of mercury formed a part all these changes cleared up completely, leaving only vestiges of some granulation at the site of the patches. The vision which had been less than one fourth rose to normal in the left eye, and to three fourths in the right.

Dr Edgar S. Thomson said the capillary circulation of the normal nerve varied to such an extent that it was very difficult under certain conditions to say whether we had an atrophy or a hyperemia unless the condition was very marked. The speaker said that in the two cases shown by Dr. Clark he would be unwilling to say that a hyperemia was present now. The present appearance of the disc in these cases did not appear to him to be especially striking. In any case where we had a condition of the optic nerve which was suspicious of either hyperemia or atrophy, the central vision and the visual field should be carefully measured.

Dr. George W. Jacoby said that in addition to the ocular symptoms and the neuroretinitis, the important points were the analgesia of the cornea and the other symptoms mentioned. In this general connection the question arose whether we were willing to accept dementia præcox as an absolute clinical entity? If so, then these symptoms would be indicative of an organic brain affection rather than of a toxemic state.

Dr. H. H. Tyson said that only high degrees of errors of refraction proved an appearance of slight optic neuritis and that among 109 cases examined, only about two per cent. showed high refractive errors. In many of the cases vision was slightly diminished, but in all there were marked limitations of the visual field. In one or two instances the atrophy of the optic nerve was very marked; in other cases the pallor might have been termed incipient atrophy. As regards an absolute diagnosis of atrophy of the optic nerve that could only be made by the microscope which of course was impossible under the circumstances. The clinical diagnosis of atrophy was based upon the recognized signs of pallor of the nerve, contracted arteries, some diminution in visual acuity and contracted visual fields.

In their series of cases they found only one with hemorrhages on and about the disk and from Dr. Koller's description of this case it so much resembles the one that Dr. Tyson saw, that he would not be surprised to discover that it was one and the same. But he did not consider the spot at the macula pathognomonic by any means. In other respects the case is quite representative of the congestive type.

In regard to the point made by Dr. Thompson about the visual acuity, he did not consider it in itself a reliable test at all. As we know, one may find normal vision in eyes showing marked fundus changes such as choked disk, etc. In making a diagnosis the entire eye symptom complex viz. anesthesia of cornea, contracted visual fields, enlarged pupil, negative psychic, sensory, and Piltz pupillary reflexes and the appearance of the disk described should all be considered and not merely the fundus alone.

A FAMILY TYPE OF COMBINED SCLEROSIS ASSOCIATED WITH GRAVE ANEMIA

By Charles L. Dana, M.D.

Dr. Dana had recently had under his care a patient in whom it might be said that pernicious anemia and combined sclerosis was a family disease. The patient was an unmarried woman, 58 years old, coming from old New England and Pennsylvania stock. She had lived a temperate and fairly healthy life, though subject rather easily to diarrheas, and having at times what Dr. Delafield termed "morning diarrhea." Very early in life she had a severe attack of dysentery, but had had no subsequent attack until eight years ago. She had other attacks seven, six, five and four years ago, but none since. Besides the dysentery, within the past eight years, she had had a distinct tendency to mild attacks of enterocolitis, and two years ago she was treated for a long time for this trouble, during which period she passed a great deal of undigested food and gas in her stools. Her physician detected an ulcer in the sigmoid flexure. She recovered from this, however and had been fairly well for nearly a year. Within the past six months she had noticed a peculiar numbness of the feet, with weakness and unsteadiness in walking, and a similar condition in the hands. When Dr. Dana first saw her, she had a decided pallor, with an almost yellowish tinge to the skin. She was rather thin, but not much more so than her ordinary habit. Subjectively, she felt fairly well excepting for the weakness and uncomfortable numbness of the extremities. She had an ataxic gait, and the Brauch-Romberg symptom. There was also some ataxia of the hands, so that she could not sew or do fine work with them, but she was able to write and dress herself. There was some general weakness of the extremities, but no distinct paralysis. The knee jerks were present, but there was no ankle clonus and no Babinski. While she complained much of the paresthesia, she had no actual cutaneous anesthesia. Examination of the patient's blood showed 3,200,000 red cells; 8,800 white cells and 66 per cent. hemoglobin. She had a curious and persistent inflammatory condition of the gums, which kept her mouth rather sore. Cultures made of this region by Dr. F. M. Huntoon showed the ordinary microörganisms of the mouth, excepting that there were certain spirillæ. An examination of the stools made by Dr. Christian A. Herter showed indications of excessive putrefaction, with an abundance of the bacillus aerogenes capsulatus. There was an excess of hydro-bilirubin and skatol, with absence of indol. A second examination of the blood, made three weeks later, showed a further decrease of the red cells to 2,780,000, with a similar condition of corpuscles, excepting that there were no megaloblasts. The hemoglobin was reduced to 58 per cent.

In this case, Dr. Dana said, the patient presented a very clear-cut type of grave anemia, with combined sclerosis. As to the family history the patient stated that her mother had also suffered from "morning diarrhea," and had a tendency to weakness of the bowels. Her father died of apoplexy. One brother died at the age of 52 from a trouble similar to her own, namely, he had a great deal of diarrhea followed by sensory disturbances, anemia, and weakness of the extremities. One sister died in the same way, and one maternal grandmother had a similar trouble. The disease, apparently, ran through three generations. In the second generation it produced only a moderate disturbance. In the third generation, out of four children, three had been affected. The case might be classed as a family form of pernicious anemia, or a family form of combined sclerosis, or, more properly, it was a family form of enteric weakness which secondarily led first to an anemia, and then to a spinal cord disorder. There were some authorities, however, who still asserted that the enteric trouble was not a primary one, but was secondary.

Dr. Joseph Fraenkel said that in connection with the subject presented he recalled a case which he had for some time under his observation at the Montefiore Home. It concerned a young man of about 25 years, who, while in South America had contracted an obstinate form of entero-colitis, which resisted treatment for quite some time. At the time of his admission to the Montefiore Home, he presented undoubted evidences of an organic disease of the spinal cord which fitted easiest a conception of a combined degeneration—exaggerated reflexes and hypotonia were interpreted in this sense. During the acute recrudescences of his colitic trouble, which were observed at the Montefiore Home at intervals of a few months, the spinal cord symptoms showed considerable amelioration. At the moment the speaker did not recollect whether there were any blood examination made, and if so, what the findings were.

Dr Abrahamson said he could conclude the history of the case mentioned by Dr. Fraenkel by stating that the patient finally died of tuberculosis. Among the later spinal symptoms he exhibited was analgesia of the toes. He had seen the patient at the German Hospital, where the case was regarded as one of amebic dysentery.

Dr. B. Sachs said he had never observed any of the family type of these cases of combined sclerosis, but the supposition thrown out by Dr. Dana that the condition was of gastro-enteric origin was very interesting. The speaker recalled the case of an extremely hysterical woman who for many years invariably vomited shortly after each meal. About two years ago, after a trip to Europe in a vain search for health, she returned in a much worse condition than before so far as her general nutrition was concerned, and developed marked spinal symptoms, with radiating pains in the upper and lower extremities, with more or less flaccid atrophy, and some slight involvement of the bladder. Blood examinations were made by two different men, and they could not agree whether the case was one of pernicious anemia or not. It was undoubtedly a case of grave anemia. The patient became weaker and weaker and finally died without any change in her paralytic condition. The upper extremities became almost completely paralyzed, while the power of the lower extremities was much impaired, and there was great pain, particularly in the arms. The speaker said he had regarded the case as one of spinal origin, due primarily to grave anemia.

Dr. Smith Ely Jelliffe, in discussing the fact that certain spirillæ were

found in the cultures taken from the inflamed gums in the case reported by Dr. Dana, called attention to the fact that general spirillosis had recently been regarded as a cause of pernicious anemia.

Dr. Jacoby referred to the possible relationship of these cases to those in which there was a complete absence of hydrochloric acid, and in which a pernicious anemia developed. In the absence of gastric juice and hydrochloric acid, any pathogenic organisms that might be present would have a free field for action, and from that point of view we should attribute some importance to the presence of achylia gastrica in connection with grave anemia. This theory would perhaps tend to corroborate the gastro-intestinal etiology of the disease.

Dr. Dana, in closing, said that pernicious anemia could be associated with extreme atony of the gastric wall and achylia gastrica, and also with a similar condition of the intestinal walls. In the latter condition, the food was not absorbed, and passed through almost unchanged. Combined sclerosis, the speaker said, had been observed without the presence of gastric symptoms, and the toxemia in this spinal disease was usually of intestinal rather than gastric origin. The pernicious anemia that occurred in association with the gastric cases was in his experience less often associated with cord symptoms. Some authorities did not agree with Herter that skatol was a dominant factor in pernicious anemia.

THE RELATION OF TRAUMA TO ORGANIC DISEASES OF THE SPINAL CORD. SPINAL CORD TUMORS

By Pearce Bailey, M.D.

The author stated that the ultimate effects of injury to the spinal cord could be divided into two classes: a first class, in which the trauma was followed, sooner or later, by a chronic progressive degeneration of the spinal cord elements, identical in symptoms and course with the same degeneration when it occurred when no injury had been received. The classical and much discussed type of this class was tabes; other types of this class which seemed to challenge less acrimonious discussion were progressive muscular atrophy, multiple sclerosis, ataxic paraplegia, syringomyelia, and, in fact, all the many chronic degenerative diseases. Any of these diseases might be inaugurated by injury or made worse by it, but for none of them was an exclusive traumatic origin demonstrable. Opinions for or against the view might be justifiable, but when all was said and done they were nothing but opinions, and the wisest might be in error.

Of the group mentioned, locomotor ataxia was the disease in which a traumatic origin was most improbable, and the speaker said he saw no means by which such an origin could be proved for it. Multiple sclerosis and progressive muscular atrophy, on the other hand, in which the onset was less insidious and the early symptoms more unmistakable, and which lacked the great underlying cause of tabes, lent themselves more easily to the assumption of trauma as a point of departure. Syringomyelia, also, from its close analogies to hematomyelia, offered a still more profitable field of study from the point of view of trauma. This disease merged toward the second class of diseases he had in mind, namely, those in which an injury was followed, not by a generalized degeneration, but by focal disease. Of course, he did not refer to acute surgical injuries of the

cord, in which the symptoms ensued immediately, but rather to those cases in which, after a definite injury to the back, there appeared, at some later day, distinct evidences of loss of function in the cord at and confined to the level of the external injury. Another variety of this class was that in which a focal cord lesion appeared weeks or months after the injury, and was secondary to vertebral disease. Tuberculosis and sarcoma of the vertebræ were the common examples of this class. Kümmel's disease, or a non-tubercular spondylitis occurring after injury in the dorsal region, was another interesting member of this group. In all these cases care must be taken not to be misled into taking the injury as the cause, when, in reality, the disease preëxisted and was of independent causation.

In two cases which Dr. Bailey reported in detail a severe and local injury to the back was followed by the development of symptoms of intraspinal tumor, a diagnosis confirmed at operation. The injury did not, in either case, directly damage the spinal cord, its roots or its membranes; at least, there were no symptoms of such damage, and in neither case did the tumor involve the bone. The pain of the original injury gradually merged into the pain characteristic of nerve irritation, and this within a reasonable length of time, so the general relationship of cause and effect seemed close. In both cases the injury was in the lumbo-sacral region.

In closing, the speaker emphasized the necessity of painstaking surgical examination of the back in all cases of injury. We should use the X-rays more, especially in the cervical and lumbar region, but at the same time, too much weight should not be placed upon them. The whole subject required great conservatism and moderation in its discussion.

Dr. M. Allen Starr said that a very careful review and tabulation of his private histories had emphasized in his mind the great difficulty of postulating any one factor as a cause in the production of organic nervous disease. There were patients who gave a distinct history of trauma of the back followed by an interval of comparatively good health and then the development of various signs of an organic nervous affection, and while we might believe that the trauma was an etiological factor in the case, still it was only suppositional and incapable of absolute proof. Yet there were other cases that seemed to prove conclusively the causal factor of the trauma. Of 154 cases of locomotor ataxia seen in private practice which he had recently reviewed, he had found only three in which the development of the symptoms followed immediately upon the occurrence of severe injuries to the back, and in those three instances there was absolutely no history of syphilis. The speaker said that as he did not believe that locomotor ataxia was always of syphilitic origin, he always took pains to inquire as to whether there was any history of trauma as a possible etiological factor. One of the cases he had in mind was that of a young man who slipped on an icy porch, falling down the entire length of the stairway of fourteen steps, and striking his back severely a number of times in his descent. He was brought back into the house, complaining of intense pain in his back, which confined him to the house for about two weeks. Subsequent to the fall, on walking, he complained of pain all over his trunk and legs, with a sense of tingling and prickling, and more or less irritability of the bladder. There were no pains in the arms. At the end of a month, when Dr. Starr first saw him, the knee jerks were absent, Romberg's sign could be elicited, and he complained of lightning-like pains. In the course of two months he gradu-

ally developed an ataxia, and he now had all the typical symptoms of locomotor ataxia. In that case there was no history of syphilis, and all his symptoms developed immediately after his fall, which was a pretty severe one. In another case the injury to the spine was due to a railway accident, while in the third the patient was thrown off a car. Neither of these two were litigation cases. In both the symptoms of locomotor ataxia developed within two months of the time of the accident. Both developed into typical tabes cases.

Dr. Starr said that a search of his records revealed only two cases of tumor of the spinal cord in which there was any probability that an injury had anything to do with the development of the tumor; these two had been selected from the records of 27 cases. One case was that of a woman who was afterwards operated on by Dr. A. J. McCosh. The history she gave was that she had sat down very hard on the floor, buising both buttocks severely. Her pain dated from that time, and developed into a tumor of the cauda equina, which proved to be sarcomatous. She failed to recover after the operation on account of the extensive involvement of the bones.

The second case of spinal tumor reported by Dr. Starr was one which he saw in consultation in Cleveland. The patient was a young man, who, while playing football, was injured by another player who came up behind him, planting his knee against the spine directly between the scapulæ. The injury was followed by intense pain in the back, and when Dr. Starr saw him, four months later, he had well-developed symptoms of a spinal tumor, the presence of which was subsequently verified by the X-ray. The tumor was very large, and an operation for its removal was deemed inadvisable.

Dr. Starr said he could recall other cases where injuries to the back were followed by hemorrhages within the membranes. In four such cases, which were originally diagnosed as cases of rupture of the cord, there was an immediate and fairly complete paraplegia, but the subsequent history of the cases showed that the cord could not have been injured, and that there must have been a hemorrhage about the cord, which was gradually absorbed.

In closing, Dr. Starr said it was very difficult to be sure that a trauma was really the origin of the development of organic disease of the cord, but in some of the cases he had seen he thought it could be positively stated that it was. He could not recall any case of progressive muscular atrophy or syringomyelia which he could attribute to trauma.

Dr. Joseph Collins said that in his opinion tabes bore no relationship to trauma save that it may bring into active evidence symptoms of the disease that are already present. It is beyond human credibility to believe that the symptoms of tabes, dependent as they are upon definite lesions, which from its very nature is one of gradual development, that develop within a few days or weeks after trauma are indicative of a lesion initiated and caused by such trauma. Expression of individual opinion doesn't count for much, but his was that if syphilis ceased to exist locomotor ataxia would not occur. Individual cases like those reported by Dr. Starr did not negative this view. It does not follow that because the patient denies having syphilis and because there are no evidences of its existence or because the patient is of such a social stratum that it seems inexpedient to discuss the question, that the infection has not existed. If in such cases as those reported by Dr. Starr a lumbar puncture had

been done and no lymphocytosis found, then the speaker said he would be willing to accept the statement that there was no syphilis, but the absence of the somatic evidences of syphilis and the patient's statement to that effect did not prove that he was free from that disease.

Dr. Collins said he had never seen a case of spinal progressive muscular atrophy that was legitimately attributed to injury alone, and the same was true of disseminated sclerosis. On the other hand, he believed that such diseases as syringomyelia and hematomyelia and various forms of myelitis and meningomyelitis might follow injury, but apart from them it was his conviction that no disease of the cord had any definite relation to trauma.

As regarded the occurrence of spinal tumors, the speaker thought that trauma was both a predisposing and exciting cause. Statistics went to show that trauma was an incidental factor in the majority of these cases, but it should not be looked upon as the final cause.

Dr. Graeme M. Hammond said he could not indorse the views expressed by Drs. Starr and Collins about the relationship of trauma to progressive muscular atrophy. He recalled three or four cases of that disease in which he felt sure that it was very closely related to trauma. One of these cases, which was also seen by Dr. Joseph Fraenkel, was a boy of nineteen years who received a blow with the fist in the cervico-dorsal region, knocking him down. Twelve days later he developed his first symptoms of progressive muscular atrophy, which rapidly advanced to quite a profound degree. In the other cases related by Dr. Hammond the relationship between the trauma and the occurrence of the symptoms of progressive muscular atrophy was equally close.

Dr. Starr, in reply to Dr. Hammond, said he had not made the assertion that he did not believe there was any relation between trauma and progressive muscular atrophy; only, he had never seen it.

Dr. B. Sachs said that while he was probably as strong a believer in the specific origin of tabes as was Dr. Collins, he saw no reason why we should regard it as the exclusive cause. While it was the most important cause of tabes, it was not necessarily the sole cause. Personally, he had seen but a single case of tabes in which he really believed that trauma was the cause, and if we had but one positive case of that character, it was worth any number of negative ones. The patient was a young man who who was the victim of a bomb explosion, receiving a violent blow in the back. Within a few weeks he developed characteristic shooting pains, and a short time afterwards there were bladder symptoms, difficulty in gait, Argyll-Robertson pupil and loss of knee jerks. There was not the slightest evidence of syphilitic infection in the history or condition of the patient.

Dr. Sachs said he did not quite agree with Dr. Collins that a lymphocytosis could always be regarded as evidence of a specific etiology in tabes; it might be satisfactory evidence in a certain number of cases, but could not be depended upon. As regarded the influence of trauma upon the development of tumors, that question was a very difficult one to decide. We were all under the impression that in such cases, as a rule, the trauma simply constituted a landmark, and that there might be a remote connection between the two. In hematomyelia, he was ready to concede the importance of traumatism. The speaker said he had never been able to satisfy himself that progressive muscular atrophy was of traumatic origin in any of the cases that had come under his observation,

and in the cases reported by Dr. Hammond he suggested that possibly a traumatic meningeal hemorrhage had given rise to symptoms which in the beginning closely simulated progressive muscular atrophy.

Dr. William M. Leszynsky said that several years ago he saw a traveling salesman who up to a week prior to that time had claimed to be in perfect health. He then fell and struck his back. Several days later there was complete paraplegia, with retention of urine. At this time, both knee jerks were present, but disappeared soon after. He recovered from the paraplegia within four weeks, and the symptoms gradually developed into a typical case of tabes. After a number of months there was some improvement in the ataxia. Since then the case had been lost sight of.

Dr. Starr, in reply to a question, said that the Argyll-Robertson pupil was one of the later symptoms to develop in the cases of tabes of supposed traumatic origin that he had reported.

Dr. Joseph Fraenkel said the discussion of this subject was particularly interesting on account of the apparently diametrically opposed views that had been expressed. While trauma, as the etiological factor in a case of hematomyelia, with subsequent development into syringomyelia, could be readily understood, it would be difficult to explain the pathogenic connection between traumatism and the purely degenerative forms of disease of the central nervous system, *e. g.*, tabes. In fact, such an explanation could only be accepted with considerable violence to our present pathologic conceptions. Of course we knew that some cases of tabes show an acute onset, but even that fact failed to admit trauma as an exclusive etiologic factor. The speaker said he could not agree with Dr. Hammond that the case of progressive muscular atrophy referred to by him was of traumatic origin.

Dr. Abrahamson mentioned the case of a boy who forty-eight hours after an injury to the back developed a total paraplegia. Examination showed a fluctuating mass about the level of the sixth dorsal vertebra, and below this the boy was totally paralyzed. Upon lumbar puncture, pure pus was withdrawn. Careful questioning elicited the fact that the boy had complained of pain in the right nipple region for some time prior to his injury, and further investigation proved that the case was one of old-standing osteomyelitis communicating with the spine which the injury to the back brought to an acute culmination.

The President, Dr. Dana, said he though we should take a most conservative attitude towards the relation of trauma to diseases of the spinal cord except as "exciting" causes. His personal convictions in that direction were very strong, and he recounted one experience which seemed to him extremely illuminating in that direction. The case he had in mind was that of a woman, about fifty, the mother of healthy children, who had been comparatively well all her life. One day, while walking along the street, she sprained an ankle and fell. She was under treatment for this injury for two or three weeks, and when she left her bed she found that she could not walk. Upon investigation it was found that she had developed a perfectly typical locomotor ataxia. The knee jerks were absent, she had the Argyll-Robertson pupils and other symptoms which left no doubt as to the diagnosis. There was no history obtained of syphilis. If this woman had sustained an injury to the spine instead of the ankle, and the case had been brought into litigation, it would probably be looked upon as one of locomotor ataxia following injury. As a matter of fact,

her injury had nothing to do with her spinal symptoms. Possibly Dr. Dana said, trauma might in exceptional cases be the cause of tabes, but if so, he had never seen an example of it.

THE PHILADELPHIA NEUROLOGICAL SOCIETY

December 23, 1907

The President, Dr. Alfred Gordon, in the Chair

STEREOGNOSIS IN LESION OF LEFT PARIETAL LOBE

By Alfred Gordon, M.D.

The patient, a man of 56, while in an attack of acute melancholia, shot himself, the bullet penetrating the right temple. It was arrested beneath the left parietal lobe and was removed by operation. The surgeon had to destroy a large portion of the superior parietal lobe. Before as well as after operation repeated examinations showed no change in the stereognostic function or in the general sensorium. The present case argues against the absolute view of parietal localization accepted by the majority of observers.

SEGMENTAL ASTEREOGNOSIS IN PARIETAL LESIONS

By W. B. Cadwalader, M.D.

Segmental astereognosis resulting from partial lesions of the parietal cortical area was first reported by Drs. Mills and Weisenburg several years ago. The present case is a similar one, and is that of a colored boy of 22, who two years ago was injured on the right side of his head, this producing unconsciousness. When he regained his senses he was paralyzed in the whole left side, with considerable sensory disturbance. The weakness on this side has almost totally disappeared but there still remains increased reflexes with Babinski phenomenon. At one time the patient was unable to recognize any object placed in his left hand, but the stereognostic function is gradually returning, especially in the thumb and forefinger. At the present time he is unable to recognize objects placed between the three ulnar fingers, but he can do so with the thumb and forefinger, but not so well as with the corresponding fingers of the right hand. There is besides ataxia in the finger-to-nose test which becomes more marked as the ulnar fingers are used. There is also disturbance of the sense of position, of movement and of localization in the whole upper limb, this being more marked on the ulnar side. Touch and pain are also involved in this upper limb, more over the ulnar side of the hand and arm. The wound is over the right parietal area.

Dr. C. K. Mills thought, as Dr. Gordon reported, that the man had no astereognosis, unless he had what he (Dr. Mills) had not been able to demonstrate, some little diminution of the various forms of sensation. Dr. Mills did not think, in the light of the numerous cases reported, that this case could be regarded as at all conclusive against the position of the so-called stereognostic center in some part of the superior parietal lobule.

He did not know exactly what had been destroyed, but from the position of the wound he thought it probable that the most destruction was at the junction of the superior and inferior parietal lobules and the occipital convolutions which border on them. Probably the explanation of the case, if the present theories of localization are correct, is that there is sufficient of the superior parietal convolution and the quadrate lobule left to carry out the function of stereognostic conception. The view of Dr. Mills and one advanced by him early in the discussion of this subject is that the stereognostic center is located in the superior parietal lobule or in a portion of it, and also in the superior part of the quadrate lobule or precuneus. The latter is probably intact in this case and also much of the superior parietal convolution. He does not think the case could be used to absolutely controvert the theory of the presentation of the stereognostic function in the superior parietal lobule or quadrate lobule.

Dr. W. G. Spiller said he had reported one of the first cases in this country (JOURNAL OF NERVOUS AND MENTAL DISEASE, Jan., 1899, p. 43) of ataxia produced by a parietal lobe lesion although the case of Starr and McCosh antedated his. Since then he has recorded several cases with the symptom complex of parietal lobe lesion. Similar cases have been reported by others. These symptoms are usually so distinct that it is well to be cautious in denying their importance. The wound in Dr. Gordon's case was evidently at the junction of the parietal and occipital lobes, and therefore we could hardly expect parietal symptoms from a lesion in that region.

Dr. Weisenburg said he was sorry that Dr. Cadwalader's patient was not present for he showed so well where the stereognostic center is located in opposition to the case shown by Dr. Gordon in which it seemed to Dr. Weisenburg that the parietal lobe was not at all involved. In the above discussion on astereognosis it seemed to him that one very important point was not touched upon, and that was that Dr. Gordon in the discussion of his own case talked of astereognosis as if it were an entirely distinct function without involvement of such other senses as those of localization, of pressure, of movement and of position. In Dr. Gordon's case there was no disturbance whatever of sensation, and this fact in itself is proof that the parietal lobe could never have been involved.

Dr. Gordon, in closing, quoted several authorities from his paper, to support his hypothesis. As to the case he presented, Dr. Gordon said that the direction of the bullet was oblique and an incision was made at the level of the parietal lobe. The direction of the bullet was such that it forced the operator to penetrate in the same direction and destroy a large part of the parietal lobe. Dr. Gordon called Dr. DaCosta's attention to the case before the operation and asked him to make a special effort to tell him particularly what lobe he was going to operate on. He said the superior parietal lobe and had to destroy a large portion of the cortical tissue with white matter in endeavoring to extract the bullet.

Even if the patient presents slight symptoms of sensory disturbances, the destruction of the cerebral tissue is entirely out of proportion with the mild symptoms Dr. Mills alludes to as possibly present in this case. Dr. Gordon said he had tested the patient on many occasions for touch, pain and temperature, the slightest touch, the pressure touch, temperature, the extreme degrees of temperature, the mild, and the man could tell every time exactly what was done. In Dr. Gordon's judgment, in spite of the remarks made by the speakers, this patient presents a contradiction to the

localization theory, that is as to the generally adopted view. Then in literature we have recorded cases where astereognosis was found corresponding to lesions of portions of the brain other than the parietal lobe. Dr. Mills himself in an article speaks of the limbic lobe as a possible center for astereognosis.

HEMIANESTHESIA TO PAIN AND TEMPERATURE AND LOSS OF EMOTIONAL EXPRESSION ON THE RIGHT SIDE, WITH ATAXIA OF THE UPPER LIMB ON THE LEFT. THE SYMPTOMS PROBABLY DUE TO A LESION OF THE THALAMUS OR SUPERIOR PEDUNCLES

By Charles K. Mills, M.D.

The following case presents such an unusual combination of symptoms as to make it worthy of being presented to the Society and permanently recorded. It is difficult to fix the site of a single lesion capable of producing a symptom complex which includes heminanesthesia and loss of emotional expression on one side of the body and ataxia of the upper extremity on the other, but it is probable that such lesion is situated in the thalamus of one side, and perhaps extends into the superior peduncles of both sides.

G. S., thirty-four years old, barber, was admitted to the men's nervous wards of the Philadelphia General Hospital, November 29, 1907, complaining chiefly of weakness on the left side of his body and numbness on the right. The patient's mother died at the age of 39 from "typhoid pneumonia." His father is living and well. He has two brothers and one sister who enjoy good health. There is no history of renal, cardiac, tubercular or malignant disease in the family. The patient's history as to nervous or mental disorders is negative. The patient has always been an excessive drinker, principally using whiskey. He averaged about one-half pint of whiskey daily. He has had two attacks of gonorrhea and he had a sore on his penis, but no secondaries.

Ten weeks ago he was suddenly seized with vertigo, accompanied with nausea and vomiting. He was confined to his bed for a week and was under the care of a doctor. When he tried to resume his work as a barber, he found that his left side was somewhat weaker and the left arm more awkward than before his attack. He also found, while cutting a boy's hair, that this arm was tremulous, and he was not so proficient in his trade as heretofore. In addition he experienced a sense of numbness in the entire right side. His friends have noticed that since his illness, when laughing he only smiles with the left half of his face.

Examination of the patient shows that the pupils are moderately dilated, equal and regular. They react promptly to light, accommodation and convergence. Extraocular movements can be well performed. Associated movements are normal. There is no ptosis, nystagmus, exophthalmus or hemianopsia. When asked to look upwards, he does so and his forehead wrinkles equally well on both sides. The nasolabial folds are as well marked on one side as on the other. The palpebral fissures are about the same on the two sides. He can purse his mouth fairly well, as in whistling, and he can move the two sides of his face equally well. In spite of the fact that this man can perform all movements of the

face voluntarily, without any difference between the two sides, on involuntary laughter he only smiles with the left side of his face, the opposite side being perfectly passive. He protrudes his tongue in the midline and can move it from side to side. There is no atrophy nor tremor. The jaw drops in the median line. The actions of the temporals and masseters are well preserved.

The arms are of good development. Power, by the grip test, is somewhat diminished in the left side. Resistance to passive movement is equally good on both sides. He can perform all the normal movements well. In the finger to nose test there is a marked ataxia on the left side, almost amounting to an intention tremor. In the right extremity no ataxia is apparent. When he extends his hands there is a coarse tremor of both hands, that in the left being much more marked than that the right. The oscillations are upwards and downwards. There is no tenderness over nerve trunks. Atrophy is absent. Biceps and triceps jerks are exaggerated, and equally on each side. The legs present a good musculature. All normal movements can be well performed. In the heel to knee test no ataxia is demonstrable in either extremity. No atrophy or tenderness is present over nerve trunks. Knee jerks and ankle jerks are exaggerated on both sides. Plantar stimulation causes plantar flexion of the toes on both sides. Ankle clonus is absent. The patient shows no alteration of station with eyes open or closed. He also exhibits no abnormality of gait with eyes open, but when closed incoördination is present to a slight extent.

In the right half of the body to the median line absolute loss of sensation to pain and temperature is present. In testing he calls the pin prick dull, except on the right side of the forehead, where he says he feels the sharp touch. Although he feels here, the sensation on the opposite side is much more acute. Touch and muscle sense seem to be well retained. There is no astereognosis. The sphincters are apparently normal. No defects of speech are noted. The mentality of the patient is good; no delusions nor hallucinations being present. The chest of the patient is well formed and he has good expansion on both sides. Pulmonary resonance is good throughout. On auscultation normal breath sounds are heard. No adventitious sounds present. Examination of the heart shows that the apex beat is not observed on inspection. On palpation it is found to be diffuse, although not forcible in the sixth interspace. Cardiac dulness is slightly enlarged. The first apex best is weak. No murmurs can be detected. The spleen and liver are not enlarged, and no tumors or ascites are apparent.

Dr. Spiller agreed with Dr. Mills in his diagnosis. He believed the lesion must be in or near the optic thalamus. Numerous cases are recorded in the literature of unilateral ataxia on the side of a lesion in the brain stem, and tremor is believed to be a sign of a lesion in the cerebral peduncle, as in Benedikt's symptom-complex.

ASCENDING POSTEROLATERAL SCLEROSIS

By T. H. Weisenburg, M.D.

Some years ago Dr. Mills first described a new symptom complex which consisted in an ascending degeneration of the pyramidal tracts. Dr. Weisenburg now reports two cases in which there was an ascending

degeneration not only of the lateral columns but also of the posterior. The first case was that of a man of 56 who seven years ago noticed a weakness and stiffness in the right lower limb which was followed in three years by a similar condition in the right upper limb. Two years ago, or five years after the beginning of his disease he developed a similar condition in the left lower limb. He now has a typical, spastic paretic gait with ataxia of both upper and lower limbs, more marked in the right side, increased reflexes and bilateral Babinski phenomenon. He has besides a disturbance of sensation for touch and pain in all the limbs, more marked in the lower. There is an uncertain history of syphilis with some pupillary inequalities.

The second case was a young man of 29 with no previous history who four years ago first noticed a stiffness in the left lower limb. This increased and two years afterward involved the left upper limb and for the past six months the right lower limb has become similarly involved. He complains of numbness in all of these parts and has disturbance of sensation for touch and pain in the same areas. The reflexes are everywhere exaggerated and the Babinski phenomenon is obtained on both sides. He is distinctly ataxic in all the limbs, more so in the lower and with the eyes closed will have a tendency to stagger.

In both of these cases there is a distinct history of a gradual involvement of the lateral and posterior columns, first in the lower part of the spinal cord or one side and then the upper and then on the other side of the cord. Evidently here we have an ascending posterolateral sclerosis. The etiology in these cases is rather difficult to determine. In the first case, as there was a history of syphilis, it might be the result of this, but it is curious that such systematic degenerations should result. In the second case there was no history of disease, but the fact that it occurred in a young man probably suggests a congenital weakness in these parts.

Dr. Mills said in regard to one of the cases of Dr. Weisenburg it would seem in some respects like a case of disseminated sclerosis with a tendency, as in some cases of this disease, ascending or descending, unilateral or bilateral, to some degree of systematization.

Dr. C. S. Potts said in reference to one of Dr. Weisenburg's cases, that it occurred to him that it resembled one of the atypical forms of multiple sclerosis. The fact that the patient had diplopia at one time would bear this out. Some years ago Dr. Potts reported a case which somewhat resembled this although the posterior columns were not affected. There was also some paralysis of the ocular muscles and a temporal atrophy of the discs. That case was regarded as one of probable multiple sclerosis, though it was never proven by autopsy.

Dr. Alfred Gordon said that in the last two weeks he had an opportunity to see a case of the type of ascending paralysis described by Dr. Mills. It was a child of eight, a boy. He had made an effort to bring him before the Society but failed, the mother living out of town and not caring to come in at night. The striking symptom is this: The child was taken a year and a half ago with sharp, severe pain in the right foot, the pain persisted for some time, then paralysis developed. The mother noticed that the child walked lame, then dragged the leg as in hemiplegia. Yet later, while paralysis persisted in the leg the child developed pain in the right arm, then shortly after that paralysis of the right arm set in. The mother also told Dr. Gordon that at the time the paralysis of

the leg developed a nystagmus appeared. Recently Dr. Gordon examined the boy and found typical hemiplegic gait. He presented exaggerated knee jerk, Babinski's sign, the paradoxical sign, etc. When the patient is told to walk he presents the gait of a hemiplegic individual. He also presents a very marked nystagmus for upward and downward movements and in a milder degree for lateral movements. The history of the case is very interesting from this standpoint, perphaps of pathogenesis of disease. The disease began with pain in the leg and then pain in the arm, when paralysis set in. Perhaps there is some connection between the two phenomena. In considering the pathogenesis of the disease a possible neuritis should also be thought of instead of referring the paralysis to motor tract involvement. The question of multiple sclerosis comes up. He did not present any other symptoms except the marked nystagmus which came on at the same time as the paralysis of the leg. The clinical picture of the case is that described by Dr. Mills originally—an ascending paralysis.

Dr. Weisenburg said that he had had multiple sclerosis in mind in both of his cases. The examinations, however, denoted no changes in the optic discs, no nystagmus, and most important of all the symptoms appeared in such regularity that it would be difficult to reconcile them with the diagnosis of multiple sclerosis.

MENTALLY DEFECTIVE CHILDREN IN THE PUBLIC SCHOOLS

By Walter S. Cornell, M.D.

The recognition of the presence of mentally defective children in the regular grade classes of the public schools is not of recent date, but official cognizance and effort to aid these children is comparatively so, and the work along these lines has been conducted in a scientific manner only in a few of our largest cities. The function of this paper is simply to record a number of cases which have recently come under notice without systematic effort to discover them, and all existing in four public schools. Dr. Cornell then described certain illustrative cases.

A consideration of this subject would not be complete without reference to the special classes for backward children in New York City. Here large numbers of such children have been gathered, those from the East Side displaying an especially low grade of mentality. Dr. Cornell recently visited one of these classes, organized in the public school at 160 Christy St. Of the eighteen children in the class, all had physical defects. Defective vision was possessed by every child, three were deaf and six had nose and throat defects. Malformed skulls were noticeable in the majority of cases, some showing hydrocephalus and still others asymmetry of the cranium. Defects in the motor apparatus were evident, many of the children showing not only weakness in gait but also a pronounced weakness in one limb, which unilateral weakness is rather common in actually mentally feeble children from cerebral paralysis or functional weakness.

Dr. John K. Mitchell asked whether he were correct in understanding Dr. Cornell to state that chorea was commonly associated with mental defect. If so, whether other gentlemen present agreed with the statement. Dr. Mitchell's experience was directly opposed—that is that choreic children were rather unusually bright.

Dr. G. E. Price referring to the percentage of syphilis and tubercu-

losis in the parents of defective children, stated that in the examination of a large number of imbecile children at St. Christopher's Hospital, he had been impressed with the fact that in many of them he could find no etiological factor except alcoholism of the parents, indeed, sometimes of both parents and grandparents.

Dr. John K. Mitchell believes that adenoids and enlarged tonsils may do harm in one case and the eyes in another. Is not the application of a little common sense to the whole subject called for? If the child is moderately backward, as some of the high grade imbeciles are, and has a further handicap of adenoids, enlarged tonsils, or defective eyesight, he has not a fair chance to execute his task reasonably well. The question is altogether one of degree. One cannot say that the adenoids have nothing to do with it; the tonsillar changes may or may not; defective vision, of course, has an influence. The child can't see as it should; it can't see the board; can't see its books. Some of us have been talking as if it were possible that correction of these gross physical defects would affect the brain. Others have been saying that they could not affect the brain. Their improvement does remove some additional nervous burden from the child and may give him his opportunity. Instead of taking such extremely narrow and opposite views Dr. Mitchell believed the subject should be treated with common sense.

Dr. John H. W. Rhein does not believe that the removal of adenoids and tonsillar enlargements has much effect on the development of feeble-minded children. He believes that when a child is born feeble-minded it remains so all its life, and the most that the removal of the adenoids and tonsillar enlargements can effect is to help along the physical condition, and thus permit of a slight improvement in the mental condition.

Dr. Charles S. Potts said it seemed to him the point Dr. Cornell made about medical students having their attention called more to this subject was very important. Dr. Potts does not think Dr. Cornell meant that we ought to have a chair for the teaching of this subject exclusively. Dr. Potts thinks the average practitioner does not appreciate that adenoids and enlarged tonsils (which he believes do cause trouble in children) and eye-strain may cause children to appear defective and they therefore do not appreciate properly that these things ought to be remedied and this he thought was one of the main advantages of having a skilled corps of examiners for school children.

Dr. Alfred Gordon said he had an opportunity to examine many hundred children for the Society to Protect Children from Cruelty. He kept the records and he has yet to find a case of a mentally deficient child to be improved mentally or to become bright mentally and able to take up his studies alongside of normal children from correction of eyesight, from removal of adenoids or removal of any other apparent deficiencies. He has seen brilliant children with adenoids, with deviated septum. He has seen brilliant children with astigmatism, myopia. He has also seen children, backward children, who can't keep up their studies alongside of normal children without defects. He thinks the term mentally defective is loosely employed. When we use the term mentally deficient that means a deficiency which is inherent, which is in the makeup of the individual. There is a special arrangement, anatomical or otherwise, of the nervous system. When a child cannot see well and cannot learn quickly, that does not make him a mental deficient. If a child has a deficient hearing through poor tonsils or adenoids, or ear disturbance, from the

common-sense standpoint these material deficiencies should be corrected as in any other individual, but not because this will have a great deal to do with the development of the brain itself. It seemed to Dr. Gordon incomprehensible and to be inconsistent, to put any special relation between mental development and physical deficiencies of that kind. He has always believed that the reports on the marvelous improvement following the removal of those physical deficiencies have been exaggerated and misrepresented. He thinks it is time for us to take up this subject a little more fully and to place the public, especially the reporters of the newspapers, on the proper ground.

Dr. Cornell, in closing, said that he was afraid that some of his remarks, which were informal, had been misunderstood by one or two of the speakers, or that he had not put the case completely enough. What he meant to say was that the cases he had mentioned were actually mentally deficient children. Certainly a hydrocephalic child may be classed as mentally deficient, and, searching of the public schools will show many of these and of similar cases. Comparison of these children with children of the highest grade in custodial institutions will show that they are considerably behind the latter class intellectually. The modern accepted classifications of these children are sufficient to clear up the points involved in the discussion just ended. There is a very admirable institution for feeble-minded children in Plainfield, N. J., in which the backward class is primarily classified into pseudo-backward and truly backward.

Those classes which are actually feeble-minded are found to show a very large proportion of physical defect, such as undescended testicle, flat chest, malformed skull and sensory defects. In these cases the defective brain is only one part of a generally deficient makeup. Naturally you can put glasses on a child with deficient brain and it will make little or no difference. On the other hand, those who are apparently defective simply from deprivation of full vision can be greatly benefited by correcting errors of refraction. Such children exist in large numbers. It is true that eyestrain does not cause very much retardation in school, yet the study of a large number of children and a comparison of the children with good eyesight, with the children possessing poor eyesight, shows a decided difference in the average of their school studies. In an investigation made recently by him in the Allison School, the children were classified on the basis of visual acuity into three classes—those with three fourth vision or more were taken as possessing good eyesight, those between three fourths and one half as fair eyesight, and those with one half or less as poor eyesight. The scholastic averages of each of these groups of children were obtained and averaged and comparisons then made. The children with good eyesight attained an average of 76, with fair eyesight 73, and with poor eyesight 69, showing that a difference of six points only existed between the extreme groups. Nevertheless this is something, and illustrates the relation between physical and mental defects. It is ridiculous to say that adenoids or enlarged tonsils do not have a profound influence on the mentality. The majority of such cases are associated with the lymphoid diathesis, including enlarged tonsils, adenoids, congested Eustachian tube, and resultant catarrhal deafness together with deficient breathing producing flat chest and poor nutrition and deficient oxygen supply. Such a child is not going to develop to the highest type, and will not have an equal chance with a child properly nourished and with sound sense organs. Dr. Cornell said he had heard

Dr. Cronin of New York state that out of 150 defectives, in certain of the New York special classes, 81 had adenoids. He had had them operated upon with the result that 76 were put back into the regular classes the next year. Dr. Cornell has at least half a dozen photographs of children before and after the removal of adenoids, to show the remarkable improvement resulting.

The point of view one gets of these cases largely depends on the grade of children habitually dealt with. If you are a neurologist you examine actually feeble-minded children, make an autopsy and of course often find the brain to be morphologically imperfect. If you are a general practitioner, a nose and throat specialist, and especially if you are not trained in medicine, you never see these cases, and consequently are more disposed to dwell upon adenoids and sociological conditions as causes of backwardness.

Dr. Cornell said he had not mentioned syphilis as a frequent causative factor. He said that he had incidentally mentioned the fact that in a poor school, near 9th Street and Montgomery Avenue, it was a frequent cause of physical defect, the disease being present to a marked extent in the parents of the children. In his book, Barr mentions a neurotic taint, insanity of the parents, alcoholism, and tuberculosis as the four primary causes. He called attention to the boy, the one of the negroid type, who possibly had inherited syphilis. This boy had a dusky skin and curly hair, indicative of this class of defect, together with a corneal opacity and internal strabismus.

Dr. Cornell said he could not answer Dr. Mitchell's question as to chorea, as he had not made a systematic study of the subject. He has seen children with chorea and a considerably larger number of cases of habit spasm. These are frequently incorrectly classed as chorea, with the result that accurate information upon the subject is not obtainable at the present time.

DEGENERATION IN THE PERIPHERY OF THE SPINAL CORD

By S. D. Ludlum, M.D.

Changes shown by cutting longitudinal sections of the cord and staining with a silver stain, which changes are not shown by the usual methods of procedure.

Periscope

Neurologisches Centralblatt

(Vol. 26. 1907. No. 1. January 2.)

A Contribution to the Knowledge of Myohypotonia or Myatonia Congenita. M. Bernhardt.

This disease was first described by Oppenheim in 1900, who thought that the condition is due to congenital lack of development of the muscles. Spiller was the first to obtain a postmortem and found normal nerves but a lack of development of the muscles. In some cases a lesion of the thymus gland was found. Secondly, Bernhardt called attention to the fact that in many cases where there has been no lesion of the muscles there may be obtained a reaction of degeneration to the electrical current and that this is due to a form of neuritis described by Gombault as pre-Wallerian in which there was degeneration only of the medullary sheaths, and he thought that this condition of myatonia congenita was really one of polyneuritis of early infancy and not due to a congenital lesion of the muscles.

(No. 2. January 16.)

Neuritis the Result of Creosote. W. G. Huet.

The author described seven cases of multiple neuritis resulting in ingestion of phosphate of creosote. The symptoms were those typical of multiple neuritis. The quantity ingested varied from 25 grams to a bottle. The occurrence of neuritis resulting from such causes should be borne in mind especially in the treatment of patients with tuberculosis.

(Nos. 3 and 4. February 1 and 15.)

The Vessel Changes in the Brains of Congenital Syphilitics. Otto Ranke.

The author gives the results of his examinations of fifty brains of children from the third fetal month to several months after birth. Thirty-eight of these range from the fifth fetal month to full birth. The principal changes are found in the interstitial vessels and secondarily in the parenchyma of the organs. They can be divided first, into the characteristic vessel diseases, second, into diffuse small cell infiltration, third, into circumscribed round cell collection, the so-called miliary syphiloma, and lastly into circumscribed and diffuse changes in the connective tissue in the vessel cells. Not the least of his findings in his excellent paper is the demonstration of the spirochaeta pallida in two of the cases.

(No. 4. February 15.)

Cerebral Hemiplegic Phenomenon. Z. Bychowski.

The author describes a certain phenomenon found in hemiplegics with the patient lying in a supine position. If an attempt is made to flex either limb this cannot be done without difficulty, but if an attempt is made to flex both lower limbs at the same time, it will be impossible to move the paralyzed limb. The author believes this to be due to the fact that the cerebrum is accustomed to alternate actions and because of this there will be coordinate phenomena as flexion of both lower limbs.

This phenomenon has been described by Gasset and Gaussel who explained this by the failure of the action of the pelvic muscles, a much better explanation.

(No. 5. March 1.)

Etiology and Specific Therapy of Basedow's Disease with the Application of Moebius Antithyroidin. Dr. Rattner.

The author first discusses the different theories of the disease and believes that it is probably due to a disturbance in the secretions of the thyroid gland. He gives the practical results obtained in treatment of this disease by the antithyroid serum of Moebius. In all of these cases there was an improvement of the cardinal symptoms for a time but the results were not permanent and he states that he knows of no cases in which good permanent results have been obtained by this serum.

(No. 6. March 16.)

The Pathology of Continuous Rhythmic Movements of the Musculature Concerned with Swallowing. Dr. Klein.

In 1904 the author reported these two cases clinically. He has since obtained postmortems and the present report concerns these with the study of these two cases. In the first case there was difficulty in speech and swallowing and unilateral rhythmic movements of the musculature concerning swallowing. The second case only differed from the first in that the rhythm of the contractions was bilateral. In both there was a complete study made. The author found, however, in both cases multiple areas of softening throughout the hemispheres but the pons and medulla in both cases were normal. The important finding, however, was that in the first case there was found an area of softening in the cerebellum, in the outer and middle part of the dentate nucleus, as well as in the substance of the superior, posterior and median lobes. In the second case the lesions were bilateral. The author believes that the disturbance in swallowing was probably due to the cerebellar lesion, although he is by no means certain of this.

(No. 7. April 2.)

A Further Case of a Temporary Loss of the Patellar Reflexes in Hysteria. Dr. Wigand.

The author reports a case of traumatic hysteria in which there was for four years either a diminution or loss of both patellar reflexes. He quotes other cases in literature reported by Nonne, Marie, Dejerine. The patient had besides all of the usual symptomatology of hysteria.

(No. 10. May 16.)

Myasthenia Gravis. A. Borgherini.

Borgherini studied carefully three cases of myasthenia gravis and was able to obtain the muscles in these cases and study them. His conclusions are that there is a relation between myasthenia gravis and progressive muscular dystrophy, inasmuch as he found them associated in the patient and the anatomical changes were the same; the electrical muscular reactions in this disease are positive and can also be obtained by galvanic irritation; the whole electric muscle reaction does not depend upon an ordinary increase of the muscle nuclei and of the destruction of the sarcoplasm but also the existence of a certain chemical substance which is the product of the life of the cells in the diseased muscle and lastly

concludes that the clinical picture of poliomyelitis and encephalomyelitis can in many cases resemble those of myasthenia gravis.

(No. 13. July 1.)

Concerning the Resulting Symptoms of Lesions of the Central Nervous System. Max Rothmann.

In a rather long article the author discusses the recent views expressed by von Monakow regarding the so-called diaschisis in which the latter expressed the opinion that a lesion in one part of the nervous system must have a resulting action in other parts. Rothmann believes that while this is true in lower animals it becomes less so as we go higher up in the scale and in the human being where special functions become more highly localized there is very little action at distance resulting from lesions.

(No. 14. July 16.)

Irritation of the Cerebellum. A. Lourie.

The author in some experiments upon the cerebellum on lower animals disagrees with most of the experiments performed by others, agreeing, however with Ferrier in the fact that irritation of the declive occasionally produced movements of the eyeballs on the same side. In opposition to Ferrier he also found unilateral movements of the eyeballs. The experiments of the author are of negative value.

(No. 18. September 16.)

A Case of Tumor of the Hypophysis with a Roentgen Ray Photograph. Dr. Schuster.

The value of the Roentgen ray in cranial surgery is yet to be demonstrated. It is therefore with great interest that we note that in a case of tumor of the hypophysis the author was able to demonstrate a tumor in this area.

A Case of Progressive Facial Hemiatrophy with Ocular Symptoms. Dr. Salomon.

The author details a case of this type with no sympathetic symptoms, but involvement of the internal and the external rectus on the same side. A neurotic origin is suggested by the author.

(No. 20. October 16.)

A Case of Rhythmical Continuous Cramps of the Swallowing and Respiratory Muscles Resulting from Functional Traumatic Neuroses. E. Ernst.

The author describes an interesting case of traumatic neurosis in which, besides the usual symptoms of hysteria there were present spasmodic rhythmical cramp-like movements of the muscles concerned with respiration and swallowing. The author discussed the two similar cases reported by Klein in this Journal and abstracted above in which the latter ascribed the cause as being due to cerebellar lesions. He discussed Klein's cases and disagrees with him, believing the cause to be that of hysteria.

(No. 23. December 1.)

Sacral Form of Multiple Sclerosis. H. Oppenheim.

The author reports an interesting case of a man 40 years who twelve years ago had sudden weakness in his lower limbs and bladder disturbance

which terminated in incontinence. Some years afterwards he had sharp pains in his limbs followed by impotence. No cerebral symptoms excepting some dizziness. Examination showed no motor symptoms in the limbs or anesthesia; total loss of reflexes, sphincter and anal reflexes and a circumscribed anesthesia for touch and pain. On the left side there was besides increased patellar jerks with the Babinski reflex. Oppenheim believes that there are two diagnoses, possibly either spinal syphilis or multiple sclerosis, and believes it to be a sacral form of multiple sclerosis.

T. H. WEISENBURG (Philadelphia).

American Journal of Insanity

(Vol. LXIII. No. 3, 1907.)

1. Embryonic Pia. C. B. FARRAR.
2. Hysterical Insanity, with Ganser's Syndrome. H. P. FROST.
3. Addison's Disease with Terminal Mental Symptoms. HARRY W. MILLER.
4. Cerebellar-Vestibular Syndrome. I. H. CORIAT.
5. Cerebral Arteriosclerosis. J. B. AYER.
6. History and Uses of Term Dementia. G. ALDER BLUMMER.
7. Insanity and Suicide. CHARLES W. PILGRIM.
8. Family Care of the Insane in Massachusetts. O. COPP.
9. Case of Loss of Memory. CHAS. W. BURR.
10. Is Dementia Præcox the New Peril in Psychiatry? J. T. ROWE.

1. *The Embryonic Pia.*—Some studies carried out chiefly on the chick embryo. The author shows that the membranes, both dura and pia-arachnoid, are developed from a mesoblastic structure which very early surrounds the epiblastic cerebro-spinal axis. The differentiation between the dura and pia takes place quite early, the pia being characterized especially by the formation of numerous vascular channels. The constituent elements of the embryonic pia-arachnoid are all derivatives of the branching embryonic connective tissue cells. The author differentiates these into four categories. (*a*) Border epithelial cells bounding the membrane within and without. (*b*) Arachnoid cells forming the trabeculæ bounding the so-called arachnoid spaces. (*c*) Reticulated cells, the wandering cells of the arachnoid stroma disappearing with the maturity of the membrane. (*d*) Mural elements building the walls of the blood vessels. The finding of the reticulated cell he thinks of interest in view of its importance in certain pathological processes. It is not found in the normal adult brain.

2. *Hysterical Insanity.*—Clinical history of a youth of 18 years of age, who subsequent to a fall on the head, after a period during which he acted foolishly and committed several thefts and forgeries, developed a psychosis marked by confusion, foolish answers and queer ideas more or less connected with his business as an agent for an arc light said to contain radium. He was much prostrated physically, showed complete analgesia of the skin and in his replies to questions gave a symptom-complex suggesting that described by Ganser. After about two weeks, sudden clearing of consciousness in the middle of the night, and gradually complete recovery.

3. *Addison's Disease with Terminal Mental Symptoms.*—A woman of 47 years of age after two years' illness from Addison's disease became fretful, discouraged, showed diminution of volitional impulses, incapacity for mental effort, and memory defect. She also had ideas apparently

dependent upon paresthesia of the skin, animals were crawling upon her, a dog had bitten her upon the arm, a search-light was being played upon her back. The patient dying after a sojourn of 18 days in the hospital, the author was able to make a complete autopsy with microscopical examination. There was healed tuberculosis in the lungs, and the adrenals showed advanced tuberculous degeneration, bacilli being found in the debris. An examination of the central nervous system and of the semilunar ganglion disclosed nothing abnormal beyond increased pigment deposit in the cells.

4. *The Cerebellar-Vestibular Syndrome.*—After a brief discussion of the literature of this subject, the author reports the following case. A man of 42 years of age had had for years a chronic ear trouble and a polypus was removed under ether. Shortly after this he developed a condition of delirium, confusion and disorientation and had some difficulty in walking. On admission to the Worcester Insane Hospital he was restless and disoriented, could not remain upright but fell over backwards and in walking crossed one leg over the other and swayed considerably. No history or evidence of past syphilis could be obtained. There was no complaint of headache or vertigo, no hemianopsia or diplopia, and no tenderness of the scalp on percussion. Fine horizontal nystagmus, right pupil larger than the left, both reacted rather sluggishly to light and accommodation. Watch heard at 5 cm. but no bone conduction. Smell normal, taste impaired in anterior portion of the tongue. No disturbance of sensation, no astereognosis. Knee jerk and Achilles tendon reflexes lively and equal. Slight ankle clonus on the left, none on the right. No Babinski. Tongue median, facial movements symmetrical. Coarse tremor of the hands more marked on the left and greatly increased on rotation. Left arm showed marked ataxia. Marked swaying on standing even with the eyes open. Walks rapid, heels brought down heavily, legs crossed, and great swaying from side to side. Cannot turn quickly or pick up objects. No ataxia of the legs when lying in bed. Speech slow but not like that of a paretic. Writing shows a coarse jerky tremor. Physical examination of heart and lungs negative. Slight arteriosclerosis. Urinary examination negative. Right ear showed old perforation, no mastoid tenderness. The confusional condition gradually became more marked, gait grew more uncertain, there was incoördination of movements of the left arm, constant complaint of vertigo, beginning optic neuritis was disclosed by an ophthalmoscopic examination, the knee jerks became exaggerated, the pupils rigid, and occasionally there was a spasmodic tic on the left side of the face. Towards the end of his life there was double ptosis, tremor of the right arm, reflexes increased in the lower extremities, Babinski on the right. The patient finally died in a condition of stupor which had been preceded by great difficulty in swallowing and periods of apnea. An autopsy performed shortly after death disclosed a large hemorrhage under the dura over the right hemisphere, which was much compressed, the convolution being flattened, while over the left hemisphere posteriorly there was also a subdural clot about the size of a silver dollar. Microscopical examination disclosed no further changes except a moderate chromatolysis of cells in the paracentral lobule, the medulla and the anterior horns of the spinal cord. Middle and internal ears normal. Discussing the mechanism of the production of the cerebellar-vestibular syndrome, the author thinks that the symptoms in this case are best explained by the fact that the pressure of the large subdural hemorrhage so interfered with the central path-

ways of the brain connected with the vestibular nerve that disturbances of equilibrium and coördination could occur. He thinks that the term "central ataxia" would be less cumbrous and better descriptive of the symptoms observed.

5. *Cerebral Arteriosclerosis.*—Résumé of the author's views as to the importance of this widespread condition and the desirability of further investigations as to its etiology, and especially of the changes in the nervous system dependent upon it.

6. *History and Use of the Term Dementia.*—A discussion of the term Dementia, its definition as given by different authors from ancient times to the present day. The author shows that the definitions given are conflicting, and in no case complete. As applied to terminal conditions Kraepelin seems perhaps purposely to avoid the word. Wernicke essays a definition, but in the opinion of the author his definition is incomplete. So on for the other authors. In conclusion he makes a plea for greater exactness in our terminology, since we have arrived at a place in psychiatry where for further progress toward unity in our conceptions, sharp and clear cut definition of terms used must be possible.

7. *Insanity and Suicide.*—Insanity and suicide are increasing out of proportion to the increase in population. In New York State the proportion of insane has increased from 1 in 377 in 1892 to 1 in 299 in 1905. A similar increase is reported in Great Britain. According to statistics compiled by an officer of one of the great life insurance companies for fifty American cities the ratio of suicide has increased from 12 in 100,000 in 1890 to 20 in 100,000 in 1904. San Francisco shows the enormous rate of 72 per 100,000 while the New England mill towns as Fall River, New Bedford, Lynn and Lowell stand lowest in the scale with a proportion of from 3 to 8 per 100,000. The high rate for San Francisco is attributed to the high percentage of foreign-born citizens, especially Germans and Chinese, whose tendency to suicide is unusually great, and the many engaged in speculative enterprises, while in New England there is a high percentage of women and a large French Canadian element, this race being notoriously averse to suicide. The rate in New York does not exceed the average, but that in Hoboken comes next to that of San Francisco, being 38 per 100,000. These figures are explained by the author by the high proportion on the one hand of a Hebrew and on the other of a German element. Strahan has divided suicides into the rational and the irrational. Among the latter class come the cases with which the alienist has chiefly to deal. After giving some of the characteristics of "rational suicide," the author reviews his experience of twenty years with insane suicides. He finds that more than half the cases whose record he was able to examine occurred in April or September. Hanging was the means employed in more than half the cases, while nearly all the rest cut their throats. As would be expected, melancholia is the form of insanity in which the suicidal tendency is most marked and especially is it present in women. It may be present in any form of insanity, however, and the term suicidal insanity is unwarranted. As to the combatting of suicidal tendency among those not insane the author can see no other measures than the endeavor to raise the moral tone and to cultivate a feeling of hope in the community.

8. *Family Care in Massachusetts.*—The boarding out of such cases of chronic insanity as are found suitable after long residence in an asylum, has now been in operation in Massachusetts for twenty years, but so far no other state has seemed inclined to follow the example.

During this time 762 patients, chiefly women, have lived in 465 families without doing any serious act of violence, and rarely attempting any. One suicide occurred at the start, then none for nearly twenty years until recently a hypochondriacal woman who was hardly regarded as insane destroyed herself. During the whole time only one pregnancy occurred On the other hand, almost universally the patients have gotten along well with their caretakers and neighbors, and in many instances mutual attachments have resulted. None of the predicted demoralizing affects upon the families of caretakers have resulted. Of course great judgment in the selection of cases suitable for life in families has been found necessary, and a rigid system of inspection has had to be observed. On the whole a surprisingly small number of complaints have been made. Taking all cases, the average cost for boarding patients in private families has been $3.40 per week as against $4.92 in institutions. This latter, of course, inclusive of interest upon original cost of plant and charges for repairs and improvements. Of the 762 patients, in twenty years 160 became self-supporting, and in 70 per cent. of these the result is attributed to family care. The demand for patients to board has steadily increased as the public has become familiar with the workings of the system. While the author can readily see certain advantages of the management of the insane in institutions, providing proper care and judgment are used in the selection of suitable cases, of families adapted to each case, and a proper system of inspection and investigation is maintained, he thinks that the relief from crowding, and the return of patients to a more useful and normal mode of life amply justifies the resort to family care, which in Massachusetts has gained popular confidence and has apparently come to stay.

9. *Loss of Memory.*—A man apparently about 55 years of age, was found wandering about the street and taken to a general hospital. He could give his name and occupation but did not know where he lived, or where he was. Though confused he was not violent. After some days, however, he suddenly became violent and was transferred to the insane wards where after a few days he quieted down and passed into a condition in which he has now remained for six years. He shows absolutely no sign of physical disease, and is not aphasic. He has, however, lost memory for many years of his life, and cannot recall anything happening at the present time, after a few minutes. He tells where his early life was spent and some of its incidents, where he learned his trade, and for whom he worked, knows that he has been married, but cannot recall what his wife looked like or whether she is living or dead. Mention of her rouses in him deep grief, which, however, is in a few minutes passed by. He understands everything which is said to him, can write from dictation if the sentences are not too long, but forgets the end of a long sentence while he is writing the first clause. He makes few errors in spelling and can do simple arithmetical problems well. He reads and understands the newspapers, but forgets what he has read before he reaches the end of a paragraph. He does not know where he is though he has been told many times, has no idea of year, month or day, cannot find his way to the dining room or pick out his own bed at night but must always be shown. He cannot even remember whether he has eaten or not but will reply, "I suppose I must have done so as I do not feel hungry," or vice versa. He does not recognize doctors or attendants, but is able to reason fairly well, for when asked at a clinic held on him, by the lecturer, "Who am I?" he replies, "You must be a professor

because you are lecturing to a crowd of young men who must be students." He also knows that he must be in a hospital because there are sick people there, but when in the hospital office does not know where he is except that it is a public building of some kind. He has no hallucinations, illusions or delusions, is scrupulously neat and correct in his conduct. He has no interest in the outside world and seems indifferent to his condition and satisfied with his surroundings. The author enters into an interesting discussion of this case in its medico-legal bearings, whether the man could properly be considered as insane or not and as to his responsibility should he commit a crime. He would certainly know at the time what he was doing but immediately after would forget what he had done. He certainly could not make a valid will.

10. *Is Dementia Præcox the New Peril in Psychiatry?*—The author properly observes that much harm can be done by too gloomy a prognosis, hence, since the term dementia præcox in general implies serious or entire mental failure greater caution in making a diagnosis of this condition should be observed. That there are plenty of such cases he does not deny, but thinks, that while called by other names perhaps, they were perfectly well recognized years ago before the present day nomenclature had come into use. He would restrict the term to those groups of insanity occurring in the adolescent period and terminating in dementia, making it a sine qua non that mental deterioration be reached rapidly. Psychical enfeeblement, with relative integrity of memory, disappearance of the effective feelings or emotional tone, apathy, dissociation between memory and judgment and a primary normal mental condition ending in early breakdown. The physical signs he does not regard as reliable as they are likely to be observed in other forms of mental disease. Especially does he thinks more accuracy needed in the use of the term "deterioration" and in the interpretation of the anatomical stigmata of degeneracy. The classes of insanity have not changed, only our names for them. In this connection he urges that American observers should not follow too slavishly the opinions of European observers. At some of the New York institutions for example the largest and most varied clinical material in the world is handled, and at the present time the author claims is studied in a manner not inferior to that carried out at any institution, hence opinion based upon what is observed there have a right to make themselves heard. The alarming increase in the number of cases of insanity especially among the young, the author thinks is due to the enormously increased stress of living, falling especially in the case of the new arrivals in this country, upon people ill fitted both by constitution and by previous habits of life to bear it. If as much public effort was expended in the relieving of the causes of insanity as is now directed to combatting tuberculosis and the other infectious diseases, he thinks a large number of people could be preserved to lives of usefulness, and the ever-increasing demand for more asylum accommodation would be much abated.

C. L. Allen (Los Angeles).

Miscellany.

Manganese Intoxication and Phobia. R. Jaksch (Muench. med. Woch., 54, May 14, 1907).

The writer reviews briefly the literature on the subject, and gives the clinical history of several cases, observed by him. The inhalation of manganese compounds by workers in factories, where manganese is used,

is considered as the cause of the intoxication. He is of the opinion that manganese forms an albuminate compound, which gets to the nervous system, and there produces the disease picture, analogous to mercury and lead. The symptoms were irrepressible laughing and crying spells, muscular weakness, and tremor of lower extremities, increased patellar reflex, peculiar spastic gait, changed facial expression, and scanning speech. The prognosis of these cases was favorable when the cause was removed, however, the symptoms, which influenced the gait of the patient remained more or less stationary. His treatment consisted in hydrotherapy, galvanization, faradization, exercise and the use of high frequency currents, which were especially useful. One of the author's patients did not have all the characteristic symptoms of manganese intoxication, but was psychically very much disturbed, he had been working in a factory where manganese was used, and also knew Jaksch's other patients. The patient recovered under treatment with high frequency currents. The author considers it a case of phobia.

F. J. Conzelmann (U. S. Army).

Acute Cerebral Polyneuritis, Involving the Auditory Nerve. Dr. Schoenborm (Muench med. Woch., 54, May 14, 1907).

The author reports an interesting case of multiple neuritis of the cranial nerves, especially affecting the eighth nerve on both sides. Only twenty cases of this kind have been reported in the otological literature. Frankl-Hochwart has reported several cases under the name of polyneuritis cerebralis menieri-formis. The etiology in the writer's case was entirely obscure.

F. J. Conzelmann (U. S. Army).

Treatment of Delirium Tremens. Dr. F. Eichelberg (Muench. med. Woch., 54, May 14, 1907).

The writer's experience covers 1,574 patients, 1,043 were uncomplicated cases with a death rate of 1 per cent. 531 cases were complicated with diseases, other than pneumonia, with a death rate of 1.4 per cent. 173 cases were complicated with pneumonia with a death rate of 33 per cent. He has no specific treatment for D. T. He withdraws all alcohol, and pays particular attention to the heart, which he supports with digitalis, strophanthus, camphor and coffee. In cases complicated with pneumonia he gives digitalis and alcohol. His results have been as favorable as those of Ganser.

F. J. Conzelmann (U. S. Army).

Serum Diagnosis in Syphilitic Diseases of the Central Nervous System. Felix Plaut (Münch. med. Woch., 54, 1907, July 23).

After Wassermann, Neiser and Bruck had succeeded in demonstrating syphilitic antibodies in the serum of luetic patients, it was certain that soon investigations would be made in diseases of the central nervous system, especially general paralysis and tabes dorsalis which are so closely allied to lues by numberless clinical observations. Wassermann and the writer have made examinations of the serum and spinal fluid of paralytics and have found in most cases antisyphilitic bodies. The negative or positive history of syphilitic infection was in no way of importance as to the result of the reaction. In a large number of cases

where syphilitic infection was denied antibodies were demonstrated. They were also found whether specific treatment had been carried out or not. The time between the infection and the occurrence of paralysis fluctuated considerably. Positive results were obtained in cases of 6 to 20 years duration. The severity of the diseases did not have any influence upon the production of antibodies. No parallelism could be ascertained between the content of lymphocytes and antibodies in the spinal fluid. All control experiments on non-syphilitic patients, made by the different investigators were universally negative. The blood serum and the spinal fluid should be examined in each case. Marie and Levaditi are of the opinion that the production of antibodies begins in the advanced stages of progressive paralysis and that they increase as the disease progresses. These authors consider that the "antibody reaction" is specific for paralysis and tabes dorsalis. Plaut has not found that the production of antibodies are in any way an index for the intensity of the disease. He is of the opinion that at present the reaction is not specific for general paralysis and tabes dorsalis. We do not know what syphilitic antigen is and accordingly know nothing of its reaction product, the antibody. One may say that syphilitic antigen is a substance which exists in the organs of syphilitic patients. It has only been demonstrated, with fitting amboceptores, in the fluids of patients suffering from syphilitic and meta-syphilitic affections.

F. J. Conzelmann (U. S. Army).

Hysterical Sweat. Hans Curshmann (Münch. med. Woch., 54, 1907, August 20).

The author reports two cases, mother and daughter, who had suffered for one year from profuse perspiration three times a day, always at a definite hour. There was no fever present. Both were confined to bed. The perspiration occurred without tonic or clonic convulsions, and was always accompanied by a subfibrial temperature. The patients were mentally quiet. Not even at the time of sweating did they show any psychical unrest. Physical examinations revealed nothing abnormal. No organic changes of the nervous system. Hysterical stigmata were absent, except both mother and daughter showed a marked psychical suggestability. The anamnesis and the psychical behavior of the patients led the author to the conclusion that it was a purely hysterical disturbance. The suggestive treatment for the patients proved the diagnosis to be correct. Mother and daughter promptly recovered. Vulpian, Binswanger and Siredey have reported cases of hysterical sweating in the literature. In neurasthenia the psychical origin of a profuse sweat is of a local nature. In many nervous cases with hyperhidrosis of the hands, the sweat paroxysm occurs at a time when they are especially afraid of it, as for instance in society when they are obliged to shake hands. Hyperhidrosis may become a phobia in these cases as erythrophobia, nervous diarrhœa and the desire to urinate. The writer considers his cases as purely hysterical hyperhidrosis. He is of the opinion that his cases speak in favor of the probability and reality that purely hysterical fevers may occur in certain patients. Typical fever reaction has been observed by investigators with tuberculin in patients entirely free from fever by the mere suggestion and introduction of the hypodermic needle without injecting tuberculin.

F. J. Conzelmann (U. S. Army).

Book Reviews

Traité des Torticollis Spasmodiques, Spasmes, Tics, Rythmies du Cou, Toricallis Mental, Etc. Par Rene Cruchet. Medecin des maladies nerveuses à l'Hopital Suburbain des Enfants. Preface de M. le Professeur A. Pitres. Masson et Cie, Editeurs, Paris. 15 Fr.

The study of those convulsive disorders allied to tics has received a great amount of attention of late years, particularly at the hands of French clinicians. It might have seemed that the study of Meige and Feindel would have exhausted the subject, but not so, for here is a monograph three times as large, 836 pages, on a portion of the subject alone.

The author first considers the historical side of the question and then takes up successively neuralgic, professional, paralytic, spasmodic, and rhythmic torticollis, then those tic-like movements of the neck and chin and the mental or habit tics.

The monograph is very thorough. It is a mine of bibliographic research and collects almost everything that has ever appeared in the general subject. It is well printed, amply illustrated and deserves a place in the specialist's library.

Howard.

The Hygiene of Mind. By T. S. Clouston, M.D., F.R.S.E., Lecturer on Mental Diseases in the University of Edinburgh, Physician-Superintendent of the Royal Edinburgh Asylum. Second Edition. E. P. Dutton and Company, New York.

It is a strange fact that for years science has been worrying men's minds about the laws of hygiene of the body, has been telling us what we should eat, what we should drink, and wherewithal we shall be clothed; it has analyzed our drinking water and our air; told us how disease is propagated by the rat, the fly, the mosquito and the bed-bug, has created an immense library of facts concerning the factors of the conservation of the body from physical disease, and yet with all this has been almost silent in its teachings for the prevention of disorder and breakdown in the most important of man's possessions, his mind.

How this may be said to be true in the face of six thousand years of discussion concerning the mind is difficult to show, yet there is little doubt that these centuries have been filled with fruitless thrashing of the air by reason of the metaphysical character of the arguments. The wise men of the pre-scientific era have given us flashes of anticipation of later teachings, some of the very best of which have been gathered in this volume.

The author's aim has been to put in popular language some of the known facts thus far ascertained concerning mental hygiene and to extend the conclusions from these facts in regard to mental betterment through physiological, psychological, and medical means. In this he has succeeded and has given an exceedingly readable and interesting work. We cannot here give an analysis of its contents, but can commend it as

the ablest of the works that have sprung up in the last few years bearing on the subject of nervous and mental hygiene. It is a volume for the neurologist not only to read and study as a guide in his daily work but one that he can safely put in the hands of the better educated of his clientele.

JELLIFFE.

TEXT-BOOK OF PSYCHIATRY, A PSYCHOLOGICAL STUDY OF INSANITY FOR PRACTITIONERS AND STUDENTS. By Dr. E. Mendel, A. O. Professor in the University of Berlin. Authorized Translation by William C. Krauss, M.D. F. A. Davis Company, Philadelphia, Pa.

The original edition of Mendel's Lehrbuch has been reviewed in these columns and we can only call attention to the fact that it is made more available to English readers by means of a very excellent translation very free from errors of interpretation.

It is to be recalled that Mendel wrote his book late in life and yet it represented the same ideas that he taught when he began some twenty years previously, showing little advance in the author's point of view of psychiatric problems.

The work is particularly valuable to students for its able discussion of the symptomatology of mental disorders; that part of the work dealing with special psychiatry, or disease forms, however, being antiquated even in so recent a volume. The didactic features of the work render it useful to a certain class of students, but the lack of perspective shown in the volume is an initial defect of the author's method.

As for the translation itself Dr. Krauss has done it well and the defects are very few. He is to be congratulated on giving to the English students of psychiatry a work which though not commanding a topmost rank in German psychiatry, is a worthy exponent of the individual teacher's thoughts.

K. M. V.

CLINICAL PSYCHIATRY. A Text-Book for Students and Physicians. Abstracted and Adapted from the Seventh German Edition of Kraepelin's Lehrbuch der Psychiatrie. By A. Ross Diefendorf, M.D., Lecturer in Psychiatry in Yale University, etc. Second Edition, Revised and Augmented. The Macmillan Company, New York.

In our review of the first edition put forth by the author we expressed ourselves that students of psychiatry in the United States were under obligations to Dr. Diefendorf for his excellent effort in bringing Kraepelin's thoughts before us in so acceptable and able a manner.

This second edition augments that weight of obligation for in it, by the marked increase in pages the author has been better enabled to bring out the value of the German original than before. It is of course an extremely difficult task to take a text-book of 450,000 words and compress it to less than one third of this size and do the author full justice, especially when the original is regarded as a model of compact descriptive psychiatry, and no one knows better than the writer of this new edition how difficult it has been to choose material which has seemed to him of most importance. He has done his work well, however, in spite of the difficulties, and we venture to say that they have been more difficult even than if an original work had been attempted.

What criticism may be launched at the present edition concerns itself with the inherent difficulties of the situation. We feel that the author's breadth of concept; his philosophical outlook on the problems of psychiatry have suffered the most in the condensing process, and that Kraepelin has been interpreted in a much narrower and more didactic manner than his admirers would wish. This is no fault of the abstracter but is perhaps a necessary evil arising out of conditions which publishers impose upon authors.

We congratulate Dr. Diefendorf on the excellent manner in which his work has been done. Students of psychiatry will welcome the work and our hope is that it may receive the hearty support that it so richly deserves and that a further edition may reach nearer the goal of the ideal of the German edition. Readers of the first edition should certainly have this later one if they would hope to obtain a better idea of the thoughts of the leading spirit in German psychiatry.

JELLIFFE.

DISEASES OF THE NERVOUS SYSTEM. Edited by Archibald Church, M.D. Authorized Translation from the Die Deutsche Klinik under the General Editorial Supervision of Julius L. Salinger, M.D. D. Appleton and Company, New York and London.

Our readers are acquainted with the volumes on Nervous Diseases in the Deutsche Klinik, reviews of which have been published. Our medical public is to be congratulated that they here appear, well edited and translated into English, affording a comprehensive series of monographs written by the best of German authors, who, for the most part may be said to have specialized particularly on the subject on which they write. Rothmann and Rosin take up the Anatomical and Histological Aspects of the Brain, laying full stress upon the physiological aspects. Redlich, of Vienna, treats of the Modern Aids to Diagnosis, while Quincke, whose name is associated with Lumbar Puncture, writes on this subject. R. Geigel writes on Cerebral Hemorrhage and Embolism, Wernicke, on Aphasia, Gutzmann on Disturbances of Speech, while Schultze, of Bonn, takes up the Tumors of the Cord. Leyden and Lazarus write the article on Myelitis, while Erb again appears with Tabes; Redlich on Multiple Sclerosis and Schultze on Syringomyelia then follow; H. Lütlye, of Erlangen, has a chapter on Freidreich's Disease, Redlich another on Spastic Spinal Paralysis, so-called, while Schultze takes up the Poliomyelitis Group. Bernhardt is again at work on the Peripheral Paralyses, Cassirer on Neuritis and Polyneuritis. Eichorst on Neuralgia. Edinger discusses Headache and Migraine, and Erb writes on Paralysis Agitans, offering nothing new; Frankl-Hochwart writes on Athetosis, Myotonia and Tetany, in which latter affection he has advanced to the possible relation of the parathyroids to some forms of the affection. Remak describes Localized Spasms; Eulenburg, Grave's Disease and Neurasthenia; Vonkastner, of Berlin, Epilepsy; Ziehen, Hysteria; Schuster, Traumatic Neuroses; Cassirer, Vasomotor Trophic Neuroses. The last article by Cassirer deals with the Occupation Neuroses.

It is well known that this series of monographs although shorter than many that are available, are among the best that we possess. Some are of unequal value, and did not represent, at the time of their appearance, the full knowledge up to that time, but as a collection, it cannot be excelled.

JELLIFFE.

Notes and News

Preliminary Program of Papers to be read at the Meeting of the American Neurological Association.—Progressive Hemiplegia Due to Vascular Lesions and Glioma of the Right Centrum, Dr. Frank R. Fry, of St. Louis. A Case of Cortical Blindness, Exhibiting Agnosia, Apraxia, and Loss of the Sense of Position, Dr. John H. W. Rhein, of Philadelphia. The Cerebral Arterial Distribution, Dr. Charles E. Beevor. A Special Diagnostic Phenomenon in the Attitude of the Head in Cerebellar Diseases, Dr. A. Gordon, of Philadelphia. An Interesting Form of Traumatic Aphasia Relieved by Operation, Dr. B. Sachs, of New York. A Brain Tumor Localized and Completely Removed, with some Discussion of the Symptomatology of Lesions Variously Distributed in the Parietal Lobes, Dr. Charles K. Mills, of Philadelphia. Hysterical Insanity, with Report of some Illustrative Cases, Dr. Theodore Diller and Dr. George J. Wright, of Pittsburgh. Localized Epileptic Convulsions without Gross Lesions of the Cortex of the Brain, Dr. Herman H. Hoppe, of Cincinnati. Treatment of Facial Spasm by Means of Injections of Alcohol—Report of Three Cases, Dr. Hugh T. Patrick, of Chicago. Sane Prototypes of Insane Mental Processes, Dr. Theodore H. Kellogg, of New York. The Course of Sensory Impulses in the Spinal Cord, Dr. Carl D. Camp, of Ann Arbor. A Contribution to the Diagnosis of Epilepsy and Dementia Præcox, Dr. L. Pierce Clark, of New York. The Symptom-Complex of Occlusion of the Posterior Inferior Cerebellar Artery: Two Cases with Necropsy, Dr. William G. Spiller, of Philadelphia. Facial Paralysis, Dr. G. A. Waterman, of Boston. Report of a Case of Myasthenia Gravis with Negative Pathological Findings, Dr. J. Arthur Booth, of New York. Tabes Associated with Acromegaly, Dr. F. X. Dercum, of Philadelphia. Tumor of the Frontal Lobes with Mental Symptoms Simulating Paresis, Dr. F. X. Dercum, of Philadelphia. The Types of Encephalitis, Dr. E. E. Southard, of Boston. Occupation Neuritis of the Deep Palmar Branch of the Ulnar Nerve. A Well Defined Clinical Type of Occupation Atrophy of the Hand, Dr. J. Ramsay Hunt, of New York. The Topographic Relations of the Cerebral and Olfactory Portions of the Brain Among Vertebrates, Illustrated by Specimens, Photographs, Models, and Charts, Dr. Burt G. Wilder, of Ithaca. Certain Needless Neurologic Synonyms, Dr. Burt G. Wilder, of Ithaca. Confusional Insanity and Dementia Præcox, Dr. Philip Coombs Knapp, of Boston. Cavity Formation in the Spinal Cord Associated with Tumor Formation, Dr. D. J. McCarthy and Dr. Milton K. Meyers, of Philadelphia. Pathology of the Spinal Dura, Dr. D. J. McCarthy, of Philadelphia. Demonstration in Studies of Secondary Degeneration: The Auditory Pathway and the Relation of the Auditory Cortex to Sensory Aphasia, Dr. Adolf Meyer, of New York. The Motor Cortex in a Case of Apparent Duplication of the Central Fissure, Dr. Burt G. Wilder, of Ithaca, and Dr. Adolf Meyer, of New York. A Case of Tumor Limited to the Arm Area in which the Babinski Reflex Did Not Become Apparent until the Fibers in Association with the Leg Center Became Diseased, with Autopsy, Dr.

C. S. Potts and Dr. T. H. Weisenburg, of Philadelphia. A Case of Right Hemiparesis with Cheyne-Stokes Respiration and Loss of Voluntary Breathing which Lasted for Twenty-six Days with Retention of Consciousness. Autopsy Report, Dr. T. H. Weisenburg, of Philadelphia. Nerve Involvement in Ischemic Paralysis and Contraction of Volkmann, Dr. J. J. Thomas, of Boston. Acute Anterior Poliomyelitis, Dr. Eugene D. Bondurant, of Mobile. Delayed Apoplexy (Spätapoplexie) with Report of a Case, Dr. Alfred R. Allen, of Philadelphia. Prognosis of Spinal Cord Tumors with Operation, Dr. William C. Krauss, of Buffalo. Three Spinal Cord Tumor Cases Seen within a Period of Ten Days. Operations in First and Second Cases. Autopsy in Third Case, Dr. William C. Krauss, of Buffalo. Brain Abscess, Dr. B. Sachs, of New York. Poliomyelitis with Special Reference to Certain Obscure, Apparently Related Infections, Dr. E. W. Taylor, of Boston. Treatment by Rest, Seclusion, etc., Criticisms upon it and its Relation to Psycho-Therapy, Dr. S. Weir Mitchell, of Philadelphia. The Weight of the Brain as Influenced by Nutrition or Disease, Dr. Henry H. Donaldson, of Philadelphia. Occlusion of the Posterior Inferior Cerebellar Artery, Dr. H. M. Thomas, of Baltimore.

Dr. Tom A. Williams has taken up his residence in Washington, D. C., after spending two years in the study of nervous diseases in Paris and other European Countries. He has been requested to give a course, which will begin on May 25th, embodying more particularly the recent researches of the French School in the diagnosis and treatment of the psycho-neuroses. The course will consist of anatomo-pathology in the morning and clinical work in the afternoon, while every week will be held two seminars at which the work will be revised and special points discussed. The number will be limited.

Dr. George H. Kirby has just resigned as Associate in Clinical Psychiatry of the Pathological Institute of New York to accept the position of Director of Clinical Psychiatry in Manhattan State Hospital, Ward's Island, New York.

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Original Articles

PRESIDENTIAL ADDRESS[1]

THE MENTAL STATE IN CHOREA AND CHOREIFORM AFFECTIONS

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I have chosen this subject for two reasons; because it is of interest in itself and because study of the mental symptoms occurring in physical diseases will eventually throw some light upon the nature of the so-called mental affections, the pure insanities. It is incorrect to divide off insanity arbitrarily as a thing to be studied apart from all other diseases. There is no doubt that if we studied the mental condition of physically ill persons with the same care as we study their physical symptoms much would be learned which would throw light on psychiatry. Much has, of course, already been done along this line but very much remains to be done. For example, investigation of the mental states in typhoid fever, even in patients not very ill, throws some light on the clinical occurrence of delirium, and some time we may learn something of its chemical causes.

It is unfortunate that the word chorea can not be dropped from use. It means so many things and includes such diverse conditions that it carries with it no clear connotation. Thus it includes not only acute chorea of childhood (Sydenham's chorea)

[1] Read at the thirty-fourth annual meeting of the American Neurological Association, May 20, 21 and 22, 1908.

but also Huntington's chorea, another form of chronic choreiform disease beginning in middle life but in which heredity plays no part, hysterical chorea, pre- and post-hemiplegic or apoplectic chorea, congenital chorea, always due to gross cerebral disease, and habit spasm. Generalized tic is also sometimes described as a chorea.

I shall speak mainly of Sydenham's chorea, but shall refer to some of the other forms, especially to Huntington's chorea, chronic non-hereditary chorea coming on in middle life, and the chronic chorea of childhood ending in imbecility of greater or less degree. Of these four the chorea of Sydenham is certainly a disease apart, separate and distinct, having an entity as defined as typhoid fever or pneumonia and probably having a specific exciting cause. The other three, it seems to me, have much in common. Huntington's chorea has as its characteristics the heredity and the onset in middle life, but cases occur without heredity which so far as symptoms go are identical. Not a few of the chronic, slowly oncoming cases of chorea of children ending in imbecility resemble the cases beginning in middle life. The resemblances in all three types point not to identical but surely to similar cases. Huntington's chorea is a good example of Gowers' abiotrophy, occurring in the nervous system, and evidence favors the assumption of the same thing in the other types.

It is often said and has been put in many text books that everyone suffering from Sydenham's chorea presents mental symptoms. This is true in so far that patients suffering from even mild attacks show peevishness, fretfulness, some loss of the power of fixed attention, and increased selfishness, but it is not true that every case presents mental symptoms of such moment as to raise doubt of the patient's sanity. The majority of the patients are entirely sane throughout the course of the disease. The severity of the mental symptoms varies greatly in different cases, and a series can be made showing each case a little more severe than the preceding and a little less severe than the succeeding one. Patients in whom the motor disturbance is very slight rarely if ever present serious mental symptoms but the converse is not true, for even though the motor symptoms are very severe, the mental upset may be relatively slight. I have never seen a patient with so-called paralytic chorea, I of course do not mean post-paralytic, in whom severe or permanent mental symptoms oc-

curred. My personal experience, however, with this type of the disease has not been large enough to justify me in making any dogmatic statements.

Though there are no sharp lines dividing the cases they may be separated, so far as the mental symptoms are concerned, into the following groups: First (and this includes a large majority) patients in whom there is peevishness, fretfulness, some loss of the power of fixing the attention and a slight loss of the moral sense shown by disobedience and selfishness. Second, those showing in addition to the above symptoms night terrors and transitory visual, auditory, or other hallucinations. Third, those with distinct delirium, wild or mild, accompanied with fever. Fourth, and this group is very small when we remember how common chorea is, those showing stupor or rather stupidity and an acute dementia which may follow the condition described under three or appear without any preceding mental symptoms at all severe, and which is usually accompanied with trouble in articulation not caused by choreic movements of the lips and tongue but the result of mental hebetude. Fever is usually present for a time at least. Patients of the first and second groups almost always recover mentally and physically; those of the third group frequently die, and those of the fourth usually either die or, recovering from the chorea, remain demented.

Wild delirium was shown in the following case I reported some years ago.[2] The patient, a boy 17 years old, was seized with chorea while convalescing from scarlet fever and suffering from acute Bright's disease. A short time after the appearance of the choreic movements he became dull and at the same time irritable, *i. e.*, he remained quiet and dull as long as he was left alone but resented being talked to or examined. Within a day he became wildly delirious shouting "murder," seeming to see horrible visions, throwing himself violently about, and refusing all food and medicine. From then on there was incontinence of urine and feces, not on account of paralysis but because of indifference. A week later the mental symptoms passed away, the movements ceased almost entirely, and the dropsy, which had been considerable, was much lessened. The improvement was only temporary and two weeks later all the symptoms returned

[2] Archives of Pediatrics, January, 1898.

with extreme severity and after about five weeks he died. The choreic movements ceased two days before death and were followed by stupor. While he was at the hospital there was no fever (except one day when the temperature suddenly and without known cause rose to 101° F., falling to normal on the following morning), until the day before death, when it rose to 100°, fell again, again rose to 103.2°, and finally a few hours before death fell to 101°.

Sometimes there is no delirium even at the onset but dullness and stupidity from the beginning with clouding of consciousness, not coma but mental apathy and emotional deadening. This condition rarely comes on until the choreic movements are well established. It is well shown in the following case:

T. S., a white boy, 17 years old, began to twitch about the middle of December, 1907. He was admitted to the Philadelphia General Hospital on December 20, 1907. The choreic movements were general and so violent that he had to be put in a crib lest he should throw himself out of bed. Study of his mental state was difficult because his speech was affected by the chorea so that he stuttered and spluttered very badly and any attempt at talking greatly increased the jerkings. He answered simple questions requiring only "yes" and "no" fairly well, but needed quite a little time to do so and sometimes would have to be asked several times before he could be roused sufficiently to understand and fix his attention long enough to answer. At first he indicated when he wished to use the bed pan but later paid no attention to his bladder or bowels, becoming incontinent and indifferent to the dirt he made. He was stupid and dull all the time but the intensity of the dullness varied quite a little. He would swallow food put in his mouth (he was on soft diet) but never indicated that he desired food or drink. He gave no evidence of having any hallucinations or delusions. He was entirely indifferent to his surroundings and paid no attention to what happened around him. On January 16 he began to have an irregular fever and at the same time an abscess appeared on the right arm. He died from septicemia on January 20, 1908, the temperature an hour before death having risen to 108° F.

The next case shows the rapidity with which mental hebetude may follow the onset of the movements.

F. S., male, 17 years old, was admitted to the insane depart-

ment September 21, 1907. There is nothing of interest in his family history save that his mother had chorea in childhood and committed suicide when 36 years old. He himself had his first attack of chorea when a child. Five weeks before admission he began to twitch and soon the movements became very severe. At first he was very fretful and peevish, easily annoyed and angered and a few days later became dull and stupid. On admission the movements were general and somewhat severe, but he could walk after a fashion, talk a little and, though with some difficulty, feed himself. Speech was very choreic. He was silly in manner. He had neither hallucinations nor delusions. There were no signs of organic visceral disease. At my last examination (February 7, 1908) all movements had ceased and he had gained in weight and color. He was apathetic and content to sit and do nothing, had no interest in anything, understood what was said to him and answered questions with some intelligence but all his mental processes were very slow.

Though the chorea of childhood is an acute disease lasting only a few months, sometimes we see patients whose symptoms are at the beginning indistinguishable from chorea but who continue ill for several years or indeed for life without intermission. In these chronic cases severe mental degradation does not always follow but frequently there are marked and permanent mental and emotional disturbances. Sometimes the movements cease entirely without mental improvement. They may be classed as instances of the chorea of degenerates. The next two cases are examples.

M. Mc., a child, ten years old when I first saw her, began to jerk during her sixth year. The only event of importance in her life before the onset of the chorea was an attack when she was seventeen months old of what was called brain fever and from which her parents stated positively she recovered completely. While she was under my care the movements were general but not very severe. There was neither palsy, muscular rigidity nor increase of the deep reflexes, in short no evidence was found that the choreic movements were secondary to any gross cerebral disease of infancy. She presented no sign of cerebral diplegia. Mentally she was in many respects below children of her age, not in ability to understand but in lack of self control and power of fixed attention. She could not receive

intellectual education. She was very disobedient, fretful and peevish. After about a year I lost sight of her but I fear that as time progressed mental degradation went on.

The next case was more severe and showed the gradual unfolding of the symptoms before the final acute outburst of serious mental disturbance.

J. R., white, male, blacksmith's helper, was admitted to the insane department of the Philadelphia Hospital in June, 1907. His father's brother had been an epileptic since childhood. His father ever since an attack of influenza eight years ago had been depressed and irritable. From the description I suspect he (the father) suffers from agitated melancholia. Sometimes he refuses to speak to anyone for a day or two. Neither he nor any other member of the family has ever had any disease showing choreiform movements. The patient, himself, was normal in all ways till his thirteenth year. Until then he did well at school, was obedient, affectionate, and fond of reading. Between his thirteenth and fourteenth year his character began to change. He ceased to make friends with other boys and indeed to associate with them. He became intractable and disobedient. At times he would be very depressed and would speak to no one for three or four days. Soon he became slow and dull in thought and could not fix his attention for any length of time. About one year before admission he began to be nervous and fidgety. At first there were only twitchings of the muscles of the face and speech became spluttering. He tried to continue work but three months before admission was discharged for incompetency because he could not handle his tools and soon after rapidly grew worse. The movements became general and severe and he was excited. He believed that people were against him and were talking about him. He masturbated and once struck his mother in anger. Later the movements became so violent that he could not stand.

Examination showed a fairly well nourished youth with general and quite severe choreic movements. He was emotionally apathetic showing neither exaltation nor depression but mere indifference and intellectually he was dull and stupid. He answered simple questions slowly though intelligently but complicated questions he could not answer because he could not fix his attention on them. I could discover no hallucinations nor

delusions and as a matter of fact consciousness was too clouded for any mental process to go on save when he was roused by a question. He was entirely indifferent to his surroundings and had no desire to hear of or see anyone he knew. Physical examination showed no signs of organic disease in brain, spinal cord, or the thoracic and abdominal viscera. At my last examination (February 7, 1908) the choreic movements had almost ceased though there was a little habit spasm of the face and shoulders. He was quite imbecile. Thought and speech were slow and he was apathetic. These cases beginning as chorea or at least with choreiform movements and later showing more or less pronounced signs of permanent mental degradation resemble very much agitated imbecility except that the course of events is reversed. In every home for the feeble-minded are found imbeciles who show great motor restlessness. Sometimes it is mere restlessness, sometimes it is a real tic, the constant recurrence of some complex movement that looks as if it were entirely voluntary, sometimes a habit spasm, which after all is closely related to a tic, and sometimes though rarely the movements resemble true chorea. Ordinarily the mental breakdown precedes the motor symptoms but occasionally the reverse obtains and the choreic movements come first.

There are three interesting questions concerning mental symptoms in Sydenham's chorea. First, are they really a part of the disease or merely a complication; second, in the patients who entirely recover is there any later effect on the mental health of the patient—in other words—what is the prognosis as to future mental health? Third, are the mental symptoms which occur in chorea pathognomonic, *i. e.*, could chorea be diagnosed from the mental symptoms alone? The last question may be answered first and negatively. The same combination of mental symptoms may and does occur in and after the acute infectious fevers and sometimes primarily.* (Parenthetically I may say that this fact is one, but of course not the most important, argument in favor of the theory of some infection being the exciting cause of chorea.) The answer to the third question helps to answer the first. I do not believe that serious mental perturbation is part of the disease but that it is a secondary result. It means inability of the patient to withstand strain. There is not a disease "chorea insaniens" but insanity occurring in chorea.

As to the second question, I have no accurate statistics. I have endeavored to discover how many of my adult nervous and insane patients had chorea in childhood but owing to the inaccuracy of histories the figures are of little value. It seems to be proven, it surely has been my own experience, that children who have had chorea rarely develop epilepsy, certainly no more frequently than would happen from mere chance. I believe, however, that the occurrence of chorea means inherent nervous instability and is a signal of possible danger in the future. Every child who has had chorea needs the wisest and most careful teaching in self control.

Though Sydenham's chorea is ordinarily a disease of childhood and adolescence, cases occasionaly occur even in the aged. More frequently, however, than true chorea in old people is the appearance of localized choreic movements caused by focal brain disease. Thus before or after an attack of apoplexy there may be in the paralyzed part or the part later paralyzed choreic jerkings. These when typical differ from athetoid movements but sometimes it is hard (I have more than once found it impossible) to decide which to call them. The lesion is usually immediately behind or in front of the motor tract in the internal capsule or else involves the optic thalamus. Sometimes, as in the case I am about to describe, the choreic movements become general.

G. S., 77 years old, was admitted to the Philadelphia General Hospital on July 21, 1903, with twitchings of the right arm and leg. The trouble had commenced three months before without an apoplectic attack or hemiplegia. Examination revealed quite severe choreic movements of the right arm but none of the leg. His gait was a little spastic. There was no hemiplegia. The knee jerk was somewhat increased and ankle clonus was present on both sides. He was very garrulous but somewhat difficult to understand because his speech was thick. He was very noisy and restless and rather hard to manage. Some time later the movements extended to the whole body and affected arms, legs, and face so that on simply looking at him the diagnosis would have been Sydenham's chorea. His arteries were very hard and the urine contained a trace of albumin and a few granular casts. He died on the following November from chronic myocarditis and nephritis, having been demented for months. I

have no doubt that the choreiform movements were really a result of widespread cerebral arterial disease.

Sometimes even long standing true chorea in the aged may be unaccompanied by any mental symptoms. Thus I saw with Dr. John G. Heisler in 1898 a woman 80 years old, who for six years had had quite violent general chorea. So far from being demented she was very bright and witty. She had no other sign of disease except that the palpable arteries were quite hard. Dr. Heisler recently told me that the movements continued till her death two years later and that she presented no mental symptoms till she developed a fatal pneumonia accompanied by low delirium.

In contrast to Sydenham's chorea, in Huntington's chorea mental symptoms ending in dementia are an essential factor of the disease. No description of it surpasses in accuracy of statement, clearness of style and brevity of language the original account published by Huntington in *The Medical and Surgical Reporter* for April 13, 1872. His statement that the disease never skips a generation but when it ceases ceases permanently apparently has been disproved in a few families but still remains an almost constant rule. He had no knowledge of isolated cases.

The following family history presents typical examples. In the last generation there were three brothers and two sisters. One sister and all the brothers developed the disease. The father of these patients died at 46 years of dropsy and heart disease, the mother at 73 of some lung trouble presumably pneumonia. It is asserted positively they never had chorea or any mental disease. One of the sons married and had three daughters and three sons. One son died when four years old, another is well and 28 years old, another at 42 years is affected. One daughter 20 years old and another 40 are well. One daughter 36 years old is affected. The father (Case I) of the second diseased generation was admitted to the Philadelphia Hospital in November, 1892, when he was 46 years old. His trouble began about seven years before and followed an injury to the skull from a kick by a horse for which the right frontal bone was trephined. At first there was a little tremor which gradually became worse and more extended. No mental symptoms were noticed till five years later when his disposition changed. He began to be easily angered, absent-minded and fretful. When admitted to the hospital he was aggressive, delusional and quite excited. The dementia increased

rapidly and most of the time he was very easily managed but at times was stubborn and excited. He was transferred to the Norristown Hospital where he died in 1894 of heart disease.

His brother (Case II) was admitted to the Philadelphia Hospital in March, 1895. He was then 47 years old and had been ailing three years. The first symptom was tremor of the hands. Later memory loss came on. He became alienated from his wife and had delusions of persecution and auditory hallucinations. At times he would become confused, vulgar and profane. When admitted there was marked dementia and general choreiform movements. At times he had outbreaks of excitement. He soon became bed ridden and died in June, 1898.

Case III (a son of patient No. I) was admitted to the Philadelphia Hospital in April, 1903. There were then slight choreiform movements in the legs, arms and face. Voluntary movement and emotion increased the jerkings. His gait was slouching and not paralytic. Both knee jerks were increased. The pupils reacted well to light and with accommodation. The admission paper stated that he had been "insane one year and that he had delusions, was very quarrelsome, and had threatened to kill his wife who fears for her life." He, himself, stated that he was "insane like his father." He knew that his memory was getting poor and that at times he acted in a silly way. He further said that his trouble began four years before with twitchings of the body. He was quite a little demented. Most of the time he was sillily happy but had outbursts of causeless anger. The choreiform movements increased somewhat.

Case IV (daughter of patient No. I) was admitted to the Philadelphia Hospital in January, 1906. She was then 42 years old and married. Twitching began in her thirty-eighth year. She had quite marked general choreiform movements. She was silly, happy and good natured. Her condition has remained unchanged.

The occurrence of chronic chorea of middle life without heredity or at least without the disease having appeared in the parents is shown in the following case. As to the grandparents nothing is known. The patient M. B., a woman 58 years old, was admitted to the Philadelphia General Hospital in July, 1905. Her father died of pneumonia at 67 years, her mother after childbirth. Neither of them had chorea of any kind. The patient

has had ten children. Five died in infancy. Five are living, aged 37, 35, 32, 25 and 20 years. None of them have had chorea of any kind. The one we saw was of low mental grade, little more than a high-grade imbecile. No positive statement was given us as to the exact time of the onset of the patient's illness. It was clear however that she had been ill for at least ten years. The thing the family first noticed was that she would jerk and stutter. Later she would have periods of excitement and become confused. She would fret and scold. She wandered away from home and got lost several times. Twice she turned on the gas without lighting it, not with any suicidal intent but because she was confused. She frequently refused to change her clothes, saying someone would steal them. Her memory became very bad.

Examination showed a somewhat demented and emotional woman, laughing and crying without cause. For the most part she was sillily happy but at times querulous. She had no delusions or hallucinations. Her speech was thick. There were marked choreiform movements involving the whole body. The knee jerks were very large but not spastic. The pupillary movements were normal. There was no palsy or ataxia. Now (February 7, 1908) she is very choreic, very emotional, easily pleased and easily frightened.

Paresis is far removed from Huntington's chorea yet once, at least, I was at first misled and was unable to satisfy myself till I had considered the matter for quite a time. The patient, a woman 47 years old, was admitted to the Philadelphia General Hospital in August, 1907. Her father had died of apoplexy when 47 years old and nothing was known of her mother. Her illness began, or rather she was first noticed to be mentally ill, two years before admission while convalescing from pneumonia. She became forgetful and slovenly in dress. For a time she seemed to have visual hallucinations of people. She ceased to do any work about the house, talked to herself, wandered away from home, and sometimes got lost. She imagined she was very wealthy. She began to twitch some months after the onset and later speech became affected.

Examination showed quite marked general choreiform movements. Speech was slurring. Station was good. The pupils were sluggish in response to light, normal for convergence. The

right pupil was a little larger than the left. The outline of each was irregular. Her mentality was too poor to develop any delusions. She was happy and silly. It is not very infrequent in the last stages of paresis to see occasional clonic spasmodic twitchings of different groups of muscles but general choreiform movements are very rare. The diagnosis in this case rests on the physical signs, the mode of onset and the grandiose state.

THE SYMPTOM-COMPLEX OF OCCLUSION OF THE POSTERIOR INFERIOR CEREBELLAR ARTERY: TWO CASES WITH NECROPSY*

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The symptoms of occlusion of the posterior inferior cerebellar artery are so pronounced and usually so sharply defined, that the clinical diagnosis should be easy, although it may be difficult to exclude implication of the vertebral artery. Three American investigators have made a careful study of obstruction of this artery, but with the exception of these papers by Hun and Van Gieson, and H. M. Thomas, I have been unable to find any thorough consideration of the subject in the American or English literature. It is possible I may have overlooked some paper containing investigation in this line.

The symptom-complex of occlusion of the posterior inferior cerebellar artery is as follows: Sudden onset, usually without disturbance of consciousness. Motor power in the limbs and fifth nerve distribution not affected, or at most temporary and slight weakness of the limbs on the side opposite the lesion. Diminution or loss of pain and temperature sensations in the limbs of the side opposite the lesion, and in the fifth nerve distribution on the side of the lesion, or also in the side of the face opposite the lesion; the disturbance of sensation in the face may be only in the first, or first and second branches, or in the whole distribution of the nerve, depending on the upper level of the lesion. Spontaneous pain, or some form of paresthesia in the area of disturbed objective sensation. Tactile sensation usually intact in all parts. Sense of position usually intact but sometimes

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affected. Occasionally a zone of intact sensation in the neck between the zone of disturbed sensation in the limbs of one side and the face of the opposite side. Ataxia in the limbs on the side of the lesion (involvement of restiform body, cerebello-olivary fibers), indicating that the fibers of coördination do not decussate below the medulla oblongata. Tendency to fall toward the side of the lesion; tendency for the head to drop toward the side of the lesion. Nystagmus bilateral, more intense when the eyes are directed toward the side of the lesion (Deiters' nucleus (?)). Ménière's symptom-complex: vertigo, revolving vertigo, auditory hallucinations, vomiting (Deiters' nucleus, vestibular nerve and nucleus, cochlear and vagus nerves). Intense headache occasionally. Disturbance of micturition occasionally. Paralysis of the muscles of deglutition on the side of the lesion (vagus) but causing complete inability to swallow, with impaired sensation of the pharynx. Paralysis of the soft palate on the side of the lesion. Paralysis of the larynx on the side of the lesion, voice hoarse, and speaking is in a whisper (loss of phonation with preservation of articulation). Tongue possibly a little weak on the side of the lesion, but the weakness usually not persistent, as the hypoglossus nerve and nucleus usually escape. Taste lost in anterior two thirds of tongue occasionally. Paresis of the sixth nerve on the side of the lesion occasionally. Paresis of the facial nerve on the side of the lesion occasionally. The paresis of the sixth and seventh nerves is often absent in a lesion confined to the posterior inferior cerebellar artery, and possibly it may be caused by further extension of the area of softening. Sympathetic disturbance; smallness of pupil, narrowing of palpebral fissure, retraction of eyeball, on the side of the lesion. Loss of sweating in the face on the side of lesion occasionally, or increase of sweating in the face on the side opposite the lesion, occasionally. Obstinate hiccough. Hemiasynergy on the side of the lesion (Babinski and Nageotte). Pulse may be rapid from paralysis of the vagus. Loss of tendon reflexes, including the patellar tendon reflex, or in other cases exaggeration of the tendon reflexes. Disturbance of hearing.

With few exceptions I have confined my citation of cases in the literature to those in which a necropsy was obtained.

Dumenil's[1] case 8 reads like one of occlusion of the posterior inferior cerebellar artery, but from the symptoms he describes it

is hard to understand his statement that the area of softening certainly was confined to the restiform body.

Leyden's[2] case 1 might have resulted from thrombosis of the posterior inferior cerebellar artery, but he supposed the cause to be embolism of a small artery, and yet the embolus was not found. The lesion was a small myelitic focus in the right half of the medulla oblongata. Objective disturbance of sensation was not detected. The patient had pain in the forehead on the side of the lesion. Leyden's case 2 presented a myelitic focus nearly in the middle of the medulla oblongata. It was supposed to be from senile softening.

Senator's[3] patient had vertigo, but was too ataxic to stand or walk, though he could move all his limbs, and was obliged to go on "all fours," and had a tendency to fall to the left. He had a sensation of cold in the left side of the face, and speech was disturbed as if by some obstruction in the throat or larynx. The left eye was smaller than the right. Every attempt to move the patient caused vomiting. He had temporary diplopia, also difficulty in swallowing. The pulse was 120. Sensation was much disturbed in the left side of the face and in the right limbs and right side of the trunk. Tactile sensation also was involved. The patellar reflex was lost on each side.

The necropsy showed thrombosis of the left vertebral and posterior inferior cerebellar arteries, and softening in the outer and posterior lower part of the left half of the medulla oblongata.

Eisenlohr[4] reports a case which had much the appearance clinically and pathologically of thrombosis of the posterior inferior cerebellar artery, as the lesion corresponded to that caused by occlusion of this artery, and yet he says no large vessel was occluded, and he regarded the lesion as subacute encephalitis.

In Senator's[5] second case the lesion was left-sided and extended from the lower end of the abducens nucleus not quite to the lower end of the hypoglossus nucleus. The left vertebral artery was thrombotic, and the thrombosis extended 2 to 3 mm. on the pons in the basilar artery. The attack began with vertigo. The patient had paresthesia in the right limbs and the left side of the face. Pain sensation was lost in the face only in the distribution of the second branch of the fifth nerve. Associated ocular movements to the left were lost, and sensation for touch pain, temperature, position and passive movement, and motor

power, disappeared in the right extremities after a second attack. The case was therefore a complicated one from partial thrombosis of the basilar artery.

In Van Oordt's[6] case the lesion was on the left side and at the level of the middle third of the lower olive, and did not extend into the pons. Hemianesthesia was present on the entire right side of the body, limbs and head, but was not complete everywhere. Tactile sensation on the right side was only slightly affected, and sense of location was intact. Sensation was normal on the left side. The lesion was thrombosis of the posterior inferior cerebellar artery.

Reinhold,[7] case 2: A man had suddenly some disturbance of sensation in the left upper limb, a few hours later complete hemiplegia of the left limbs developed without any disturbance of consciousness. The face was not paralyzed. Nystagmus was present in looking to either side, but more intense in looking to the right. Sensation was much diminished in the left side of the body. The movements of the right upper limb were ataxic. There was complete inability to swallow and right-sided laryngeal palsy. The right vertebral artery was thrombotic from the point of union with the left vertebral artery to a distance of about 3 cm. The basilar artery was not thrombotic. The lesion extended from about the lower end of the inferior olive to the upper part of the hypoglossal nucleus, and almost the entire right half of the medulla oblongata was implicated, including the right pyramid. Much mental excitement was present from the beginning of the paralysis, and the patient was unable to sleep even with narcotics. Reinhold believed this could be explained by interference with the circulation of the anterior part of the brain by the thrombosis of the vertebral artery. Inasmuch as the patient was a physician, and doubtless realized fully the gravity of his condition, it may be that the explanation given is not necessary. Although swallowing was impossible, only one side of the pharynx was paralyzed.

In Wallenberg's[8] case the lesion was on the left side, and was occlusion of the left posterior inferior cerebellar artery and frontal part of the left vertebral artery. Vertigo, and pain in the left eye occurring in the beginning of the symptom-complex, and pain later implicating the entire left side of the face, are explained by irritation of the restiform body, vestibular nucleus,

Deiters' nucleus, and the spinal root of the fifth nerve. Slowing of pulse was observed. Diminution of pain and temperature sensations was noted in the right half of the face, and there was also disturbance of the sensations of pain and temperature in the first and second branches of the fifth nerve in the left side of the face. The tactile sensation was not affected. Complete paralysis of deglutition was present. Herpes developed in the areas of disturbed sensation.

The manner in which the symptom-complex of occlusion of the posterior inferior cerebellar artery develops is remarkable. One might well suppose that the destruction of so large an area in the medulla oblongata would be attended by a general disturbance of function. Hun's[9] patient is an example of the manner in which the symptom-complex is likely to develop. The man went to bed feeling fairly well. He awoke during the night with a feeling of a lump in his throat, as if he had swallowed something. In the morning he remained in bed by his wife's advice and not from any feeling of inability to rise. He found he was unable to swallow his breakfast. His voice was hoarse, pulse 80, temperature 99.5°.

In Hun's case there were three areas of softening, but the one which interests us in this connection was in the left side of the medulla oblongata, in the region affected by occlusion of the posterior inferior cerebellar artery. The pathological study was made by Van Gieson. Analgesia and thermic anesthesia were present in the left side of the face and right side of the body. The man was ataxic in the left upper and lower limbs, not in the right. The "muscular sense" was lost only in these limbs. The man showed a tendency to fall toward the left and for the head to fall on the left shoulder, *i. e.*, toward the side of the lesion. This was explained by a lesion of the left restiform body. There was a decided increase in the secretion of sweat on the right side of the face and on the right hand. The fillet was not degenerated, "muscular sense" was lost on the left side, but returned after the first month. Hun attributed the lesion to occlusion of the posterior inferior cerebellar artery.

Ransohoff's[10] case is unsatisfactory, as the clinical history is very deficient. The lesion was on the right side of the medulla oblongata.

In Breuer and Marburg's[11] cases the symptoms were:

Case 1. Right side: hypalgesia on the trunk and limbs. Left side: hypalgesia of face, falling toward the left, ataxia of the upper and lower limbs, disturbance of sense of position in the upper limb, loss of triceps reflex, and sympathetic ophthalmoplegia. Bilateral symptoms were: paralysis of soft palate, paralysis of deglutition.

The left vertebral artery was thrombotic, both posterior inferior cerebellar arteries were intact; therefore the symptoms must be attributed to the occlusion of the vertebral artery and not to occlusion of the posterior inferior cerebellar artery.

Case 2. Right side: mild disturbance of pain sensation on the trunk and limbs (?). Left side: falling toward the left; slight diminution of temperature sensation for cold in the fifth nerve distribution; slight facial paresis; disturbance of hearing; slight weakness of tongue; sympathetic ophthalmoplegia (myosis, ptosis); vasomotor disturbance in the face, and ataxia of limbs. Bilateral symptoms: paralysis of soft palate; paralysis of deglutition; disturbance of senses of position and of passive movement in the upper limbs, slight impairment of lateral movement of the eyes; diplopia, and disturbance of vision.

Thrombosis of the left vertebral and basilar arteries was found following embolism of the vertebral artery.

In the first case the disturbance of pain sensation was in all three branches of the fifth nerve, but was more intense in the third branch. Temperature sensation was unreliable.

The case of Babinski and Nageotte[12] with necropsy was a complicated one. The focus they designate as F.1 corresponds to the area of degeneration caused by occlusion of the posterior inferior cerebellar artery, but three other minute foci were found in the medulla oblongata on the same side. The basilar and left vertebral arteries were thrombotic.

In Mai's[13] case, without necropsy, pain and cold sensations were lost on the left side from the second intercostal space and spine of the scapula downward, and in the right side of the head in the distribution of the first and second divisions of the fifth nerve. The muscles of the throat and larynx were weak, although it seems uncertain whether the larynx was affected. The right palpebral fissure was smaller than the left, and right enophthalmus was present. Sensations of touch, pressure and location were normal everywhere. Sensation of heat was increased in the area

of altered sensation. Herpes of the right upper lip was observed. In the affected areas a sensation of warmth was felt.

H. M. Thomas[14] observed two cases of occlusion of the posterior inferior cerebellar artery. In his first case the symptoms were sudden vertigo, pain in the right side of the face, tendency to fall toward the right, vomiting, slight ptosis of right eye, narrowing of right pupil, loss of sweating on the right side of the face, transient lateral nystagmus, some difficulty in swallowing, disturbance of pain and temperature sensations on the right side of the face and in the left limbs and left side of trunk, and ataxia of the right upper limb. The lesion was thrombosis of the right vertebral and posterior inferior cerebellar arteries. There were no marked microscopical lesions. In his second case the symptoms were: recurrent attacks of numbness in the left side of the face, vertigo, difficulty in speech, tendency to fall to the left, dissociated sensory disturbances in the left side of face and right limbs and right side of trunk, paralysis of left vocal cord, loss of sweating on left side of face, diminution of conjunctival, nasal and pharyngeal reflexes on the left side, and slight ataxia in the left upper limb, more marked ataxia in the left lower limb.

It seems strange that in Thomas's first case, in which a necropsy was obtained, no softening was found in the medulla oblongata, and he explains its absence by the rich collateral circulation, but his seems to be the only case in literature of positive occlusion of this artery without softening in the medulla oblongata.

In Müller's[15] first case the symptom-complex developed suddenly during the night. The patient was awakened with vertigo and a feeling that his bed was moving to the right, and with headache soon followed by vomiting. He had paresthesia (sensation of warmth) in the entire left side, including the left side of the face. The cool bed covering felt hot to the left hand, but warm water was cold to the left hand and left side of the face. In urinating he was obliged to wait ten minutes, and he had constipation, and frequent and painful erection during the first few days. He had also dysphagia, and paralysis of the soft palate and larynx on the right side. Sympathetic paralysis was present on the right side of the face; the palpebral fissure and pupil were smaller than those on the left side. The man had a somewhat hoarse and nasal speech. He was not ataxic, but was weak at

first in the left side, and after three days the right lower limb became weak for a time from the nearness of the lesion to the pyramidal decussation. The triceps and patellar reflexes were more active on the left side, and there was an indication of the Babinski reflex on this side. Pain and temperature sensations were lost on the left side (diminished in the left side of the neck), but tactile sensation and senses of position and of movement were perfectly normal everywhere on the left side. The sensation in the distribution of the right trigeminus was intact, except that in the distribution of the first branch tactile sensation and pressure sensation were diminished. The pulse was a little rapid.

Müller's second patient awoke one morning with the symptoms, having been well the night before. He was probably syphilitic. The symptom-complex was very similar to that of the first case. Anidrosis was present with the ocular signs of sympathetic paralysis. The facial and hypoglossal nerves were not involved in either case. The pain and temperature sensations in the second case were diminished in the distribution of the first branch of the fifth nerve on the same side as the disturbance of those sensations in the limbs and trunk, but tactile and pressure sensations were intact. The trigeminal nerve of the opposite side was not affected. The corneal reflex was diminished in both cases on the side opposite the disturbance of pain and temperature sensations in the face. In both cases the distribution of the upper branch of the trigeminus was more affected than that of the second and third branches. The mucous membranes of the head in both cases seemed to show no disturbance of sensation. The motor power of the limbs was not affected in the second case, but the patellar and Achilles reflexes were prompter on the side of the sensory disturbances. Ataxia was not present.

The symptom-complex in these two cases was: Dysphagia, paralysis on the side of the lesion of the soft palate, larynx and sympathetic supply, and weakness of the corneal reflex; on the opposite side dissociated sensation involving the entire side, and especially of the first branch of the fifth nerve in the face, and perverse temperature sensation. The absence of ataxia in these cases is noteworthy. Death did not occur in either case. Hiccough is not mentioned. The lesion in one case was evidently left-sided; in the other, right-sided. In the second case glyco-

suria and albuminuria were present a short time. The intense headache was attributed to the involvement of the fifth nerve. The vesical symptoms in both cases were regarded as bulbar, they were forcible and continued pressing before the stream could be started after the necessity of urination was felt. The painful priapism in one case was noteworthy (both patients were males).

Duret is said to have been the first to state that usually the left vertebral artery is the seat of obstruction, although he had only embolism in mind, but Senator in 1881 expressed the opinion that the left vertebral artery seems more liable to thrombosis, possibly because its course is more in the direction of the subclavian artery, and also because it has a greater blood pressure.

Wallenberg[16] traced the course of the injected posterior inferior cerebellar artery. He found that frequently there is only one posterior inferior cerebellar artery, and when that occurs it is usually the left that is present. Several smaller arteries, branches of the vertebral, take the place of the missing cerebellar artery, but only for the medulla oblongata; and a larger anterior inferior cerebellar artery replaces the missing posterior cerebellar artery. The absence of the right posterior inferior cerebellar artery I have repeatedly noticed.

Wallenberg sums up Duret's investigations on the supply of the vertebral arteries. Each of these arteries, the left the larger, gives origin about 2 cm. below their union to the posterior inferior cerebellar artery, and higher to the anterior spinal artery. Branches from the latter artery enter the raphe and nourish the interolivary bundles, posterior longitudinal bundles, hypoglossus nuclei and other nuclei below the floor of the fourth ventricle. The pyramids are nourished by the anterior spinal arteries and frequently by the vertebral arteries. [It is understandable therefore that when the anterior spinal arteries are not occluded the central and anterior parts of the medulla oblongata are not affected.] Branches from the posterior inferior cerebellar or vertebral artery supply the lateral portion of the medulla oblongata. The posterior inferior cerebellar artery supplies the restiform body. These are all terminal arteries. According to Wallenberg the posterior inferior cerebellar artery is given off 12 to 20 mm. below the union of the vertebral arteries. The free anastomosis of the terminal portion of the posterior inferior cerebel-

lar artery explains the escape of the cerebellum in occlusion of this artery. I have, however, observed an abscess in the terminal distribution of this artery in the cerebellum, without any evidence of implication of the medulla oblongata.

The study of Breuer and Marburg[17] shows that clinically the diagnosis as to whether the vertebral artery or the posterior inferior cerebellar artery is occluded cannot be made with certainty, as the anterior branches of the vertebral artery may supply much the same area (lateral and posterior part of the medulla oblongata) as does the posterior inferior cerebellar artery. Great variations occur in the distribution of the blood vessels. Thus in Breuer and Marburg's first case the vertebral artery was occluded and the posterior inferior cerebellar artery escaped, and yet the lesion occupied about the same region as in those cases in which the latter artery was thrombotic. Breuer and Marburg state therefore that in the majority of cases the diagnosis cannot be made more exactly than that of a lesion of the vertebral artery. When symptoms indicative of pontile lesion occur, the vertebral artery is probably occluded rather than the posterior inferior cerebellar artery. Thrombosis of the vertebral artery, however, may give a different clinical picture from that of thrombosis of the posterior inferior cerebellar artery, in that it presents certain additional symptoms. Especially is this true when the thrombus extends into the basilar artery.

Eisenlohr[18] reported two cases, nos. 3 and 4, in 1879; in one (case 3) the symptoms were suddenly developing right hemiplegia, without loss of consciousness, with disturbance of speech and swallowing, complete paralysis of the tongue eight days later, of the pharynx, complete loss of speech, paresis of the left facial nerve, and partial paralysis of the larynx. In the other (case 4) the symptoms were: Sudden paralysis of the right arm, paresis of the right leg, disturbance of articulation and swallowing, complete loss of speech a few days later, inability to swallow, and paralysis of the larynx.

Eisenlohr mentions that Lichtheim regarded as diagnostic of occlusion of a vertebral artery sudden loss of speech and of the movements of the lips and tongue, paralysis of the larynx and paralysis of the limbs of one side. In Reinhold's case of thrombosis of the vertebral artery hemiplegia occurred.

In my cases of thrombosis of the posterior inferior cerebellar

artery, and in most other cases with this lesion, hemiplegia, complete loss of speech, and paralysis of the tongue were not symptoms.

In 1888 Eisenlohr reported the findings in his two cases. In case 3 the vertebral arteries were narrowed and obstructed and numerous branches of the basilar artery entering the pons were obliterated. It seems uncertain whether either vertebral artery was completely thrombotic, but it is so implied. In case 4 the important lesion was old thrombosis of the left vertebral artery with several small foci in the lower half of the pons.

In some of the papers it is distinctly stated that prodromal symptoms preceded the apoplectic attack. Thus Senator's patient felt badly for a few days before the apoplectic insult, and this probably was caused by the beginning thrombosis, or at least, disturbance of the circulation. In the clearly marked clinical picture weakness is not great, but inability to walk may exist because of the marked ataxia (Senator).

The ataxia may be caused by the lesion of the inferior cerebellar peduncle, or of the olivary cerebellar fibers, or of the lower olive, or of the fibers of the vestibular nerve and its nucleus, or of Deiters' nucleus. In some cases ataxia is very striking, thus Senator's patient was obliged to go on all fours, as he could not stand; in my first case also standing at first was impossible, probably largely because of ataxia. The ataxia is usually on the side of the lesion, and may be nearly confined to this side (Reinhold, Hun, Breuer and Marburg, Thomas).

The thrombosis was found on the left side in the cases of Senator (two cases), van Oordt, Wallenberg, Hun, Breuer and Marburg (two cases) and Babinski and Nageotte; and on the right side in the cases of Reinhold, Ransohoff, and Thomas.

The diplopia mentioned in some of the cases is usually temporary, and probably is caused by an interference with the function of the sixth nucleus. My second case shows that at least the lower part of this nucleus may be implicated in the softening. In Senator's second case the lesion extended to the lower part of the nucleus. The abducens nerve was weak on the side of the lesion in Henschen's clinical case.

Weakness of the face occurred in the cases of Breuer and Marburg (case 2), and in my case 2. Degeneration of fibers

in the facial nerve explained the facial weakness in the second case of Breuer and Marburg.

Weakness of the tongue was observed in Breuer and Marburg's case 2.

Difficulty in swallowing probably exists in every case, and probably is caused by implication of the nucleus ambiguus. The paralysis is probably unilateral, but the loss of function is complete (Reinhold, van Oordt, Wallenberg, Spiller, two cases). It was complete in a case of unilateral fracture of the base of the skull with paralysis of the vagus on only one side reported by me.[19] The patient was entirely unable to swallow.

Vomiting in some cases is very distressing. Every attempt to move Senator's patient produced it, and in my case 1 it was very intense and could not be controlled.

Slowing of the pulse was observed by Wallenberg, and was attributed by him to irritation of the vagus nucleus.

I have been unable to find any satisfactory explanation for the loss of the tendon reflexes in some cases, and the exaggeration in others; but this variation in these reflexes occurs also in cases of cerebellar tumor. The patellar reflexes were lost in the cases of Senator, van Oordt (except feebly present on reinforcement) and Spiller. In Hun's case the patellar reflex was normal on the right side and a little exaggerated on the left side. Rossolimo[20] mentions diminution of the patellar reflex and of other tendon reflexes, unilateral or bilateral, in five cases of lesions of the brain stem.

The disturbance of the sympathetic fibers in the face on the side of the lesion is very common, and consists of narrowing of the palpebral fissure, narrowing of the pupil, and retraction of the eyeball. Disturbance of sweat secretion probably belongs to this symptom group. All these symptoms are not present in every case. They occur usually on the side of the lesion, and therefore demonstrate that the sympathetic fibers entering the lower part of the cervical cord do not decussate below the upper part of the medulla oblongata. In Hun's case sweating was increased in the face and hand opposite to the lesion. Vasomotor symptoms in the face on the side of the lesion were observed by Breuer and Marburg in their second case. Ptosis also has been observed.

In both cases of Breuer and Marburg the left side of the soft

palate was more affected, but both sides were weak. In Hun's case and in van Oordt's the paralysis of the soft palate was bilateral, and this is explained by Breuer and Marburg as the result of bilateral innervation from each nucleus. The nucleus ambiguus, they believe, controls the muscles of deglutition, the larynx and the soft palate.

Disturbance of speech has occurred in most of the cases. It is explained by the implication of the nucleus ambiguus, as this nucleus usually forms the center of the lesion.

The nucleus ambiguus is probably the center for the laryngeal muscles. Van Gehuchten and de Beule believed that it had no connection with the larynx, inasmuch as they found degeneration of the dorsal nucleus after section of the laryngeal nerves. Kohnstamm and Wolfstein[21] have cut the recurrent laryngeal nerve on one or both sides in rabbits and dogs, and have always found the dorsal nucleus intact, but the nucleus ambiguus on the side of the division was degenerated. They therefore regard Van Gehuchten's views as incorrect.

Vertigo is not uncommon (Senator, van Oordt, Thomas, Müller). In Henschen's case the symptoms were those of Ménière's disease—revolving vertigo, hallucinations of hearing, and vomiting—and were explained as a result of lesion of the vestibular nerve or of Deiters' nucleus, cochlear nerve, and vagus.

Subjective sensory disturbances are common. Senator's first patient had a sensation of cold in the face on the side of the lesion; his second patient complained of paresthesia in the limbs opposite to the lesion, and in the face on the side of the lesion. Wallenberg's patient had pain in the eye and entire side of the face, on the side of the lesion. In Hun's patient tingling was felt spontaneously in the face on the side of the lesion, and in the limbs opposite to the lesion following slight friction, *i. e.*, in the analgesic regions, and was evidently a symptom of irritation. Pain was complained of in the face on the side of the lesion in one of Thomas's two cases.

Deep sensation was lost in Hun's case on the side of the lesion, about one month and then returned. The fillet was not found degenerated at necropsy, but the transitory disturbance of the deep sensation may have been due to transitory implication of the fibers forming the fillet.

Sense of position was disturbed in the upper limb on the side

of the lesion in one case of Breuer and Marburg, and this they explain by the involvement of the fibers from the nuclei of the posterior columns, especially those from Burdach's nucleus, as these are more exterior.

Tactile sensation usually escapes, but temperature and pain sensations are likely to be diminished, or lost, usually in the face on the side of the lesion, and in the limbs and trunk opposite to the lesion (Senator, Hun, Breuer and Marburg). Tactile sensation may in some instances be impaired, and was so in the cases of Senator. It is possible that the explanation for tactile anesthesia is to be found in implication of the fillet, as some investigators believe that this fasciculus contains tactile fibers.

Hemianesthesia, partial in places, was present in the entire side, including the face, opposite to the lesion, in van Oordt's case; the tactile sensation was only slightly affected. The statement he makes is "Hemianesthesia vom Scheitel bis zur Sohle."

In Wallenberg's case the diminution of pain and temperature sensations in the face were on the side opposite to the lesion, but only for a few days, and this was attributed by him to implication of the central tract of the fifth nerve after the fibers had decussated. In this case pain and temperature sensations were diminished also in the face on the side of the lesion in the first and second branches of the fifth nerve, and this was explained by the involvement of the spinal root of the fifth nerve on the side of the lesion.

The central tract of the fifth nerve, according to Wallenberg, is very near the median fillet in the medulla oblongata, and in front of the hypoglossus nucleus. A lesion to involve this tract must extend nearly to the raphe. The tract passes from the substantia gelatinosa dorso-medially, ventral to the hypoglossus nucleus, across the raphe to the opposite side.

The pathology of the spinal cord has shown with certainty, according to Mai, that a lesion of the spinal root causes complete loss of sensation, whereas a lesion of the central tract causes dissociated sensation. This statement, I think, is questionable.

According to Wallenberg and Schlesinger the first branch of the fifth nerve descends lowest in the spinal root. A lesion corresponding to the lowest and middle thirds of the spinal root of this nerve would give disturbance of sensation of the first and second branches of the nerve.

Henschen,[22] in reporting a clinical case with symptoms such as are observed following occlusion of the posterior inferior cerebellar artery, remarks that tactile fibers ascend in the posterior columns and are next the raphe, therefore they usually escape in these bulbar lesions. He says that he has demonstrated by several cases that destruction of the fillet causes a loss of tactile as well as of pain and temperature sensations, therefore the latter fibers enter the fillet more proximally than the medulla oblongata. If ataxia is on one side and disturbance of pain and temperature sensations is on the other, the lesion is more caudal than when the disturbances are on the same side.

Henschen's patient had normal taste on the posterior part of the tongue, but taste was much affected in the anterior two thirds of the tongue. The entire distribution of the left sensory fifth nerve was affected in his case, so that pain and temperature sensations were lost in this area while tactile sensation was preserved.

Analgesia and thermanesthesia in the trunk and limbs of the side opposite the lesion is explained by Müller by the crossing of the spinal tracts for pain and temperature soon after their entrance into the posterior horns, and the ascent of these fibers in the tractus spinotectalis and tractus spinothalamicus.

From his two cases Müller concludes that the substantia gelatinosa must be regarded as a continuation anatomically of the cervical posterior horn, its disturbance must give sensory symptoms of the posterior horn type, *i. e.*, implication of temperature and pain sensations. The central fibers of this trigeminal nucleus must decussate soon, and the crossed tract would be implicated in the lesions of Müller's two cases. His second case showed the Brown-Séquard symptom-complex in the distribution of the fifth nerve; deep sensation and tactile sensation were diminished in the territory of the first branch on the side of the lesion, and pain and temperature sensations were diminished on the opposite side. This indicates, he thinks, that the fibers of deep sensation and tactile sensation in the spinal root of the fifth nerve have their central tract uncrossed in the medulla oblongata. The first branch has been shown to be connected with the distal end of the terminal nucleus, and this Müller explains by the fact that in the lower vertebrates the first branch (the forehead) does not extend forward as far as the second and third branches. In the conus

the lowest roots supply the anal region, which in animals is posterior to the limbs.

In case 1 of Kutner and Kramer the sensation of the neck was not disturbed, and this escape is explained by the gradual crossing of the sensory fibers from this region, so that they were not all caught in the lesion. In occlusion of the posterior inferior cerebellar artery there may therefore be a sensory zone intact between the disturbed area of the face and that of the opposite side of the body.

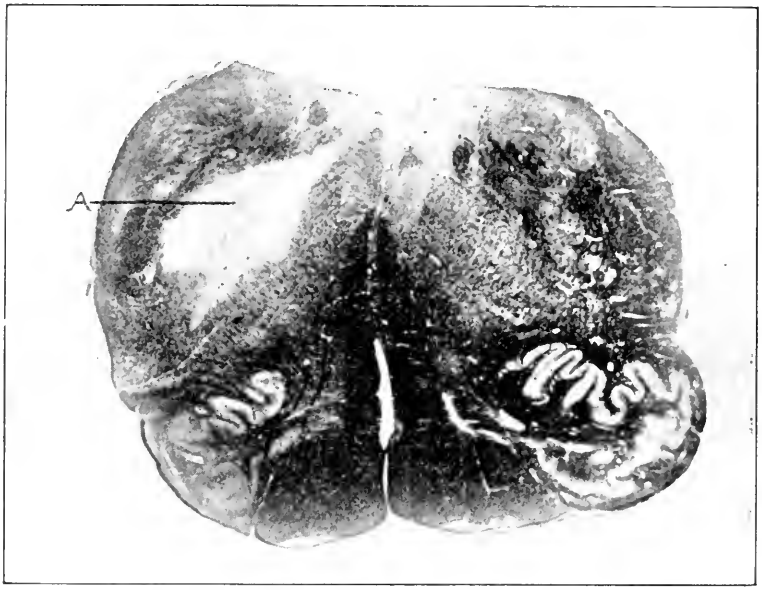

FIG. 1. Case I. Showing the lowest level of the lesion A.

My two cases are as follows:

CASE I. Douglas, male, was admitted to the Philadelphia General Hospital, to my service, March 6, 1907. He died March 12, 1907.

Pathological Diagnosis.—Catarrhal and ulcerative enterocolitis, hemorrhagic fibrinous peritonitis; localized chronic adhesive pleurisy, emphysema and edema of lungs; dilatation of right heart; chronic perisplenitis; fatty metamorphosis of liver; cloudy swelling of kidneys; chronic interstitial cystitis.

Notes taken on March 6 are as follows: Chief complaint: Inability to swallow, weakness in left upper and lower limbs. He had a chancre fifteen years ago. Alcohol and tobacco have been used to excess. He has not felt well for three weeks, and

stopped work about a week ago, but returned to it again and worked several days. The bowels had not moved for a week; he began last Sunday to take salts after having had his usual cathartic dose on Saturday, and took large doses all day Sunday and during the night. His bowels did not move until Monday morning, when they acted repeatedly, and he had a severe chill. At nine o'clock Monday morning he drank a glass of hot milk and this is the last time he remembers swallowing anything.

At eleven o'clock he tried to get out of bed, but found that he was unable to stand or walk. He slid downstairs to the second

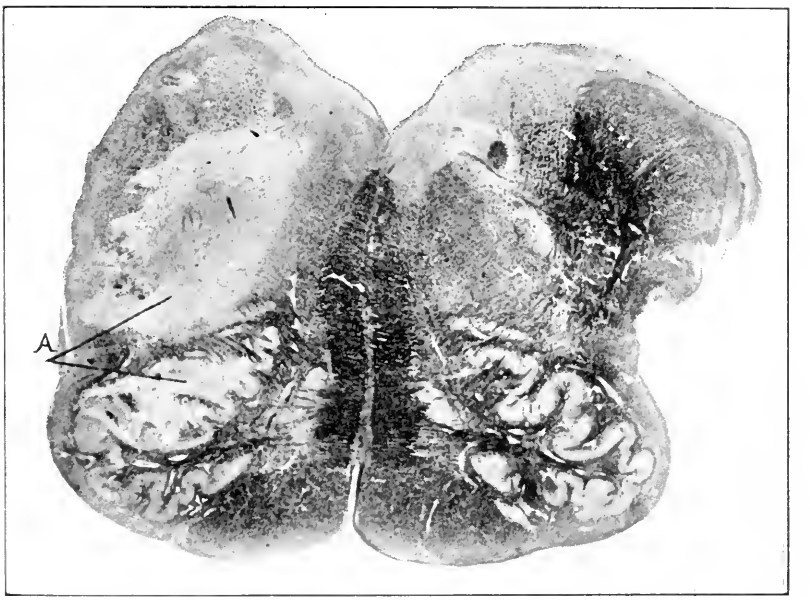

FIG. 2. Case 1. Showing the middle level of the lesion *A*.

floor and called to the family and was carried back to bed. He then found he was unable to drink the milk which was brought him; although he states that he noticed that he was unable to swallow saliva when he first tried to get out of bed. Before he got out of bed he felt dizzy, things swam around, he saw specks before his eyes and then couldn't see at all, although he did not lose consciousness. He had a spasm of the right arm followed by numbness. At present he is unable to swallow anything. He has numbness and some loss of power on the left side. He complains of seeing double and has persistent hiccough. There is no involvement of the bladder or rectum. He is not able to speak above a whisper, although he articulates distinctly. The facial muscles seem to be normal.

My examination was made March 8, 1907, and gave the following results: Ptosis of each eyelid is slight and equal on the two sides. He wrinkles the forehead well on each side, closes his eyelids, shows his teeth, draws up well either corner of the mouth separately. The right pupil is a little larger than the left. Reaction to light is present in both eyes but slow. Reaction is prompt in convergence and accommodation. Movements of extraocular muscles are normal. No double vision is noticed at present. The masseter muscle contracts well on each side. In opening the mouth the jaw does not deviate toward the right

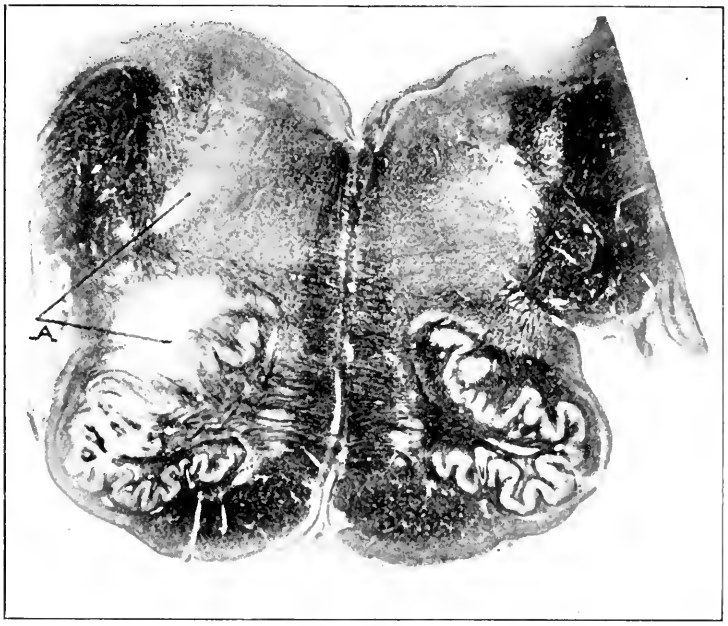

FIG. 3. Case 1. Showing the highest level of the lesion *A*.

until the mouth is quite widely open, when the lower jaw goes suddenly to the right, as though there were some defect in the articulation of the lower jaw on the left side. The tongue may deviate slightly to the right, but this is questionable.

Sensations of pin prick and touch are normal on the two sides of the face. The soft palate when the man says "ah" is drawn up more on the right side than on the left, this is distinct. He is entirely unable to swallow. The nasal tube is passed without difficulty to a distance of 62 cm. and there seems to be no obstruction to it. He speaks distinctly, but merely in a whisper and expectorates constantly. There is no deafness to the voice and he hears a low ticking watch at a distance of six inches in

each ear. The tongue while in the mouth deviates toward the right, more to the right while in the mouth than when it is protruded. The hiccoughing, while constantly present during the observation, has a tendency to occur in attacks of increased severity. The grasp of each hand is good. Biceps tendon reflex and triceps tendon reflex are present, but not very distinct on either side. The movements of the upper limb are free on each side. Sensations of touch and pain are normal in both upper limbs and there seems to be no involvement of either upper limb. Resistance to passive motion is equally good in either upper limb. There is no wasting of the upper limbs. He has little or no tend-

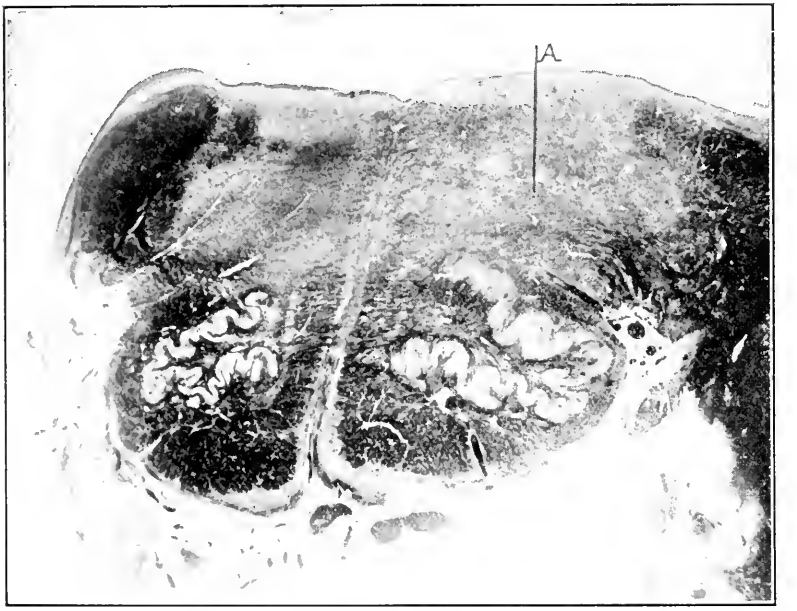

FIG. 4. Case 2. Showing the lesion *A*. The section is reversed in the photograph.

ency to cyanosis of the fingers at present, although Dr. Evans, the resident physician, said the cyanosis was pronounced yesterday morning.

There is no cardiac murmur and no accentuation of the second pulmonic sound. No distinct weakness of the lower limbs is detected. It is very doubtful whether the right lower limb is weaker than the left. The patellar tendon reflex is lost on each side, even on reinforcement. Heel to knee test shows some ataxia on each side. Achilles jerk is lost on each side. Sensations of touch and pain are probably normal in each lower limb, although

sensation of pin prick may be somewhat diminished over the right sole. The gait and station are somewhat ataxic. The ataxia is especially pronounced when he is standing with his feet together and eyes closed. He has never had any severe pains in his legs. He says he did not stagger when he walked on the street.

I was unable to see the larynx on account of the epiglottis. Dr. Grayson later made an examination, but the paralysis of the epiglottis prevented him from seeing the vocal cords. He intended to bring suitable instruments and make another attempt, but did not succeed in doing so before the death of the patient.

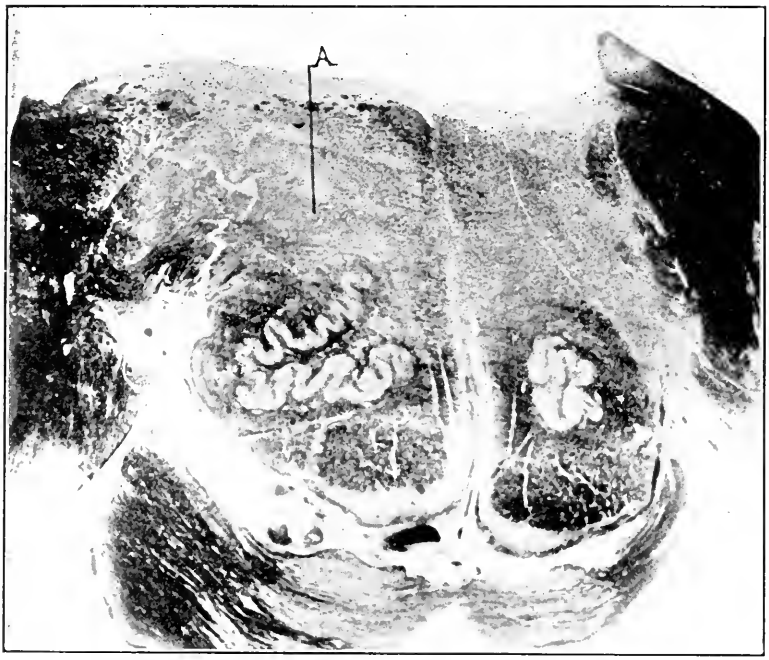

FIG. 5. Case 2. Showing the lesion *A*.

Pulse is 100 at present. Unfortunately the temperature sensation was not tested. Dr. Knipe reported on March 9, 1907: O. D., pupil oval, 4 to 5 mm.; O. S., pupil, 3 mm.; both pupils react faintly to light and convergence; O. D., media clear, fundus negative; O. S., media clear, disc has slightly pale yellowish cast, but no haziness; normal-sized vessels; tension normal.

March 11, 1907: Pulse is 124. Hands and feet are very cyanotic. The vomiting continued last night and this morning; he is weaker. The radial pulse is weak on the right side and is scarcely detectable on the left side. The throat is not very sen-

sitive to manipulation. 11.30 P. M., pulse is imperceptible. He died 1.45 A. M., March 12, 1907. His pulse while he was in the hospital ranged from 80 to 160, and frequently was as high as 120 to 130. Respiration 20 to 30 or 35; temperature on several occasions reached 101 or 102.

The softening with hemorrhagic infiltration is on the left side of the medulla oblongata and extends to the descending root of the glossopharyngeus. It implicates a large part of the restiform body, the anterior part of the descending root of the eighth nerve, a part of the spinal root of the fifth nerve, and the posterior part of the lower olive. It does not extend quite to the periphery of the medulla oblongata, nor does it implicate the lemniscus or nucleus of the hypoglossus. The area of softening in its upper part almost disappears at the level where the restiform body begins to pass into the cerebellum, and in its lower part it is very small at the upper part of the hypoglossus nucleus. The escape of a portion of the sensory root of the fifth nerve, of the fillet, and of the periphery of the medulla oblongata explains the preservation of sensation. The ease with which the nasal tube was passed suggests anesthesia of the pharynx from implication of the vagus and glossopharyngeal nerves.

The diseased area stains a light brown by the Weigert hematoxylin method and contains here and there small recent hemorrhages. The intramedullary portion of the hypoglossus nerve is intact. The nucleus ambiguus is in the degenerated area, and some cells in the position of this nucleus are swollen and greatly degenerated. The anterior pyramids are intact. The nucleus of the hypoglossus on the affected side shows a few degenerated cells, the hypoglossus nucleus of the opposite side is intact. A slight perivascular round cell infiltration is seen here and there in the medulla oblongata. The vessels in front of the pyramids are not notably thickened, but probably are a little so. The pia shows a slight round cell infiltration. Many of the axis cylinders in the necrotic area are much swollen, and this area contains many fatty granular cells.

The left ninth and tenth nerves do not show any degeneration by the Marchi method. No change is detected in a piece of muscle from the left vocal cord, even by the Marchi stain, nor can any change be detected in muscle from the left side of the soft palate by the Marchi stain. It is probable that the duration of life after the lesion occurred was not sufficient for degeneration to be detected even by the Marchi method.

The left vertebral artery is occluded by a thrombus and distended just below where it joins the basilar. The left posterior inferior cerebellar artery shows a partly organized clot.

Case II. Dawson. Male, age 30 years, bartender, was admitted to the Philadelphia General Hospital, to my service, March 27, 1907. He died the same day.

Pathological Diagnosis.—Chronic catarrhal gastritis; active hyperemia of kidneys; edema and congestion of lungs; chronic fibrous pleurisy; right omental hernia.

A history was not obtained from the patient, as he was unable to talk sufficiently, and lived but a few hours after he came to the hospital. He came with an ambulance history of facial paralysis. Duration of illness was one week.

March 27, 1907. Last Monday, three days previous to his admission, just after the patient had arisen in the morning, he felt weak and dizzy. As this feeling passed he found that he was unable to swallow anything, even saliva, and that the left side of the face was very weak; that when he opened his mouth it was drawn strongly to the right. Since then he has been unable to eat or drink and has taken no nourishment for three days.

He has ptosis of the left upper lid. When he shows his teeth his mouth is drawn strongly to the right side. His tongue is well protruded, but deviates apparently to the right, although this may be due to his mouth being drawn so far to the right. His pharynx is anesthetic, as is shown by passing the nasal tube without causing him any annoyance. He is unable to swallow, the fluid passes to a certain distance and then is coughed out. He is able to speak only in a whisper and with much difficulty. His upper and lower limbs on each side are apparently unaffected, as are also the sphincters of the bladder and rectum. His mind is clear, and he is a man of fair intelligence.

The lungs seem clear. He expectorates a considerable amount of grayish mucus. Heart sounds are fairly good. Pulse is 120. Respiration, 40; temperature, 97.

The area of degeneration extends from the level of the upper part of the twelfth nucleus to that of the lower part of the sixth nucleus, and implicates the posterior half of the fillet. A section made at the lower part of the pons where it joins the medulla oblongata shows that a large part of the posterior part of the left side of the section is in recent degeneration. The sixth nucleus is slightly implicated at its lowest portion. The necrotic area stains faintly by the Weigert hematoxylin method. It extends to, but not beyond, the raphe in the pons, and almost to the floor of the fourth ventricle. The restiform body is not implicated, and the spinal root of the fifth nerve only slightly so. The area extends to, but does not invade the lower olive; but implicates the nucleus of the facial nerve. The degenerated area contains numerous small hemorrhages, and is like that seen in the former case, although it is a little higher. The necrotic area contains numerous swollen axis cylinders. The perivascular round cell infiltration is very intense within the medulla oblongata, as is also the round cell infiltration of the pia. The sections present the appearance of syphilis. The basilar and left vertebral arteries are thrombotic, and both are partly occluded by pro-

liferation of the intima. The left vertebral artery is much larger than the right. It is doubtful whether the clot in the basilar artery existed during life. The left posterior inferior cerebellar artery is filled with red blood corpuscles which do not form a distinct thrombus.

The head of the right caudate nucleus is in intense acute degeneration and is filled with fatty granular cells.

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NEW YORK NEUROLOGICAL SOCIETY

January 7, 1908

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

A CASE OF PROBABLE ENCEPHALOMYELITIS

Wm. M. Leszynsky, M.D.

A girl, seven years old, on September 5, 1907 (four months ago), developed a chill, occipital headache, vomiting, fever 105° with mild delirium lasting five days. She was taken ill three days after her brother was attacked (see report of case following). She had pain and tenderness in the right shoulder and arm, and both legs. Within twenty-four hours a flaccid paralysis appeared in the right arm and both lower extremities. Improvement began on the tenth day and was progressive. Four weeks later there was slight difficulty in arising from the floor on account of weakness of the lumbar and hip muscles. She walked well without assistance. There was paralysis of the left tibial, peroneal and crural groups. Both knee jerks were present. On the right there was pseudo-clonus. There was paresis of the right deltoid, triceps and shoulder girdle, with pronounced rigidity (adduction of arm, flexion and pronation of forearm and hand) which could be overcome by force. There was no local atrophy. The faradic irritability was preserved in all the paretic muscles. There was no disturbance of sensibility. On December 26, the lower extremity had recovered and there was only slight paresis and rigidity in the shoulder muscles. This was probably a case of encephalomyelitis.

A CASE PROBABLY OF MENINGOMYELITIS

By Wm. M. Leszynsky, M.D.

A boy, aged two years and eight months, a brother of the patient above reported. On September 2, 1907 (four months ago), he had a chill, fever 104° with mild delirium, vomiting, occipital headache. Within six hours he had pain in the legs and lumbar region. Twenty-four hours later, both lower extremities were paralyzed, including the lumbar and hip muscles. He gradually recovered at the end of the second week. The improvement was progressive. Four weeks later, he was well nourished, his gait was waddling and of a peculiar oscillating character, the muscular power and resistance good in both lower extremities. There was paresis of the lumbar and hip muscles, both knee jerks and plantar reflex were normal and there was a very slight lordosis. The child got up from the floor by climbing up his legs as in pseudo-hypertrophic paralysis. Otherwise he was in perfect health. On January 3, 1908, he was very much improved. This was probably a case of meningomyelitic process.

Dr. Leszynsky presented these two cases, a brother and sister, in order to demonstrate an anomalous clinical type of infectious disease

occurring during the recent epidemic which had been loosely characterized as an epidemic of poliomyelitis. In this epidemic, as was well known, many cases appeared which, under ordinary circumstances, would never have been classified in the category of acute anterior poliomyelitis.

PSYCHOTHERAPY

By C. L. Dana, M.D.

Dr. Dana said he was conscious of a serious responsibility in bringing before this society the subject of psychotherapeutics. One reason is that the subject, according to certain Boston observers, when mentioned to New York neurologists, arouses not thought but emotion, not intellectual consideration, but emotional explosion.

Some subjects are so essentially trivial, and so beyond the realm of sense and scientific treatment, as to deserve this attitude, and invite only this emotional complex. Psychotherapy is not, however, quite deserving of such treatment, for if it is not worthy of scientific study it demands attention as a social question.

There is already a wave of public interest in the matter, which we at least must watch if we take no definite attitude toward it. It seems as if a certain large group of minor psychoses are to be taken in hand by the clergy, coöperating with medical men, and it is seriously proposed to have psychotherapeutics a part of the religious work of our churches. Already centers of this work exist in Boston, Chicago and this city. Perhaps the best attitude to take towards this movement is aloofness, believing that it will die out.

His own feeling, however, is that the best way would be to take a decided position against it, certainly as a general measure. We can reasonably assert that the care of the sick is safest in the hands of those trained for the purpose. But if we say this, will we not have to assert also that medical men are using the forces of therapeutics, and using them more skilfully and effectively than clergymen, or non-medical therapeutists can do. Dr. Dana is persuaded that this is not now the case. We do use psychotherapeutics in our daily work in various formal and informal ways, but in the casual work of the office, it is not always possible, or at least it is not always the practice to bring out to the utmost the power of hopeful attention, or to dislodge the demon of the subconscious effectively. By studying and analyzing the different methods of psychotherapeutics, we may be able to use them better, and to show its limitations, for they are very great, and its possibilities, for they are important.

THE REEDUCATION METHOD OF DUBOIS

By Smith Ely Jelliffe, M.D.

In his opinion faith was one of the corner stones of psychotherapy, but it should be a faith, separated from superstition, with which it had always been associated, a faith raised to the level of knowledge, so far as the physician might be able to see it, and so far as the intelligence of the patient might be able to grasp this should be the aim of the psychotherapist in using the reëducation method. In the earliest phases of the so-called temple sleep, he took it that one saw one of the most primitive expressions of a medico-theological therapeutic application of

the principles of psychotherapy. He purposely limited his remarks to the consideration of one symptom group which, although hampered by the misinterpretation of ancient, and even our own times, must need blindly grope about in its protean and bizarre aspects as hysteria. Hysteria considered as an entity appeared like a gigantic proteus and escaped all definitions. Bernheim, Schnyder, Dubois, and many others had protested against this nosological conception and the former reserved for the crises alone the term hysterical. They were with Bernheim when he said "Hysteria is not a morbid entity," but, in his opinion, the crises constituted the abnormal psychical process to which one could not without inconvenience apply the denomination hysterical. All in all, hysteria was the psychopathic manifestation par excellence, the most primitive expression, the most common of the inherent weaknesses of the human personality. After speaking of the psychical characteristics which almost inevitably showed the hysterical personality, he said the hysterical manifestations appeared in mentalities very different one from another. It was for this reason that hysteria presented itself under different aspects according to the ethnic and social condition of the individual. They should note the different characters offered by infantile, male, and female hysteria. Hysterical manifestations might appear in connection with the more frank insanities, and they had been noticed along with general paralysis. They had also been found associated with other organic affections of the nervous system, and they might take part in the clinical picture of certain intoxications. Finally they impressed upon certain purely neurasthenic conditions certain characteristics which justified the term hystero-neurasthenia. One of the most striking features of Dubois' point of view was to deny to hysteria the character of a morbid entity, and to describe it as one of the psychic changes which were included under the name psychoneuroses. Dubois was not alone in this, since this tendency was evident in Babinski's definition as well as in that of Crocq and others. One hysterical sign did not make hysteria. Hysterical conditions arose by insensible transitions from ordinary mentality. The hysterical modifications of the mentality were established by exaggeration, and the perversion of the psychic and psycho-physical reactions of suggestibility. Hysteria in an adult was closely allied to certain psychical conditions which represented, in fact, a mental retrogression towards the infantile type, of which the chief characteristic was a lack of logical judgment. The importance of moral causes in the development of hysterical states could not be overestimated. In a general way, one might say then with Schnyder that the hysterical modifications of the mentality had as their starting point a defect of judgment, or lack of mental synthesis, which led to an erroneous conception of real situations, and a consequent inability to adapt oneself to them. Hysteria in its pure form was a disease of youthful individuals; one rarely met it in an old man whose mentality had reached its full development. Schnyder believed that he could show philogenetically that hysteria had been the attribute of evolutionary phases of humanity; it appeared in history each time that the aspirations of the human mind were held back and repressed by the laws of the established order of the age. With Schnyder and Moebius, he did not believe that hysteria could be considered as the attribute of a people whose civilization was too refined, unless it was the expression of the decadent period of a people. Certain classes of individuals likewise presented a mentality

which offered a favorable soil for the culture of hysteria. There were, on the one hand, the dwellers in rural communities and, on the other, the workmen of the proletariat. There might still be found in some remote village, an epidemic of hysteria which recalled the classic instances of the kind described in the Middle Ages. Another type written about, particularly by the French school, regarded hysteria only as a manifestation of mental degeneracy. There was no question but that one might say it was a manifestation of mental insufficiency.

He had laid considerable stress upon that aspect of hysteria which might be called *evolutive*, forming, in fact, only an accident in the mental evolution of the individual or of the species. There remained to be discussed a second group developing not on a simple healthy soil, but on that of a distinct neuropathic heredity, a group which they had for a long time termed the degenerative hysterias. The degeneracy might lead the mentality toward an inferior type which it corresponded to in some particular, as the infantile type. The psychic change was often partial. It was in this group that they found the great disproportions in mental capacities. The kind that made the demi fous of the world. Symptomatic hysteria constituted one of the chief stumbling blocks in the path of psychotherapy. He asked if hysteria constituted a morbid modification of mentality more frequently at the present time than formerly, and what was the future of hysteria, questions that interested both the physician and the psychologist. Statistical study of hysteria permitted them to state that certain forms of hysteria had diminished in frequency as the result of the progress of civilization. Among the disturbing features for the modern individual were impatience, irritability, immoderate ambitions, etc. Modern nervousness was translated into neurasthenic or psychothenic forms more than by purely hysterical forms. Hysterical conditions were still strongly impregnated by mysticism, imagination, fantasy, naivete and puerility which belonged, so to speak, to the golden age of nervousness. Hysteria still remained the appanage of primitive mentalities. He believed that the era of the grand manifestations of collective hysteria might be considered as closed. The moral education of man still rested in a large degree on the principle of authority transmitted by the Church; it made the practice of duty depend upon the fear of punishment and the hope of reward. If psychology was absolutely indispensable for the analysis of the mechanism of hysterical phenomena, it could not in itself alone resolve the complex problem of the origins of the hysterical modification of mentality. To all psychological conceptions of hysteria there must be associated a moral conception of it. It was chiefly this moral conception which ought to inspire all rational treatment of hysteria, such as had been indicated by Dubois in maintaining for the psychoneuroses a *moral treatment* in the highest sense of the word.

FREUD'S METHOD OF PSYCHOTHERAPY

By L. Pierce Clark, M.D.

He said that at the present time Freud limited himself to the so-called analytic method for subjective reasons. He believed it was the only method capable of going beyond itself and leading to other fields of therapy. Freud's analytic method was often confounded with hypnotic suggestion. In reality analysis was directly opposed to suggestion.

Freud's method was inconvenient for the average physician and should be employed only by the expert and then only on the most desperate and rebellious cases, refractory to other plans of treatment. Freud's method showed poorly in the neurotic degenerates. It was not applicable to patients without keen insight into their disorders and therefore had limited use in the pure psychoses. In a general way those above fifty years of age were not applicable. The usefulness of the method was not so limited as one was apt to think when he bore in mind that all chronic hysteria, impulsive states, aboulias, etc., were amenable to this special treatment. Analytical psychotherapy of Freud was grounded on the unconscious psychical process of the patient. The treatment consisted in translating the unconscious to the conscious and thereby in correcting deviations from normal psychical processes, and in removing from the patient the impulsions which he labored under. The sex idea of Freud's analytical method was grounded on his firm conviction that the sexual element was in the last analysis supreme as a factor in causing hysteria; in tireless reiteration he emphasized the necessity of considering both the sexual alteration and sexual repulsion together. The technique was as follows. Freud usually had the patient fully relaxed in a favorable environment, cutting off all sense stimuli. Then with hand on patient's brow he urged the patient to search the memory for any forgotten painful experience, usually involving sex. Under favorable circumstances such experiences are mentioned early in the examination, or if at first the patient resisted, the "confession" when it came, came all the more sharply and clearly. Freud's method was of great value in many cases, particularly the hysterias, but it was not widely applicable in general disorders of the minor neuroses until the sexual idea was eliminated. The advantage of Freud's method over hypnotism in eliminating the curiosity of the physician to the patient's disadvantage, was quite obvious. Freud's method was cumbersome. It might be noted that hardly any one, according to Freud's sexual theory, could escape the charge of being neurotic, either having too much or not enough sexuality.

Psychotherapy in Mental Diseases, was presented by August Hoch, M.D.

SOME RESULTS OF PSYCHOTHERAPY

By B. M. Hinkle, M.D.

By the term psychotherapy she included all form of mental medicine whether accompanied by hypnosis or without. According to continental authorities, Bernheim, Forel and Moll, although 95 per cent. of all people are hynotizable only about 13 per cent. are amnesic on waking. In her practice only about 10 per cent. of her cases experienced loss of memory, the others all declaring that they heard everything and were conscious of their surroundings. The power to induce hypnotism in greater or less degree was inherent in the majority of the people, and the authorities already quoted agreed that the physician who could not influence 90 per cent. of his patients was not competent to criticize psychotherapy. In her own experience with suggestive treatment she had found that the most intelligent Americans yield more readily to mental therapy than the foreign-born clinic cases. There was no question, however, that there was a great variability in the susceptibility of indi-

viduals as there was in their varying temperaments and characters. Her method of treatment was based on that of Bernheim and Leibault and followed very much the plan of Bramwell in that she always gave her intelligent patients a preliminary explanation of what she expected from the treatment, the probable sensations they would experience the first time, and the fact that sleep, as generally understood, was not necessary in order to experience the benefit of the suggestion. There were two distinct classes of patients, those who were afraid they would go to sleep, and those who were afraid they would not. Both had to be met and dealt with accordingly. In a few of the cases when they could not be reasoned with she had used hypnotic drugs for their quieting influence and to heighten the verbal suggestion. It was not wise to give drug therapy at the same time as suggestive therapy. Dr. Hinkle then presented three definite cases from her own practice which illustrated the success that had been attained by the use of this method of treatment.

Dr. Joseph Fraenkel said that he was mindful of the admonition of the President to appear ready to discuss the question, not with emotional, but intellectual weapons. He would limit his remarks to psychotherapy only—its applicability, limitations, indications and management. He bases his remarks upon the fact that he has been more or less interested in the various phases of the question during the last 16 years or more. The speaker's interest in the question was first aroused at the time he was interne at the Austrian Insane Asylum, when Krafft-Ebing took the chair of psychiatry in Vienna and brought with him the wave of psychotherapy. The speaker has seen many of the men mentioned to-day at work and has seen their results.

Of course we all recognize the fact that there is such a thing as hypnotism · that there is an influence of the mind over the body and the body over the mind. At the same time we must understand that psychic methods of investigation of the genesis of disease are not psychotherapy but psychodiagnosis. Refined psychoanalysis may or may not add to our understanding of the genesis of mental or somatic symptoms in neurotic individuals, but to the present time, little definite data are here to make it a basis for rational therapeutics.

Psychotherapy is used either as an adjuvant therapeutic measure, or as an exclusive method of treatment. As an adjuvant measure it has been used by all physicians of all times, and is used by us. Each one has his own method, and the best have no fixed method, but change it according to the nature of the case and the idiosyncrasies of the individual. Here the speaker referred to the story told in the Bible of the leper, to whom the Lord said: "Go thy way; faith will cure thee," and he got well of his leprosy. Modern Bible criticism reports records testifying to this occurrence.

As an exclusive method of treatment psychotherapy is applicable only to a very small group of diseases. Only to the psycho-asthenic conditions, in which the impulsive ideas or impulsive acts are a predominant feature, and to the purely psychogenetic forms a mono- or polysymptomatic variety of hysteria. According to the speaker, the latter form is a very rare disease. Exclusive psychotherapy should be handled and practiced, in the opinion of the speaker, only by the best and most experienced minds and in the manner of the schools, *i. e.*, in a continuous form, so that the patient is not only given occasional suggestions, but that he live and breathe in a psycho-theapeutic atmosphere, away from the milieu which was productive of disease.

Dr. B. Onuf said there was no question but that the faith element entered also in therapeutics of persuasion, for persuasion also counted on credulity to a certain extent. He who is to be persuaded has to take many things as they are presented to him, simply because he is not sufficiently familiar with them to form an opinion of his own. For instance a patient is told that the functions of the stomach are such and such, and in order to keep his stomach in good condition he has to follow such and such rules. In other words he is given a certain explanation of facts partly theoretical and which therefore must be taken in good faith by him. Nevertheless persuasion and conviction should rest chiefly on solid facts thus giving the faith element the least possible share.

With regard to Dr. Jelliffe's remark about hysteria, that it was not usually found in the developed mature mind, such perhaps, on the whole, held true; but there is no question that it can occur at very mature age, as witnessed the case of a man seen by Dr. Fraenkel and the speaker, in whom pronounced hysteria developed at 45 years. That hysteria was found only in infantile minds is contradicted by competent experienced observers who found it frequently in extremely developed minds. Therefore Dr. Onuf thought it was wrong to make such a general classification.

He said he wished to correct a statement made by Dr. Clark in his description of Freud's analytical method. Dr. Clark stated that Freud placed his hand on the patient's brow; Dr. Onuf said that this was something that Freud avoided. In his psychoanalytic method he wished to do away with anything that was suggestive of hypnotism in treating these patients. It is true that in the method which Freud had formerly employed, the so-called cathartic method, hypnotism was made use of to enlarge the field of consciousness, thus bringing facts to the patient's consciousness of which he was unconscious in the normal state. He had, however, abandoned the method and applied the psychoanalytical method in which hypnotism and everything that could suggest it, was carefully avoided. In order to apply Freud's method correctly one should be able to interpret the phenomena of the conscious and the subconscious life. The patient must be made conscious of certain processes which had absolutely no meaning to him. He is made to see things he was blind for, so to speak. To affect this, Freud made ample use of the processes of dream-life. The latter had a great deal of relationship to hysterical phenomena and their interpretation made it possible to understand many of the hysterical phenomena.

Dr. Onuf said he wished to call attention to one point that Dr. Jelliffe had not brought out in the reëducation method of Dubois. Dubois, far from using the faith element, was very much against suggestion at all in treating patients. He concluded that it was important to free the patient of auto-suggestions. He said that patients might, by not being treated hypnotically, lose the advantage of one or the other symptom, but would gain an advantage in not falling victim to auto-suggestion. In Dubois' method, far from encouraging the faith element, consciously at least, he attempted to free the patient from its fetters.

Dr. Pearce Bailey said he considered Freud's theory of hysteria the most valuable contribution to the neuroses in many years, but he was surprised at the great popularity attained by Dubois' book. Not at the lay popularity, as the public always welcomes a book of this character, but the avidity with which the book had been welcomed by physicians. It seemed to him that the book was written in an unscientific way and that

it was extremely incomplete in its details. Whatever Dubois might say, his method of reëducation was the method of suggestion. Like the pilgrims to Lourdes, the patients come from long distances and take what Dubois tells them as gospel, whether they understand it or not. It seemed to him that for true psychotherapy, the physician must get to thoroughly know the individual and then apply appropriate means, either suggestive or persuasive. As time goes on, there will be less spoken of classification of hysteria and the various neuroses and more on the differentiation of various types of individuals, what their evolution had been and how they reacted to the misfortunes of life to which all are exposed. A better classification than that of diseases is that of individuals, the depressed, the gay, the timid, the dreamers, etc., and the way each class may be expected to respond to various psychic traumata. Such a classification will some day give a more rational basis for psychotherapy. Adherence to any one system of psychotherapy is to be avoided. There is danger of the physician thereby forgetting to look out for the patient's other needs which normal association and employment alone can supply. Any psychotherapy which partakes of the crowd movement will probably have only an ephemeral and uncertain success.

Dr. Edward D. Fisher spoke of psychotherapy as being a wave which came up occasionally and which lasted but a short time.

With regard to the combination of the church and the physician it seemed to him about as faulty a combination as one could imagine. Psychotherapy was applicable as a form of treatment, but only in addition to the well-recognized forms of medical treatment. He could not conceive of a pure psychotherapeutic treatment; he could not conceive of a treatment that left out all drugs. He thought that if this practice was to be carried out, it should be carried out in the hands of the best trained men. He had seen harm done with hypnotism. Mental disturbances might follow the use of this practice in untrained hands.

As a rule, psychotherapy would never be generally used by neurologists, for the reason that its proper application required too much time. Therefore, it should be applied by men who have the time to devote to it, as in institutions. For the same reason the ordinary practitioner could not use this method. So he feared that unless carried out in institutions not so often found in this country as abroad, the use of psychotherapy would fall into the hands of the untrained or worse yet the charlatan.

Dr. Edward W. Scripture said that about one month ago he attended a meeting of the German Medical Society in which took place a discussion on the same subject and one prominent neurologist made the statement that hypnotism was a dangerous procedure, while another gentleman said that Americans had dropped hypnotism twenty years ago. Dr. Scripture thought that these gentlemen failed to realize that they were quite out of date.

Four or five years ago he started in Europe to be cured of a trouble by being hypnotized, and he met with men who were famous in both France and Germany. He got his cure and at the same time picked up a number of points regarding the methods which he found very useful indeed. Hypnotism as at the present day practiced in Germany was nothing sensational, like the procedures of Charcot. It was as simple in its application as a hypodermic injection would be. Tonight it was refreshing to hear such common-sense and sane view taken of the subject as by Dr. Hinkle. Hypnotism in trained hands was free of danger, sensational-

ism and quackery, and was quite different from the notions many Americans have of it. It was a valuable procedure.

Dr. Leslie Meacham said he had been interested in this subject for sixteen years and had had much experience in experimental hypnotism, in teaching that and principles of psychotherapy to physicians and the practice of psychotherapy. Men failed in trying to use this treatment because untrained. They cannot judge the suggestibility of the patient or give suggestions properly. In his own practice for the last five years he had found hypnosis rarely necessary. To succeed in this work one should understand psychology and pedagogics as well as nervous and other diseases, and all other therapeutic agents should be employed in the general treatment of the patient and care of the underlying neurasthenia. People using psychotherapy sometimes ignore the physical needs. His own methods and principles were similar to those of Dubois, considering the work properly to be one of reëducation. As a preliminary it is necessary to explore the utmost depths of the patient's mind, determine all the psychic errors and their origin. In the majority of cases systematic suggestions, earnestly repeated, logically given, are sufficient. Build up his hope and courage and self-control, develop his powers and enable him to help himself. The only cases in which he found it necessary to use hypnosis were those exhibiting morbid fears, fixed ideas, etc., *i. e.*, the pure psychoses, habits, some forms of hysteria and periodic alcoholism. Chronic alcoholism readily yields to systematic suggestion. Many fail in their attempts to use psychotherapy because of insufficient preparation for the work. They do not know the limits of suggestion, the frequently slow progress to recovery, and expect to cure serious cases in two or three treatments. Physicians and patients often expect only actual hypnosis by the psychotherapeutist. The pedagogic side of pschotherapy has not been sufficiently insisted upon.

THE BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

November 23, 1907

WITH

THE NEW YORK NEUROLOGICAL SOCIETY AND THE PHILADELPHIA NEUROLOGICAL SOCIETY, AS GUESTS

The President, DR. W. N. BULLARD, in the Chair

Dr. Knapp showed a young man of eighteen who had had hemiatrophy of the left side of the body, associated with scleroderma, for two years.

A CASE OF DIVERGENCE PARALYSIS

By G. L. Walton, M.D.

The case was that of a young man (seen in consultation with Dr. Clap), who was thrown from the running-board of an electric car by striking a post. While still in bed he found that he had double vision for objects directly before him at a distance. There was a hematoma over the anterior parietal region on the right and a bruise of the left temple. The genuineness of the double vision was shown by the fact that the two

objects fused with eight degrees of prism, no matter whether used in one glass or in various combinations with two glasses. When the patient looked to the right or to the left the diplopia disappeared. This is characteristic of so-called divergence paralysis in distinction from diplopia due to paralysis of ocular muscles. Again, when an object was brought within two feet of the patient the diplopia disappeared. This is another characteristic feature. There are about twenty cases on record of this variety of diplopia. It has been attributed to paralysis of the divergence center, the theory being that just as there is a center for convergence (presumably in the mid-frontal lobe) there is a center for divergence which is inhibited when we converge, but called into play together with inhibition of the convergence center, when we look at distant objects.

TRACTION-SPLINT FOR MUSCULAR SHORTENING

By G. L. Walton, M.D.

Dr. Walton also showed the splint of Sir William Gowers, for use in cases of toe-drop to obviate tendency to shortening of calf muscles. The objection to the use of an elastic muscle attached to the toe and to a band about the leg is that the local pull is irksome and cannot be borne continuously. This apparatus was described and figured in a lecture in the *Clinical Journal*, March 6, 1895, and reprinted in Gowers' "Clinical Lectures," Blakiston, Philadelphia, 1895, in a lecture on the treatment of muscular contracture, but it seems not to have attracted in this country the attention which it deserves.

This apparatus is admirably adapted to remedy this condition by causing the pull to be extended over the surface of the boot. The boot consists of a single piece of sole leather "connected by a narrow part about two inches wide, behind the heel, which allows flexion of the ankle and yet affords sufficient vertical resistance to prevent any pressure on the skin at the source of the pull." The leather having been soaked in cold water is allowed to harden about a cast of the leg and foot in such a way that the narrow strip of leather forms a hinge at the heel. Lacings are inserted, and the elastic is stretched from rings attached to the back of the foot sheath and the sides of the leg piece. In the model shown (which Sir William Gowers had forwarded Dr. Walton at his request, together with directions for its manufacture) the latter was continued to the thigh and cut out for another hinge at the knee. This extension is for use in case of tendency to contracture of the hamstring muscles, in which event the elastics are extended crosswise over the knee. This appliance may be worn continuously day and night for months, and if desirable, the patient can walk about the room with the apparatus on.

BRAIN SHOWING CIRCUMSCRIBED CORTICAL ATROPHY.

By Dr. McDonald

Since Marie's views regarding aphasia have been so widely discussed and received with such marked favor in many quarters, one runs the risk of being regarded as behind the times in attempting to find special centers in the brain having for their function the focalization and preservation of concrete memory concepts; that is, concepts dependent for their complete

arousal not alone upon general mental powers which—as Marie would have us believe—are inherent in the brain as a whole and cannot be further localized, but concepts which can be aroused only through the activity of special cortical areas.

As old-fashioned as these theories of localization may seem to many, Dr. McDonald confessed to a hesitancy in abandoning them entirely in favor of a chaotic agnosticism which robs us of a most powerful incentive to closer clinical study. And he had been guilty of continued temporization with these kindergarten methods in the fascinating endeavor to find in disease of particular portions of the brain the explanation for the occurrence of certain symptoms of mental disorder. Despite the alleged futility of such attempts he had, by chance perhaps, in one case at least, hit upon a correct guess as to the situation of a more or less circumscribed brain lesion.

At two medical meetings, one of which was that of the American Medico-Psychological Association, at Washington, in May, 1907, he made reference to the occurrence of localizing symptoms in so-called diffuse mental diseases such as senile dementia, and endeavored to show that such signs could probably, by close and systematic study, be found in a large majority of senile dements.

The attempt to diagnose ante mortem such areas of especially severe brain atrophy is not new and Pick has described a so-called "left-sided temporal lobe complex" in senile dementia, a complex associated with a particularly marked atrophy of a certain area of the brain which lesion he was able correctly to locate in several cases before death.

In the two cases which Dr. McDonald had in part reported the symptoms differed from those observed in Pick's cases and briefly summarized were as follows:

Without motor or sensory symptoms there was partial auditory agnosia, partial auditory asymbolia, almost complete auditory anomia, partial visual agnosia, almost complete visual anomia, partial astereognosis (tactile agnosia) in both hands alike, stereognostic anomia, olfactory agnosia and anomia, paraphasia, agraphia for numbers. In addition there were more general symptoms of senile dementia, such as loss of impressionability but which in turn did not involve all sensory functions alike; *e. g.*, loss of visual impressionability was much greater than that for auditory experiences.

By a process of reasoning too complicated to permit of rehearsal at this time, the conclusion was reached that there is much evidence of the existence in the brain of a highly specialized center which concerns itself with the unification and concentration of related sensory experiences permitting them to appear in consciousness as concrete concepts. But instead of regarding such an area as being composed of and inclusive of all the memory fields of the various senses as does Mills, Flechsig and others, it seemed more probable that if such a center for concrete concepts existed at all it would be found separate from the centers of primary sensory experience though closely related with all of these and particularly accessible to the visual and the kineto-kinesthetic centers.

Such an arrangement was strongly suggested by the fact that, whereas with these two patients no defects in primary sensory experience could be found and no evidence of inability to associate, as coming from one source, the various impressions aroused by an object, there was nevertheless a distinct failure to arouse the memory concept of the object itself.

Thus with a match, for example, the patients could describe the shape and size, criticize the odor of the fumes, show disgust at the taste of the phosphorus, repeat the word *match,* spell it, tell that it was made of wood. withdraw from the heat and sight of the blaze and yet declare that it was an unfamiliar object, they had never seen anything like it, and moreover could neither describe its uses nor name it. There was no arousal of a concrete memory concept of the object itself despite the accurate association of its many qualities. The same was true of coins, bills and other objects.

Assuming the existence of such a center, it seemed not improbable that it lay in the left brain and in the great unknown territory lying at the junction of, and composed of, adjacent portions of the occipital, the parietal and temporal lobes where it would be especially accessible to all sensory fields and especially close to the visual and kineto-kinesthetic cortical areas. It was accordingly stated that, if autopsy should be permitted in the cases mentioned, an area of especially intense cortical atrophy would be looked for in the locality indicated. Such a lesion—a roughly circular moderately deep excavation from 2 to 2.5 cm. in diameter—is here to be seen in the brain of one of these patients and at that part of the cortex in which it was assumed that the centers for concrete concepts might be located. Of course it would be quite unscientific, rash and unwarranted to deduct great significance from the chance coincidence of a lesion situated at a point indicated in a lucky guess—for such reasoning as led to the above conclusion is not to be exalted beyond the realm of guessing.

It is meet, however, that the examination of the brain of the second case should be awaited with respectful patience though with eager interest for the details which, should they prove to agree with those here to be seen, would afford a basis for one of those tentative theories which occasionally lead the way to a more certain knowledge.

THE CYTOLOGY OF THE CEREBROSPINAL FLUID: RÉSUMÉ OF STUDIES AND DEMONSTRATIONS.

By Dr. Cotton and Dr. J. B. Ayer, Jr.

Alzheimer's method of treating the cerebro-spinal fluid, published in the *Centralblatt für Nervenheilkunde und Psychiatrie,* June 15, 1907, goes far to obviate the faults of the earlier methods. It depends for its good results upon fixation of cells in the fluid by alcohol, and the subsequent treatment of them so that they may be mounted and cut by a microtome. The method is briefly as follows: To one third volume of cerebrospinal fluid (3 c.c. were used) two thirds by volume of 96 per cent. alcohol was added in a centrifuge glass with conical bottom, and the whole thoroughly mixed. A thin white cloud of precipitated proteids will result; this is centrifugalized for one hour at high speed, when a small firm coagulated mass containing the cells will be found at the bottom. The supernatant fluid is decanted and absolute alcohol added for one hour, followed by alcohol and ether equal parts, and ether, each for one hour. Now by touching the side of the coagulum just enough to loosen it, a tap on the bottom of the glass will dislocate it, when it is immediately placed in thin celloidin. The coagulum is later mounted in thick celloidin on a block and kept indefinitely in 80 per cent. alcohol. Sections were cut uniformly

at 14 m. and the routine stain employed was Pappenheim's pyronin-methyl green mixture, permanent slides being thus obtained.

By this method cells are found pretty evenly distributed throughout the coagulum, thus giving us a basis for a fair quantitative as well as qualitative count.

1. *Qualitative.*—The following cells may very readily be recognized in the fluids studied. Where there are many "unclassified" cells the fluids have always been from post mortem punctures (and the cells may therefore have undergone autolytic and other changes). Group I: Cells normally found in the cerebrospinal fluid are (1) *endothelial cells*, these are cells having an oval or horseshoe-shaped nucleus with slight amount of chromatin substance, and a moderate amount of pink staining protoplasm (with pyronin); these are considered as normal findings because found in almost every fluid examined, and because they simulate cells found in the normal pia; (2) *lymphocytes*, resemble in every detail the lymphocyte of the blood—lymphocytes were found in every fluid studied; (3) *connective tissue cells*, while not found in every case should, however, not be considered an abnormal finding because found in the normal pia mater.

Group II.—Cells found under pathological or abnormal conditions are (4) *Phagocytic endothelial cells.* These phagocytes are undoubtedly similar cells to those described under class (1). The inclusions are almost invariably lymphocytes, though rarely a blood globule or another endothelial cell is recognized as an inclusion. These cells are also recognized in the pia of cases the fluids of which show them; (5) *Plasma cells*, the nucleus eccentrically placed resembles that of the lymphocyte, but the protoplasm is greater in amount, is stained a deep red with pyronin, having a blotchy appearance. A light area in the protoplasm at one edge of the nucleus is characteristic. These cells have been found only in cases in which they occurred in the pia, and in all of these cases, and these have only been general paretics. Transitional (?) and giant forms of plasma cells are also described; (6) *Polymorphonuclear leucocytes* are perhaps abnormal findings, though a few may be accounted for by blood contamination; (7) *Fibroblasts*, or young connective tissue cells are probably recognizable. They would seem to indicate an active meningeal process. (8) *Körnschen*, or fatty granule cells, have been found only post mortem in cases of cerebral softening. They are cells apparently of the endothelial class with fatty granule inclusions, as demonstrated by Sharlach R.

Besides the above cells, ependymal and possibly neuroglia cells were recognized. By this method also other extraneous cells such as tumor cells, could easily be recognized.

2. *Qualitatively* this method offers a fair standard of accuracy for the individual worker, and perhaps the results of many workers could be correlated with as much accuracy as by use of the other methods.

The work was taken up to determine its diagnostic value in insanity, and especially in general paralysis, with entire success as regards the latter.

From the table of living cases, the speakers were led to assign a certain characteristic cell picture to the fluid of general paresis, namely a large total cell count, approximately 75 per cent. of which are lymphocytes, presence of plasma cells, and frequently of a few phagocytic endothelial cells. Other living cases show no characteristic cell picture, and probably represent nearly normal fluids. From the study of the post

mortem fluids they were led to believe that the Körnchen cell and perhaps the phagocytic endothelial cell may be of diagnostic value in organic conditions. The value of this method then lies in the possibility of obtaining by it beautiful cell pictures by which the diagnosis of general paresis is greatly aided, and by which cells characteristic of cerebral softening or even of tumor might be recognized if seen.

A MEANS OF DETERMINING THE "ALEXIC" OR REACTIVATING CONTENT OF HUMAN BLOOD SERUM

By Dr. Gay and Dr. Ayer

Attempts have been made to determine this reactivating power of human blood in order to gain some idea of the resistance of the individual to bacterial infection, but hitherto no reliable method has been offered by which the sera of different individuals may be compared. It was found that red blood cells which had been saturated with inactivated specific immune serum from an animal of another species offered in a given dosage a relatively fixed unit for the determination of the reactivating power of the blood. By means of this method the alexic content was determined in a number of patients suffering from different types of insanity, but physically well. No constant differences in alexic activity were noted in relation to forms of insanity. The marked correspondence of alexic power in these patients will probably serve as a basis for a normal mean by comparison with which similar determinations in cases of acute infectious disease may offer results of prognostic value.

Dr. Southard mentioned briefly a case of general paresis developing in an alcoholic. The patient was always spastic in legs during observation and some weeks before death showed signs of fresh lesions in the motor zones. The microscopic examination of the spinal cord showed a long-standing bilateral pyramidal tract sclerosis (Weigert's myelin sheath method) as well as a fresh degeneration, also bilateral, in the same region (Marchi method). The recent Marchi degeneration differed qualitatively in the different segments, gradually altering from groups of fat and myelin containing perivascular cells below to wholly intrafibrous blackening in the upper cervical segments. Dr. Southard employed these findings as logical support for the idea that the nerve fibers were undergoing a chronic progressive atrophy rather than the effects of local destruction. The cortical findings were somewhat confirmatory of this idea, since the cells of origin (such as had survived the original lesion) showed "neuronophagia" rather than axonal reactions.

THE ATTITUDE OF THE MEDICAL PROFESSION TOWARD THE PSYCHOTHERAPEUTIC MOVEMENT

By E. W. Taylor, M.D.

The object of the paper was to discuss the following propositions: First, the past and present attitude of the medical profession toward mental therapeutics; secondly, the present state of therapeutics in relation to the psychotherapeutic movement; thirdly, the general attitude of special students of the subject; and, finally, the means by which a knowledge of the movement may best be disseminated.

It was pointed out that the general attitude of the medical mind, now as heretofore, has been one of antagonism to methods of treatment which appeal to other than physical means, and that this attitude of the profession was in accord with the scientific revival of the last century. The change which is now becoming manifest is toward a more generous recognition of mental means of treatment as worthy the close study of the physician. Attention was called to the fact that the word "psychotherapy" as a heading occurred in the Index Medicus for the first time in May, 1906. Reference was made to the still prevailing idea in the medical profession at large that drugs constitute the essential means of treatment, as illustrated by a recent address by Dr. Horatio C. Wood, Jr., of Philadelphia. In general, however, it was apparent that a great widening of the therapeutic outlet has taken place and that the physician of the future must alter in a measure his relations to his patients and to the community at large. After a general statement of the more recent work which has been done on the subject, it was pointed out that certain definite methods of procedure of a rational sort had taken the place of vague statements and generalizations and that the time was at hand for a statement of results through the publication of cases based on such definite methods of procedure. The general enthusiasm of special advocates of psychotherapeutic methods of treatment was touched upon with a warning lest the cause be damaged by an excess of zeal. It was maintained that the subject had now reached a stage when exact scientific methods might be applied to the elucidation of the problem, and that the success or failure of mental methods of treatment must stand on the same plane as any other series of facts, if real progress were to be made. The attempt must be made to rationalize the psychotherapeutic movement if we are to expect to overcome the legitimate skepticism of those whose interests lie in other fields of research. It was maintained that the immediate aim of special students of the subject should be to publish cases illustrative of definite methods, to state facts simply and without recourse to over-much speculation, and to maintain an attitude of conservatism in the interpretation of results, such as would be demanded in any problem of physical science.

Finally the attempt was made to lay special stress upon the means of disseminating a comprehensive knowledge of the present movement toward practical psychotherapeutic methods. Public lectures to the laity were on the whole regarded as unwise. Carefully prepared articles in popular journals by men of high standing in the profession were considered a legitimate, if somewhat doubtful, method of spreading such knowledge. The alliance of physicians with church movements, such as have recently sprung up in Boston, was regarded as wholly unwise, and as likely to put back the general cause of psychotherapy in the ranks of the profession itself. On the other hand, articles and communications addressed to medical men through the medium of medical societies was strongly urged, since one of the first duties of those particularly interested in the movement was to enlist the coöperation and sympathy of fellow practitioners. It was also urged that medical schools should recognize mental therapeutics as a legitimate branch of study, and that certain studies bearing on the mind primarily should constitute a part of the medical students' preliminary training.

The general conclusions to which the paper led were as follows: In our enthusiasm for the development of a highly significant therapeutic

advance we should not allow the traditions of the profession to which we belong to be submerged. Our enthusiasm should not lead to the antagonism of any reasonable member of the profession. In our efforts toward what we regard as progress we should proceed with great deliberation and by recognized logical methods. To this end we should quote cases and exploit methods which have stood the test of experience. We should hesitate in appearing before the public with a detailed expression of our views and theories until we have secured the acquiescence of the conservative members of our own profession. We should not ally ourselves with movements over which we have no immediate control. We can ill afford misinterpretation, especially at the hands of our brother physicians. Our essential aim should be to develop a permanent interest in the psychotherapeutic movement within the ranks of the profession, and to do nothing which can in any way retard this effort. To this end we should urge reasonable but adequate recognition of the claims of psychotherapy as a legitimate and necessary element in medical education. Finally our one object as members of a liberal profession should be to insist upon the importance of psychotherapeutic methods with an enthusiasm which should only be increased by the legitimate limitations set upon its action. By these means it was believed that the beneficent results which were destined to result from a complete acceptance of psychotherapy by the medical profession would soonest be attained.

Dr. Dana spoke of his own experience in connection with psychotherapy in its various forms. He had not encountered any opposition to the use of hypnotism in the city of New York. Twenty years ago Dr. Beard had brought the subject of hynotism before the medical world, and his demonstrations had awakened interest more than opposition in hypnotism at that time. Psychotherapy by means of hypnotism had been used more or less regularly by himself and his associates ever since. For several years he had had a "hypnotic room" in connection with his clinic at the Post-Graduate College, and had obtained some results, but it was finally abandoned because about the same results could be gotten with less trouble in other ways. In fact psychotherapy, by means of formal hypnotization had been generally abandoned except for some specific and rare cases. He did not think that it formed a very useful part of the therapeutic armamentarium. The speaker thought perhaps Dr. Taylor had dwelt with more emphasis than was just, upon the neglect of psychotherapy. He had for several years an assistant at his clinic at Cornell, who had been specially assigned to that kind of work, and who had had a special training for it. He had kept an open mind for all cases in which it could be used. The trouble was as with all methods of technical psychotherapy, the treatment required a certain setting for it, in order that it might be fully effective. The essential thing in all forms of systematic psychotherapy, was to have the patient properly staged. This did not mean that he must have the procedure surrounded with any dramatic associations, but simply that he must be placed where he is removed from all influences but those of the doctor and his assistants. The atmosphere must be such that the doctor's personality, views, and counsels especially affect the patient. This throws a certain limitation to the practical utility of the formal methods because it makes them time-consuming and often expensive. The speaker thought that psychotherapy on the whole was more effective with rather weak and foolish people, or at least with people whose education and point of view were narrow. He thought that

the introduction of instruction as to what was meant by psychotherapy, its limitations and its various technical methods was a wise measure. In fact the endeavor of Dr. Taylor to advocate a systematization of the various methods of psychotherapy was to be heartily encouraged.

Dr. Charles K. Mills said the subject of psychotherapeutics can be approached from various directions, and can be handled in diverse ways. When the wheat is sifted from the chaff however, it will be found that what might be termed mental medicine is usually dispensed in three ways, namely, (1) by the employment of hynotic procedure; (2) by calling upon normal suggestibility; and (3) by educational or disciplinary methods.

Hypnotic procedures have, he believed, a limited although a real place in neurological medicine. Hysterical palsies, anesthesias, amauroses, and the like may in special instances be more quickly relieved through the use of hypnotization than in other ways. Dangers however attend the use of hypnotization either for the purpose of study, diagnosis or treatment, and in his practice he had seldom found it necessary or wise to make use of it.

Every successful neurologist or alienist, indeed every successful physician, plays with more or less skill upon the normal suggestibility of his patients. His success is often in large part measured by the manner in which he is able to make use of suggestion. It is not merely in hysteria and other so-called functional neuroses that this psychotherapeutic method is of value. In many organic affections it is a strong adjuvant to other means.

The most important application of psychotherapy in connection with nervous and mental disorders is through the use, by the physician, of educational or disciplinary suggestions.

This is perhaps not saying much, and is perhaps repeating what has been said by others, here and also in recent works like that of Dubois. Nevertheless each physician has his own way of educating or training his patients out of their moods and their fancied ills, as well as their real and partly remediable ailments. The good doctor studies his patient as an individual as well as a case, and tries in a certain sense to teach that patient to study himself. By this is not meant that the patient is made introspective and self-analytic in the morbid sense, but rather that he is taught to consider his own limitations and possibilities. The patient, in brief, is made by psychic methods to help himself.

In organic and incurable nervous affections is a large field for the exercise of psychotherapy. Many patients suffering from locomotor ataxia, from diabetes, or from some slowly advancing or abeyant form of renal or cardiac disease can be so guided and so stimulated by the physician who knows how to influence the body through the mind that his life will be sweetened and prolonged, and his health so improved that those dependent upon him can be maintained for years instead of losing their means of support. We should not be "foot in the grave" doctors even with our incurable patients, but should teach them to get all out of themselves that is possible, notwithstanding their disease.

Dr. Sachs agreed in substance with the position taken by Dr. Dana, and felt that too much importance was being attached to methods of treatment embraced under the heading of psychotherapy. As a matter of fact, the various methods of psychotherapeutists, so far as one could make them out, had been followed by each and every one of us for many

years past. There was, to be sure, some satisfaction in having all these methods and practices described under one heading, but giving a new name to old methods does not imply that the methods are in any sense new. The speaker saw the danger of over-estimating the importance of psychotherapy, and if the matter was already to be taken up as a subject for the instruction of students, he hoped that the instructor would point out the very limited applicability of such methods. The chief use of such methods would be in the handling of patients suffering from hysteria, neurasthenia, the neuroses and in some few psychoses. He related an experience in trying to argue a patient, suffering from delusion of persecution, out of this delusion; if anything was to be included under psychotherapy, such an argument addressed to the patient could well be included under that term. But, in view of the very serious task before the neurologist, and the many important questions that he is asked to solve with regard to the care and treatment of patients suffering from organic nervous diseases, he feels that altogether too much time and energy are being expended upon psychotherapy. While hysterical, neurasthenic patients, and others of the same order are numerous enough, their ailments and sufferings are, after all, less important than the sufferings of those who are afflicted with various forms of organic spinal disease, say tabes, primary lateral sclerosis, and the like. Let us try to do more for these patients; let us try to discover methods of hastening the cure of multiple neuritis, for instance, and do not let us waste too much time and energy on what people are now pleased to call psychotherapy.

Dr. F. X. Dercum had carefully listened to the paper by Dr. Taylor but had failed to gather from it any fact which would lead one to infer that under the term psychotherapy any new thought or idea is conveyed. Suggestion made its appearance with the dawn of civilization. It was practised alike by the Egyptians, the Hebrews, the early Greeks and Romans. One need hardly allude to the Pythonism of the Greeks, of the Shamanism of the Urals or of the similar practices among the barbarous and savage people of our own day to show how universal suggestion always has been and still is. How it made its appearance in special forms within relatively recent periods as Mesmerism, hypnotism, metallotherapy and the practice of Perkins's tractors is a matter so well known as to need no more than a passing mention. Similarly is it with the decadent Doweyism and the still flourishing Eddyism of our own day. Psychotherapy does not convey a new idea despite its clothing of a new phraseology; we cannot but discern the lineaments of the most ancient of all of the arts of healing. Let us not deceive ourselves in the assumption that it is something new, it is literally as old as the human race, the term alone, high-sounding and impressive, is new, that is all. Much might be said of psychotherapy as to its usefulness and as to its limitations but the time afforded by a discussion such as this is by far too short. It is important, however, to state that psychotherapy has not only its limitations but also its dangers and that these dangers are twofold. They pertain first to the physician who, placing too great a reliance upon suggestion, fails to make an accurate clinical study of the pathological condition from which his patient suffers and secondly there is the still greater danger to the patient whose mental integrity may be placed in jeopardy. This danger lies in developing in the patient a "pathological suggestibility" exactly like that seen in subjects the victims of frankly

hypnotic methods; in other words the danger lies in the inducing of hysteria. What is gained by compounding the nervous affection from which a patient suffers by adding to it hysteria? Surely the induction of "positive" or still worse of "negative hallucinations" concerning the affection from which a patient really suffers, can only be regarded as an injury—an injury which when frequently repeated, may lead to a permanent lesion of the mental integrity. Such a patient ceases to be able to distinguish accurately if at all between his impressions as they really are and the substituted conceptions or hallucinations which have been induced.

Suggestion inasmuch as it was practised by physicians in all ages early found its limitations. It is equally true that within these limitations it is practised by all successful practitioners of medicine, usually without special forethought or design, even often unconsciously. Even its special application in functional nervous disorders is not new, for it has been long practised in connection with the full feeding, massage and bathing of the rest cure, in the isolation and retraining and reëducation of the patient—in the pointing out of the unessential character of his symptoms and in the "untying" of his pathological associations. The keynote, however, it will be remembered, in the rest cure has ever been the raising of the patient's physical health to the highest possible level and that under these conditions normal suggestion is of most avail, goes without saying.

Dr. John K. Mitchell remarked that Dr. Taylor had said it was not long since any one who practiced any form of what he preferred to call mind-cure was considered "without the pale." Perhaps he might speak with the more knowledge, as a member of a family which has been for seventy years without the pale, for it was in 1840, or thereabouts, that his grandfather was asked by the trustees of the Pennsylvania Hospital to resign because he had used Mesmerism, as then understood, in his treatment of patients.

At present psychotherapy is in danger chiefly from its friends. Excessive zeal, if it stimulates at first, can kill afterwards. A new name for an old acquaintance does not make it a new thing, and yet it seems as if many of the men who talked about psychotherapy thought that they had a new application or a new science. Over-zealous, indiscriminating use of it now may discredit it in the way abuse and misuse discredited hypnotism.

While it would not be borne out by minute examination, still the first impression that one gets from reading Dubois' excellent book, as in reading Freud, would be that neither of them believes that there is *ever* any physical foundation for nervous disorders. A man of one idea is often a useful reformer or propagandist, but he is also a danger, and here we have men constantly stating well-known facts as if they had made a grand discovery of something new and hitherto unheard of—in short, endeavoring to enforce upon us the obvious as if it were the recondite.

The general limitations of psychotherapy as a treatment cannot well be set. They are very precise in one case; very wide in the next; they vary with the force, capacity and knowledge of the treater, and with the possible receptivity of the treated. They are affected again by the personal relations of the two, but in the last analysis they depend upon the power, strength of character,—in short, what we call *personality* of the physician,—a gift, this, which cannot be taught or learned.

To make good use of mind-cure a man must have perception of human

nature, appreciation of character, comprehension of all the forces that move man—matters which, though experience helps them, cannot be acquired by any study any more than by taking thought one can add a cubit to his stature. Nothing human must be strange to him, since there is literally no motive in the whole range of the possible springs of human action that may not be called to our aid—no desire, no emotion that may not be made use of, from religion to vanity. In short, the physician who is capable of and suited for the use of this tremendous engine has been practicing the method since the time of the Pharaohs. For a concrete example one may cite Aaron's rod. The man who needs to be *taught* its value will probably be doing as much good, with less likelihood of harm, if he sticks to belladonna plasters.

Dr. Joseph Collins said that the subject of psychotherapy was one that he was glad to hear discussed because of the important place which it held and was destined to hold in the treatment of the class of patients with which we dealt. He did not experience either indifference or anything that might be construed as antipathy on the part of general practitioners to accept the offices of mental healing, or to endeavor to employ it. That which he did experience and that which, in reality, is the most difficult feature of the whole problem, is a personal incapacity to set forth the formulation of psychotherapy. What does it consist of? What shall we tell our students or fellow-practitioner we do to our patients or offer our patients, the sum total of which constitutes mind-cure? We may discuss the expediency of giving systematic instruction in this matter and reach the conclusion that it is desirable that we should give such instruction, but it is necessary to formulate the principles that we are endeavoring to base our work upon before we begin to teach it. Surely systematic instruction in psychology, experimental or otherwise, is not instruction in psychotherapy, nor does familiarity with the association method of Jung, or the method of psychic analysis of Freud, or mode of inducing hypnosis, constitute it, unless the term psychotherapy be used in a different sense than it is customarily used, viz., the cure of disease through the operation of the mind. Of course, it may, and legitimately, be used to include modes of diagnosis as well, but when we speak of therapy in conjunction with some distinct measure, such as hydrotherapy, electrotherapy, and mechanotherapy, etc., we have no reference whatsoever to modes of diagnosis that had been employed to elicit the disease for which such measures are utilized in the treatment of them. As a matter of fact, the trouble is that no method of psychotherapy has as yet been formulated and unless such formulation has resulted it cannot be taught systematically, and personally Dr. Collins was of the opinion at the present time that all that will ever be successfully done in this direction is to teach the principles upon which it is founded, and then the individual who essays to use it must work out a plan of application for himself. If Dr. Collins were mistaken about the fact that there is no formulation of the principles of this plan of treatment, he would like to ask the gentlemen who close the discussion to state specifically what they do in the treatment of their cases, not how they elicit their fundamental complaints, but what is their plan of treatment and what are its specific details. In closing he would like to say that he was a believer in the efficacy of psychotherapy, not only in functional diseases, such as psychasthenia, hysteria, constitutional headache or idiopathic headache, but in organic diseases as well.

Dr. Smith Ely Jelliffe, of New York, said that he felt that he could add little to the discussion, although, as joint translator with Dr. White of Dubois' work on the psychoneuroses, he was perhaps partly responsible for much of the psychotherapy movement which was now so manifest. The general argument that psychotherapy was very old did not impress him greatly. So is the knowledge of opium and of belladonna—both were used probably before man could read or write, but that does not imply that we are not better acquainted with the more intimate structure of these drugs to-day and cannot use them to greater advantage at the present time than our forefathers did, notwithstanding the ages that they have been known. It is so with psychotherapy. Certainly the "dream sleep" in the temples of the priests in the days of the Pharaohs was an application of some kind of psychotherapy, but we hardly feel that the methods then in vogue have not been improved on since those days.

It is somewhat confusing, Dr. Jelliffe thought, to speak of psychotherapy as a single thing—as the expression of only one method of the application of therapeutic principles along mental lines. In reality that which constitutes the real advance in psychotherapy is the crystallization, out of the general mass, of a number of differing and quite different principles, each of which has its special application. It is just as illogical to assume that pharmacotherapy, for instance, makes use of the principle of astringent action alone, as to hold that psychotherapy is hypnotism, or suggestion, or any one mode of influence alone. The researches of the last ten or twelve years demonstrate that the mode of approach to the psychical problems are numerous and that one must use the methods at hand with just as much discrimination as one would use material drugs. To attempt to argue out a fixed idea is as illogical in the application of psychotherapeutic principles, as to get rid of a deep-seated abscess by arguing with the patient that it does not exist. Hypnotism, waking and sleeping suggestion, psycho-analysis by means of Freud's hypnoidal or distraction method, Sidis' hypnoidization principle, Dubois' reëducation methods, Weir Mitchellism and others all embody different psychotherapeutic principles which have application in different cases. The exact mode of application, the limit of availability and the appreciation of the right means to the end—these are the problems in the foreground of modern psychotherapy. He who argues that he sees very little hysteria, and that psychotherapy has very little application confesses that he does not understand its principles and thus accounts for his not seeing that class of patients. Such patients have "suffered of" many physicians who for the most part apply the method of villification or brow beating and wonder why the patients do not recover. Which of the newer methods is applicable in the greater number of cases it is difficult to say, but from his point of view Dr. Jelliffe felt that the general principles laid down by Dubois, which in reality are a refinement, elaboration, and combination of Weir Mitchell's rest and isolation methods, with special attention paid to reëducation of the emotional life, had offered the most help. In spite of all our knowledge of method some patients escape to be helped by the individual who establishes by art or by natural gift the special rapport necessary to treat some individual cases. This is the element in psychotherapy which cannot be taught—the rest is teachable and understandable.

Dr. Leslie Meacham said that psychotherapy is now placed upon a more rational and scientific basis. This fact is not understood by the medical profession generally and even by the older neurologists. Patients who can be benefited by psychotherapy, are all those suffering from the

various minor psychoses, including obsessions, imperative ideas, habits, perversions, neurasthenics with psychic symptoms, and even some organic conditions such as tabes. Sufficient time cannot be given to psychotherapy in regular medical courses to enable the students to become expert in its use. Instruction should be limited to fundamental principles, knowledge of which will help in the management of all classes of cases, the conditions relievable, the real nature of psychotherapy, and its limitations as a therapeutic agent. Its successful employment demands natural fitness and special training even more than the various surgical specialties. There must be a knowledge of psychological principles, and the philosophy of education, sufficient experience to give self-confidence, and enable one to judge the suggestibility of the patient, and therefore the methods to be employed. Equally if not more important, are common sense, a knowledge of life and of the ways of the world, that comes only with years and experience. These are far more essential than medical knowledge. Other essentials are enthusiasm, and special interest in psychic conditions. Inexhaustible patience is not far behind.

All medical students should be required to have previous training in psychology as well as physical sciences.

Physicians now in practice should be stimulated to inform themselves about minor psychoses and psychic phases of neurasthenia. They should be made to realize that such conditions require careful management, and cannot be helped by telling them to use will power. They should know that time and patience are essential to the relief of such conditions, and that psychotherapy in its present use means chiefly reëducation, and only in exceptional cases actual hypnosis. All should understand that psychotherapy is employed only after most careful examination and diagnosis and in connection with such medical, hygienic and dietetic measures as may be indicated.

Dr. J. Ramsay Hunt felt that the large group of the psycho-neuroses is a most important one; and one which had been neglected in the past. The brilliant results achieved in this field during the past few years show what careful scientific methods can accomplish in this direction.

Naturally with a greater facility in diagnosis and a more complete understanding of the underlying mental and nervous states, more rational methods of treatment will be evolved.

The subject is now receiving the attention of men of high scientific training and large clinical experience, and has acquired a new impetus and dignity. In such hands its future progress is assured, and its development will follow the same conservative lines as in other branches of the medical sciences.

Dr. Wm. A. White said that the subject of psychotherapy is such a broad one, and there is so much that might be said upon it, that it is difficult to choose. He was surprised that the antiquity of psychotherapy should be urged as an argument against its use. We might as well argue that the modern methods of cement-construction are of no value because we have found cement work that had been done by the Chinese some 4,000 years ago, but to-day cement-construction work is carried out with all the added accuracy that has been placed at our disposal by chemical and physical laboratories and by methods of testing the strength and durability of materials. Although it may be true that psychotherapy was practiced in the time of the Pharaohs, still we are to-day utilizing it with all the added information that comes to us from psychiatry, psychology and the psychological laboratory. It has been said that we cannot teach

psychotherapy in our medical schools. He could not understand the force of this statement. Surely if we can practice psychotherapy, and if we know what we are doing when we do practice it, we can tell other people how to do the same thing. It is perhaps true that a course in psychotherapy could not be outlined with the same systematic precision as a course in the practice of medicine, because the principles involved are vaguer, and there is less unanimity of opinion regarding them. And though such a course could not be outlined, and though perhaps the time is not yet ripe for the teaching of psychotherapy in the medical schools, yet the medical student should be taught the importance of the mental factor in the disease. He should not be permitted to go forth, as has been suggested in the speech of President Eliot, with the idea that he is dealing with the human animal solely from the point of view of the naturalist. He should be made to understand that the man must be considered from a broader standpoint, as having characteristics different from the characteristics of the plants and animals that are usually studied, namely, as having a mind, and that after all it is perhaps the mind that is the most important—this at least should be taught in the medical schools and Dr. White believed the time has come to do it.

Dr. James J. Putnam said that as some reference had been made to the Emmanuel Church movement and his connection with it, he would like to say a few words about the matter.

He stated that Dr. Worcester had come to him about a year before saying that persons frequently appealed to him for advice with regard to their troubles which were sometimes of a serious nature and involved questions of mental as well as physical discipline and health. Dr. Worcester had felt both an obligation and desire to meet these calls in the wisest and most thorough manner and desired to have medical aid and support in so doing.

Dr. Putnam believed that this sentiment was a sound one. He did not feel himself called upon either to criticize or endorse every detail of the movement but he thought that the problem of illness had religious as well as medical bearings and that if individual ministers felt a qualification and desire to tackle this problem they should be helped to do so in the way which would be productive of the best results. No one can foretell exactly how far this present movement will reach or what form it will eventually assume, but it would be unwise for physicians to adopt towards it an attitude of complete aloofness.

In regard to the general subject of so-called psychotherapeutics, Dr. Putnam felt that the reason that the mention of it sometimes aroused unfavorable criticism was that feelings were excited which took the place of reason. No one objects to rational discussions on "education," and it is just as reasonable to discuss the methods of "psychotherapy" as it is to discuss the methods of pedagogy. If psychotherapy is old so is education, and yet both are new and as time goes on lose nothing of their importance.

It has been said that most of the patients to whom the methods referred to by Dr. Taylor had proved beneficial were foolish persons whose disorders were of much less significance than those of sufferers from tabes and the like. From this proposition the speaker entirely dissented. It is only necessary to glance even through the table of contents of such a book as that on psychasthenia by Dr. Janet to obtain an idea of the severity of the suffering to which neuropathic patients are exposed.

It had been intimated that the methods of psychotherapeutics were

easy of application, but this also seemed to him an error. Everyone who has seriously tried to apply the recently exploited methods of analysis of subconscious memories, for example, must realize that to do this successfully is equivalent to a surgical operation of the most delicate sort. Yet these methods are useful not only in diagnosis but in treatment. They might be spoken of as the major surgery of neurological therapeutics. The principles indicated by the recent painstaking research of Dr. Prince (The Dissociation of a Personality) are of deep significance and call for skill of the highest order in their application. The same may be said of the principles involved in the treatment of amnesias, as in cases reported by Dr. Sidis and others, and in the elimination of psychasthenic symptoms dependent on the morbid action of subconscious processes. The best proof that this branch of investigation and treatment is in the way of progression and has passed beyond its antediluvial stage of development is that these methods, and the principles on which they are based, were unknown when Dr. Weir Mitchell, with the insight of genius, first established the value of the treatment which has since then borne his name.

It is sometimes said and with justice, as an unfavorable criticism of these methods of treatment that they depend too much on the personality of the physician. But it was partly to obviate, so far as might be, objections of this sort that, as the speaker understood, Dr. Taylor's paper had been written. We wish to train our students to rely on something more that the merely "personal" skill such as might make a successful "medicine-man" or charlatin. It is true that a man must be born with certain characteristics if he is to become an eminent practitioner just as much as if he is to become an eminent artist. Yet there are principles which can profitably be studied in both cases. It is important that we should know what we are doing when we use physical methods of treatment that really depend for their efficacy upon the mental element that goes with them.

Dr. Putnam said he was ready to go further than Dr. Taylor in recognizing that something can be learned even from methods of treatment initiated by persons of practical genius and insight who are not physicians and whose views seem in many respects eccentric. It is liable to happen that physicians get hide-bound in their prejudices and conventions, and medical men, above all, ought to keep their minds open to conviction and train themselves to extract the wheat from the chaff.

Dr. Morton Prince said that he thought that Dr. Taylor was right in describing the general attitude of neurologists towards the method of psychotherapeutic treatment of the psychoses as one of antagonism. A few years ago he would have used a stronger term, that of resentment, but this attitude has recently become somewhat modified under the influence of the wave of medical opinion which is now travelling round the world and influencing thought everywhere. He referred to the attitude of neurologists rather than to that of general practitioners, whom he had found usually more open and more broad-minded than students of our own specialty. The reason of this antagonism was easily found in the fact that the practice of psychotherapeutics requires a special knowledge and technique. The older men were not willing to take up a study of the principles and of the technique in their latter days any more than they were prepared to take up the study of the technique of bacteriology, if they had not already learned it in their student days. In this respect the older men are Oslerized and it is to the younger men we must look to make advances in this method of treatment. Being himself one of the Osler-

ized, he should not himself have been able to take up the study of psychotherapeutics afresh at this time, if his knowledge had not been the gradual growth of accumulated experience and study of twenty years.

There was a confusion of thought which he had noticed in the remarks of the speakers, all of whom had spoken as if there were some one therapeutic method underlying the treatment of all those different forms of psychosis which are open to psychotherapeutics. This is not true. Different psychoses need different methods corresponding to the difference in their pathology. To understand the principles and methods of psychotherapeutics we must have a reasonably deep insight into their pathology. The treatment of the neurasthenic symptom complex needs one method, of obsessions, impulsions, phobias, psycholeptic attacks, etc., needs another; of hysterical stigmata like paralyses, contractures, still another; and so on.

Yet in a broad sense all may be included under the principle of educational treatment. The method of treating the neurasthenic symptom complex he attempted to systematize and formulate about ten years ago, in 1898. To this method, which he then proposed as a substitute for the so-called "Rest Treatment," he gave the name "Educational Treatment." He hoped he might be permitted to call attention to the fact that this was the first time, he believed, that this name was used and the first time that the educational principle was insisted upon. It was a radical departure from the methods in vogue at that time, and the minds of the medical profession were not prepared for the principle involved, as is the case to-day.

The rules of procedure which he laid down were as follows:

After a preliminary study of the origin and grouping of the symptoms and a searching analysis of them, including the mental associations, habits and attitude and the ideas of the patient, then the treatment is carried out along five lines. The preliminary analysis will show that many symptoms constitute an association neurosis.

First.—Instruction of the patient in the nature of the symptoms and disease.

Second.—Fixed ideas, apprehension and erroneous beliefs suppressed by educational explanations with implantation of new, healthy ideas, etc.; faulty habits of temperament and character corrected by instruction and insistence on rational points of view, etc.

Third.—Individual symptoms suppressed by electricity, suggestion, and other therapeutic agents.

Fourth.—Rules given for the daily conduct.

Fifth.—Improvement of nutrition, moderate rest, and, in extreme cases, isolation from previous surroundings only.

The treatment of obsessions, phobias, psycholeptic attacks and some types of hysteria demands a different procedure. Here we require a technique that involves considerable experience and knowledge of mental pathology. Such psychoses often depend upon mental experiences or accidents which lie buried in the forgotten past. The memories of them are often dissociated and can no longer be brought into the synthesis of consciousness. The attacks, whether of fears or impulsions, or psycholeptic, are often recrudescences, automatic recurrences of the original experience; or automatisms which have lost their original associations and now recur from moment to moment, like epileptic attacks freed from their original exciting cause.

For the treatment of such conditions a preliminary psycho-analysis is essential in order to get at the fundamental psychical mechanism and persisting thorn in the mind. Such an analysis requires special knowledge and methods. It is commonly necessary to put the patient in a condition in which the buried memories of the past are awakened. Such a condition is one of abstraction, or what Sidis calls the "hypnoidal" state. But there are several methods of doing this. Having obtained a complete knowledge of the genesis and mechanism of the attacks, the treatment consists in the revival of the dissociated memories and the re-education of the patient by the formation of new healthy mental syntheses, and so on.

In many hysterical conditions the pathology will be found to be subconscious fixed ideas. These must be treated, by similar methods of analysis and educational procedures. In some persons (especially the unintelligent, such as apply to the hospital), calling upon the blind faith of the patient—faith in the power of a given remedy to effect a cure—is sufficient. But these are the exceptions. Dr. Prince often used, at the hospital, a fictitious magnet for this purpose.

Holding these views, it is apparent that he was not at all in accord with what some of the speakers had said. He could not agree with Dr. Dana that the treatment required a "certain setting" and that it was "essential" "to have the person properly staged." On the contrary, this is just what we do not want. All that sort of thing should be gotten rid of. All that was needed was to tell the patient the truth, after first learning, by a thorough analysis, what the truth in each case was. He did not know what Dr. Sachs meant by saying that "altogether too much time and energy are being expended on psychotherapy" and that the sufferings of hysterical and neurasthenical patients are less important than the sufferings of those who are afflicted with various forms of organic spinal disease. There are one hundred persons suffering from functional disease to one from organic spinal disease, and from the point of view of numbers as well as from that of our power to relieve suffering, the former are far more important. We can do little or nothing for organic disease of the spinal cord; we can do everything for functional afflictions. Nor does it matter, in principle, which is the more important.

He disagreed with Dr. Sachs' point of view in other respects, but as he always had done so, he thought he should always continue to do so.

He could not agree with Dr. Collins that the principles of the treatment could not be formulated. They could be formulated. But after this has been done, the formulas would be of no use unless they were applied with intelligence; and to apply them with intelligence one must know the psycho-pathology of disease. One might just as well try to use drugs in accordance with formulas instead of in accordance with our knowledge of the diseased conditions with which we are dealing. No formulas, however, would be intelligible to anyone who is not familiar with the facts with which the formulas have to do.

Psychotherapy may be "as old as the hills," but the educational treatment, as such, was not old but modern. In making allusion to the antiquity of psychotherapy it was evident that many of the speakers had a very slight conception of its principles and less of its methods and technique.

Above all, we must have a knowledge of the subconscious if we are to

understand and cure grave hysteria and allied states, many obsessions, psycholeptic attacks, etc. We may make classifications of symptom complexes and give them names, but this is not a knowledge of pathology. Such a method can have no greater use than any classification can have.

The pathology of many of the conditions he had referred to was to be found in the subconscious. There was no law of the country which compelled a person to study the subconscious if he did not wish to. It was a matter of medical culture, and if a person did not wish to obtain that culture it was his own look-out, but he surely was no more qualified to express an opinion upon the pathology and psychological treatment of those diseases of the mind which involve the subconscious than would a person who had no knowledge of bacteriology, to express an opinion on that subject.

The story runs that an American, visiting Pasteur in his laboratory, said to him that some American bacteriologists did not accept the results of his experiments on hydrophobia. "Have they repeated my experiments?" said Pasteur. Being told that they had not, he replied: "Then I have no time to discuss their opinions. Let them repeat my experiments first."

Dr. E. W. Taylor, in closing the discussion, said that he thought there was considerable evidence of the prejudice in the medical profession of which he had spoken in his paper against psychotherapeutic methods exemplified in the discussion of the evening. The discussion had been characterized by a certain acrimony out of keeping with the ordinary calm and judicial attitude met in dealing with medical problems. Dr. Dana stated that he had met with little opposition on the part of the medical profession towards psychotherapeutic methods and that the reader of the paper had dwelt with too great emphasis upon the neglect of psychotherapy. Dr. Taylor thought that the neglect of psychotherapy, as a distinct and recognized therapeutic measure in the profession at large, was a fact and that as yet no widespread and intelligent interest had been aroused. Hypnotism, and suggestion in the narrow sense, had been left wholly out of discussion in the paper, as on the whole superseded by other and simpler methods, appealing more directly to the reason of the patient. Dr. Taylor did not agree with Dr. Dana in his idea that "staging" of the patient was an essential of treatment, and that more may be accomplished with "weak and foolish people" than with others. On the contrary, he believed that the chief significance of recent methods lay in the fact that no "staging" was necessary, and that decidedly better results could be obtained with persons of culture and general poise than with those whom Dr. Dana had designated as "foolish." The general criticism made by several of the speakers that we are merely rehabilitating an old method of treatment under a new name was in a measure true. No claim whatever was made that psychotherapeutic procedures were a recent discovery. On the contrary the very significance to be attached to the present active movement lay in the fact that the principles underlying psychotherapy were deeply founded and widely recognized. What the present active movement claimed was merely a more minute analysis of the general principles and a reapplication of our knowledge based on closer study freed in great measure from the mystery and superstition of the past. The fact that psychotherapy is as old as the history of medicine should be the strongest argument toward a restatement of the facts in the light of most recent knowledge. Dr.

Sachs' point of view seemed to the writer narrow and unprogressive. Whether or not the application of psychotherapeutic measures was limited it deserved our most painstaking study. It was not likely that the statement that the neuroses were unimportant would be widely accepted, nor was it relevant to the question that we should expend our efforts in finding a cure for organic diseases. The careful study of one therapeutic measure should encourage rather than exclude an equally careful study of others. The dangers of psychotherapy as practiced by intelligent members of the profession, to which Dr. Dercum alluded, could not be regarded as significant. In fact, the paper was designed to show that the rational, common sense, educational methods, with careful analysis of the mental life, were destined rather to remove the dangers to which Dr. Dercum alluded than to foster them. Dr Mitchell and others alluded to the personality of the physician as an important factor in successful psychotherapy. This was in a measure true, but here again the significance of recent studies on the subject lay essentially in the proof it was giving that the personality of the physician was an element of secondary importance, and that men who were willing to devote time and study to the subject, whatever their personalities might be, could use psychotherapeutic methods effectively. A main object of the present agitation was to get away from such vague generalities as the much talked of "personality of the physician." Dr. Collins' interesting objection to the possibility of adequately teaching psychotherapy, on the ground that no methods had as yet been formulated and that diagnosis was not treatment Dr. Taylor thought was not altogether sound. The correct diagnosis of a condition amenable to mental treatment constituted in itself an essential part of the treatment, and certainly there could be no question that methods of arriving at the causes of certain nervous affections amenable to psychotherapeutic treatment had been greatly extended of late years. Dr. Taylor was in general accord with what he regarded as the more liberal and progressive attitude expressed by Dr. Jelliffe, Dr. Meacham, Dr. Hunt and Dr. White. He felt that they had more completely grasped the significance of what he had attempted to state in his paper, the essential object of which was to express the significance of psychotherapeutics not as a new method of treatment, but rather as one which had survived for many years and which now gave evidence of renewed vigor in directions amenable to exact observation and capable of wide applicability. In using the term "teaching psychotherapy," it was hoped that students might hereafter be instructed in the general principles underlying the application of mental means of treatment and in methods of diagnosis which are significant as means of cure.

Book Reviews

Disease of the Nervous System Resulting from Accident and Injury. By Pearce Bailey, A.M., M.D. D. Appleton & Co., New York and London.

This book, which is practically a second edition of "Accident and Injury, Their Relations to Diseases of the Nervous System," has been brought down to date and very capably enlarged and revised.

It has been lately rather harshly criticized because it only touched briefly upon themes which the critic considered deserved long and minute treatment.

We take issue with such a criticism in the case for several reasons. In the first place, if there be any virtue in a single volume of some six hundred pages on a general subject, it lies in the fact that it not only classifies the subject matter, but gives concisely the most generally accepted opinion as to etiology, pathology, etc., without burdening one with every theory held by the myriad of observers each one of whom together with a considerable following proves himself right at the same time as he proves all the rest of the world wrong. This method of treatment may seem *ex cathedra* to one who gets any satisfaction from wading through dreary wastes of conflicting testimony, but to the general medical man, to whom, we take it, the book in question is addressed, it is not only clarifying but relieving.

Let us take, for instance, the part in the introduction anent the subject of blood pressure. The increase in arterial tension is dismissed with very few words but not in a way to belittle its significance. Now the subject of arterio-sclerosis in all its grades is one of the most demanding and important interest, yet it cannot be otherwise than briefly treated in a book of this nature.

The subject of fracture-dislocations of the spine is treated in two brief chapters, yet is this part of the book, excellent in its conciseness, to be condemned because it does not reach the great magnitude of the monograph by Wagner and Stolper?

The book is divided ino two large subdivisions: "Part I, Organic effects of injury to the nervous system;" "Part II, Functional effects of injury—the nervous disorders which most frequently follow railway and allied accidents—the traumatic neuroses."

A rather doubtful gain over the first edition is the printing of the bibliography at the end of the book, instead of having the references of each chapter immediately follow it. The bibliography is very rich and has been brought down to 1906.

It is a very excellent work of its kind, which is that to which the general medical man must turn for instructions and guidance.

There are two species of medical writing: the text-book on a more or less general subject and the exhaustive monograph along one particular line. The former is not the place for the discussion *ad nauseam* of a thousand and one theories, but the latter may well take up a careful consideration of its bibliography, and the more analytical thought brought to bear on the different opinions the better. But the critic who can make no distinction between these two widely separated classes, and who insists in charging up as a shortcoming the absence of a characteristic not rightly belonging to the class in question is singularly obtuse or truculent.

Alfred Reginald Allen.

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Original Articles

DISEASE OF THE PRIMARY MOTOR NEURONES CAUSING THE CLINICAL PICTURE OF ACUTE ANTERIOR POLIOMYELITIS: THE RESULT OF POISONING BY CYANIDE OF POTASSIUM

A Clinical and Experimental Contribution to the Toxic Effects of Cyanide of Potassium upon the Peripheral Motor Neurones [1]

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The peripheral motor neurones, that is, the ganglion cells in the anterior horns of the gray matter of the spinal cord and their processes, particularly the neuraxones or axis-cylinders, are susceptible to injury by many toxic and infectious agencies. When such agencies affect the cell-bodies or intra-spinal processes of the cell-bodies, the resulting disease is known as poliomyelitis. When the extra-spinal processes, or neuraxons, are diseased the condition is called neuritis. For a long time it was thought that the same agency which produces disease of the cell-body was not likely to produce disease of the neuraxone, and in a measure that belief is founded upon fact. The agencies that are most prone

[1] Read before the New York Neurological Society, March, 1908.

to cause inflammation of the motor nerves constituting motor neuritis, are probably not those responsible for inflammation or decay constituting poliomyelitis, save perhaps in very exceptional instances. Numerous investigators have shown that certain metallic substances, such as lead, cause degeneration of the motor nerves constituting a true motor neuritis. Such cases coming to autopsy have shown distinct degeneration of the anterior horn cells of the spinal cord. Moreover, it has been abundantly shown by many investigators, particularly by Stieglitz,[1] that experimental lead poisoning produces degeneration of the entire motor neurone. The question which awaits reply in those cases is the same as we shall raise in the present article, *i. e.*, which part of the primary motor neurone is first and principally affected in certain intoxications?

We have been unable to find any cases of neuritis or poliomyelitis produced by cyanide of potassium poisoning in a fairly careful search of the literature.[2] This article is offered as a contribution to that subject.

We report herewith a case of acute inflammation or degeneration of the peripheral motor neurones, which constitutes clinically a peripheral motor neuritis, but which, however, can scarcely be distinguished from an anterior poliomyelitis. In fact, so far as the reporters of the case are concerned, it cannot be distinguished.

The patient, an Italian, 38 years old, was admitted to the City Hospital, December 3, 1906. A brief synopsis of the history of his illness is as follows: Since coming to this country, in 1904, he had been working as a silver polisher in a hotel. The method of keeping silver bright in such establishments is to drop it into a solution of cyanide of potassium and then dry it. He says that his hands and forearms were in such solution so much of the time that they took on a deep brownish red color and he frequently complained of a distressing itching sensation in them. In addition to this the finger-nails were quite black. He does not know how strong the solution of cyanide of potassium was that he used, but he says that he had to be careful to keep his hands away from his nose or his mouth, for otherwise he would get very dizzy. On the seventh of September, 1906, he was

[1] Arch. f. Psych., Vol. 24, p. 1.

[2] Dr. Larkin, in discussing this paper said he had seen one instance of disease of the entire peripheral motor neurone following cyanide of potassium poisoning.

seized with diarrhea which soon became very severe, the passages containing large amounts of mucus but no blood. The following day he complained of severe headache, of pain and stiffness in the back of the neck and of feeling ill. He was then taken to a hospital and it is said that for a few days he was mildly delirious and had such meningeal symptoms as stiffness of the neck, retraction of the head and sensitiveness on being handled. These symptoms continued for four or five days. Then there developed a sensation of stiffness of both ankles and severe pain in the legs below the knees. Within forty-eight hours his legs and arms were so weak that he could scarcely move them, and from that time he was unable to stand or walk. About this time, *i. e.*, eight days after the onset of his symptoms, he had retention of urine and had to be catheterized for more than a week. After that he had difficulty in expelling the urine, but he got on without having a catheter passed. At this time he did not complain of pain or paresthesia, the symptoms were entirely of the motor apparatus, nor were there any trophic symptoms apparently. His best recollection concerning the atrophy of the muscles, which was so conspicuous on his entrance into the City Hospital, was that it was first noticed about eight weeks after the onset of his disease.

Examination at this time showed that the patient was bedridden, unable to move the lower extremities and to perform very few movements with the upper extremities. The atrophy of the muscles was the most conspicuous objective symptom, and the distribution of this atrophy was as follows: The trapezii were involved to a slight extent. The supra and infraspinati muscles, the serratus magnus, the deltoids, the biceps to a very considerable degree, and the triceps very conspicuously atrophied. The plantars and the extensors of the forearm were also very much shrunken. In the lower extremities the muscles chiefly involved were the anterior tibial group and the posterior calf muscles, the vasti and quadriceps. The left leg lies in a position of external rotation, and it is impossible for him to turn it inwards. Both feet are in a position of drop-foot, the right being more profoundly so than the left. The dorsal surfaces of the feet are slightly cyanotic; the nails show transverse ridges at their bases. The knee- and ankle-jerks are absent. There is a flexor plantar jerk. There is marked atrophy of the glutei over the left hip. In the upper extremities the left deltoid and the trapezius are atrophied and this atrophy throws the shoulder lower than on the opposite side. There is some atrophy of the muscles of both forearms, and there is marked shrinkage of the thenar and hypothenar eminences of the left hand.

Tactile, thermal and pain sensibility are normal. There is no disorder of function of any of the cranial nerves.

Electrical examination shows that the anterior tibial muscles contract on both sides with a strong faradic current. There is

a slight response to the faradic current in the quadriceps, but the reaction is sluggish and vermiform. In the other atrophied muscles the faradic contractility is lost apparently in proportion to the intensity of the atrophy. In all the muscles there is a slight reaction to the galvanic current with polar alteration characteristic of reaction of degeneration.

For upward of six months there seemed to be no indications of recovery. Then gradually the atrophied and paralyzed muscles of the upper extremity began to display slight functional capacity. In the autumn of 1907 braces were put upon his legs and now he is able to walk with the aid of crutches. Examination of him at the present time shows that the muscles of the neck and trunk are of good volume and capacity. Both deltoids are atrophied, the left more profoundly than the right. External rotation of the shoulders is weak. The muscles of the arm and forearm seem to be in good condition, save the supinator longus and the biceps of both sides. The triceps, which was very much affected in the beginning, now seems to be approximately normal. The extensors of the right wrist are weaker than those of the left, but there is no evidence of atrophy in the muscles. The cranial nerves are intact.

Flexion of the thighs is accomplished very indifferently, but better on the left side than on the right, while extension of the legs upon the thighs, which is also very weak, is accomplished better on the right than on the left. There is double foot-drop. He can flex the toes of the right foot but not of the left. The crural muscles and the calf muscles show the greatest amount of atrophy.

The knee-jerk is present on both sides but extremely weak and sluggish. The abdominal and cremasteric jerks are elicitable. The right Achilles jerk is present but sluggish, and the left absent. Tactile, pain, and thermal sensibility seems undisturbed. There is no tenderness of the nerves to deep-seated pressure. Distinct fibrillary movements are occasionally to be seen in the gluteal and deltoid muscles.

In other words, the patient has made a fairly good recovery and he is still improving. The only symptoms that tend to indicate the dependence of some of the atrophy upon spinal cord lesion is the fibrillary phenomenon to be observed in the muscles in which the atrophy is still most pronounced.

Taken in conjunction with the experimental work that has been done to determine the effect of cyanide of potassium upon the peripheral motor neurone, it is obvious that the entire motor neurone was affected by the poison but that the brunt of the lesion was borne by the axones at their periphery (see Plate I).

The important features of the disease as they presented themselves in this case were: (1) The rapidity with which the symp-

toms came on and their intensity. In character they were of an overwhelming intoxication or infection; (2) the occurrence of

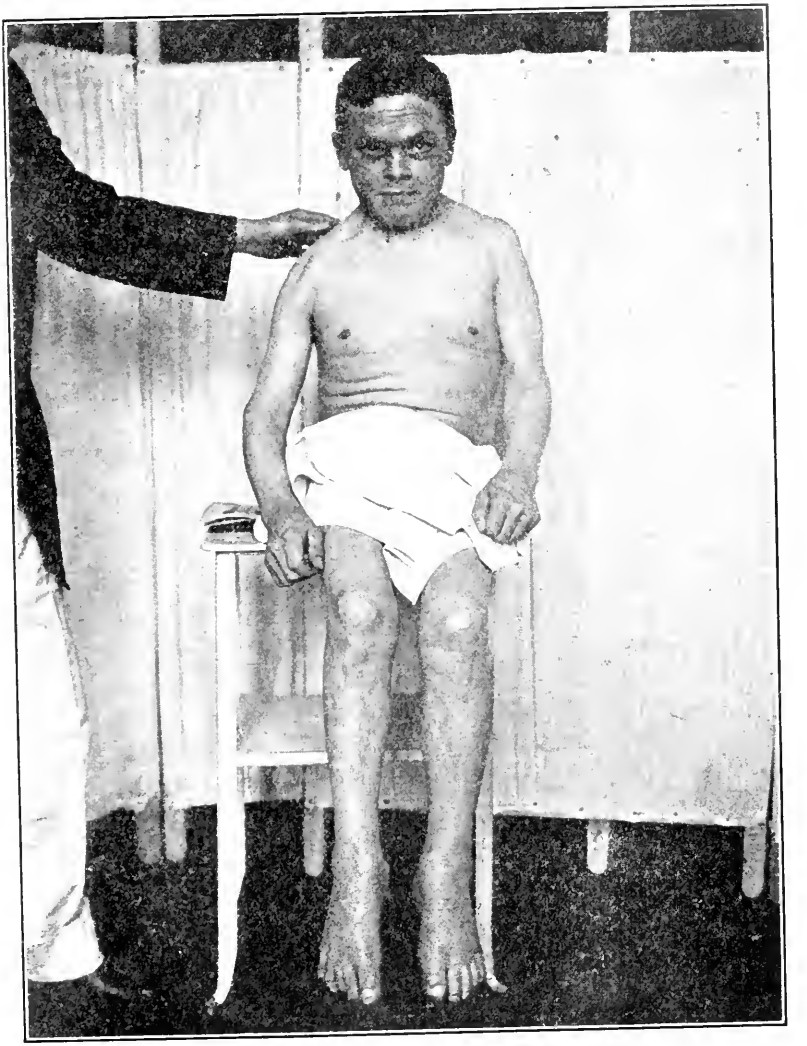

FIG. 1. Picture of patient showing drop foot atrophy, etc.

spinal cord symptoms; (3) distribution of the atrophy and partial recovery.

Unlike the neuritis that is caused by other metallic poisons such as lead and arsenic, there seem to have been no premonitory

symptoms whatsoever, although there are cases of lead neuritis in which there has been no history of colic, digestive disturbances, etc., and there are cases of arsenical neuritis which are not preceded by the peculiar garlic odor of the breath; such cases are very uncommon. In our patient there seemed to have been no premonitory symptoms except the symptoms of gastro-intestinal irritation. The extent and intensity of the atrophy as well as the intensity of the motor incapacity which preceded it, bespeak the profound involvement of the motor neurones in their entirety.

From the experimental work which we have undertaken to show the effects of potassium cyanide upon the peripheral motor neurones, we are led to the belief that the entire motor neurone is affected, the neuraxone, however, more than the cell-body, but the participation of the cell-body in the destructive process is fairly certain, and this participation accounts for the occurrence of spinal cord symptoms in our patient. That the atrophy, however, that is, the degeneration in the muscles, is not primarily dependent upon spinal cord lesion, is best testified to by the distribution of the atrophy. It would have to be an extremely extensive process in the spinal cord and such extensive lesion would cause symptoms that did not occur in this case. Moreover, the degree of recovery which has taken place indicates that the cell-bodies of many of the peripheral motor neurones, which at first seemed to be very profoundly diseased because of the amount of atrophy, are in reality in possession of sufficient vitality to assist in the regeneration of the diseased neuraxons.

The following experiments were done for the purpose of studying the effects upon the nervous system of cyanide of potassium administered in poisonous doses:

Four healthy rabbits were used in the experiment. The two first died, the two last recovered.

The salt of hydrocyanic acid used was the potassium salt, which was made in a sterile water solution, 1 c.c. equaling 2 mg. of the pure salt. The injections were made with a sterile hypodermic syringe, usually into the posterior auricular vein. The wound was immediately sealed with collodion.

All spinal cord specimens were hardened in 10 per cent. formalin, followed by alcohol, imbedded in celloidin and cut at about 6 microns; sections were then stained in 1 per cent. aqueous methylenblue, being gently heated until the dye steamed; they were then differentiated in absolute alcohol, 90 parts, and anilin oil 10 parts, washed in alcohol and cleared in xylol.

Rabbit No. 1 received 2 mgs. of the drug every other day for a period of sixteen (16) days, or a total of 16 mgs. On the sixteenth day a complete flaccid paralysis of both hind legs was noticed, accompanied by loss of reflexes, and bladder and rectal incontinence. Sensation was apparently normal. On the seventeenth day the rabbit was much worse; both forefeet were now completely paralyzed, and there was slight twitching of the neck muscles. On the eighteenth day, as the animal lay in his cage, he was totally helpless, unable to hold up his head, and paid no attention when food was offered. Sudden disturbances, such as noise, were followed by convulsive movements of the head. Towards evening he died—sixty hours after the appearance of paralysis.

The following is a summary of the autopsy findings: Dilatation of the left ventricle of the heart; pulmonary edema, with hemorrhage in bronchi; parenchymatous degeneration of kidneys.

The following tabulates the distribution of the lesion found in the spinal cord: At the level of the oblongata, the anterior horn-cells were normal. Sections from the upper portion of the cervical enlargement were also normal; sections from the mid-portion of the cervical enlargement showed some neurones in various stages of degeneration. Sections from the mid-dorsal showed marked destruction of the anterior horn-cells of both horns. Sections from the lower dorsal also showed extreme destruction. Sections from the lumbar region showed scattered cells in various stages of destruction. Sections from the extreme lower lumbar region showed apparently normal neurones.

The changes observed in the anterior horn-cells were central chromatolysis with nuclear eccentricity, leading to entire destruction of cytoplasm, with vacuolization, shrinkage, and almost complete solution of the cell.

The lesion apparently commences in the chromatophilic granules which do not stain with the same intensity as they normally do; they appear pale and have ragged edges. At the same time, the nucleus also becomes swollen, and its nuclear membrane becomes well marked and distinct.

The chromatophile flakes now become granular and disintegrate, causing a great diminution in the number of bodies; this takes place first around the axone hill and nucleus. The cytoplasm around the nucleus becomes vacuolated, filled with fissures, or even entirely devoid of matter.

While the chromatolysis is going on, the nucleus also shows changes. Owing to a loss in substance of cytoplasm, it is left unsupported and falls to one edge of the cell—it becomes eccentric. The nucleoplasm becomes granular, causing the nuclear membrane to become puckered and crenated. The nucleolus remains clear and sharply defined, it being retained when all other structures of the cell are lost. The nuclear membrane is finally lost and complete destruction of the nucleus soon follows.

With such great loss in bulk of cell, it, as a whole, shrinks, leaving an open cell space; the cell may finally break up and disappear (see Plate II).

A portion of the right anterior tibial nerve, when teased out in glycerine and stained with osmic acid, showed extensive degeneration, with destruction of the myelin sheath, and the presence of irregular-sized fat droplets (see Plate III).

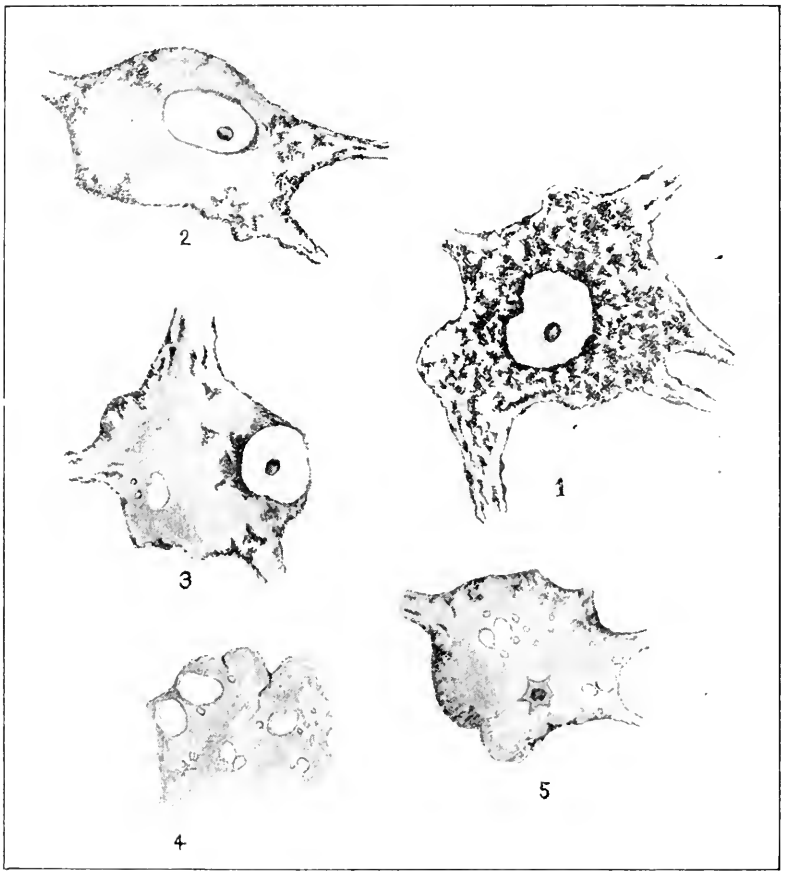

FIG. 2. Illustrating changes in ganglion cells of cord of rabbits.

Rabbit No. 2 received 2 mgs. every other day for ten days. At the end of this time the animal became very quiet, respirations and pulse were rapid, food was refused. On the morning of the next day the animal was paralyzed in both hind legs; at noon his forefeet were involved, and he died during the night—about twenty-four hours after paralysis had started.

The spinal cord was taken out immediately and studied, with the result that the anterior horn-cells, throughout the mid-cord, and in the cervical enlargement, showed the various stages of chromatolysis and neurone degeneration above described. Teased portions of the anterior tibial nerve showed fatty degeneration of myelin sheath.

Rabbit No. 3 received 12 mgs. over a period of fifteen days. Except for slight diarrhea he had no ill effects.

Rabbit No. 4 received as an initial dose 4 mgs. He immediately went into severe convulsions with rapid panting respirations, which soon stopped. Artificial respiration revived the ani-

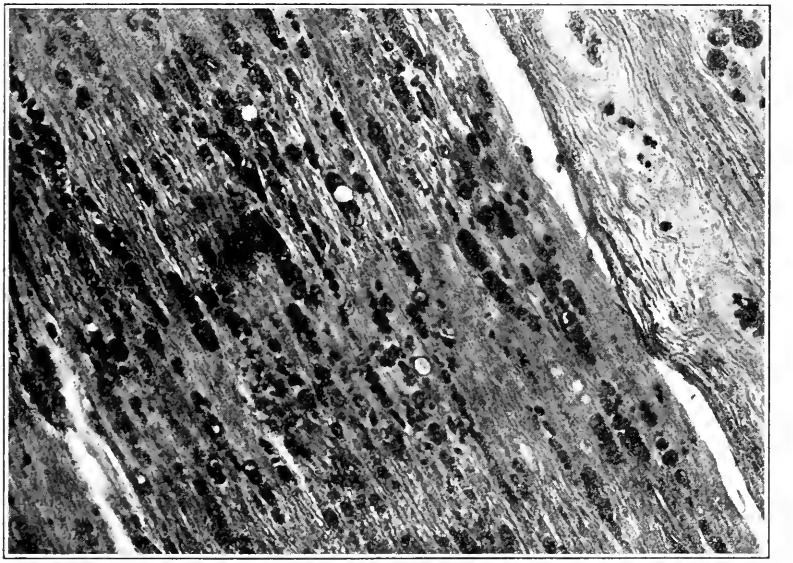

FIG. 3. Section of a peripheral nerve from a paralyzed rabbit, stained with osmic acid. Showing a severe parenchymatous neuritis, with formation of fat globules in the myelin sheath.

mal from the acute cyanide poisoning. After this he received a total of 14 mgs. in fourteen days, but no paralysis developed.

In the two last rabbits many of the injections were not made directly into the vein, but in the surrounding subcutaneous tissue.

There has been a great deal of controversy as to the nature of chromatophile bodies. F. B. Bailey (*Journal of Experimental Medicine*, 1901) concludes that "Nissl bodies are not present in the living cell, and that the chromatophile granules seen in the Nissl preparations represent a precipitation of the substance previously held in suspension in the cell protoplasm, caused by post-

mortem change, or action of fixatives." These bodies are best observed in tissues fixed in acid fixatives.

It is also not certain that the cytoreticulum is present in the living cell (as it can be seen only after fixation) or whether it, too, is a precipitate formed before, or at the time of the formation of the Nissl bodies.

These observations, however, make little difference in the case in question, as we know the chromatolysis (in our present conception of the term) is not the rule in the normal neurone and that a disappearance of the chromatophile bodies, as seen in these cases, associated with vacuolization and nuclear disintegration, is caused by a neurone injury, which leads to a partial or complete degeneration.

In conclusion, it would appear that the changes observed in the anterior horn-cells were due to a further extension of a peripheral multiple neuritis, that is, an axone degeneration which is so severe that it finally affects the neurone itself. In a chromatolysis, due to toxic material in the blood serum, which bathes the periphery of the cell, the chromatophile bodies are first destroyed around the periphery of the cell, and they last disappear around the nucleus, or, in other words, a peripheral chromatolysis is more common than a central one. A nuclear eccentricity is also very uncommon. In these cases, the foregoing, however, is not true. Here we have a chromatolysis which takes place, first, around the axone hill, and around the nucleus (a central chromatolysis), indicating that the cell has suffered internally, so to speak, by the extreme degeneration that has already taken place in its axone.

THE SIGNIFICANCE OF PHRICTOPATHIC SENSATION

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During the process of recovery from a hysterical anesthesia a great variety of abnormal sensations may be experienced by the patient when the part in question is stimulated. Certain features characteristic of these sensations occur in such constant association that it seems justifiable to include them in a single group, which I have proposed to designate by the term phrictopathic (14). So far as I am aware the unity of the group has not previously been recognized, though, as will presently be shown, indications of several of the individual features are to be found in the literature. A description will first be given of the six features characteristic of the symptom and then a discussion of its pathological significance. The symptom may be met with in all degrees of intensity, and naturally its characters are most easily to be studied in cases in which it is most marked. As the most marked instance of it I have observed was in a patient in the achiric stage of dyschiria, a syndrome I have elsewhere described (15), I shall refer particularly to this case at first and point out later the milder forms in which the symptom presents itself. The individual features of the sensation may now be considered in detail.

1. *Abnormal Persistence.*—Instead of the sensation ceasing to be experienced immediately the cutaneous stimulus is withdrawn, as it does in the normal, it here persists in unabated intensity for a variable time. In the case referred to above the sensation persisted for 50–60 seconds; the shortest period I have known it to persist has been—in other cases—six or eight seconds, and between these two extremes all gradations may be observed. The dying away of the sensation is a rapid process

occupying usually only a second or two. Many of the after-sensations which are well known sometimes to follow cutaneous stimulation in hysterical anesthesia are probably of this nature. Binswanger (4) has described these "pathologischen Nachempfindungen" as "brennende, ätzende, kriebelnde Schmerzen," which might well pass as a description of the milder forms of phrictopathic sensation.

2. *Delayed Reaction Time.*—Any reaction time involving a conscious process is unduly delayed. An interval of between two and six seconds elapses before the stimulus is consciously appreciated. This fact has been noted in cases of allochiria by Bose (6), Janet (10), and others. It will be remembered that, as was first demonstrated by Onanoff (20), reaction times in cases of hysteric hypoesthesia are shorter than normal, provided the mental process is subconscious, as in a simple motor response.

3. *Non-perception When a More Normal Sensation is Present.*—I have not met with any previous account of this observation which I believe to be of importance in connection with the theory of the symptom. Two sets of facts have to be distinguished according as the normal sensation is evoked simultaneously with or subsequently to the application of the stimulus evoking the abnormal sensation; this may be illustrated by taking the case of the above patient who had achiria of the right limbs. (*a*) If stimuli were applied simultaneously to the right and left arms, only that applied to the latter was appreciated. (*b*) If the abnormal and long-lasting sensation was first evoked by stimulation of the right arm, and then, while he was still feeling this, a stimulus was applied to the left arm the abnormal sensation on the right side was instantly abolished.

Two further observations are necessary to complete the description of this point. In the first place sensibility in this patient's right arm was absolutely intact even to the most rigorous of the usual tests, so that although he could appreciate the lightest Von Frey hair when this was applied alone he felt nothing at all in the right arm, however strong the stimulation, provided the left arm was simultaneously touched. In the second place the observations were perfectly constant, and in some thousands of tests I have never seen any exception to the above rule.

In a case of bilateral allochiria in which the defect in auto-

somatognostic feeling (see below) was more marked on one side, a stimulus applied to the more normal side acted precisely in the same way as a stimulus applied to the quite normal left side in the unilateral case; that is to say, it prevented sensation being evoked by a stimulus applied to the less normal side if applied simultaneously, and abolished it if applied subsequently.

4. *Tendency to Immediate Motor Response.*—The stimulus is followed by an instantaneous start which the patient is quite unable to prevent; this sudden jerk is so fatal and irresistible as to give the appearance of a direct reflex. This has also been noticed by Pitres in connection with a symptom called by him haphalgesia, which we shall presently have to discuss. He states (21) that immediately on experiencing the sensation in question the patient "se recule vivement." It is also met with in connection with what Head (9) has termed protopathic sensation. Like the latter, phrictopathic sensation is also very disagreeable, but there is no further resemblance between the two either in their other attributes or in their origin.

5. *Disagreeable Quality.*—This is the most striking feature of the group. The sensation is never one of pure pain. When only slight it is mainly one of "queerness," "unnaturalness," "strange disagreeableness"; when more pronounced it is one of unpleasant tingling or shuddering; when very marked this shuddering has an intense quality that can be described only as horrible. In other words, this peculiar quality of the sensation is always unpleasant, is often very disagreeable and sometimes constitutes a horrible shudder, on account of which feature I have described it as phrictopathic. The sensation is essentially diffuse and may radiate not only widely over the limb stimulated, but even over the whole length of the body. In the achiric case referred to above it was most striking to witness the way in which the patient, sweating with horror, was relieved from his misery by the lightest touch applied to any part of the left side of his body.

Indications of this feature, though in a much less marked degree, are given in many cases described in the literature. Thus Janet, in a case of bilateral achiria, states (11) that cutaneous stimulation "provoque seulement une sensation vague de quelque chose de gênant, sans que le sujet sache ce que c'est. Ce n'est

pas une douleur proprement dite, c'est surtout une sensation indistincte." Pitres (21) describes the sensation in haphalgesia as "une sensation indéfinissable de vibration chaude, plus désagréable assurément que celle que provoque du côté opposé le passage d'un fort courant faradique." Elsewhere (22) he describes it as "une sensation très vive de brûlure." Gilles de la Tourette (32) also described haphalgesia in one of his cases as "une sensation douloureuse qui ressemblait à une brûlure." Sollier has given a more detailed description of the quality in several of his cases. Thus in Observation V (25) he describes the return of sensibility in a previously anesthetic limb as being accompanied by "des sensations de fourmillements, de crampes, de tiraillements, de brûlure, etc." Under like circumstances in Observation VI (26) the patient says "qu'elle a des fourmillements, et des élancements, des crampes, que ça tire," and (27) that "Cela me picote, me brûle comme si on m'avait frottée avec des orties"; on another occasion (28) she describes the sensation thus, "Ça tire; ça picote; j'ai des crampes et des brûlures en même temps; c'est comme des caoutchoucs; il y a quelque chose qui coule dans mes jambes; ça fourmille," etc. In connection with this case and also with that of Observation XVII (29) the tendency to immediate motor response is also fully described.

An observation I have frequently made is that the patient "tries to rub the touch off"; this no doubt is due partly to the tendency to motor response, but mainly to the disagreeable irritating nature of the sensation. Sollier (27) remarked the same fact in one of his cases.

6. *Impairment of the Sense of Personal Ownership.*—The sensation gives to a patient a curious feeling that he usually describes as "being touched on some part that doesn't belong to me." Like the other attributes mentioned above this varies greatly in degree. When slight it feels as though the part touched is "strange"; "funny," "hardly belonging to me"; when it is more pronounced the part touched feels decidedly foreign, and when very marked the part touched is definitely repudiated by the patient as in any way belonging to him; it is then as though the information about the stimulus, which may be perfectly correct as regards its nature, position, etc., comes to him from nowhere. To give an instance of this, one of my patients

touched on the forefinger would say "You are touching the back of some forefinger with a blunt pin; it isn't my finger and I have no idea where it is, but it causes an intensely disagreeable shudder to run all up one side of me."

We thus see that there are plentiful indications in the literature of similar attributes to those grouped here under the term phrictopathic. I have little doubt but that the symptom described by Pitres (23) under the name of haphalgesia is identical with phrictopathic sensation. Pitres, who at that time was unfortunately under the influence of Charcot's erroneous teaching on the subject of metallotherapy, wished to indicate by this expression a disagreeable sensation that a patient experienced in an otherwise anesthetic part when it was "stimulated" by contact with various precious metals. He described the symptom in several cases (24), certain metals being considered to be efficacious in some cases and other metals in others. The only references that I have found to the phenomenon besides those of de la Tourette just mentioned are in the writings of Janet (13), Loewenfeld (19), Bardonnet (2), Binswanger (5), Decoux (7), and Lannois (18). The latter describes the case of a female tabetic with extensive anesthesia of the left arm in which the symptom was produced by contact with copper only. All these authors seem to accept Pitres' description and explanation, though Janet would restrict it to only certain cases. It need hardly be said, however, that this supposed dependence of the symptom on contact with metals was due to the fact that at that time metals were thought to be powerful esthesiogenic agents, the importance of suggestion in this connection not being recognized. As Dr. Jung once drily remarked, it is not only hysterics whose hands are excited by contact with precious metals.

We come now to the pathogenesis of phrictopathic sensation, but before we discuss this a few remarks are necessary concerning the two fundamentally different types of hysterical anesthesia. These are best understood by considering for a moment the different sets of mental processes that have to do with a given part of the body in the normal. These are sharply divisible into two main groups. *First* there are the mental processes that depend on the incoming excitations flowing in from the bodily mem-

ber at a given moment and which may be called the esthesic[1] sensibilities. This group is composed of two subgroups, first the sets of common sensibilities (touch, pain, etc.) that have to do with the immediate relation of the member with the external world, and secondly the sensations, largely subconscious, which have their origin in the functioning of our internal organs; a good description of the latter subgroup, which is called cenesthesia, is given by Sollier (30). *Secondly* there is a group of mental processes that are essentially of the nature of memory feelings. All the mental processes of diverse origin that in the past have had to do with the member in question go to form this group. To enumerate only a few of these, there are the memories relating to its functions, both motor and sensory, memories relating to its appearance, "sidedness," position, and all that the member stands for to the individual; in short, all the complex of memory feelings that may normally be aroused by the mention, touch, sight, or thought of the member in question. This second group might be thus described by the name autosomatognostic; in the past it has often been confused with the totally different group of cenesthesic sensibilities.

From the standpoint of pathology perhaps the most important difference between the esthesic and autosomatognostic groups is the outstanding fact that whereas only the former can be affected by organic disease, either or both may be affected by functional disease. Affection of the second group is a form of amnesia, of either a retrograde or antero-retrograde type, and localized amnesia bearing on one set of memory processes is a form of psychical disaggregation highly characteristic of hysteria. On the other hand it is most striking that complete abrogation of all esthetic sensibilities has no appreciable effect on the autosomatognostic group. Every nerve in the limb may be severed, the limb may even be amputated, and the patient still has the liveliest memory of what it feels like to have such a limb and enjoy its functions and has a normal knowledge of all that such a limb means

[1] We seem compelled to employ this etymologically indefensible term, for the regular derivative formed from the Greek genitive has become so infused with meaning foreign to the sense here intended that its use here would tend to lead to confusion; to speak of "esthetic sensibility" in the present connection might be ambiguous. We have the precedent of "cenesthesic" to support the use of the term "esthesic."

to the individual; in the case of amputation in infancy such memories never form, but that is of course a different matter.

We thus see that in hysteria two entirely different forms of anesthesia occur, according to whether only the sensibilities of the part or the sensibilities plus the memories of the part are dissociated. One important difference between these two anesthesias concerns the amount of distress caused to the patient. With the ordinary hysteric anesthesia in which only the first group is affected the direct result so far as the patient's well-being is concerned is almost negligible, and in fact is considerably less than the result of an anesthesia of organic origin. This is strikingly illustrated in Janet's well-known case (12) of a girl who came to the Pitié hospital complaining of the inconvenience she suffered from a patch of anesthesia which was due to an injury of the median nerve. In the course of examination it was demonstrated, to the patient's surprise, that she was completely anesthetic on the whole of the opposite half of the body. In fact the patients' ignorance of their anesthetic stigmata was, as De Lancre mentioned three hundred years ago (17), widely known at a time when these were regarded as marks of the devil's claw, and the notorious frequency with which they are overlooked by the patient has led to the erroneous inference, by Babinski (1), Bernheim (3) and others, that they are usually artificially created by the observer.

In sharp contrast to the patients' ignorance of this form of anesthesia is the great distress and misery suffered by patients who have in addition the rarer autosomatognostic defect. Autosomatognostic memories seem to be intimately connected with our feelings of capacity, adequacy and well-being, but above all with our sense of personality. In fact the condition in question is usually described by authors under the name of "partial depersonalisation" and the importance of it to the feeling of personality and well-being has frequently been noted. When it is very pronounced the loss of the sense of personal ownership of the part concerned is very striking. The patient may have so thoroughly forgotten what it feels like to have a given member that he becomes totally incapable of imagining the normal feeling in this respect or even of in any way understanding what is meant when it is referred to. Thus the patient of mine referred to

above had so completely forgotten what it felt like to have a right arm that he did not understand one when one used the expression "right arm." He could as readily have imagined the feeling of a wing growing from his right shoulder as that of an arm and would have felt as competent to use the one as the other; the amnesia for all that a right arm stood for was complete.

When now in this graver type of anesthesia recovery comes about it often does so only gradually. In the majority of cases both groups of mental processes, *i. e.*, sensibility and autosomatognostic feeling are recovered synchronously. If however sensibility begins to return first then the dawn of sensation is a vague impression that something is being felt, but where, what, or how is to the patient a mystery. He has forgotten what such sensations mean and only slowly recovers the memories they normally evoke. He not only feels a touch on a part that is not accustomed to feel touches, but he knows nothing of this part even, as is shown by the sense of foreignness it conveys. It is thus like feeling a touch with a part that has no previous mental history, a feeling that a normal individual can try to appreciate only by imagining that he were to experience a touch with some newly added part of a body, the nature, functions, shape and position of which were entirely unknown. The whole process is to the patient such a novel, strange and uncanny process as amply to account for the bizarre and disagreeable feeling experienced. An excellent descriptive account of these various feelings experienced on the "réveil de sensibilité" is given by Sollier (31).

This consideration seems to me to be of fundamental importance in relation to the problem of phrictopathic sensation. In the study of any case showing this symptom one of the most striking facts observable is the close correlation between the intensity of the abnormal attributes above described and the depersonalization or loss of the sense of personal ownership of the part concerned. If the patient above referred to came one day and told me that his right arm felt "funny," "strange" or "sleepy," I could be quite certain that the sensation elicited by a touch on the arm would persist for from eight to twelve seconds, that the delay in the reaction time would be two seconds and that the disagreeable quality would be of only a moderate degree. If on the other hand it was found that such a sensation

persisted for sixty seconds, I could predict that the patient would not describe his right arm as "sleepy" or even as "dead," but that he would declare that he "hadn't a right arm" and that he could feel nothing whatever beyond the shoulder joint. The phrictopathic symptom is therefore intimately correlated with and probably dependent on what I have termed the autosomatognostic defect.

The essential point in the pathogenesis of phrictopathic sensation seems to me to be the occurring of a cleavage between the esthesic sensibilities and the autosomatognostic memory feelings, and the intensity of the characteristic attributes to be a measure of the extent of this cleavage. This explains why in most cases of the severe type of anesthesia, where the recovery of sensibility is only a little, if at all, in advance of the recovery of autosomatognostic feeling, the attributes are so elusive and faintly marked as almost to escape the observer's attention. In order that the cleavage should be pronounced it is necessary that the loss of autosomatognostic feeling should be profound, or, put in more modern language, it is necessary that the inhibiting or suppressing force of the underlying painful feeling-complex, which Freud (8) demonstrated thirteen years ago to be the cause of the resistance to becoming conscious that characterizes disaggregated mental processes in hysteria, should be unusually great; this is so when the autosomatognostic feeling bears a peculiarly intimate association to the central complex, as I have been able to show by the psycho-analysis of my cases. A further point worthy of remark is that the cleavage in question is usually, and perhaps always, accompanied by the presence of one or other of the dyschiric manifestations, which I have described in detail elsewhere (15).

Let us now briefly review the individual features of the symptom in the light of this hypothesis, keeping well in mind the difficulty with which the sensation has to contend before reaching consciousness, this being due to the resistance caused by the suppressed complex with which it is associated. The delayed reaction time is a direct consequence of this difficulty, and has been amply demonstrated by Jung (16), in association experiments, to be characteristic of the stimulation of a submerged painful complex. The abnormal persistence of the sensation and the

irresistible tendency to an immediate motor response are attributable to the uncontrollable, impulsive and automatic activity of a mental process associated with a complex that is unassimilable in consciousness, a well-known psychical phenomenon. The obviously defensive nature of the motor response is a typical instance of the resentment elicited by irritation of a painful feeling-complex, and is a measure of the resistance to the assimilation of this. The non-perception of the sensation in the presence of a rival normal sensation is similarly attributable to the difficulty with which the mental process contends in reaching consciousness; the resistance can be overcome only when no competing mental processes are present. The mental state of hystericals used to be described some years ago in terms of diminished power of attention; stated in this language we may say that the least opportunity to attend to some other normally experienced sensation is automatically seized upon and the painfully associated one is once more suppressed. The disagreeable quality of the sensation I would attribute directly to the amazingly strange and bizarre nature of the mental process, which is so indescribably foreign from any previously experienced by the patient. The impairment of the sense of personal ownership of the part stimulated is of course easily explicable by the defect in autosomatognostic feeling that is at the base of the whole phenomenon.

Conclusion.—Sensations showing the six features here grouped together under the designation phrictopathic are due to a cleavage between the esthesic sensibilities and the autosomatognostic memory-feelings of a part of the body, which results from hysterical disaggregation implicating the latter group of mental processes; the degree to which the features are marked is an accurate measure of the extent of this cleavage.

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TUMOR OF THE FRONTAL LOBES WITH SYMPTOMS SIMULATING PARESIS.[1]

By F. X. Dercum, M.D.

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D. P., white, age 50, clergyman; unmarried, first presented himself for examination on September 14, 1905.

The family history was unimportant. The father died at 82; mother at 69 of some affection of the heart. There are two brothers and three sisters, all of whom are in good health.

Personal History.—The personal history is as follows: The patient was healthy in early life and had never had an illness of moment save that nine years ago he had congestion of the lungs from which he made a good recovery.

Three months ago he became aware of an impairment of vision in his right eye. No ophthalmoscopic examination was made at the time, the patient merely buying a pair of glasses which he himself selected. Some time subsequently he began to suffer from headaches, more marked upon the right side. The headaches were not continuous and were never accompanied by vomiting or nausea.

The patient denies that he has suffered from any seizures or attacks of any kind. The brother, however, who is present during the examination, states that he thinks the patient has had spells in which "he could not do what he wanted to do." His friends have also noted the following changes in his demeanor and manner. He appears to be easily pleased and his friends express it by saying that he is "of an easy disposition." He was at one time exceedingly active and interested in his clerical duties but now is indifferent to his duties. He never worries about anything. He used to be very conscientious but now is apt to take everything "as a joke." He is never serious. Formerly he was very punctilious, but now he is careless in regard to keeping his appointments and indifferent as to beginning his services at the

[1] Read by title at the Thirty-fourth Annual Meeting of the American Neurological Association, May 20, 21 and 22, 1908.

right time. At one time he assigned as a reason for not beginning his services at the proper time by saying that the congregation was not present, which was not correct. He has also done various erratic things, such as visiting his friends and forgetting altogether the proprieties as regards the length of his stay. Instead of remaining for a short time as was his wont, he would remain seated an entire evening, indeed until far into the night. At another time he visited some friends and instead of remaining for a day or two, remained to their surprise for a number of weeks. This conduct contrasted strongly with his former habits. His housekeeper stated to his brother that he appeared to be dizzy at times and that at such times his face became purple. He had had considerable worry because of his clerical duties; the duties had been arduous and he had been under considerable strain. Of late it was also noted that he was somewhat somnolent.

The physical examination reveals his station and gait to be normal. At times he appears to stand less readily upon the right leg alone than upon the left; but this symptom is not definite in character. He holds his head a little to the left but his friends who accompany him say that this is a habit, that he has always done so. His grip is good and equal upon both sides and there is no intention tremor. There is, however, a distinct tremor of the tongue. The lips are held steadily, though some tremor also is noted. The angles of the mouth are retracted equally well. The palpebral fissures are equal in size and normal. The knee jerks are both equal and perhaps a little plus. There is no ankle clonus and both plantar reflexes are normal. There are no sensory anomalies. The visceral examination also is negative.

The patient's expression is that of indifference. His answers are not always responsive to the questions asked, and his enunciation is somewhat indistinct though whether this is due to indifference or to actual difficulty of articulation, does not appear.

When asked to remove his clothing, it was noted to our surprise that he had not a single undergarment upon his person. He had neither stockings, drawers, undervest nor shirt. He had simply put on his shoes, trousers, vest and coat. When asked as to why he had dressed himself this way, he did not seem to realize that he had done anything unusual. His manner was very much like that of a paretic. He had no realization of the fact that he was ill, indeed his attitude was that of denial of illness,

declaring repeatedly that he felt good, that there was nothing the matter with him save that he had some trouble with his eyes. His answers were only elicited by repeated questioning and then were usually unsatisfactory.

An eye examination, made by Dr. Charles S. Turnbull, revealed optic atrophy apparently secondary to a neuritis. It was most marked in the right eye. The pupils were equal and reacted feebly to light. The fields could not be satisfactorily studied because of the inability of the patient to give the necessary attention and because of his disposition to jest.

Hearing appeared to be normal. Taste also appeared to be preserved. The sense of smell, however, was lost; at least his answers were unsatisfactory and confusing. The diagnosis of coarse encranial lesion, probably a tumor, the location of which was pre-frontal, was made. Because of the condition of the optic nerves, a decompressive operation was discussed, but not seriously considered by the patient's friends. It was decided to place him in the St. Agnes Hospital where he remained, under observation, a number of weeks. Although no specific history was elicited, a trial was made of both iodides and mercurials with a negative result.

He now passed from under observation. His brother, who is a physician, informs me that subsequently the diagnosis of paresis was made. The patient had deteriorated morally and had gone so far as to behave improperly with a servant. Some weeks before his death, he began to have seizures with loss of consciousness. The seizures would be very brief and would not cause him to fall to the ground. They rather resembled petit mal. He was brighter, said his brother, toward the last and was full of anecdotes of his past life. At times he would be a little tottering in his gait. At other times again he would seem very strong. Two days before his death it appears he had an epileptic attack. He was missed, was found in the bath-room and had the appearance of having had a fit; he had also vomited. Later in the evening of the same day, his brother found him quite alert and without signs of any paralysis. There was, however, some incontinence of urine. His pulse was 106, respirations 24. He was restless but responded well to sedatives and the next morning seemed to be in about his usual condition. That evening, however, he suddenly passed into a state of collapse. His respirations increased

to 36 or 38 and his pulse to 124. He was unconscious and could not be roused and there was present some edema of the right lung. The next morning he rallied. He seemed quite rational and knew everyone. His respirations now became quite rapid, 50 to the minute and his pulse 140. He gradually became weaker, became unconscious and died the morning following. During the night his temperature rose to 102. For some time previous to his death his eye-sight had deteriorated so much that he could hardly more than distinguish night from day.

The autopsy was made June 6, 1907, by Dr. A. H. P. Leuf, to whom I am indebted for the following notes:

Short, stout man. Face discolored by small hemorrhagic extravasations into the skin, especially marked over lower half of right cheek, and more or less over other parts of face. These were not post-mortem lividities. The latter were present as commonly in dependent parts of the body. Hair scant on top of head and mostly gray.

Scalp was very thick and stiff. Veins fully distended. Skull cap thick, rather hard and diploic layer very thin or absent. Dura very adherent to calvarium and pacchionian bodies thick and wart-like, more on left than right side and on both sides indenting the skull one sixteenth of an inch with abrupt (sharp) edges.

All the veins of the pia and the sinuses were full of blood.

The right side of the brain, parietal and frontal lobes, felt unusually firm and tense and the left frontal lobe correspondingly soft and yielding. There were many adhesions between both anterior lobes and the roofs of the orbits and crista galli. There was also an inch-long firmly adherent band just above the posterior margin of the lesser sphenoid wing on the right side, beginning at the clinoid process. In the middle and posterior fossæ the brain was not adherent.

The pia slipped away from the brain without effort on the right side and the sulci opened wide on slight manipulation.

A little rough handling of the anterior lobes was unavoidable, because of the adhesions and for the further reason that the skull cap was taken off wedge-shaped, leaving rather a small space for extraction of the brain.

The general autopsy revealed little of interest save a congestion and edema of the lungs.

When the brain is turned base upward, an enormous tumor

is revealed involving both frontal lobes. It is dense and firm and has widely separated the frontal lobes. It appears to have involved both lobes to about an equal extent. It is but feebly adherent to the surrounding brain tissue.

The specimen having been hardened in formalin, it is found that the growth can be readily lifted out of its base, leaving an enormous cavity. The tumor has the shape of an irregularly flattened ball with numerous nodular masses of varying size upon its surface. In its longest or antero-posterior diameter it measures $2\frac{3}{4}$ inches, in its transverse diameter $2\frac{3}{8}$ inches and in thickness $1\frac{3}{4}$ inches. The frontal lobes have suffered extensively from compression and loss of the white substance, while the convolutions, especially the anterior and orbital portions of the first and second frontals upon either side have been much compressed and thinned. The most mesial portions of the orbital surfaces have been destroyed and with these the olfactory lobes; the latter, if present, can no longer be distinguished in the specimen. The mesial and orbital surfaces of the frontal lobes have suffered most, the white matter next, and the lateral surfaces (or convexity) of the lobes least of all. Indeed, the latter could only have been interfered with indirectly by the pressure. Further, it was clearly the orbital portions of both lobes that had suffered most.

The tumor upon microscopic examination proves to be a sarcoma. Though firm to the touch, it cuts readily upon section. The cut surface is whitish in appearance and solid looking. A small wedge-shaped piece was taken for microscopic study and prepared according to the usual methods.

Upon microscopic examination (made by Dr. Radasch) the capsule is seen to consist of loosely arranged fibrous tissue, in which can be seen quite a number of blood-vessels of various sizes; these seem to be normal in structure and appearance. Quite a few round and spindle cells are to be seen in the capsular tissue but the fibrils are of the adult form.

The mass of the tumor consists of spindle-shaped cells almost in its entirety; these form a very dense meshwork and run in various directions. Near the middle of the sections are scattered round and oval cells. Here and there in the sections are seen pearl-like formations that are probably peritheliomas and might represent obliterated blood-vessels. They measure about fifty to

sixty microns in diameter, and consist of cells that are flattened and arranged more or less concentrically. The nuclei stain deeply but the protoplasm does not respond well to the stain. These structures are more numerous near the capsule than centrally.

Stroma is practically absent; that which is noted is homogeneous and contains no fibrils. The vessels do not seem normal in the deeper portions of the tumor.

The pituitary body at first sight seemed to be enlarged, but this did not prove to be the case upon subsequent examination. Its histological structure also failed to reveal anything abnormal.

The above case is doubtless to be grouped among the inoperable tumors because of its enormous size and location. Perhaps if greater weight had been given to the symptom of anosmia a local exploratory operation would have been justified, and yet the size and location would probably have led to a fatal result had removal been attempted.

The mental symptoms, which in a way suggested paresis, were extremely interesting. Particularly was this true of the undoubted sense of well-being that manifested itself in the refusal of the patient to regard himself as ill. It was also true in the change in his habits and conduct. The tendency to joke and to take everything humorously was, of course, suggestive of a frontal growth.

Society Proceedings

NEW YORK NEUROLOGICAL SOCIETY

February 4, 1908

Dr. Charles L. Dana, the retiring president, in resigning the chair to Dr. B. Sachs, the president-elect, expressed his pleasure at the successful work of the society during the past year, and his gratification at the support which had been given him in his efforts to make the society a working organization.

Dr. B. Sachs, the president-elect, expressed his deep appreciation of the confidence his fellow-members had reposed in him by electing him a second time to the presidency of this society. After referring to the good work done by the society in the past, and urging the members to continue to give their best efforts in the future, the speaker said that if he read the signs of the times correctly we were passing in neurology and psychiatry from a period in which we were swayed by purely anatomical and physiological considerations, from an era in which localization and the clinical definition of disease played a most important rôle, to an era in which the etiology of disease and of disease-processes would claim our chief attention. We would be more interested in the questions of the bearing of heredity upon the development of disease; in the question of degenerative disorders, of bacterial infections, and in the aids given to diagnosis and treatment by a study of the blood, of the cerebro-spinal fluid, of the efficacy of organic extracts, etc. Changes in the ductless glands and their relations to various conditions in which neurologists were interested would also come up for special consideration, and there was much yet to be done in connection with the proper recognition and treatment of brain abscess, tumors at the base of the skull, and even in the matter of spinal surgery.

A CASE OF SUSPECTED HUNTINGTON'S CHOREA

By M. Allen Starr, M.D.

The patient was a negro, 26 years old, who had apparently enjoyed perfect health until his seventeenth year. His family history was unimportant and contained no facts bearing on the present case. The patient was born in eastern Maryland, and a fairly good knowledge of his ancestors was obtainable. About nine years ago his mother noticed that he was awkward in his movements. This was first noticed in the hands, and gradually extended to the arms, legs and body, and finally affected the muscles of the jaw and mouth. In connection with this loss of control, there were peculiar arhythmical movements of the muscles. There was a dropping of the hands, with a certain amount of contraction of the wrist, together with a shuffling, peculiar gait with high, irregular steps.

On account of the involvement of the muscles of the mouth and jaws he was unable to make himself understood, although his mother stated that she still could understand him at times. On attempting to speak, he made extraordinary grimaces. He apparently understood what was said to him. Accompanying his physical symptoms there had been a gradual mental impairment. When he was sixteen years old, according to his mother, he could read and write and was fairly bright at school; now he was stupid and took little interest in what was going on around him. There was no nystagmus; no ankle clonus; no Babinski; no sensory symptoms. The knee jerks were slightly exaggerated.

Dr. Starr said that on account of the intentional tremor he was first inclined to regard the case as one of progressive multiple sclerosis, but Dr. Richard H. Cunningham, who saw the patient at the Vanderbilt Clinic, diagnosed it as one of Huntington's chorea. Personally, Dr. Starr said, he had never seen an example of that affection, and he could find no record that it had ever been observed in a negro, nor was there a family record of the disease in this instance.

Dr. Richard H. Cunningham said that in 1893, while practicing in the South, he had under his care a family affected with this trouble. They were not negroes, however. The history obtained was that the paternal grandfather died of apoplexy. The father developed these peculiar choreic movements at about the age of thirty, although he lived until the age of 56. He was the father of six childern; two of these were apparently healthy; the other four, two of them girls and two boys, all suffered from this disease. In the eldest son it began when he was 18 years old, and when Dr. Cunningham saw him he was 25. In the next brother and in one sister it began at the age of 14, and in another sister at the age of 18. One paternal aunt had also developed this trouble when she was about 20 years old. They all presented these typical incoördinate movements, and in the eldest son the speech was involved as in the present patient. In the other children the speech was affected but not so noticeably. The progress of the disease was apparently extremely slow. The speaker said he had lost sight of the cases after they were under his observation about a year.

Dr. Edward D. Fisher said he had under his observation at present a family several members of which had suffered from Huntington's chorea. The father had died of the disease. One brother while suffering from it shot himself, and there were two other brothers who were now suffering from it. While the case shown by Dr. Starr certainly suggested Huntington's chorea, it was also somewhat suggestive of a double hemiplegia, with degeneration and athetosis. The reflexes in this case were a little more marked than those usually observed in Huntington's chorea. In the cases he had seen, the disease began later in life than in this one.

Dr. Pearce Bailey said that in a case of Huntington's chorea lasting as long as this one, the characteristic mental changes should be more pronounced. The speaker said that in one of the two cases of Huntington's chorea he had seen the mental condition became such that the patient had to be committed to an institution. The slow synergic movements in this case, however, were quite characteristic of Huntington's disease.

Dr. Charles L. Dana said that in a case of Huntington's chorea which had been under observation for a year or two at Bellevue Hospital the patient died and the brain was examined. In that instance, as in

those referred to by Dr. Fisher, the disease developed much later in life than in this case. The patient had the characteristic clonic movements and the peculiar gait, but there was no disturbance of speech. In the case shown by Dr. Starr there were certain symptoms which were not usually present in Huntington's chorea; there was a distinct tremor of the limbs and tonic movements of the muscles which suggested a spastic tic. On the whole, however, he thought it could be classed as a case of hereditary chorea of the Huntington type.

A CASE FOR DIAGNOSIS

By Edward D. Fisher, M.D.

The patient was a man, 45 years old, who for the past three years had noticed a gradual weakening of the right side of the body, especially involving the hand and arm, and later the leg on the corresponding side. Accompanying this loss of power there was an excessive intentional tremor affecting the muscles of the right hand and arm. The speaker said he was first inclined to regard the case as a slowly progressive lesion of the crus cerebri, but an examination of the left arm showed a distinct wasting of the muscles below the elbow, especially involving the intrinsic muscles of the hand, with considerable loss of power. This condition, according to the statement made by the patient, was of much longer standing than that on the right side. The patient denied syphilis. The pupils showed some irregularity; the reflexes were exaggerated; there was no change in the electrical responses; no disturbance of speech; no nystagmus. Dr. Fisher thought the case was possibly an irregular type of amyotrophic lateral sclerosis.

Dr. Starr called attention to the fact that the atrophy of the left arm as well as the contracture of the fingers pointed to a lesion of the ulnar nerve on that side.

Dr. Frederick Peterson said the case shown by Dr. Fisher reminded him somewhat of the condition known among the French and German writers as Benedikt's syndrome, which consists of a hemiplegia on one side, with clonic spasm or tremor and oculomotor paralysis of the opposite side. The condition had been described by Oppenheim and Charcot under the name of Benedikt's symptom-complex or syndrone.

NERVE BRIDGING, WITH REPORT OF A SUCCESSFUL CASE

By Alfred S. Taylor, M.D., and L. Pierce Clark, M.D.

The patient was a child upon whom the operation of nerve bridging for brachial birth palsy had been done two years and eight months ago. At the time of the operation the child was eleven months old and had complete paralysis of the left arm as the result of a rupture of the brachial plexus. Upon operation, it was found that the distal portion of the nerves could not be brought into apposition with the nerves as they emerged from the spinal foramen, and nerve bridging had to be resorted to over a space of 2 cm. Six months after the operation, a certain amount of motion was noticed in the triceps and the pectoralis major, and the fingers showed the redness that was characteristic of trophic changes. Eleven months after the operation there was consider-

able motion in the arm, and the trophic disturbances had nearly disappeared. At the expiration of two years and four months the child was able to raise the arm, and since October, 1907, the increase in the degree of motion in the muscles of the affected extremity had been very marked. The child could now bend the elbow, and had fairly satisfactory use of an arm which was at one time absolutely palsied.

Dr. J. Ramsay Hunt said that in one case of attempted nerve bridging that had come under his observation, in a child aged 20 months, the wound unfortunately became infected, suppurating for several weeks, and the arm was left in a worse condition than before the operation.

A NEW PLATE FOR DEFECT IN SKULL

By Charles A. Elsberg, M.D. (By invitation)

The patient was a boy of ten years who was admitted to Mt. Sinai Hospital on May 22, 1907, with the history that half an hour before admission he had fallen out of a second story window. He was brought to the hospital in a semi-conscious condition, with tonic and clonic convulsions of the right side of the face and of the right upper extremity. There was a large hematoma in the left parietal region. At the operation, which was done by Dr. Howard Lilienthal, there was found a depressed fracture, with considerable splintering of the fragments and an irregular fissured fracture running across the median line to the right parietal region. A large amount of bone had to be removed. There was an extensive extra-dural blood clot. Forty-eight hours after the operation there was complete paralysis of the right side of the body. In the belief that there was blood underneath the dura, the boy was again anesthetized, the wound reopened and the dura incised. A very profuse hemorrhage followed, which could only be controlled by tight packing with gauze. The patient recovered from this operation after a long and complicated convalescence, and was finally left with a large defect of the skull. There was a depression so large and deep that the greater part of the closed adult fist could be inserted into it. The deformity was a very ungainly one, and the parents of the child were anxious to have something done to remedy it.

On August 26, 1907, Dr. Elsberg made a large flap over the defect in the skull, turned down the skin and inserted an aluminum plate of his own design. The result was a very satisfactory one. The wound was entirely closed and healed by primary union. The boy was discharged cured on September 21. The splint employed in this case was made of aluminum, which could be cut into the shape desired without trouble. The plate had a number of arms which could be cut off at different lengths to fit into the irregular defect. The ends of the arms were split longitudinally for a short distance and one part of the arm then bent downwards. The entire plate was then bent to conform to the general shape of the skull. When in place, one part of the extremity of the arm rested on the outer surface of the skull along the edge of the defect; the other part rested against the cut edge of the skull along the margins of the defect. The plate was kept in position by a few catgut sutures which attached the horizontal part of the extremity of each arm to the periosteum. The advantages of this plate were the following: It formed a perfect arch, and the more pressure was

put upon the arch from the outside, the firmer and stronger it became. It remained in place and made a firm support, bridging over the defect of the skull. It was easily made and easily inserted, and could be bent into any shape desired. It had none of the faults of plates which rested on the outside of the skull and were apt to shift their position, or of plates put inside of the bone, which rested directly upon the dura or the brain.

ACUTE TOXIC CHOREA

By B. Sachs, M.D.

In this paper, the writer called attention to a grave form of chorea which he had had occasion to study of late years, more particularly in his service at Mt. Sinai Hospital, namely, toxic or infectious chorea. So long ago as 1872 Pianese claimed to have isolated a diplococcus and a diplo-bacillus from the cervical cord and the cerebellum of a patient who had died of St. Vitus' dance, and by inoculating cultures of these microörganisms into animals, he claimed to have produced chorea in them. The findings and experiments of Pianese had not been substantiated by later observers. In 1894 Dana wrote a paper on the "Microbic Origin of Chorea," and reported a fatal case in a man 34 years old. In that case diplococci were found in the proliferating tissue between the meninges and the brain. Other authors had found staphylococci and streptococci in the blood of the heart of choreic patients. Heubner was the only one who succeeded in developing streptococci and staphylococci from the blood of a choreic patient taken during life, and by inoculation produced a streptococcic septicemia in mice. The very best work of this description was done by Cramer and Toebben, who succeeded in proving the presence of Gram positive cocci in the blood taken from one of their patients during life, and these bacteria were unmistakably streptococci.

We were only now beginning to have positive evidence of the existence of an infectious form of chorea, but the ground upon which this doctrine rested was still meagre. The most important step to be taken in every case of chorea, at least from a diagnostic point of view, was to make repeated blood examinations so as to prove the presence or absence of bacteria in the blood of the patient during the earlier stages of the disease.

The pathological and bacteriological studies suggested the infectious or toxic origin of certain forms of chorea; the clinical symptoms pointed with much greater force to the existence of a special infectious type of choreic disturbance. The clinical features of such a condition might be briefly summarized as follows: The disease might occur at any period of life, although most of those hitherto observed have been youthful persons. One of Cramer and Toebben's patients was only seven years of age, the other thirteen. Dr. Sachs said that in the cases he had seen, the patients were fifteen, eighteen and twenty-two years old, respectively. After a somewhat indefinite prodromal period of restlessness and slight twitchings, universal choreic movements of an extravagant type set in, which constituted the most prominent symptom of the disease. The jactations were so severe that the patients had to be restrained or carefully watched in padded beds. Erosions appeared all over the body,

and particularly on the parts most apt to be rubbed or bruised by contact with the bed-clothes and the bed-posts. The erosions were found chiefly on the buttocks, the elbows and the ankles. Speech was difficult at an early stage of the disease, and later became impossible. This was due in part to the extreme choreic movements of the lips and tongue, and of the muscles engaged in the mechanism of speech, as well as to the accompanying mental change which might vary from mild stupor and apathy to a condition of active delirium. The movements were lessened during sleep, but sleep was fitful and as a rule could be induced only by large doses of hypnotics and sedatives. After a period varying from one to two weeks the patient passed from the state of continued restlessness into stupor and coma. After a further period of a few weeks or possibly a week, death supervened, although the speaker said he did not claim that a fatal termination was inevitable. High fever was observed from the beginning to the end of the disease. A universal erythema, bearing all the marks of a toxic eruption, occurred in the earlier stage.

Dr. Sachs then reported in detail two cases of acute toxic chorea that had come under his observation, with post-mortem and pathological findings. Reviewing his experience with these and other cases in an impartial spirit, he said the conclusion was inevitable that the grave form of chorea he had described was due to a general toxemia caused by the invasion of staphylococci or streptococci, or of some other organisms not yet determined. The search for a single specific organism as the cause of chorea seemed to him fruitless, and, in view of what had already been discovered, entirely illogical. Some forms of chorea were evidently the expression of an acute general toxemia. There might be a number of different microbic agents giving rise to this condition, but it seemed reasonable to suppose that certain bacteria had a special predilection for the motor neurones, while other exerted their baneful influence over the sensory elements of the central and peripheral nervous system.

Dr. Charles L. Dana asked Dr. Sachs how he differentiated between this form of chorea and that known as chorea insaniens. He generally observed several of the latter type of cases in the hospital each year, and while they were usually looked upon as of infectious origin, that fact had not been demonstrated as perfectly as in the cases reported by Dr. Sachs.

Dr. M. Allen Starr said that within the past two years, in the Presbyterian Hospital, he had seen two cases of ulcerative endocarditis in which, among other symptoms, there were marked choreic movements of the face and extremities. The endocarditis was shown to be of definite bacterial origin, and the cases were a good illustration of the fact that any acute bacterial infection may in certain individuals produce choreic movements. That view might be taken in connection with the cases reported by Dr. Sachs. In both there was a general streptococcus infection of which the chorea was apparently the chief manifestation. It was questionable, therefore, whether we were really dealing with a new toxic disease or merely a manifestation or prominent symptom observed in certain infections.

Dr. Nathan E. Brill asked Dr. Sachs why he used the term "toxic chorea" in reference to the disease. As yet it has not been demonstrated that a toxemia is the cause of chorea. Nor yet could it be proven that in the class of cases presented by Dr. Sachs the toxines rather than the

mechanical effects of the bacteria in the cerebral capillaries were the causative factors.

It would seem to be more in harmony with the fact that in one of Dr. Sachs' cases there was a distinct staphylococcemia to call this class of cases "chorea associated with septicemia" or "bacteriemic chorea" rather than toxic chorea. Pathologists have called the invasion of the circulatory system by bacteria septicemia. The tendency is now to more definitely limit the term and call such a condition "bacteriemia." If this condition be associated with chorea, either etiologically or not, we have the condition which Dr. Sachs wishes to emphasize. But in this respect this form of chorea would differ but little, perhaps mainly in its clinical picture, from the chorea associated with what is called "Acute Articular Rheumatism." It is now generally conceded that acute rheumatism is a generic term covering many conditions associated with acute inflammatory joint changes; that the most of these are regarded as the result of an acute infectious process, some with known, as the tonsils, others with unknown portals of entrance. It is also a fact that endocarditis and chorea are accompaniments of this class of diseases, and likewise that in some forms a bacteriemia has been established. In some cases a diplococcus tenius and in others a streptococcus tenius have been recovered from the blood. The fact that in acute articular rheumatism, in which chorea and endocarditis may play a part, no bacteria have been isolated from the blood ought not to be considered to prove that no bacteria are present in the circulatory medium, but rather, reasoning by analogy, that thus far the cultural means at our command have been inadequate or insufficient to recover the offending organism. In the light of this view Dr. Sachs' cases might differ but little pathologically from the class of chorea associated with acute rheumatism—the clinical form, perhaps, being modified by the varying type and virulence of the invading organism.

We may say, however, that Dr. Sachs has established a fact—namely, that certain cases of chorea are associated with distinct bacteriemia. Dr. Brill does not understand that Dr. Sachs desires to establish a new form of chorea.

Dr. Sachs said he had not the slightest intention of trying to establish a new disease. His only idea was to place on record these very unusual cases, which he recognized as a special form of chorea associated with an intense general infection of which the chorea was the most prominent symptom. In some of these cases the infective agent was the staphylococcus, in others the streptococcus, or some other agent, and one object in keeping them apart was that in a therapeutic way it would help us to recognize them as very serious infections.

In reply to Dr. Dana's question as to the connection between this form of chorea and chorea insaniens, Dr. Sachs said that in recent years he had dropped the term chorea insaniens, and he did not consider it a very useful one from either a clinical or pathological standpoint. The term was applied usually to the more chronic forms of chorea, associated with mental deterioration, rather than to cases of acute origin associated with high fever.

Dr. Dana said he had seen cases of chorea insaniens which corresponded very closely to those reported by Dr. Sachs. A few of them had recovered. He could recall no good description of this type of chorea in literature.

Dr. Smith Ely Jelliffe said that some of Dr. Sachs' cases were typical cases of chorea insaniens, and that moreover there was an exceedingly rich and full literature from the appearance of Krafft-Ebing's studies on chorea insaniens in 1894 to the present time. The relations of choreic disturbance to post-microbic infections, in many instances, were established.

Dr. Sachs, in reply to Dr. Brill, said that in calling these cases toxic chorea he had not been guided entirely by his own view of the etiological factors concerned in their production. The symptoms were supposed to be due to a toxin, but whether always and distinctly bacteriemic or not he could not definitely decide.

At the annual meeting of the society in January the following officers were elected for the ensuing year: *President,* Dr. B. Sachs; *First Vice-President,* Dr. J. Ramsay Hunt; *Second Vice-President,* Dr. Smith Ely Jelliffe; *Corresponding Secretary,* Dr. Max Mailhouse; *Recording Secretary,* Dr. E. G. Zabriskie.

PHILADELPHIA NEUROLOGICAL SOCIETY

January 27, 1908

The President, Dr. Alfred Gordon in the Chair.

Dr. Weiss presented a patient from Dr. Spiller's service at the Philadelphia General Hospital with lateral movement of the foot in ankle clonus.

FRAENKEL TREATMENT OF TABES

By M. D. Bloomfield, M.D.

This paper brought out the points that the treatment should only be carried out by a physician who has had a good hospital training, who is thoroughly familiar with the disease and the status præsens of his case. The treatment can only be carried out successfully by one who has had a practical training from an experienced man, as the details of the treatment can only be acquired by practical assistantship. Dr. Bloomfield spoke of the loss of the sense of muscular fatigue in tabetics, a symptom not mentioned in text-books, and of the accidents (cardio-vascular hypotonias, spontaneous fractures, etc.) which could only be prevented or treated by a medical man; he also showed a man who was extremely ataxic when he first came under treatment last May, but who could now perform almost any movement.

Dr. T. A. Williams stated that the loss of sense of muscular fatigue in tabes is described by the French writers, of whom perhaps the most conspicuous one to record this fact is Dejerine. In regard to the mechanical treatment in connection with the education, Dr. Williams said that Faure of La Malon, an institution in the north of France, has worked a great deal recently at it and has written two papers on the subject, one of which was read before the French Congress of Neurologists in 1906, and the other was read at the Congress at Amsterdam last year, and a third paper was read at the Congress of Geneva last August, in which he advocates the supplementing of the education movements by mechanical supports in

the nature of elastic bands to assist in overcoming the hypotonia. He claims that this renders the education easier and adds greatly to the patient's comfort; in fact, he states that in some cases the only bar to locomotion is the hypotonia. These patients are not in reality so ataxic as they are hypotonic. In that connection, Dr. Williams mentioned another case described by the physician of another institution, on the border of Switzerland, in which he showed the remarkable results obtained by spontaneous efforts at the education on the part of the patient, that is to say, the patient on being encouraged to get out of bed makes a few faltering steps to the next bed and so educates himself. Dr. Williams, however, believes that it is a very important matter to insist upon the supervision of these exercises by a medical man. He thinks it is too commonly left to the attendants, who are not always educated in the necessary precautions to carry out the exercises and use too much vigor possibly.

ACQUIRED SPASTICITY AND ATHETOSIS

By William G. Spiller, M.D.

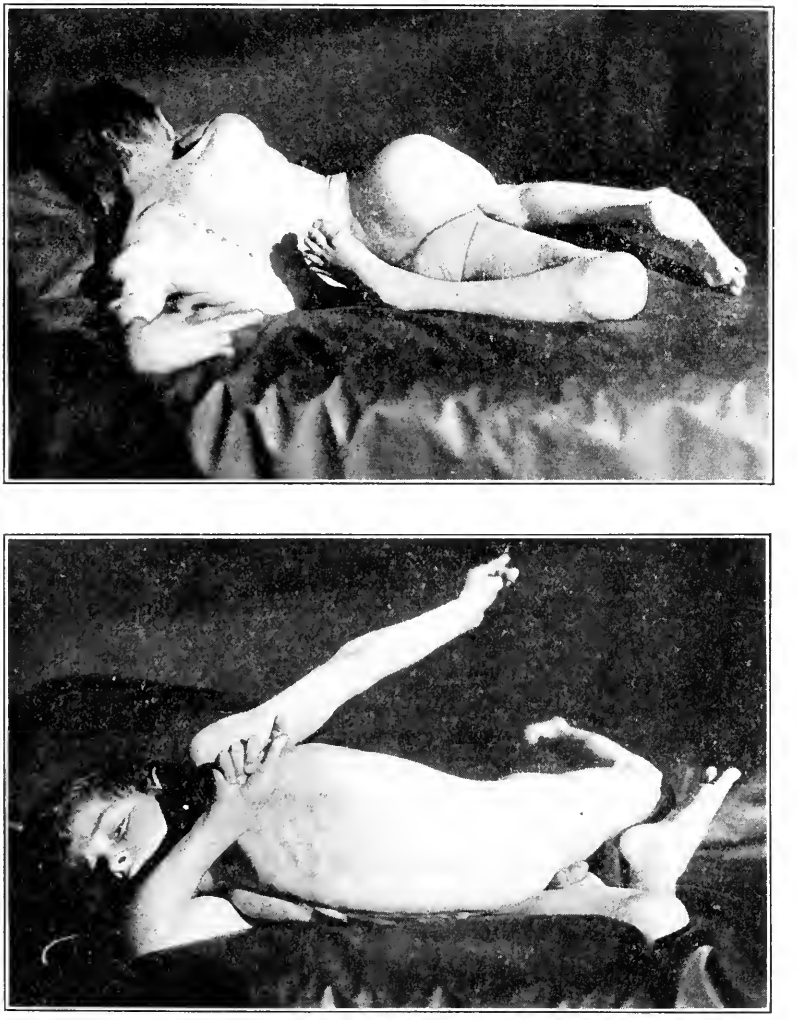
Haupt[1] remarks that idiopathic or primary athetosis is relatively rare. Lewandowsky distinguishes between acquired athetosis and similar forms developing after hemiplegia. The former is not merely a post-hemiplegic athetosis affecting both sides, or a result of infantile spastic diplegia, but is an independent peculiar disease, whose pathology is not definitely known, but probably consists of bilateral cerebral lesions. Oppenheim also makes the distinction. Previous diplegic disturbances should be excluded if the term primary double idiopathic athetosis is employed. In a case reported by Haupt small cortical foci were found in the left parietal lobe, and seemed to indicate a previous encephalitis. A few cases with necropsy in the literature are referred to by him.

Dr. Spiller presented a patient, a boy twelve years old, in whom during the past five years he had observed gradually developing spasticity of all the limbs with athetosis, reaching finally such an intensity that the patient was confined to his chair. The patient first came under Dr. Spiller's observation December 15, 1902. At that time he was seven years old. He has been under Dr. Spiller's care at intervals since 1902. The following history was obtained in 1902. He was the first born child. The birth was easy and normal. He was said to have had convulsions when four months old. The father stated that the boy walked, ran and jumped as other children until four months previously, but since that time had gradually been getting lame in the left lower limb, and had been obliged to wear a brace during the previous four weeks. He had not had any pain but occasionally had some tremor of the upper and lower limbs.

An examination showed that the boy was unable to stand without supporting himself by bending back the knee. When he attempted to walk the feet were wide apart, the knees were close together and the lower limbs became spastic. There was no spasticity of the limbs when the boy was at rest. The lower limbs were somewhat weak when he was walking, but very little if at all when he was sitting. The grip was good in each hand, and the voluntary power of both upper limbs was good. The patellar reflexes were prompt but there was no clonus. The plantar and Achilles reflexes were normal. When lying down the lower limbs showed no spasticity on voluntary movement. Sensations to touch and pain were normal. Each thigh could be moved passively freely. What weakness was present

[1] *Deutsche Zeitschrift für Nervenheilkunde*, Vol. 33, Nos. 5 and 6, p. 464.

seemed to be in the extensor muscles of the back and hip. Electrical reactions in the lower limbs were normal, but in August, 1903, a quantitative decrease was observed. Scoliosis was present when the boy was standing. The calf muscles were not enlarged.



Photographs made by Dr. A. R. Allen in 1908, with very rapid exposure, showing the extreme spasticity of the limbs.

At that time the diagnosis was very difficult; by some the condition was regarded as muscular dystrophy, a diagnosis which Dr. Spiller never accepted, and the variability in the gait from time to time suggested a hysterical element.

The notes of an examination made by Dr. Spiller January 13, 1908, are as follows: The lower limbs are very spastic but at times this spasm yields, so that the limbs can be moved at most of the joints quite freely, though not to the full extent. The right lower limb is usually kept extended, with the foot in equino-varus position. The varus position can be overcome, but the contraction of the Achilles tendon is so great that the foot cannot be flexed at a right angle with the leg. The big toe is hyperextended. The left lower limb is partially contractured in flexion at the knee, and the left foot is extended to the full degree with slight tendency to varus position. The varus deformity is not so intense as in the right foot. The contracture of the Achilles tendon here also is so great that the foot cannot be flexed at a right angle with the leg. The lower limbs are not distinctly wasted, but are poorly developed on account of disuse. The boy usually lies with the left leg flexed on the left thigh, lying upon the leg. When he is entirely at rest voluntary jerkings occur only occasionally. But any passive or voluntary movement causes involuntary jerkings of portions of the upper and lower limbs resembling athetosis, seen especially in the lower limbs in the right big toe which is slowly and repeatedly hyper-extended, very much as in athetosis. The spasms of the lower limbs are increased by passive movements. The patellar tendon reflex is exaggerated on the right side and probably also on the left. The spasm of the muscles prevents the movements of the legs. The Achilles tendon reflexes are probably exaggerated, though the full degree of the exaggeration cannot be determined. The Babinski sign is very distinct on each side, more so on the right. Ankle clonus is impossible because of contracture of the calf muscles. He moves the right lower limb in toes, knee and hip, but with much diminished power, and he has no movement at either ankle. The movements of the left lower limb are preserved in the toes and at the knee. The movements of the knee consist of slight flexion and extension. He has no voluntary movement of the left hip.

Touch, pain and temperature (heat and cold) sensations are normal in all parts of the body. The trunk is greatly deformed. When the shoulders are placed fairly on the bed the body is so distorted that the boy lies on the right hip with the left hip elevated. The scoliosis is extreme with the concavity towards the left in the lumbar region.

The right upper limb is moved at all parts and at all joints, but with much diminished power. There is spasticity of the upper limbs and all movements are exceedingly incoördinate with increased spasm on movement, resembling athetosis. There is no contracture in either limb. The limbs are not muscular but are not atrophied. The biceps and triceps tendon reflexes cannot be determined on either side because of the spasticity. The left upper limb is moved at all the joints, but with much diminished power, and is weaker than the right upper limb. Incoördination on voluntary movement is extreme, and the athetoid movement of the left hand is very pronounced on any voluntary movement, and occasionally when the patient is at rest. The left upper and lower limbs are more involved than are the right limbs.

The pupils are equal and respond promptly to light and convergence. The extra ocular muscles are normal. The tongue is normal. Facial nerve supply is normal on each side. There seems to be no positive involvement of the cranial nerves.

The boy is very intelligent.

Dr. Spiller expressed the opinion that the condition was probably the result of progressive involvement of the pyramidal tracts.

Dr. Williams regarded the case as so remarkable that he had very little to say about it, except that it is very extraordinary to note the exceedingly gradual progress of the symptoms and that they began in and were confined to the lower extremities for so long. He asked whether Dr. Spiller had remarked previously in this patient a projection of the jaw and a fixation of the forehead in the position of elevation of the eyebrows from time to time. The face gives one the impression occasionally of stiffness, being held in a rigid position. If not, whether Dr. Spiller did not think it seemed to indicate an extending of the process to that portion of the pyramidal tract which concerns the nuclei of the face? Otherwise it would seem that the process is so slow and so remarkably free from lesions of any other system than the pyamidal that it would appear to be a dystrophy of the pyramidal system beginning in the lower extremities. It would be interesting to see the case in the future. It reminded Dr. Williams a little of a very remarkable case he saw in the Salpêtrière in a boy of eleven, who acquired spastic symptoms, not so slowly as this, but in the course of a few months, which were pseudobulbar in type, but in whom the symptoms became entirely arrested, indeed improved very markedly. He did not think the cases resembled each other pathologically at all, excepting that there should be a progressive disease of that type. The case was regarded as being probably lacunar in the boy of eleven.

SOFTENING OF THE DENTATE NUCLEI CAUSING SYMPTOMS OF CEREBELLAR TUMOR

By William G. Spiller, M.D.

The patient, a male aged eighteen years, was seen by Dr. Spiller about April 23, 1907, in consultation with Dr. M. H. Bochrock, from whom the following history was obtained. The boy had been in fairly good health until about one year previously, at which time severe headache began. The pain was felt in the entire head and most severely in the occipital region. He had some ataxia in walking and would fall, especially to the right. He was very deaf, had vomited during several months, had much vertigo and divergent strabismus. Venereal disease was denied.

An examination of the eyes by Dr. James A. Kearney, April 4, 1907, gave the following results: "Media clear, the disc protrudes from the posterior wall of the eye very similar to the apex of a thimble (ampulliform). The vessels of the apex of the disc are engorged, especially the veins, and about four millimeters of their length is plainly seen. The vessels are then enveloped in the disc tissue and emerge at the base where they are of normal character. The difference in refraction between the apex and the base of the disc is two diopters. The refraction of the fundus is sphere plus five diopters. The character of the retina is normal but slightly irritable. The above examination is of both eyes."

The patient's condition at the examination by Dr. Spiller was as follows: when sitting in a chair the head was thrown far backwards, the neck muscles were stiff, the seventh, twelfth and fifth nerves were not implicated, deafness was intense and bilateral, stupor was pronounced, the iridic reflex to light was very feeble if present at all in either eye, the eyeballs

were moved in all directions, but it was impossible to get the patient to make extreme movements of the eyeballs in any directions, nystagmus was not observed, ataxia was present in each upper limb, sensation to pin prick was preserved all over the body, the limbs were not weak, the patellar reflexes were lost even on reinforcement, the Achilles jerk was feeble on each side, the boy was unable to stand alone and would fall backwards if not supported, hemiasynergia and diadococinesia could not be tested for because of the stupor, the corneal reflex was present, the Babinski reflex was very uncertain.

The diagnosis of a lesion of the cerebellum was made and as the symptoms indicated that the progress was gradual in development, a tumor was supposed to be present.

Decompression was performed by Dr. Nassau and was followed rapidly by death on April 25, 1907.

Only the cerebellum and a portion of the pons were obtained for examination. A cavity was found in the interior of the right dentate nucleus, and the left dentate nucleus did not appear to be normal. The small vessels of both cerebellar lobes near and in the dentate nuclei were much congested, and numerous small hemorrhages were found about them with some perivascular round cell infiltration. The vessels of the pons were congested and here also a few small hemorrhages and slight perivascular round cell infiltration were found. As the necropsy was necessarily so incomplete it was impossible to say whether any other intracranial lesion was present or not.

This case in its findings resembles the case reported by E. F. Buzzard in *Brain*, Vol. 29, p. 508, in which thrombosis affecting, and probably destroying the functions of, the dentate nuclei was found.

THE SYMPTOM COMPLEX OF TRANSVERSE LESION OF THE SPINAL CORD AND ITS RELATION TO STRUCTURAL CHANGES THEREIN

By Alfred Reginald Allen, M.D.

This paper is founded upon the study of the spinal cord of a woman who died from carcinoma of the vertebra secondary to a primary breast lesion. There were symptoms of transverse lesion of the spinal cord almost two months before death, yet the microscopical study of the spinal cord demonstrated practically no secondary degeneration. Some of the prevailing theories as to parenchymatous regeneration in nerve tissue are mentioned and discussed, and the bearing of the absence of marked histological change in the case cited upon laminectomy to fracture-dislocation of the spinal column with cord symptoms is considered.

ASCENDING PARALYSIS

By Alfred Gordon, M.D.

A middle-aged man noticed about two years ago a gradually developing paralysis of the left leg accompanied by pain in the perineal group of muscles. Eighteen months later the same conditions appeared in the left upper extremity. At present he shows a gait resembling largely a hemiplegic one, but there is no rigidity in the limbs. There is considerable

loss of power in the affected limbs. The knee jerks are increased on both sides. There is a faint Babinski and a slight paradoxical sign on the left. The interesting feature about the case is the presence of pain at the beginning of the paralysis.

CHICAGO NEUROLOGICAL SOCIETY

February 20, 1908

Dr. Hugh T. Patrick in the Chair

SYRINGOMYELIA WITH KYPHO-SCOLIOSIS AND UNILATERAL TRUNK ANESTHESIA

By E. W. Ryerson, M.D.

The patient is a boy sixteen years old, a patient in St. Elizabeth's Hospital. Family history of no interest. Delivery was by forceps and very difficult. The right shoulder was dislocated or fractured, and the right arm has been paretic since birth. There also was some injury to the head causing unilateral peripheral facial paralysis which lasted a few weeks. He walked when one year old, and shortly afterwards had scarlet fever followed by bilateral suppurative middle ear disease. The latter has recurred from time to time. His health was fairly good until January, 1905, when he was in bed for nine weeks with a right-sided pneumonia, possibly complicated by pleurisy. About a year and a half ago gradual onset of left kypho-scoliosis with convexity to the left. Little or no pain in the back and none in the legs. A brace was put on a year ago and worn for half a year. Then gradual onset of weakness and stiffness in left leg and arm. No sphincter disturbance at any time. No fever.

Examination shows marked diffuse kypho-scoliosis with convexity to the left. Skiagraph negative aside from deformity. No tender point in the spine. Right arm paretic with loss of tendon reflexes (old birth palsy). Left arm slightly paretic, rigid, with increased reflexes. Left leg also spastic, with exaggerated knee jerk, ankle clonus, and extensor response of big toe. Right knee and angle jerks also exaggerated, with clonus; flexor response of big toe. Abdominal and cremaster reflexes present on right side, absent on left side. Band of anesthesia around left side of the trunk from third to eleventh thoracic segments, with involvement of tactile, pain and temperature senses. Slightly impaired sensation in places on right arm, but no sensory disturbance elsewhere outside of the trunk area mentioned. No ataxia. No cranial nerve or ocular findings; fundi normal. No muscular atrophy.

Tuberculosis, syphilis or neoplasm of the spine or meninges were excluded largely on account of the painless course, and by exclusion syringomyelia was considered the most plausible diagnosis in spite of the absence of sensory dissociation, muscular atrophy and sensory disturbance below the level of the lesion.

TWO CASES OF JUVENILE DEMENTIA PARALYTICA

By Julius Grinker, M.D.

CASE I.—J. C., aged 20, was seen in the Neurological Clinic of Northwestern University Medical School, service of Dr. H. T. Patrick, on February 3, 1908. His mother stated that her son was nervous, that his memory was somewhat impaired and that his speech was indistinct and "trembling."

Patient's mother is now 51 years old; she was married at 19, has never been sick and comes from a healthy family; her mother is well at 80, father died at 86, three brothers and one sister are well. The patient's father is 54, has always had good health; his father reached old age, but the mother died young. The first two pregnancies resulted in abortions of two and three months respectively; nineteen months after her marriage a son was born who is now 30 years old and in good health; then came another son who is healthy; and two years later, a daughter, who is 25 years old and perfectly well. Henceforth the offspring showed little vitality. A daughter died at four weeks, because of difficulty in nursing; the next child, a boy, lived two hours; then our patient arrived on the scene.

He was born in normal labor and seemed well up to the age of two months. And now he developed an eruption, his lips and the corners of his mouth became ulcerated, his nose was inflamed, he had difficulty in breathing and nursing, was extremely restless and cried most of the time. At this early age (two months) he suffered from an eye disease which almost caused blindness, but which was cured in one week. There were no spasms, nor headaches. The boy was under a physician's care for six months when he completely recovered and remained well until the beginning of his present trouble. At nine months he walked, at eighteen he talked and was considered bright. While at school he was a fairly good scholar. With the exception of a suppurating wound in the lower abdomen, caused by a fall, he never suffered injuries, nor serious disease of any kind. His habits are said to have been good, though he was a moderate smoker and drinker.

About one and a half years ago his character gradually underwent a change. He became irritable, peevish and would occasionally leave the house for a few days without any assignable cause. His parents ascribed these irregularities to bad temper, until they discovered that his physical health also began to deteriorate. Their next thought was that he might be addicted to the cocaine habit, which suspicion remained unproven. A little less than a year ago his moodiness changed into euphoria; he was mostly happy, laughed over trifles, developed an enormous appetite and thirst and altogether in contrast to his former self he became very tractable.

While riding in a street car last winter he experienced twitchings in his face accompanied by loss of speech lasting for about an hour. This was followed by occasional attacks of transient loss of power in arm and leg, always, however, eventuating in complete recovery. Last winter while dancing—being a vaudeville dancer by profession—he became dizzy and fell off the stage. Since then his speech had become very indistinct and has continued to grow more so. For the past week on alternate days he has been mute, inactive, disinclined to eat, but obeyed

commands when repeatedly urged. On other days he is talkative, reads the newspapers, interrogates his parents regarding certain information asked of him at the clinic.

In appearance is rather boyish for his age, looks somewhat frightened and indulges in a peculiar stare, expressive of uncertainty. Upon attempting to speak a flickering of the lower facial musculature is produced. The pupils are large, slightly irregular, do not respond to light and but little to accommodation. Immediately upon protrusion of the tongue a coarse irregular to and fro tremor develops in it which it shares with the oral ring. The speech demonstrates a typical example of paretic stumbling, trembling and slurring utterance. He is oriented as regards time, place and persons. There are no delusions of grandeur and there is a fair degree of insight into his condition. Mathematical problems requiring but small feats of memory are beyond his reach. He is himself conscious of memory defects. An examination of the ocular fundus reveals normal findings bilaterally. There are white lines radiating from each corner of the mouth, indicating cicatrices of former lues. The teeth are not of the Hutchinson variety, but the cutting edges of the upper incisors are rough and worn away.

The reflexes are brisk in the upper extremities, the knee jerks are about normal, but the Achilles responses are absent. There are no sensory disturbances. There is a slight Romberg and very little incoördination in the upper extremities. The viscera are normal.

Case 2.—C. F., aged 23, was admitted to Cook County Hospital on February 7, 1908, in the service of Dr. Bassoe. His father is living and well, his mother died of pleurisy at 38; there are three living brothers and one sister; several died in infancy. As far as can be ascertained, he was well until the age of 9 when he developed suppurating glands on the right side of the neck, for which an operation was performed, leaving a large scar. He was a moderate drinker until five years ago when he contracted gonorrhea and chancroid. Careful probing for the existence of secondaries yields negative findings. The patient is married. There were two pregnancies: the first one resulting in abortion, the second in a healthy child, now 18 months old.

About three months ago he had what appears to have been an attack of grip of moderate severity, but which left him somewhat nervous and caused him to enter the hospital. He states that his attack of grip is responsible for his weakness and nervousness. He complains of occasional dizziness and he is certain that for some time his memory has not been good.

Examination reveals a fairly well-nourished young man with a rather insipid, asymmetrical face. Around the angles of the mouth several linear scars can be seen radiating in all directions. When he speaks, a slight tremor can be seen playing around the lips, which becomes more pronounced when he gets angry. The pupils are irregular and do not respond to light and but slightly to accommodation. The fundi are normal. The eye muscles functionate correctly and no nystagmus is seen. Abdominal and cremasteric reflexes are normal; likewise the deep reflexes in the upper extremities. The knee kicks and Achilles jerks are equally exaggerated; ankle clonus and Babinski's phenomenon cannot be elicited.

Sensory disorders are absent. Coördination is normal in the upper extremities, but there is some ataxia of station and gait in the lower extremities. There is slight tremor in hands and fingers. The protruded

tongue shows a coarse tremor which extends to the lips. The speech is hesitating and in a lengthy conversation becomes jerky in character. The usual paradigmata can be repeated, but there is noticeable an omission of syllables and inability to pronounce the "r's."

His arithmetic is exceedingly faulty: Examples: $9 \times 9 = 72$, $100 - 21 = 78$, etc. Memory tests show defects which are not very pronounced. There is complete orientation as to time, place and persons.

GLIOMA OF THE BRAIN

By Julius Grinker, M.D.

The patient 52 years old, a financial secretary of a union, was always capable of fulfilling the official duties which required a good memory; the members would pay him dues, he would give them a receipt for same, and then had to enter their names on the books. Often he would accept money on the street or wherever he happened to be and yet he seldom made a mistake. In other words, until about two months prior to his death, he was mentally sound. He attended his usual work. About two months prior to his last illness he sustained a scalp wound over the left occipital bone, caused by the fall of a brick. The wound suppurated, had to be dressed quite frequently, but at no time could a fracture be ascertained.

The patient was able to continue at work after a few days' absence from his accustomed activity. His wife is certain that ever since the accident he made mistakes in his official work, that he frequently failed to recognize old friends and was often rather confused and dull mentally. About a week before he came under observation, he had a stroke which caused a right-sided weakness and confusion of speech: he used the wrong words and appeared not to comprehend spoken or written speech. It appears that he improved rapidly for a few days and then became worse. There was a positive Babinski on the right side. Because of the conjugate deviation and the rather sudden onset, slow and full pulse and the age, the diagnosis of cerebral hemorrhage in the capsular region was made. The following day a more complete history was obtained. The patient had a slight rise of temperature (101°), and a polynuclear leucocytosis, and operation was advised in order to search for a possible deep-seated abscess. The operation revealed no abscess, and the patient died twenty-four hours later. Postmortem examination revealed a glioma situated deeply in the occipital and parietal lobes of the left side. An old hemorrhage cyst was seen, extending from its superior surface and continuous with it, also from its inferior border which broke into the lateral ventricle and thereby exerted pressure upon an old hemorrhage, and a rather recent hemorrhage of the left capsular area.

This case, like similar reported cases demonstrates several points: (1) a glioma may exist without causing symptoms; (2) cerebral hemorrhage may be the first symptom of glioma; (3) trauma of an insignificant kind may initiate serious disturbances in a quiescent brain tumor, eventuating in death; (4) mental disturbance, such as slight confusion and loss of memory may be the only symptom of deep-seated glioma; (5) glioma in which hemorrhage has occurred may simulate brain abscess; (6) diagnosis of existing brain tumor could not be made before the hemorrhage occurred because of the absence of general and local symptoms of brain tumor.

The microscopical examination reveals a typical glioma with multiple hemorrhages. In areas the cells are of medium size, closely set and show but little protoplasm. In other places the glia fibers seem to predominate. The most recent hemorrhage has extended into the left lateral and third ventricles and caused symptoms by pressure from within outward.

In conclusion this case corroborates the oft-noted observation that psychic disturbances may occur in brain tumor irrespective of its location. It will be seen that the entire prefrontal lobe is free from disease, and yet several weeks before the end there were decided psychic symptoms.

PATHOLOGY OF PSEUDOHYPERTROPHIC MUSCULAR DYSTROPHY.

By O. P. Johnstone, M.D.

The case is that of a boy between 15 and 20 years of age. Two other children in the family had previously died with typical symptoms of pseudohypertrophic muscular dystrophy. The clinical history of this case unfortunately is not available at the present time.

The splenius colli, erector spinæ, gastrocnemius and soleus muscles, and the 1st cervical, 5th cervical, 7th dorsal, and 3d lumbar segments of the cord have been examined. The erector spinæ showed the most marked changes of any of the muscles examined. Hardly a single recognizable muscle fiber remains in the sections studied, except those in the muscle spindles. A very few degenerated fibers are found, which, however, could not be recognized as muscle tissue, if the source of the section had not been known. The section is made up of a mass of fatty and connective tissue, the latter not very great in amount. Grossly the tissue appears to be a mass of adipose tissue with a few pinkish points here and there. Cellular infiltration is not much in evidence. Muscle spindles are numerous, several being found in practically all sections examined. A few muscle fibers within them are normal and show cross striations well. Fully as many have lost their cross striations, show longitudinal segementation, central nuclei, and moderate atrophic and hypertrophic changes. The atrophic and hypertrophic changes are not marked, however, the smallest fiber found measuring 10 micra in diameter, the largest 65 micra. Their cross section is round or oval. The nerves within the spindles show no apparent changes, with the hematoxylin and eosin stains.

The splenius colli was next in the extent of involvement, from two-thirds to three-fourths of the fibers having disappeared in some sections. In other sections nearly all the fibers have disappeared. The lost fibers have been replaced by fat, or the empty sarcolemma remains. Here most of the fibers remaining show good cross striation, but stain somewhat unevenly. Those which have lost the cross striation and show longitudinal segmentation are for the most part hypertrophied fibers. There is a most striking difference in the diameter of the individual fibers in this section, some measuring as little as 4 micra, while others lying close by measure from 100 to 200 micra in diameter. Most fibers measuring more than 100 to 125 micra show loss of cross striation, with granular degeneration, and longitudinal segmentation. These degenerative changes are found in but few fibers of normal or less than normal size. Many fibers show complete disappearance of the muscle substance, only the

empty sarcolemma remaining. Some of these show many nuclei within the sheath; more however show only a moderate increase or no increase in the nuclei. Occasionally centrally placed nuclei or chains of 15 to 30 such nuclei are seen in the fibers. Cross section shows the fibers round or oval, widely separated, and occasional central nuclei. Some of the cross sections of the fibers show splitting into two or more parts, others show the fibrillæ very plainly. Longitudinally different parts of the same fiber show marked difference in diameter, and in the clearness of the cross striation. Very many of the fibers appear distorted and kinked.

The connective tissue between the fibers is only moderately increased in amount; in some places the connective tissue appears to be young and richly cellular, in other places mature and with the usual number of nuclei. A few sharply outlined small masses of apparently lymphoid cells are seen. A few polynuclear leucocytes are present in these masses. There is no evidence of inflammatory reaction about these areas.

The fat is very great in amount and for the most part is in the connective tissue between the muscle fibers. Some small droplets are seen within the muscle fibers, but the amount is strikingly limited.

The walls of the blood vessels appear decidedly thickened. No changes can be detected in the nerves with the hematoxylin-eosin stain. No muscle spindles are found in the sections examined.

The gastrocnemius and soleus muscles show the least involvement, about one half to two thirds of the fibers having disappeared. The changes are very similar to the changes in the splenius colli, only less marked. In these sections, especially in the muscle bundles least involved there is a striking tendency for the fibers to disappear and be replaced by fat in the center of the muscle bundle, the periphery being comparatively normal as far as the disappearance of the fibers and replacement by fat is concerned. Apparently the fibers in the center of the muscle bundles are the first ones attacked by the degenerative process.

Sections of the cord at the first and fifth cervical, the seventh dorsal and third lumbar segments, show no changes in either gray or white matter when stained by the hematoxylin eosin and Weigert-Pal stains. Nissl's stain could not be used as the tissue had not been suitably fixed.

DEMONSTRATION OF A BRAIN SHOWING TUBERCLE IN PONS WHICH CAUSED HEMIPLEGIA ALTERNANS

By Sydney Kuh, M.D.

The patient was a boy two years old when first examined. He was first seen on October 25, 1907. His present trouble began in January of that year with a weakness of the left arm which increased very gradually, until that member had become entirely useless. His family history was negative. He had been born at full term, after a normal labor, and was breast fed. He never learned to walk, but began to talk when about a year old, although he never learned to say more than a few simple words. About April, 1907, the child was taken to a hospital for his paralysis, and while there he is said to have had an attack of both scarlatina and measles. There is no history of vomiting or spasms and nothing suggestive of dimming of vision has been noticed. The course of the disease has been afebrile.

The examination showed a well-developed child, whose intelligence appeared to be normal. His anterior fontanelle had not yet closed entirely, the biparietal diameter of the skull was wide, the lower portion of the sternum sunken. There was paralysis of the right side of the face of the peripheral type, of the right sixth nerve and of associated movements of the eyeballs to the right. Some vertical nystagmus of the right eye. The left arm was markedly paretic, somewhat thinner than the right, slightly spastic. In walking he dragged his left leg, the gait was spastic and there was some spasticity on the right side; there was a Babinski on the left side only; no Kernig. The fundus showed no pathological condition. No incoördination.

The tuberculous opsonic index was taken five times and was found to be 0.65, 1.62, 1.30, 0.86, and 0.57 respectively. The temperature was normal most of the time; occasionally there was a very slight rise, at other times it was slightly subnormal. The pulse rate was about normal. A few days before his death, which occurred on December 19, 1907, he became stuporous and the pulse became rapid and weak. A diagnosis of tubercle of the pons had been made, and the case demonstrated with that diagnosis. Autopsy revealed solitary tubercle in right side of pons, and microscopical examination of the lesion showed typical tubercles with giant cells.

That peculiar law which brings one more cases of the same type brought to Dr. Kuh no less than four cases of crossed hemiplegia in the last two months or so, the last a young man. There was a paralysis of the extremities. A peculiar matter of this case is that in this instance there was a very pronounced optic neuritis. At first the etiology was not quite clear, but a thrombosis was found in the left lower extremity. A very remarkable matter in this second case was that nearly all of the paralysis cleared up in a very short time. After four or five days no paralysis of the right leg was found. The patient died, and toward the end there was practically nothing left except paralysis of the sixth nerve.

Periscope

Neurologisches Centralblatt

(Vol. 27. 1908. No. 1. January 2)

1. Observations Concerning the Auditory Cortex of Human Brain. PAUL FLECHSIG.
2. Periodicity in the Appearance of Nervous Symptoms. H. OPPENHEIM.
3. Paresis in Catholic Clergy. K. PANDY.

1. Continued article.

2. *Periodicity in Nervous Disease.*—Oppenheim cites several cases of organic nervous disease, most of them being syphilitic, in which the symptoms were divided into two classes—permanent and transitory. Among the former were pupil rigidity, hemiparesis, anesthesias, etc.

The transitory conditions were a variety of subjective symptoms, *e. g.*, headache, nausea, vomiting, lassitude, restlessness, insomnia, depression, vertigo, disturbances of vision and hearing. These appear and disappear with almost perfect regularity and generally the patient has continuous alternation of good and bad days—one day feeling perfectly well, the next suffering from many symptoms. Two facts are emphasized:

1. Stubbornness to treatment, especially to suggestion.

2. Symptoms correspond to an organic base and generally appear in patients showing signs or sequelæ of luetic infection.

Argument is made against the probability of the symptoms being hysterical, but that "there probably exists in the organization of the nervous system of certain individuals a *disposition* to periodical, rhythmical appearance of symptoms which remains latent until awakened by disease of brain. We have no clear conception of the origin of this, but see many analogies in the rhythm of sleeping and waking, the regularity of menstruation and in many forms of insanity and neuralgia."

3. *Paresis in Clergy.*—After a study of asylum records Pandy arrives at the conclusion that general paresis among Catholic clergy is not, as is commonly considered, a rarity, but is as frequently met with as among other callings, syphilis and dissipation being frequent in the histories of those affected.

(No. 2. January 16)

1. Observations Concerning Auditory Cortex. PAUL FLECHSIG.
2. Tetany and Calcium Metabolism. W. STOELTZNER.

1. *Auditory Cortex.*—Flechsig considers the cortical auditory center to be the anterior transverse temporal convolution. His method of investigation is principally the myelogenetic, which he finds most efficient in following the fiber tracts. This method consists of the study of fetuses in different stages of development, noting order in which the tracts receive medullation, as these always follow a definite succession. In contrast to the cord in which the motor fibers are medullated before the sensory, in the sensory area of the cerebral cortex the corticopetal always precede the

corticofugal. In a fetus of 50 cm. long a medullated bundle appears extending from the internal capsule or lenticular nucleus to the anterior transverse temporal convolution (primary auditory project system). Arcuate fibers are not medullated at this time, but become so at about the maturity of the fetus, and it then becomes difficult or impossible to establish auditory radiation. So long as only auditory fibers are medullated the greater the number can be seen to enter the inner two thirds of the anterior transverse temporal convolution; the outer third contains fewer, and only a few isolated fibers go to the free-lying portion of the first temporal convolution. This tract can be traced downward directly to the region of the internal geniculate.

Corresponding results are obtained by secondary degeneration following destruction of the anterior transverse temporal convolution. This tract represents the upper tract in relation to the cochlear nerve. The course of the vestibular is doubtful. The difference in cortical topography of the right and left temporal regions is fairly constant and conditions are found reversed in the left-handed, while in three distinguished musicians both sides were almost equally developed. As to the structure of the cortex, there are certain characteristics as to number and thickness of layers, and the boundaries of the auditory cortex can be more sharply outlined in the new-born than in the adult, but this area cannot be as sharply mapped as the visual cortex. Large pyramidal cells appear early in the fourth layer similar to solitary cells of the visual cortex and post-central convolution. Cajals' special giant cells of the auditory cortex are too widely distributed over the temporal lobe to be characteristic.

Fibers of auditory radiations are interwoven and enter the cortex in an irregular tangled mass in contrast to comparatively regular parallel bundles of optic radiations. However, there is nothing distinctive in the histology of the auditory cortex by which it can be outlined.

Destruction of the right anterior transverse temporal convolution causes only slight disturbance of hearing in the left ear. Destruction of the left causes *word deafness*, usually permanent or nearly so.

Amnesic aphasia is absent in lesion of the anterior transverse temporal convolution, but lesion in the first temporal causes amnesia without word deafness.

Thus, according to the author, the common doctrine of aphasia needs revision in respect to localization of sensory aphasia.

2. *Calcium and Tetany.*—Stoeltzner defends his hypothesis that tetany is caused by excess of calcium in the body fluids. This excess may be brought about by inefficiency of excretion of calcium, and conditions favoring this are, on one side a large supply of calcium in the food, on the other a "negative balance" of the skeletal tissues. By his hypothesis he explains the predisposition to tetany of children with gastro-intestinal disturbances and rickets; also the fact that many children do not develop tetany in spite of a rich amount of calcium, while others develop it with a small amount of calcium in the food.

The so-called "negative balance" of the skeletal tissues he describes as a condition in which more calcium is liberated from the bony tissues than is absorbed by the formation of new bone. This, with the calcium absorbed from the food, accumulates in the body fluids if elimination is insufficient and tetany results.

S. D. INGHAM (Philadelphia).

Review of Neurology and Psychiatry

(Vol. V. No. 7. July, 1907.)

1. Psycho-epilepsy. Sir Wm. R. Gowers.
2. The Symptoms due to Peripheral Neuritis of Spinal Lesions in Diabetes Mellitus. R. T. Williamson.

1. *Psycho-epilepsy.*—In this brief leading article, Gowers suggests that psychic symptoms similar to those sometimes preceding an epileptic fit, may be extended and perhaps replace the fit, and he briefly cites a few illustrative cases: one of prolonged depression; one of intense vague dread; one of "thoughts passing through the mind like dreams"; and one of a condition of mental inertia, such as sometimes accompanies "vagal attacks."

2. *Symptoms Due to Peripheral Neuritis or Spinal Lesions in Diabetes Mellitus.*—In many cases of diabetes, nervous symptoms occur which are due to changes in the peripheral nerves, or in the fibers of the posterior nerve roots just after they have entered the cord. The chief symptoms of the so-called diabetic neuritis are: pain and burning sensation; tenderness and hyperaesthesia of skin and muscles; loss of vibrating sensation on the feet or feet and legs; loss of tendo-Achillis reflex and frequent loss of knee-jerks. The symptoms resemble those of tabes, in some cases, but the diagnosis from tabes is usually easy. In all cases of severe pains in the legs, in sciatica, and in cases with symptoms of peripheral neuritis, examination of the urine for sugar is urged. Slight nervous symptoms may occur in any form of diabetes; more severe in chronic forms; and usually the patients are over forty years of age.

Atwood (New York).

Deutsche Zeitschrift für Nervenheilkunde

(Band 30. Heft 3–4)

6. Disturbances of Sensation in Cerebral Hemiplegias. Sandberg.
7. Hysterical Fever. v. Voss.
8. A Rarely Described Form of Tabes Dorsalis. Lapinsky.
9. Another Case of Angiosclerotic Disturbance of Motility of the Arms. Erb.
10. Tumor of the Gasserian Ganglion, Subjected to Operation. Hofmeister and Meyer.
11. The Optic Tract, The Basal Optic Ganglion and the Fiber System in the Floor of the Third Ventricle in a Case of Atrophy of the Bulbs of Both Eyes. Herzog.
12. The Diminution of the Reflex Phenomena in the Paralyzed Portion of the Body, in Compression of the Upper Portion of the Spinal Cord. Lapinsky.
13. Brief Communication So-called Hysterical Fever. Strümpell.

6. *Cerebral Hemiplegias.*—Sandberg has made a careful study of sensation in cases of cerebral hemiplegia. He divides sensation as follows: First, superficial, subdivided into (1) touch, (2) pain, (3) heat, and (4) cold. Second, deep, subdivided into (1) deep pressure, (2) muscle sense (position and movement). Third, derived sensation, subdivided into (1) localization, (2) the recognition of objects by touch. He reports 5 cases,

particularly, in which there was marked disturbance of the stereognostic sense. Altogether there were thirty-one cases examined, of which ten had no objective disturbance of sensation. In the other cases it was found that the sense of touch and deep sensation was disturbed, and that of pain, heat and cold was well preserved. In the five cases in which the stereognostic sense was disturbed all types of sensation were defective, and there was probably a lesion in the medulla. In those cases in which deep sensation was defective various types of ataxia were present. The disturbance of the deep sensation is the chief cause of astereognosis. Sandberg concludes from his studies, that the psycho-motor tracts, and those for deep sensation, pass together through the internal capsule, and terminate in the brain, because it is in the lesions of the internal capsule that they are chiefly affected.

7. *Hysterical Fever.*—Von Voss reports the case of a woman of twenty-four who, while in the hospital for some gynecological condition, suddenly developed a high fever. She also had psychical disturbances, and a violent hysterical convulsion with loss of consciousness. A diagnosis of hysterical fever was made. There was total hemianesthesia in the left side, and sensitive points; the history showed neuropathic heredity. The hysterical manifestations continued for several months, until the patient finally apparently recovered. Another patient, a woman of twenty-seven, had weakness of the left leg, loss of voice, and convulsive attacks. There were various other stigmata of hysteria, and while under observation she suddenly developed a temperature which reached 39.5°. There was also polyuria. The hysterical manifestations continued for more than a year, and improved somewhat under hypnotic suggestion. Then complete recovery ensued, persisting for three years. Von Voss concludes that hyperthermia belongs to the symptom-complex of hysteria. It occurs only in severe cases, and often in association with convulsive attacks. This fever is not the result of increased muscular activity during the convulsions, but is a primary symptom. All the manifestations of the vasomotor diathesis, such as fever, edema, polyuria, and skin lesions, are best explained by ascribing them to some lesion of the appropriate portion of the cerebral cortex. The diagnosis can only be made when there is no organic lesion.

8. *Tabes Dorsalis.*—Lapinsky reports five cases of tabes dorsalis in which the diagnosis could be made by the typical disturbance of sensation, the ataxia, the change in the pupillary reflexes, the difficulty in micturition, and the changes in the tendon reflexes. All the cases had in common the symptom that, in the early stages, sensory phenomena were entirely absent. On the other hand, all the patients complained of weakness and diminution of power, especially in one or more of the extremities. These parts were either wasted or atrophied, or else apparently normal, with weakened muscular tone. The electrical reactions were invariably normal. The weakness of the muscles did not correspond to the distribution of the peripheral nerves. In addition, the bones and joints could be excluded as the cause, nor was there any evidence of myositis, or myopathy. The reflexes were increased, but there were no spastic manifestations, nor was there any evidence of functional neurosis. It can only be explained, therefore, upon the basis of a diminution in the functional activity of the cells of the anterior cornua, such as occurs after section of the posterior roots. Lapinsky therefore inclines to the belief that in his cases the symptom is due to an early involvement of the

posterior roots, which control the motor centres of the cord, of the affected extremity.

9. *Angiosclerotic Disturbance of Motility of the Arms.*—Erb reports the case of a woman fifty-seven years of age, who, at the age of fifty-five developed tabes. Shortly after this she had pains in the arms and legs. Finally she noticed constantly, after slight exertion with the right arm, cyanosis and weakness. The right radial was not palpable. The other arteries were sclerotic. In the right arm the blood pressure was 70 millimeters; in the left, 130 to 140. The muscles gave normal electrical reactions. There was a history of slight injury to the right arm half a year before the symptoms developed. This case of dyskinesia angiosclerotica brachii illustrates Erb's law of the duplication of cases.

10. *Tumor of the Gasserian Ganglion.*—A man of twenty-six developed pain in the right side of his face, associated with hypesthesia, hypalgesia, weakness of the muscles of mastication, diminution of vision in the right eye, general emaciation and weakness, and later, protrusion of the eyeball, paresis of the eye-muscles, and choked disc in the right eye. Operation was performed, and a tumor about the size of a cherry was removed from the Gasserian ganglion. Microscopically this proved to be a peculiar type of sarcoma. The patient was entirely relieved of his pain, but later became cachectic and died. Autopsy was not obtained. The removal of the tumor was only accomplished after many difficulties had been overcome. It consisted of chiefly connective tissue and alveolar arrangement, containing numerous plasma cells, together with some spindle and star-shaped cells. It also contained cells resembling those of the spinal ganglion, and degenerated nerve fibers. Cases of tumor of the Gasserian ganglion are exceedingly rare. Almost invariably they are either metastatic tumors, or have invaded the ganglion by contiguity.

11. *The Optic Tract.*—Herzog describes the brain of a woman forty-nine years of age, who, at the age of thirty-one, had some disease of the eye which led to complete blindness. She died of valvular heart disease, and the brain was examined in serial sections. In the optic nerves no nucleated nerve fibers were found until just before the chiasm a few were discovered in the optic nerve, grouped together in the lower portion of the section. A few faintly staining fibers were also found in the chiasm, grouped in small tracts. In each of the atrophic optic tracts there was a well-stained bundle of fibers at the lateral edge. No changes were found in the pulvinar or in the anterior corpora quadrigemina. The fibers in the peduncles of the anterior corpora were normal. The external geniculate bodies were reduced in size, and the fibers were thinner, and stained less distinctly. In the central portion of the optic tract, that is, in the radiation Gratiolet, and in the region of the cuneus nothing abnormal was found. Herzog gives a careful description of the commissures in the floor of the third ventricle. He was able to find Ganzer's and Meynert's tracts in his preparations, but no fibers corresponded to that of Gudden. He also was able to determine the position of Forel's commissure. He then describes the basal optic ganglion, of which the nucleus supra-opticus is a part; and describes the fibers associated with this ganglion. This article is really a careful study of the anatomy of some of the finer details of the cerebral portion of the visual apparatus, and the abstract is merely an imperfect statement of its contents.

12. *Diminution of Reflex Phenomena in Compression of Upper Portion of Spinal Cord.*—Lapinsky describes the case of a woman, fifty-four

years of age, who had some dull pain in the back, in the ribs on the left side, and increasing weakness of the legs. Examination showed (in the left leg) the muscle tone was slightly diminished, voluntary movement diminished in the great toe, and lost for all other parts, complete loss of the muscular sense. No change in the touch, pain or temperature senses, or in the skin reflexes, but increase in the tendon reflexes. In the right leg the muscle tone was normal; the muscular sense was normal; voluntary movement was normal; the touch, pain and temperature senses were greatly reduced, and there was inability to control the bladder. The electrical reactions of the nerves and muscles were normal on both sides. On the left side sensation was diminished as high as the sixth rib. On the right it was normal. There was hyperalgesia in the area of the third and fourth intercostal nerves. A diagnosis was made of tumor involving the left half of the spinal cord, in the neighborhood of the third and fourth segments. The patient, from time to time, had attacks of severe pain radiating over the side of the thorax and abdomen. During one of these attacks the absence of the Achilles tendon reflex was observed. Later the patient developed a bed-sore, but improved on vigorous mercurial treatment and counter irritation. Finally, she developed a tumor in the abdomen, emaciated and died. No autopsy was obtained. Lapinsky made a series of experiments upon dogs which consisted in the destruction of the plexus of the anterior extremities, and the irritation of the injured nerves. He found that during the period of greatest pain the reflexes in the posterior extremities were diminished. Examination of the spinal cords showed that the reflex arc was not injured. In his own case he believes that the loss of the reflex was due to peripheral irritation. He concludes therefore, that the law of Bruns, that loss of the reflexes occurs in acutely developing compression of the spinal cord requires further confirmation. Painful irritation involving the spinal cord may depress the reflexes above and below the affected arc. He therefore thinks that clinically it is important to classify the various forms of paraplegia resulting from compression of the cord into those with, and those without, pain, and that the absence of the reflexes in the first form may be purely functional; in the second form probably due to an organic change in the reflex arc.

13. *Hysterical Fever.*—Strümpell reports a number of cases of so-called hysterical fever, in which the high reading of the thermometer was artificially produced by the patient in order to render herself interesting, or for other reasons. Practically all the patients had hysterical stigmata.

(Band 30. Heft 5–6.)

14. Contribution to the Knowledge of Inherited Nervous Disease. KOLLARITS.
15. Contribution to the Knowledge of Traumatic Lesions of the Conus. FISCHLER.
16. Contribution to the Pathology of the Epiconus Medullaris. MINOR.
17. Extirpation of the Lower Half of the Spinal Cord, and the Subsequent Manifestations. L. R. MULLER.
18. Contribution to the Casuistry of the Neuritic Plexus Paralysis. GROBER.
19. Clinical Contribution to the Study of Hemiatrophy of the Tongue of Supranuclear Origin. MINGAZZINI and ASCENZI.
20. Diffuse Encephalitis of the Pons with Termination in Recovery. BREGMAN.

21. Nine Epileptic Absences or Brief Narcoleptic Attacks. FRIEDMAN.

14. *Inherited Nervous Disease.*—Kollarits continues Jendrassik's studies upon hereditary forms of nervous disease, reporting fifteen cases. Among these he observed changes in the bones, particularly in the spinal column, in the skull, and in the thorax. In another case the tendon reflexes of the patella and triceps were not present, but the Achilles tendon reflex was exaggerated. He believes that this condition is often present, and may be explained by assuming tension of the gastrocnemius. He also observed that, in spite of the flexion contracture the extensor muscles of the forearm were the stronger. The vegetative functions were often involved, particularly deglutition, defecation and micturition. In one case of pseudo-hypertrophic distrophy the patient was apathetic and rarely spoke. This was the only instance of defect in intelligence. In several of the fifteen cases the exciting cause appears to have been some infectious disease. Kollarits regards alcoholism in the parents rather as a sign of their degeneration, than as a cause of degenerative hereditary disease in the children. He calls attention to the frequent difficulty in classifying these cases, and the polymorphous character of the nervous effects, not only in the same case, but particularly in different members of the same family. Consanguinity appears to play an important part. In four of the fifteen cases it could be determined to be present to a greater or less degree. The difference in the age of the parents does not appear to exert much influence. In one of the families they were of the same age; in two others, the father was twenty years older than the mother. In twelve cases, however, the father was over thirty-seven years of age. It appeared that the earlier children in the family usually escaped, the hereditary manifestations of the disease affecting those born somewhat later. In many families the boys seemed to be more frequently affected than the girls. In only one instance, that of hereditary optic atrophy, were two generations affected by the same condition. Among the typical forms of hereditary degeneration Kollarits includes hereditary neurasthenia, epilepsy, arteriosclerosis, and sterility. The pathology appears to consist in an aplasia or failure of development, in the affected parts, particularly the central nervous system. The cases observed were as follows: (1) Muscular atrophy and shortening; tremor of the upper extremities; nystagmus; increase in the tendon reflexes. In one case there was found degeneration of the lateral pyramidal columns, and a slight degeneration in Goll's column. (2) The symptom-complex of Friedreich's ataxia, combined with dystrophy. (3) Two brothers with nystagmus; intention tremor; cerebellar ataxia; spastic phenomena; increased tendon reflexes; stuttering, with remittent course. One of the cases also had arteriosclerosis, intermittent claudication and contracture of the right hand. The third brother died early of arteriosclerosis; three sisters had neurasthenia, and one hysteria. (4) Divergent strabismus, spastic paralysis, genu recurvatum, small head. (5) Three sisters and one brother had pseudo-hypertrophy. (6) Two brothers had pseudo-hypertrophy. (7) Pseudo-hypertrophy, deformed spinal column; Babinski reflex. (8) Pseudo-hypertrophy; general distrophy with chypho-scoliosis; difficult deglutition, and various developmental peculiarities, and shortening of the muscles. (9) Pseudo-hypertrophy, followed by general dystrophy, divergent strabismus; extreme chypho-scoliosis; deformed thorax; drumstick fingers; increased Achilles tendon reflexes. (10) Pseudo-hypertrophy; muscular retraction; abnormal conformation. (11)

Three brothers with muscular dystrophy, beginning in the face and muscles of the shoulder girdle. In one case the leg and forearm were pseudo-hypertrophic. (12) General dystrophy, more pronounced on the left; diminution of the tendon reflexes on the left. Imperfect development. There was strabismus, and difficulty in urination. (13) Rhachischisis, spastic symptoms. (14) Various forms of nervous disease in three generations, including impaired vision. In one case, atrophy of the optic nerves was present. Three of the daughters were sterile. (15) The parents were uncle and niece. Two daughters and a son had epilepsy; two daughters were healthy.

J. SAILOR (Philadelphia).

Psychiatrische-Neurologische Wochenschrift

(Vol. 8. November 10.)

1. Contribution to the Psychology of Cataleptic States in Catatonia. (Continued.)
2. Effect of Isopral.
3. Hysteria and Religious Impressions. WITRY.
4. Forensic Significance of Paramnesia. (Continued.)

2. *Effect of Isopral.*—Isopral has the chemical name trichloriso propylalcohol. It was first used in 1904 as a hypnotic. The author of the article is not able to conclude from his observations what the value or the possible danger of drug may be as it has not as yet been long enough in use.

3. *Hysteria and Religious Impressions.*—A short account of two hysterics. Of no special interest.

(November 17.)

1. The Technic of Large Brain Sections. L. W. WEBER.
2. Freud's Mechanism in the Symptomatology of Psychoses. (Continued.) Professor E. BLEULER.
3. Forensic Significance of Paramnesia. (Continued.)

1. *Large Brain Sections.*—A purely technical paper on the methods of hardening, embedding and cutting large sections. Does not lend itself to abstraction.

(November 24.)

1. Freud's Mechanism in the Symptomatology of Psychoses. (Continued.)
2. Forensic Significance of Paramnesia. (Continued.)

(December 1.)

1. The Plaut-Wasserman Researches regarding Syphilitic Antibodies in Paresis. KONRAD ALT.
2. Freud's Mechanism in the Symptomatology of the Psychoses. (Concluded.) PROFESSOR E. BLEULER.
3. The Forensic Significance of Paramnesia. (Concluded.) PROFESSOR H. ZINGERLE.

1. *Syphilitic Antibodies in Paresis.*—It is generally conceded now that paresis is the result of a preceding specific infection. The anamnesis, however, is notoriously inaccurate partly because paresis seems to develop in those cases that showed few of the syphilitic manifestations after infection and partly because of defects in memory as paresis usually follows

infection by ten or twelve years. This latter defect in anamnesis can be well illustrated by an attempt to get a syphilitic history in well recognized cases of syphilis. This accounts for the wide variation among different investigators who give all the way from twenty-five to ninety per cent. of previous infection in paresis. A clinical method for definitely determining the presence of syphilis must therefore be most valuable. Investigations of the cerebro-spinal fluid have been made both as to cellular and albumen content—but authors do not agree on the results and then other conditions than syphilis—inflammation—give similar pictures. A specific reaction, therefore, does not exist by this method. The methods of serum diagnosis offer most in the way of possibilities of diagnosis. When an infection invades an organism it endeavors to protect itself by the development of the so-called antibodies which tend to destroy or weaken the infecting material and these antibodies are developed most in the tissues most seriously involved. The method results in the bringing together of the invading body and the antibody and the result will inhibit the hemolysis of the red blood corpuscles.

Plaut and Wasserman made lumbar punctures in forty-one cases of paresis and mixed the fluid with a clear, watery extract from the organs of luetic fœtuses. Thirty-two cases already showed inhibition of hemolysis, four cases were not so clear, the other five cases failed to show it at all. The same lumbar fluid with extract of non-syphilitic organs did not give the reaction. The luetic extract was mixed with cerebro-spinal fluid of nineteen non-syphilitic men without interfering with the hemolysis. A control experiment with three cases of meningitis was negative. Experiments seem to show that antibodies are produced for a long time in the nervous system. This is probably due to the fact that the nervous system seems to contain the tissue especially elected by the specific virus. Researches in three cases, for example, showed the cerebro-spinal fluid much richer in antibodies than the blood serum. The author of the article closes with some general considerations as to the different problems and lines of research this method of Plaut and Wasserman opens up.

2. *Freud's Mechanism in the Psychoses.*—The author speaks of the many criticisms of Freud and, on the part of some, of the wholesale denunciation of his methods. He believes, therefore, that a critical review of his work should be made so that if there is anything good in it it will not be lost. Freud maintains that our psyche has a tendency to build up the picture of the world in accordance with our wishes and our efforts. This disposition is well shown in all situations in which the connection is disturbed between the outer circumstances conditioning thought and their logical relations to reality. This is the case in dreams, in unconscious movements, inattentive speech and writing, etc. Freud has shown this tendency in the neuroses and endeavored to formulate a useful therapy on it as a basis. The same mechanism also plays a great rôle in the pathology of other mental disorders.

The author does not attempt a detailed analysis of Freud's teachings. In order to indicate their nature he picks out some simple examples where analysis gives the most essential points. The full explanation of a short dream will show many of them. Once a patient suicided in the hospital of which the author was director. He dreamed that the suicide had occurred in another hospital of which he had been formerly director and congratulated himself that he was a better director than his successor. During working hours one wishes and fears, our inclinations and antipathies

govern our thoughts, our half-conscious acts, even our comprehension. Names of people who are disliked by us are easily forgotten. In misspeaking and misreading the same influence plays a great rôle. In reading nearby words not actually seen are often falsely apprehended. Usually there is only a small change like Leitung for Leistung. This was due to the fact that while reading the mind was occupied by thoughts in which Leitung occurred, not Leistung. One who keeps a diary will find on referring to it that events are not accurately put down. Our thoughts even transform our memories according to our wishes. Persons react against or away from disagreeable ideas (*abreagirt*). Intolerable ideas are suppressed but give origin to hysterical symptoms. If it is possible to bring these ideas and their corresponding effects into consciousness often the disorder disappears. These unpleasant circumstances to which the individual has *abreagirt* often play an important part in life. The poet's phantasies may frequently be traced to them. Thus many of the most beautiful love songs have been composed by those disappointed in love. Such was their method of compensating for the loss. Nietzsche created the super-man because he was of a very tender nature. Houston Stewart Chamberlain, the great defender of the idea of the improvement of race, was himself of mixed blood. The same mechanism governs the details of artistic creation. Goethe must have his Werther die, not from artistic necessity, but because he must destroy this side of himself which had done and suffered so much. In such manner our affectivity builds up symbols which express our wishes and our fears. This thinking in symbols is of the highest importance. Owing to this symbolic method of thinking things which in reality are widely different, but may have a single point of similarity, are often confused and it becomes difficult to untangle the true meanings. In all this symbolism it is natural that the sexual should play a large and important rôle. An illustration of the symbolic character and method of association of ideas is given in a case of dementia precox. A young hebephrenic believed his father dead. He continued in this idea even after his father visited him. Examination showed that he had seen his father's death (suicide) in a dream. As a further explanation it seems his father had recently married a young woman whom the son loved. Therefore the father must make a place for the son. The contents of many delusional states are often nothing but badly retained bits of dream consciousness which often have the characteristics of wish fulfilling dreams. This is often so in delirium tremens. A patient who had been arrested for stealing food and a coat thought himself at a great fair where he could buy everything he wished and where he got two beautiful suits. The case of an embezzler is cited who all during his delirium saw pleasant visions of dancing fairies, etc. There was no relation between the actualities and his delirium of which he was merely the interested onlooker. This is not uncommon in delirium tremens.

Even in organic brain disease, such as paresis and senile dementia, the Freud principle is useful. It is found that the patient forgets the things easiest which are unpleasant to him. In summing up the author recalls the fact that interference with the realization of wishes may lead to persecutory ideas—that the sexual feeling is easily changed to anguish—and last but not least that in general the condition of our bodily organs influences our mood. On all these grounds must the patient have the tendency to produce unpleasant perceptions and moods, and it is not to be wondered at that this negative tendency is only seldom overcompensated through the positive one of the Freud mechanism.

3. *Forensic Significance of Paramnesia.*—An account of two cases with the details of both physical and mental examination. The author cautions that cases following head injury are apt to fill up gaps in the memory without noticeable disturbances of consciousness so that such cases must be very carefully examined.

WHITE.

Revue Neurologique

(No. 5. March 15, 1907)

1. A Case of Crossed Atrophy of the Cerebellum. RENE CORNELIUS.
2. New Contribution to the Study of Bone Reflexes. NOICA.

1. *Crossed Atrophy Cerebellum.*—The authors report a case of atrophy of the right cerebral hemisphere particularly marked in the frontal and second temporal convolutions. The atrophy of the cerebellum was most marked in the quadrilateral lobe of the left hemisphere and involved also the dentate nucleus.

2. *Bone Reflexes.*—These reflexes occur in hemiplegics or in spastic paraplegics. In the lower limbs the contractions are in the muscles innervated by the fifth lumbar root. In the upper limb the centers for these reflexes are probably in the seventh and eighth cervical segments.

(No. 6. March 30, 1907)

1. Some Researches on the Transplantation of Nerve Ganglia. MARINESCO.
2. A Sign of Organic Paralysis of the Lower Limb: The Possibility of Elevating the Paralyzed Limb with the Impossibility of Elevating both Limbs Simultaneously. GRASSET.
3. Oculo-motor Palsy by Labyrinthine Intoxication. PIERRE BONNIER.

1. *Transplantation Nerve Ganglia.*—The researches of Marinesco are on the histological changes taking place in transplanted nerve ganglia. For the most part the nerve cells undergo grave modifications and disappear; only those at the periphery survive for a short time.

2. The sign is sufficiently explained in the title of the paper.

3. The cerebro-spinal and labyrinthine spaces are communicating; any variation in the composition of the cerebro-spinal fluid is often manifested by oculo-motor palsies of which the most frequent is paralysis of the sixth pair of nerves.

(No. 7. April 15, 1907)

1. Peripheral and Central Neurofibromatosis. J. ROUX.
2. Alterations of the Peripheral Nerves in Fatal Anemia. (Apropos of Neuritis in the Insane.) E. MEDEA.

1. A case of neurofibromatosis in a lad of twelve years who also had epileptic attacks confined to the left side and a partial left hemiplegia. The left side was cyanotic and there was amblyopia of the left eye. The author's diagnosis was multiple lesions of the central nervous system of the same nature as the peripheral lesions.

2. The two aspects of the question considered are: the frequency of neuritis in the insane, and the relation of the psychic trouble to the neuritis. In three cases where the clinical symptoms suggested neuritis, histologic examination showed the nerve to be normal. In another case in which there was great anemia there was some degeneration of the nerves.

(No. 8. April 30, 1907)

1. Experimental Researches on the Morphology of the Cells and Fibers of the Spinal Ganglia. J. NAGEOTTE.
   1. Not suitable for abstracting.

(No. 9. May 15, 1907)

1. The Gnosic Function. MAX EGGER.
2. On an Ill-determined Meningo-encephalic Affection. HENRI CLAUDE and PAUL LEJONNE.

1. The author reports a case of loss of the ability to recognize objects by the sense of touch though the only sensory disturbance present was enlargement of the circles of Weber. As in cases of pure motor paralysis (one case given in detail) there is no interference with this function, and as in cases of tabes there may be enlargement of the circles of Weber without the difficulty in recognizing objects placed in the hands, he concludes that in the case reported the gnosic function was lost, not because of any lesion in the sensory projection system, but rather in the association tracts.

2. The patient, a female aged thirty-one years, began to have propressive psychic troubles. A month later, following a convulsive crisis, she assumed the aspect of a general paralytic. A month later the symptoms again changed and then developed fever, rigidity of the neck, Kernig's sign and contractures, but the lumbar puncture was negative. This state lasted, with remissions, for three months, when the symptoms changed to those of multiple sclerosis, intention tremor, scanning speech, exaggeration of tendon reflexes and abolition of the abdominal reflex. All these symptoms slowly diminished.

The authors regarded the case as one of meningo encephalitis.

(No. 10. May 30, 1907)

1. The Root Ganglia Lesions of Zona. J. DEJERINE and ANDRE-THOMAS.
2. Chronic Rheumatism and Thyroid Insufficiency. PEPPO ACCHIOTE.

1. A man, aged seventy-five years, developed an eruption of herpes zoster in the territory exactly localized in the distribution of the eighth cervical root. Six weeks later he died of a cardiac affection. Lesions were found only in the eighth cervical root and they consisted of partial degeneration of the fibers, an inflammatory reaction in the ganglion and degeneration of the cells. A regenerative process was apparent, proving the aptitude for rapid restoration.

2. Following eighteen treatments with X-rays for removal of superfluous hair, a woman developed myxedema and at the same time chronic rheumatism. Both conditions improved greatly under treatment with thyroid extract.

C. D. CAMP.

Book Reviews

THE PSYCHOLOGY OF ALCOHOLISM. By George B. Cutten, M.D., M.A., Ph.D. Charles Scribner's Sons, New York. $1.50.

We have had the opportunity to comment on portions of this work as it appeared in the *American Journal of Psychology* as early as 1902 and welcome it most heartily in its present more permanent and convenient form. From many points of view Dr. Cutten's presentation is one of the most satisfactory that we have had the fortune of seeing in the English language. He has grasped the meaning of the work of Kraepelin and his school in their study of alcohol on psychical functions and has presented us not only a readable account of the relation of alcohol to mental states, but an accurate one as well.

The scope of the book may best be indicated by the chapter headings, since a subject such as the psychology of alcoholism offers so broad a choice for a writer. In the introduction some historical facts are entertainingly presented—the alcoholist has been with us since the beginning of written records—the prevalence, spread, and cost to the community are also dealt with in this place and the relation to disease and mortality touched upon.

The Physiology of Alcoholic Action is then taken up in a modern manner, the newest work of Overton and Meyer receiving full attention. Then follow chapters on the effects of alcohol on the memory, on intellect, on will processes, the emotions, senses, and on morals. Chapter IX is devoted to the insanities of alcohol, Chapter X to religious conversion as a cure for alcoholism and Chapter XI to hypnotism and other cures.

The author is disposed to regard religious conversion as one of the most potent of therapeutic agents. The author's comment on the therapeutic aspects indicate the study rather than the clinic, but apart from this feature we know of no work in English that shows such a thorough grasp of the entire subject as this.

JELLIFFE.

DIE KINDLICHE PSYCHE UND DER GENUSS GEISTIGER GETRÄNKE. Von Leopold Lang. Verlag von Josef Safar, Vienna. 1.40 marks.

In this short monograph one will find an excellent discussion of the action of alcohols upon the psyche, especially dealing with its hereditary and degenerative aspects. In addition the author gives us an excellent experimental study, by means of the association methods, of the action of alcohol on psychical processes.

The work that is here presented bears the stamp of thoroughness and diligence and if few new facts have been added it is because so many investigators have preceded the author in this important field. The monograph is a worthy one and affords an excellent summary in a restricted field of research.

JELLIFFE.

DIE ERKENNUNG UND BEHANDLUNG DER MELANCHOLIE IN DER PRAXIS. Von Geh. Med. Rat. Prof. Dr. Th. Ziehen in Berlin. 2d Edit. Carl Marhold, Halle.

Ziehen gives us here a second edition of his diagnosis and treatment of melancholia which appeared some few years past. His definition of melancholia appears as in his text-book as a condition in which there is a primary, continuous, unmotived, or insufficiently motived emotional depression, associated with a primary slowing of the flow of ideas, or thought retardation. In which it may be seen that his criteria are far from being as searching as those of Kraepelin. Naturally there is little said of the relations of this melancholia to Kraepelin's manic-depressive insanity.

Although nothing is said of delusional ideas in the definition Ziehen discusses them later in his monograph. Anxious depressions are separated from the simple depressions, hallucinations and illusions are not characteristic. He distinguishes (1) melancholia passiva, (2) melancholia attonita, (3) melancholia agitata.

As to prognosis, 90 per cent. recover, but the picture is clouded by the tendency to recurrence. Ziehen distinguishes the hypomelancholia, apathetic, hallucinatory, impulsive, neurasthenic, hysterical and periodic varieties. Concerning treatment he advocates rest in bed, opium, and hydrotherapy. JELLIFFE.

MENTAL DEVELOPMENT IN THE CHILD AND THE RACE. METHODS AND PROCESSES. By James Mark Baldwin, Professor of Philosophy and Psychology in Johns Hopkins University. Third Edition. The Macmillan Company, New York.

The reviewer feels that this work is one of great value to all students of the problems of neurology and psychiatry. The author has followed so closely in the lines of biology in his interpretations of psychology that his methods of thought and conclusions ally themselves directly with those naturally arrived at by the medical thinker.

The study of the development of race consciousness is the essentially fundamental theme of the volume and in this the third edition much of the abstruseness present in the first edition of 1895 and which was reviewed here at that time, has disappeared. In the field of mental disorders probably the most fruitful point of view for the psychiatrist to assume is that adopted by the exponents of genetic psychology, of which Baldwin is one of the ablest and this volume its best expression. Genetic psychology lays the foundation for education and in faulty education the psychiatrist sees the fountain source of some psychoneurotic products and perhaps even some of the psychoses.

We have nothing but the highest praise for this new edition and can recommend it most heartily to our readers. JELLIFFE.

DIE GEISTIGEN EPIDEMIEN. Von Willy Hellpach. Rütten und Loeing, Frankfurt am Main.

A short popular monograph on psychic epidemics written in the "high" German style, not uninteresting, but replete with fine phrases and few facts. It deals with a number of general philosophical questions, notably prostitution and hysteria, painting them in a striking and effective literary manner.

JELLIFFE.

Beiträge zur Kenntnis der Heine-Medinschen Krankheit (Poliomyelitis acute und verwandter Krankheiten. Von Dr. Ivar Wickman, Privat dozent am Karolinischen Institut zu Stockholm. S. Karger, 1907.

This is a second contribution of the author to this subject made within two years. In 1905 a monumental pathological study and now a complete discussion of the various features of the disorder founded on a comprehensive review of recent Swedish epidemic conditions. Wickman proposes to rechristen the acute disease as the Heine-Medin disease, from Heine who in 1860 first gave a description of the acute disorder and Medin who in 1890 called attention to the contagious nature of the epidemic occurrence of the trouble. Wickman bases this monograph on a study of 1031 cases which have been analyzed in a cursory manner in his contribution to the *Zeitschrift für klinische Medizin*, Vol. 63, 1907, and here in a more thoroughgoing manner. He distinguishes 8 forms of the disorder. I, Acute Poliomyelitis Form; II, Landry's Paralysis Form; III, Bulbar or Pontine Forms; IV, Encephalitic Form; V, Ataxic Form; VI, Polyneuritic Form; VII, Meningeal Form, and VIII, Abortive Forms. He discusses each of these forms in extenso. He then discusses the Epidemiology of the disease in an exhaustive manner, paying particular attention to the Sweden epidemic of 1905. The incubation time he believes falls under 10 days, the period of 1–4 days showing the greatest probability. Then follows an exhaustive and extremely interesting series of analyses of the factors of spread in a number of epidemics in smaller Swedish towns. These are models of epidemiological research and should be consulted by all workers in neurology.

The month of August was the month of greatest frequency in Sweden in the epidemic of 1905, 370 of the 1,000 cases ocurring in this month. In 1906 the cases fell to less than fifty in any month of the year.

So far as etiology is concerned heredity, trauma, taking cold, etc., play practically no rôle, the chief factor is of an unknown but specific infectious nature. From the standpoint of prognosis Wickman enlarges the conceptions usually held in the text-books. He shows that the disease is dangerous to life in a high percentage, 159 in 1,025 cases, 16.7 per cent., and that conversely abortive and mild forms appear in which recovery is absolute even after severe paralytic symptoms. The greater number of abortive cases occur under the year of 14; adult cases are more severe both as regards mortality and permanency of paralysis.

This monograph adds another to the rapidly growing literature of this subject. It is a notable addition to our knowledge of the disease.

Jelliffe.

Leitfaden des Verfahrens bei Geisteskrankheiten und Zweifelhaften Geisteszuständen für Militärärzte. Von Dr. Bruno Drastich. K. and K. Stabsarzt und Chefarzt der psychiatrischen Abteilung des Jarnisonspilates No. 1, in Wein. Josef Šafář, Wein. 4.75 marks.

This work affords an excellent illustration of the specialism that is found throughout the Teutonic races. Not only is the military service well equipped with hospitals, but such hospitals have their psychiatric divisions, controlled and directed by competent psychiatrists, the evidence of such competency being manifest in the work before us.

It deals with those forms of psychiatric questions most liable to come up in military life and special emphasis is laid on the more practical questions of discipline and field service.

Imbecility, Moral Insanity, Degeneration, Alcoholism, Paresis, Dementia Præcox, Paranoia, Manic-Depressive Insanity, Infectious and Exhaustion Psychoses, Epilepsy, Hysteria and Neurasthenia, are the chapter headings in the special portion.

The author is a partial follower of Kraepelin although acknowledging his allegiance to the Vienna school, but he makes the statement that the problems to be solved in the army must be laid down in unequivocal terms and questions of different terms for the same conditions are of less importance than the prognosis, and here the Kraepelian doctrines stand paramount in their practical importance.

The detailed analysis of the work is of secondary value in this place. It is an excellent manual of psychiatry written with the needs of the military surgeon in the foreground. JELLIFFE.

PRINCIPLES OF MICROSCOPY. Being a Handbook to the Microscope. By Sir A. E. Wright, M.D. (Dublin). The Macmillan Company, New York.

"The present text-book," writes the author in his preface, "has no message to those who are content to follow a system of rule of thumb, and to eke this out by blind trial and error. It addresses itself to those who are dissatisfied with the results thus obtained, and who desire to master the scientific principles of microscopy, even at the price of some intellectual effort."

It is all of this, being a very thorough and scholarly work on the microscope and the microscopical image. There is nothing of so-called microscopical technique in the book, by which is meant the ordinary handling of objects intended for research, nor yet is it solely a work on applied optics, although the optical parts of the work occupy the foreground.

To one who would thoroughly understand the optical principles involved in the use of the microsope we know of no better book to recommend. JELLIFFE.

CONSIDÉRATIONS SUR LA MALADIE DE PARKINSON ET SUR QUELQUES FONCTIONS NERVEUSES. Par Dr. Gaston Maillard, Jules Rousset. 1 Rue Casimir-Delavigne, Paris.

This is an interesting small monograph in which the author advocates that paralysis agitans is due to an arterio-sclerosis in a mesocephalic organ situated specially in the cerebral peduncle, and probably the red nucleus. It is clinically marked by the fundamental symptoms of slowness of movements, pathognomonic tremor, rigidity, all of which are due to disturbances of static equilibrium consequent to a lesion in the organ indicated.

In addition to these fundamental symptoms there are certain accessory phenomena which may be observed and which depend largely on the site of the arterio-sclerotic process. Our author distinguishes three groups: paretospasmodic, pseudobulbar and psychic symptoms.

The interesting features of the monograph are the authors theoretical explanations of muscle tonus, of orientation, the relations of propulsion to the cerebellum, and the conception of the mesocephalic nervous centers and their relations and connections with the cortical, spinal and bulbar centers. JELLIFFE.

JURISTISCH-PSYCHIATRISCHE GRENZFRAGEN. Vol. 5, Heft. 6 and 7. Herausgegeben von Drs. A. Finger, A. Hoche and J. Bresler. Carl Marhold, Halle.

The two last contributions to the series of short monographs present to us in heft 6, the Proceedings of the Meeting for Legal Psychology and Psychiatry in Hesse, held in July, 1906. Dr. Mittermaier, of Giessen, has a communication on the needs for courses in psychiatry for those engaged in medico-legal work. He states that if any advance is to be made in the character of the care of prisoners, especially those suspected of psychoses, and if jurists are to be better equipped for their medico-legal practice, special courses must be given in the universities and clinical courses given in the prisons.

A second paper on the function of the physician especially as a medico-legal expert affords instructive reading in view of the many panaceas that have been offered by self-appointed deities during the past two years. A reading of the discussion brings out the pertinent fact that difficulties exist in Germany, just as they do here, although we are often told to the contrary. Dr. Sommer, of Geissen, draws out very clearly that without a personal examination of a defendant the opinions of an expert are not worth very much and he further suggests that if experts really knew more about the law they would be less ready to attempt to reform that with which they were not acquainted.

Heft 7 gives a full discussion of the subject of Criminal Psychology by Dr. Alfred Gross. In this the author gives us an excellent discussion of the Association method in its application to the detection of crime; an old subject, recently brought to the fore by Jung, and cribbed without acknowledgment by one of our "professors."

JELLIFFE.

ARBEITEN AUS DER DEUTSCHEN PSYCHIATRISCHEN UNIVERSITÄTS-KLINIK IN PRAG. Herausgegeben von Prof. Dr. Arnold Pick, Vorstand der Klinik. S. Karger, Berlin.

Professor Pick gives us another evidence of his industry and scholarly attainments in this recent collection from his clinic. It consists of seven studies, four by Pick himself and three by his assistants.

Those by the chief of the clinic are On Disturbances of Orientation in the Body; Circumscribed Senile Atrophy as an Object of Clinical and Anatomical Investigation; Asymbolia and Aphasia, and Symptomatology of Atrophic Occipital Lobe. Dr. Oskar Fischer has contributed a paper on the Isolated Axiscylinder Degeneration in the Cortex of Paretics; Dr. E. Sträussler, On the Symptomatology and Anatomy of Tumors of the Cranio Pharyngeal Canal (Erdheim), and Dr. M. Pappenheim has given a full description of a case of "Periodic Melancholia, combined with Hysteria and Tabes, and Characteristic Migraine Attacks, at the same time a case of Veronal Poisoning."

We cannot enter into a detailed discussion of these contributions, but call our readers' attention to them at this time. Professor Pick is to to be congratulated on this series of contributions.

JELLIFFE.

VOL. 35. AUGUST, 1908. No. 7.

The Journal
OF
Nervous and Mental Disease

Original Articles

A BRAIN TUMOR LOCALIZED AND COMPLETELY REMOVED, WITH SOME DISCUSSION OF THE SYMPTOMATOLOGY OF LESIONS VARIOUSLY DISTRIBUTED IN THE PARIETAL LOBE.[1]

From the Department of Neurology of the University of Pennsylvania.

By Charles K. Mills, M.D.,

PROFESSOR OF NEUROLOGY IN THE UNIVERSITY OF PENNSYLVANIA; NEUROLOGIST TO THE PHILADELPHIA GENERAL HOSPITAL, AND

Charles H. Frazier, M.D.,

PROFESSOR OF CLINICAL SURGERY IN THE UNIVERSITY OF PENNSYLVANIA; SURGEON TO THE UNIVERSITY HOSPITAL.

The rapidity with which the symptoms indicating serious cerebral disease developed; the presence of a well-defined symptom-complex showing a lesion situated at the junction of the parietal and occipital lobes, and the complete success which resulted from surgical interference, mark the case which is the basis of this paper as one of the most instructive that has yet appeared in the literature of cerebral localization and intracranial surgery. For the opportunity of seeing the case, we are indebted to Dr. H. A. Spangler, of Carlisle, Pa., who before it was seen in consultation, had recognized the nature and probable location of the disease from which the patient was suffering. We shall first present the clinical history of the case, including a résumé of the operation with comments, and shall then briefly discuss its bear-

[1] Read at the thirty-fourth annual meeting of the American Neurological Association, May 20, 21 and 22, 1908.

ings upon the subject of the focal diagnosis of lesions, especially tumors variously distributed in the parietal lobe.

The patient was a married woman, forty-five years old, who had enjoyed average good health until October, 1907, although two to three years before she had had several attacks of what she described as dizziness or lightness in the head. She had not however suffered with headache, nausea or vomiting. She went to Philadelphia on October 26, 1907, and while there had an attack which she regarded as a bilious spell, and in which she suffered from dizziness, nausea, and vomiting, but no headache. About a week later she had a severe attack of vertigo or dizziness, in which she did not fall or lose consciousness. One week after this she had another attack of dizziness, with a little headache, but without nausea and vomiting. She also at this time showed a little forgetfulness of some details in packing her trunk.

During the few days following she did not feel very well, occasionally being a little dizzy and having headache. On Saturday, November 30, while in Philadelphia she was in fairly good health, but on her way home the next day she was very sick at her stomach and suffered much from headache and dizziness in the cars. She reached home feeling very badly.

On November 30 she almost ran into a tree, and later into a post. These objects were on her left. On the same and the following day she failed to notice persons passing her on her left.

After her return to Carlisle, Dr. Spangler discovered the existence of something wrong with her sight, and with her nervous system and at his suggestion she went to Philadelphia to see Dr. G. E. de Schweinitz, whom she first saw December 19, 1907. The report of Dr. de Schweinitz, made at about this time, showed the presence of left lateral homonymous hemianopsia, and beginning optic neuritis. The fields are shown in Fig. 1.

She returned to Carlisle, where she continued under the treatment of Dr. Spangler, and where she was seen by Dr. Mills in consultation December 30. In the meantime, during the month of December, before she was seen by Dr. Mills, she had headache, worse at times, and was occasionally dizzy. About this time and later she had every two or three days attacks of nausea and vomiting, with dizziness and headache. During the night she would suffer with headache, and in the morning would be sick at her stomach and vomit, with some dizziness, the headache continuing but abating somewhat at noon and returning again in the night. She began to drag the left leg a little, and did not use the left hand and arm as skilfully as the right, this impairment and awkwardness varying considerably at different times. Ever since her first symptoms were noticed in October, the patient had had a peculiar puffing sound in the left ear, and a singing or buzzing sound in the right, although her hearing had not changed. Her memory and powers of attention continued good.

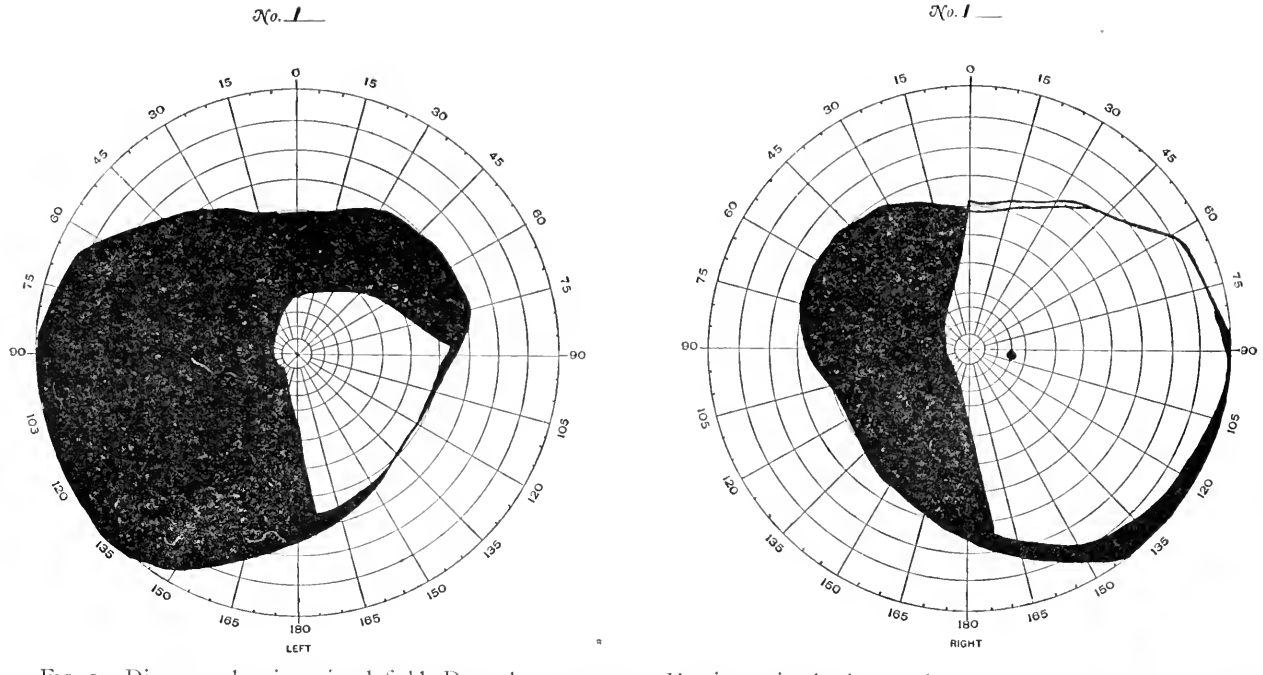


FIG. 1. Diagram showing visual field, December 19, 1907. Hemianopsia absolute; color correctly seen within prescribed fields. Vision, right eye, 6/7.5; left eye, 6/6.

On December 30, when the patient was examined by Dr. Mills in conference with Dr. Spangler, at Carlisle, the chief cerebral symptoms presented were left lateral homonymous hemianopsia; quite moderate hypesthesia of the left extremities; hypastereognosis on the same side as tested in her hand, some ataxia with atactic tremor of the left upper extremity; impairment of the ability to use her left leg and arm, this being due to incoordination rather than to loss of strength. Central vision was little if at all affected; she had no word deafness or word blindness; no aphasia, sensory, motor, or mixed. Her reflexes were not abnormal, or only slightly so, the Babinski response being absent and the response to plantar stimulation being slight or not present.

The patient continued at her home under the care of her family physician from December 30 until February 1. From time to time reports regarding her were made by Dr. Spangler, these showing a very gradual increase in the symptoms indicating the cerebral lesion. Examinations made of her urine during this time showed no albumin and only a few hyaline casts. During this period Dr. J. Walter Park, of Harrisburg, made three separate reports regarding her eyes, each revealing about the same condition, except that the last one showed some increase in the optic neuritis. The ophthalmic findings of Dr. Park summarized were as follows: Vision in the right eye was 20/30 and in the left 20/15; she read number 4 minion type with right eye and number 2 pearl with the left eye. Her field of vision of each eye showed a hemianopsia of the nasal side of the right eye and of the temporal side of the left eye. The ophthalmoscope showed considerable optic neuritis; the veins were tortuous and full, sometimes disappearing under the edematous retina, and then reappearing. At Dr. Park's second examination he found two fatty or albuminoid spots, three or four mm. in size, showing in the right eye, one slightly below, and the other above the disc. They had no well-defined edges, were flame-like in appearance, and fairly whitish.

During January she was under treatment with iodide and bromide in moderate doses. This treatment had indeed been tried previously, but her stomach did not stand it well and she made no improvement under its use.

After a conference by letter it was decided to bring the patient to Philadelphia, where she came February 1, and was at once admitted to the University Hospital. An examination made soon after her arrival showed the same conditions as were present on December 30, with some additions. The dominating symptoms were still the left hemianopsia and hemiataxia. The results of the examination in detail were as follows:

The patient's left palpebral fissure was wider than the right. The left pupil was slightly larger than the right. Both were round. Associated upward movement seemed to be limited, but was not lost. There was no paralysis or weakness of the indi-

vidual superior muscles of either eye. Lateral movements seemed to be well performed. Downward movements, both individual and associated, were well performed. Convergence movement was very slight on the first test, but after several attempts the movement improved. Nystagmus was absent. The pupils responded to light stimulation and to convergence.

The brow was wrinkled equally well on each side. The right eye was forcibly closed better than the left. Resistance to opening was greater on the right than on the left. Slight flattening of the left nasolabial fold was present, with a drooping of the angle of the mouth on that side. The teeth were displayed a little better on the right than on the left side, and puckering the lips for whistling was better done on the right than on the left. Expression of emotion, as in laughing, was equal on the two sides. Voluntary movement of the right side of the face was better than that of the left. The mouth opened straight, no deflection, as in fifth nerve paralysis, occurring. Power was well retained in the masseters and temporals.

In the left upper extremity resistance to passive movements, as compared with the right, was possibly somewhat impaired, and the patient showed a little awkwardness in some of the finer movements.

Light hypesthesia was uncertainly shown on the left, as compared with the right. As the patient herself expressed it, the difference was trifling, and she sometimes seemed to be uncertain about it. She had slight hypastereognosis on the left, at least this was the conclusion from the tests. She recognized everything, but with a little less readiness on the left than on the right. She had some loss of the sense of position and of movement on the left. If movements were made of parts of the limb on the left side, she could not repeat the same on the right side; but if the movements were made on the right side, she could repeat them on the left. The sense of location was undoubtedly better on the right than on the left. Again and again she failed to recognize the place where the finger was touched on the left, whereas when tested on the right she quickly indicated the position. She had very distinct ataxia in the finger to nose test on the left side, hovering and uncertainty being easily observed, although not pronounced. No ataxia was shown when the test was made with the right forefinger. The grip on each side was good, somewhat better on the right, but it must be remembered that the patient was right-handed.

She had no Romberg symptom. She had some awkwardness of gait, due to the impairment of muscular sense in the left lower extremity.

No Babinski was present on either side. Slight plantar flexion was shown on plantar stimulation on the left, and the same on the right. The knee jerks were prompt on both sides, probably a little more so on the right. Ankle clonus was absent.

Ophthalmic examinations were made on several occasions by Dr. G. E. de Schweinitz. He reported on February 3, 1908: "The optic neuritis of the right eye is now five diopters in height, an increase of two and one-half diopters since the nineteenth of January. The optic neuritis of the left eye, which was just beginning, is now three diopters in height, an increase of fully two diopters. An entirely new process is the development of numerous hemorrhages, which have appeared thickly on the swollen discs and in the neighboring retinal areas, and give the impression of being the result of thrombi in the retinal veins, or, in other words, thrombotic hemorrhages, although it is perfectly possible that some of them, owing to the difficulty of venous return, are the representatives of a true diapedesis. Both processes are the common etiological factors in this type of hemorrhage, I think. There is absolute left lateral hemianopsia, that is to say, the blind fields are blind for form-sense, color-sense, and light-sense (Fig. 2). This may be of some importance in your localization. Wernicke's symptom is not present; in point of fact, if there is any difference in the reaction of the pupil to the changes of light and shade, the light reaction is a little more active when the light falls upon the blind side of the retina,—certainly there is no difference. There has been marked failure of direct visual acuity, which has fallen from practically normal to 6/12, or one-half, on the right side, and to 6/7.5 on the left side, that is to say, a little better than two-thirds."

The effects of the operation (to be hereafter described) on the hemianopsia and optic neuritis, as shown by examinations made by Dr. de Schweinitz at intervals after the operation, were very interesting.

On February 13 he reported as follows:

"I examined your patient this afternoon, and have to report to you that while the hemianopsia continues, there is a distinct gain, and the blind area has lessened in size (Fig. 3). Moreover, the contraction of the preserved fields, which was present when I took the fields of vision on the nineteenth of December and the first of February, has disappeared, so that the preserved halves are now fully normal in extent. This would seem to me a very favorable sign as indicating that the pressure, on the one hand, has not been severe enough to entirely destroy the optic radiations, and that we may have fair hopes that there will be still further gain, and on the other hand, that the intracranial pressure has been so materially lessened that the preserved halves have enlarged. There is no change in the size of the discs, which are still swollen five diopters. There is one large fresh hemorrhage in the right retina, in addition to those which I have previously described. It should be remembered, however, that in many of these cases, not only as we have seen them but as they have been reported elsewhere, the real subsidence of the neuritis did

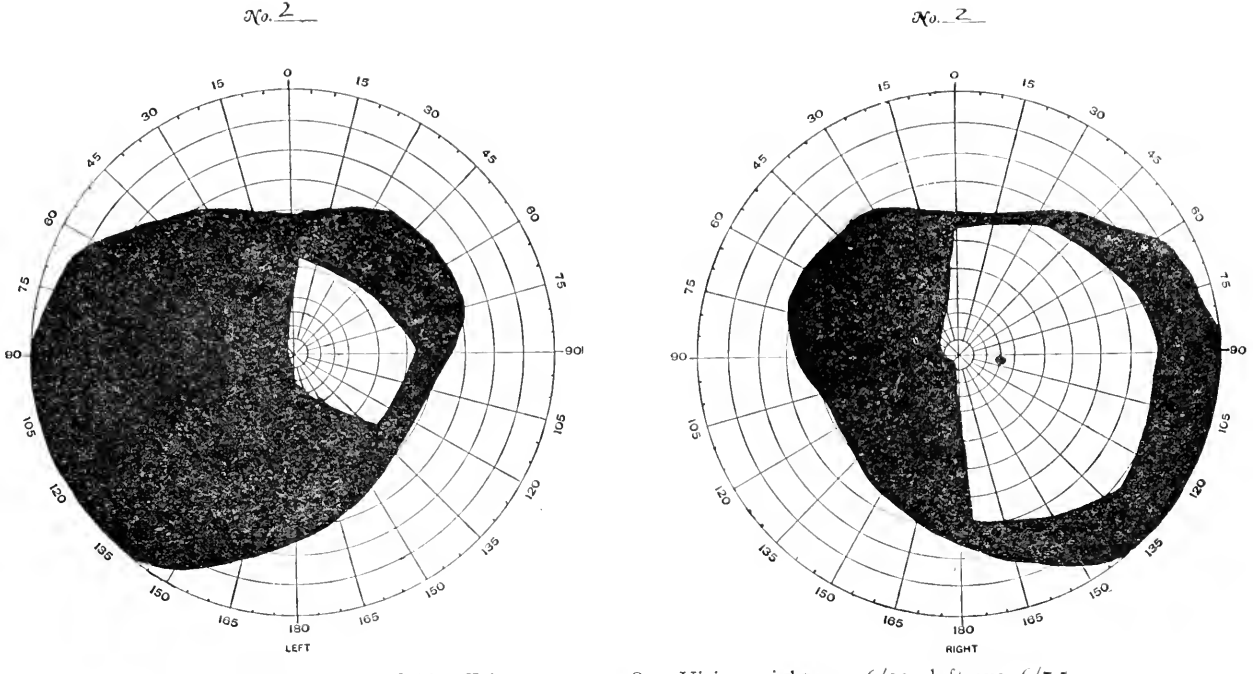


FIG. 2. Diagram showing visual fields, February 1, 1908. Vision, right eye, 6/12; left eye, 6/7.5.

not begin until the fourteenth day, and if I mistake not, in some of Paton's cases as much as five and six weeks elapsed before there was marked lessening of the neuritis."

On March 9 the report was as follows:

"I have great pleasure in reporting to you that the letter vision is normal in each eye, that there is almost complete disappearance of the optic neuritis, each disc being now swollen not more than one diopter. The margins are beginning to appear quite clearly on the temporal sides. In the right eye they have disappeared. While there are still homonymous losses of the visual fields upon the left sides, there has also been a gain in this respect, particularly of the left eye, which now in some meridians is not far from the normal boundaries."

On May 8 Dr. de Schweinitz further reported as follows:

"I examined your patient yesterday, and found the conditions the same as when I reported to you last—practically full field on the right side, with partial hemianopsia upon the left, central vision sharply normal, no hemorrhages of any kind in the retina, and only a very slight haziness of the nasal margins of the disc to suggest the former choked disc" (Fig. 4).

The operation was performed by Dr. Charles H. Frazier on February 5.

Surgical Memorandum.

The technique and method of procedure differs in no respect from the routine which we have adopted in the clinic of the University Hospital. The following is a brief description of the operation: Under nitrous oxide ether anesthesia preceded by the administration of one-sixth of a grain of morphine and one hundredth of a grain of atropine the patient was placed upon the operating chair in the erect posture. The incision was made so as to expose portions of the occipital, parietal and temporal lobes. The flap was so fashioned that its base was directed towards the temporal region, the superior margin being one inch from the median line, the anterior margin one and a half inches anterior and the posterior margin two inches posterior to the parieto-occipital fissure. The bone was sectioned with the spiral osteotome and the osteoplastic flap reflected. As soon as the dural flap was reflected the brain at once bulged considerably through the opening, thus assuring us that there was a decided increase of intracranial pressure, the cause for which we at once began to investigate. Upon palpating the exposed surface of the brain an area was discovered in the superior posterior angle of the opening, which, compared with the normal brain structure, seemed much softer if not cystic. It was noted furthermore that in the same region the surface was of a different color. The region thus described did not exceed that of a twenty-five-cent piece and its margins were so sharply defined that it was not difficult to differ-

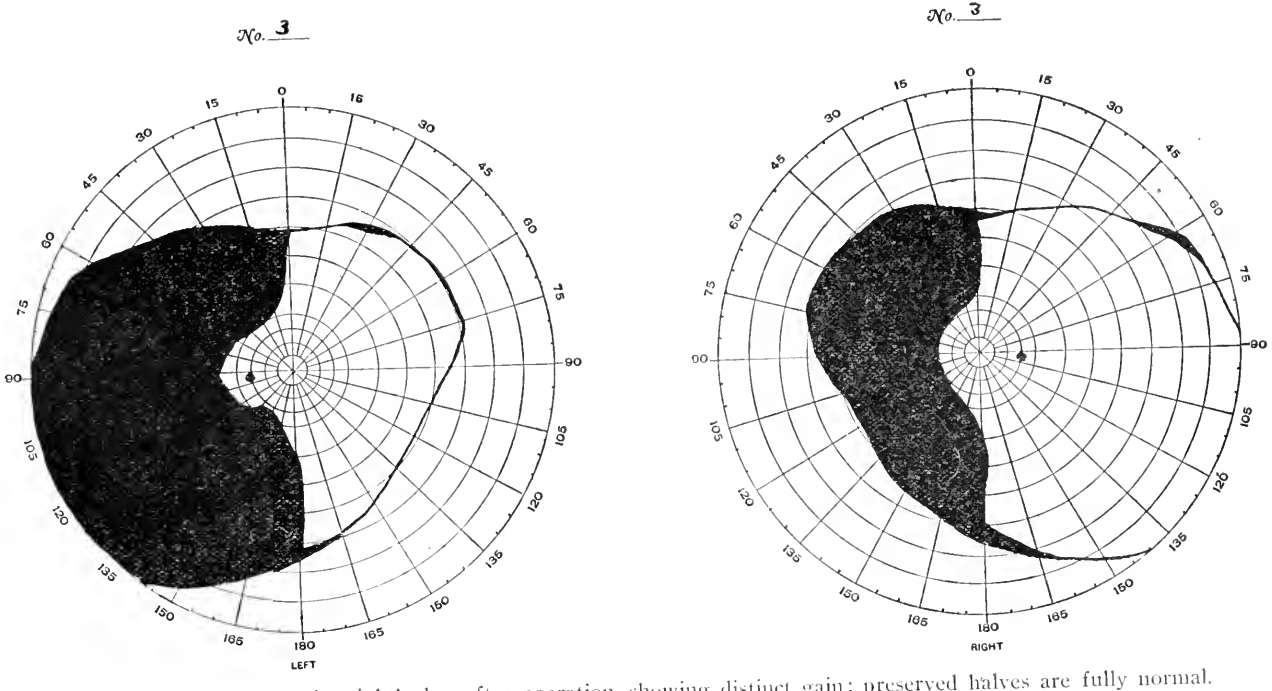


FIG. 3. Visual fields on the eighth day after operation, showing distinct gain; preserved halves are fully normal.

entiate the normal from the abnormal brain tissue. The line of demarcation was much more distinct after the pia mater had been peeled off. With the handle of the scalpel the line of cleavage between the tumor and surrounding structure was easily found, and we at once proceeded to enucleate. While thus engaged there was a sudden spurt of clear straw color fluid and the tumor was now found to be a cyst collapsed. One finger was inserted into the cavity of the cyst as a guide and with but little bleeding the entire cyst wall was removed intact. During this process there was a progressive fall of blood pressure and the table was lowered, changing the position of the patient from the vertical to the horizontal posture. A small rubber tube was introduced into the residual cavity in order to prevent the retention of blood and a possible cyst formation. The dural wound was closed with interrupted silk and the scalp with silk-worm gut sutures. A small groove was cut in the skull just to the median side of the superior margin of the flap and an opening in the scalp made to provide a means of exit for the drainage tube.

From the time the operation began until the bone flap was reflected six or seven minutes had elapsed; within ten minutes the dural flap had been reflected; five minutes more were occupied in determining the seat and margins of the lesion and fifteen minutes for enucleating it. Thus with the exception of closing the wound in the dura and scalp the operation was completed in thirty minutes.

The condition of the patient at no time gave us any cause for apprehension. By the time she had been taken to her room she had recovered consciousness sufficiently to recognize her physician and answer questions intelligently. Her excellent condition at the conclusion of the operation was due in part to the short duration of the operation, to the avoidance of exposure, and to the skill and care with which the anesthetic was administered. In Fig. 5 is shown a photograph of the patient taken four days after operation, showing the size and position of flap. It is needless to call attention to the fact that disregard to certain precautionary measures will have a deleterious effect upon the patient and may lead to a fatal issue. The advantage of conducting the operation expeditiously in a reasonably short time need not be dwelt upon, nor need we emphasize the importance of conserving body temperature, by having the patient warmly clad and last but not least of entrusting the ether always to a trained skilled anesthetist.

The operation was perfomed at a single sitting. We have never been advocates of the two step operation, so popular with some surgeons abroad because experience has shown that the actual removal of a brain tumor has little if any harmful influence, as indicated by the pulse or blood pressure. With this case the question of postponing the removal of the cyst to a

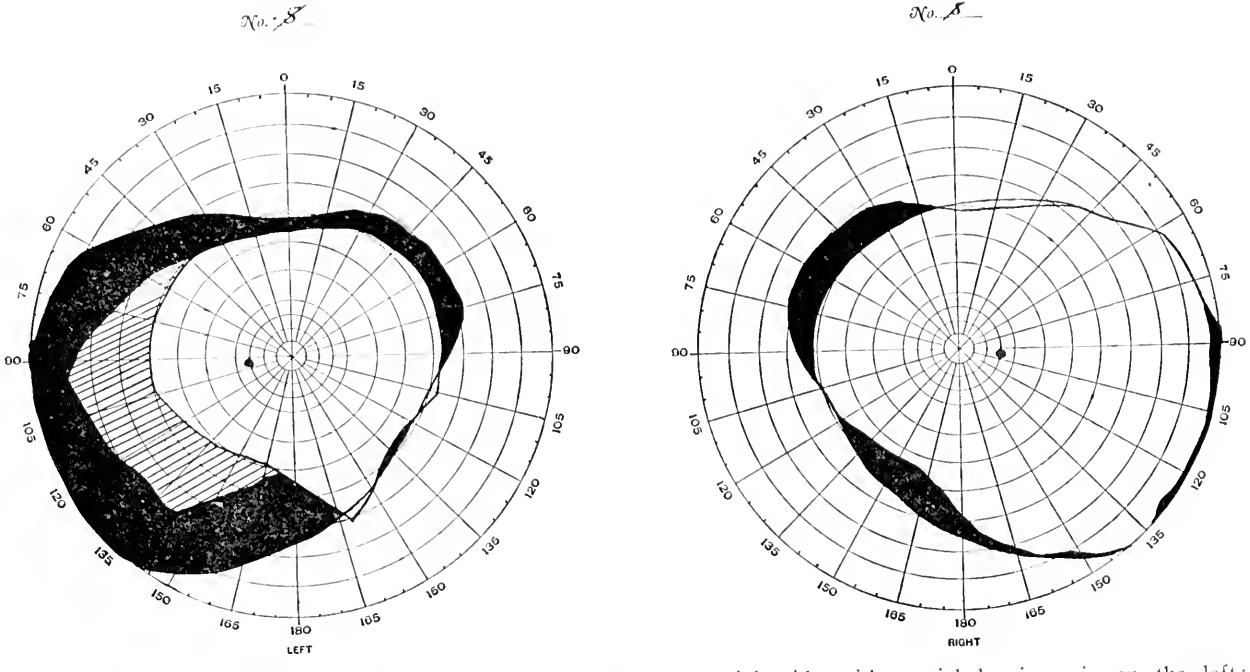


FIG. 4. Visual fields, May 7, 1908. Practically full field on the right side with partial hemianopsia on the left; cross-hatching shows dim vision; central vision sharply normal, right eye, 6/6; left eye, 6/5.

second sitting was not considered. The additional risk of a second anesthetization and of infection must be reckoned with in the two-step procedure. To be sure there are exceptional instances, particularly in cerebellar lesions, in which by the time the lesion has been discovered the condition of the patient may be such as to make it advisable not to proceed.

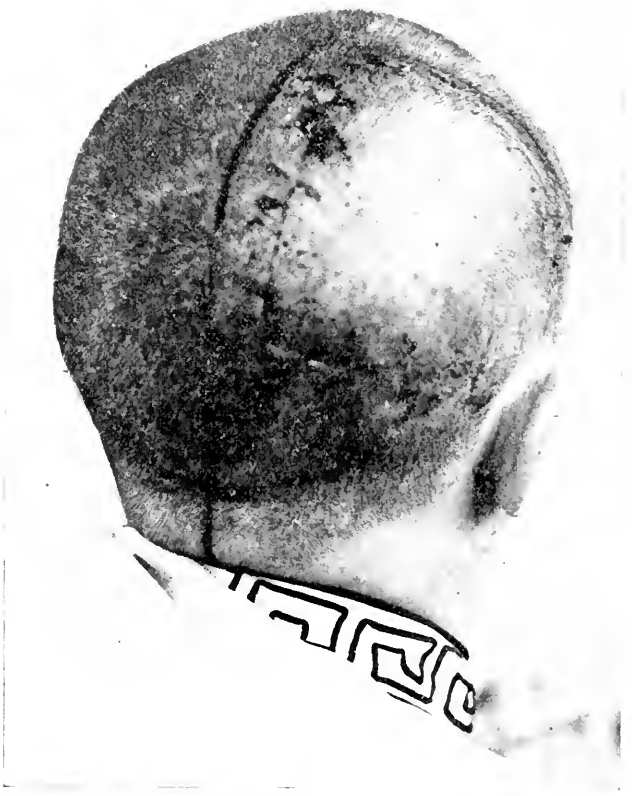


FIG. 5. Photograph of patient taken four days after operation, showing size and position of flap.

The cyst, when filled with fluid, measured eight centimeters in length and four in diameter (Fig. 6.) Its shape was not unlike that of an egg and the greater portion of it was subcortical. Fortunately a small portion of it, what might be said to be one pole, presented on the surface; otherwise it might have escaped detection. While the cyst appeared in the surface

in the superior portion of the occipital lobe the greater portion of it extended into the parietal lobe, thus accounting not only for the visual disturbances but for the hemiataxia.

The report from the Laboratory of Surgical Pathology describes the cyst as follows:

The specimen consists of a thin-walled cyst measuring about 8 x 4 cm. in size. When received in the laboratory it contained a ragged rent along one of the surfaces. The outer surface was smooth, even, and of a pinkish-gray color, with a few very small vessels ramifying over the surface. The wall was generally about one or two mm. in thickness and very friable; the interior was smooth except at three places where some soft tissue was adherent to the wall, but could be peeled from the

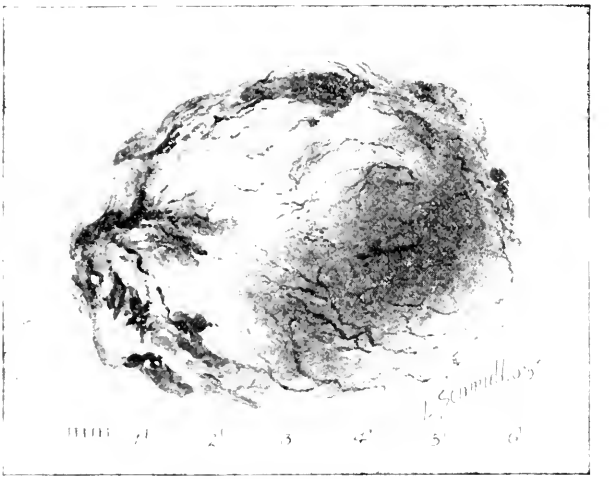


FIG. 6. Cyst, drawn to scale.

same with ease. This tissue was of a darker color than the wall. When received there was no fluid in the sac, and scrapings from the walls failed to reveal any evidence of echinococcus.

Microscopic examination of portions of the wall and of the tissue adherent to its inner wall failed to show any trace of brain tissue, or resemblance to any glioma or other tumor tissue. The wall was composed of several layers of fibrous tissue fairly rich in nuclei, and having an abundant blood supply; the inner surface was rough and ragged, while the outer layers seemed to have condensed to form the smooth outer surface. The tissue masses represented blood clot with fibrin, red cells, and pigment, and a few round cells, probably lymphocytes.

As to the pathological diagnosis there seemed to be little question from the macroscopic and microscopic features that

we were dealing with a simple serous cyst. Cysts resulting from hemorrhage and softening are much more irregular in shape and have not a well developed sac. There was not the least suspicion that we were dealing with a cyst as part of the development of a morbid growth, such as sarcoma or glioma.

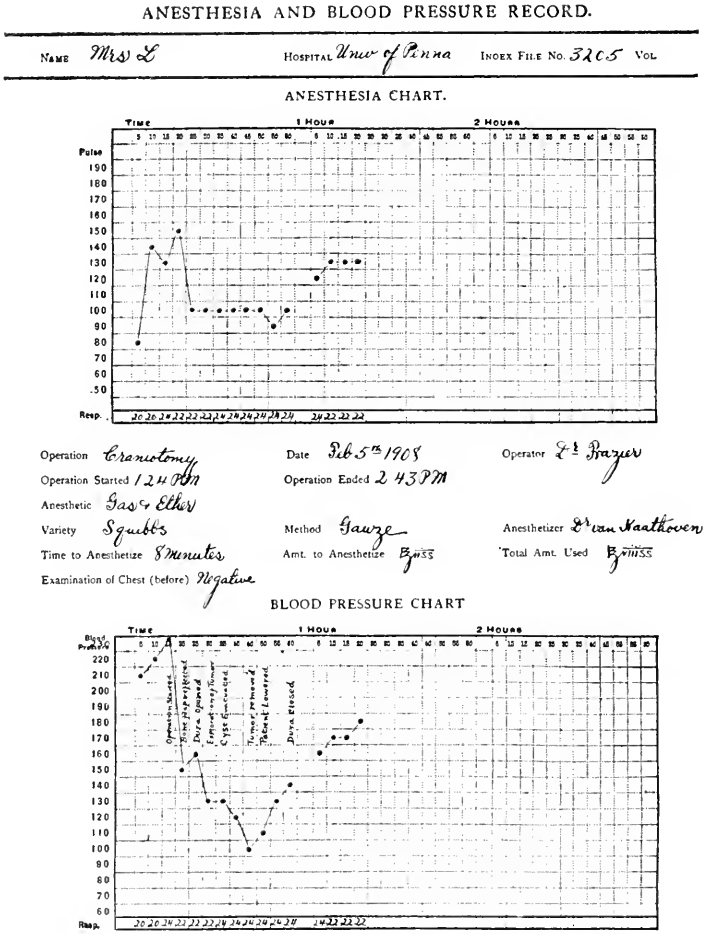


Fig. 7. Blood pressure chart, showing fall in blood pressure after removal of cyst.

The wall of the cyst throughout was composed of fibrous, not of tumor, tissue.

A word or two might be said as to the blood pressure (Fig. 7). Before the operation began the blood pressure registered

210 mm. of Hg; as soon as the flap was reflected, within five minutes, it dropped to 150, while the cyst was being removed to 100, and at the conclusion of the operation registered 180. Whether the high pressure at the beginning was due to a general arteriosclerosis or to increased intracranial tension is a matter of conjecture. On two occasions during the convalescent period the systolic pressure was 180 and 140 respectively. We are rather disposed to believe that in this case the intracranial tension was an influential factor, although in numerous and repeated observations in other cases of brain tumor or internal hydrocephalus the blood pressure has not been appreciably disturbed. When the pressure had fallen from 210 to 100 mm. of Hg during the course of the operation the position of the patient was changed from the vertical to the horizontal posture. This we have found almost invariably has a favorable influence upon the circulation and in this case the pressure at once began to rise until at the conclusion of the operation it registered 180. With the opportunity to observe the pressure throughout the operation we have no hesitation in placing the patient in the vertical posture. Apart from the fact that this position is more convenient for the operator it is very efficient as a means of controlling hemorrhage.

The recovery of the patient from the effects of the operation was prompt; during the convalescence the temperature did not rise above 99.4° F., nor the pulse above 96 (Fig. 8). The patient was sitting up in bed on the third and walking about her room on the fourth day, and with the removal of the stitches on the fifth day was concluded a brief but interesting surgical experience.

While the rapidity with which the signs of disturbed function disappeared constitutes one of the most striking features of the case, the absence of any immediate aggravation of symptoms was equally noteworthy. In many instances the trauma to the brain tissue, incidental to the removal of a growth, causes a transitory disturbance of function in the structure adjacent to the lesion. This is more particularly the case when the tumor is situated in the motor cortex. In a case recently operated upon, following the removal of a tumor the size of a hickory nut from the motor region, even though the growth was sharply defined and easily enucleated, the patient had a complete hemiplegia.

From the standpoint of the surgeon this case was of especial interest because it presented a benign, accessible and operable lesion. Perhaps the most discouraging feature of the surgery of brain tumors is the comparative infrequency of operable tumors. According to Duret's frequently quoted statistics, based

on a series of 344 cases, but 10 per cent. were said to be operable and of these in two thirds the seat of the tumor was not determined: thus reducing the percentage of operable cases to 6.5 per cent. Many of the statistical tables, however, are based upon necropsy findings, when the tumor is far enough advanced to take the patient's life. This "autopsy method" of determining the operability of tumors, as we have had occasion to say before, is

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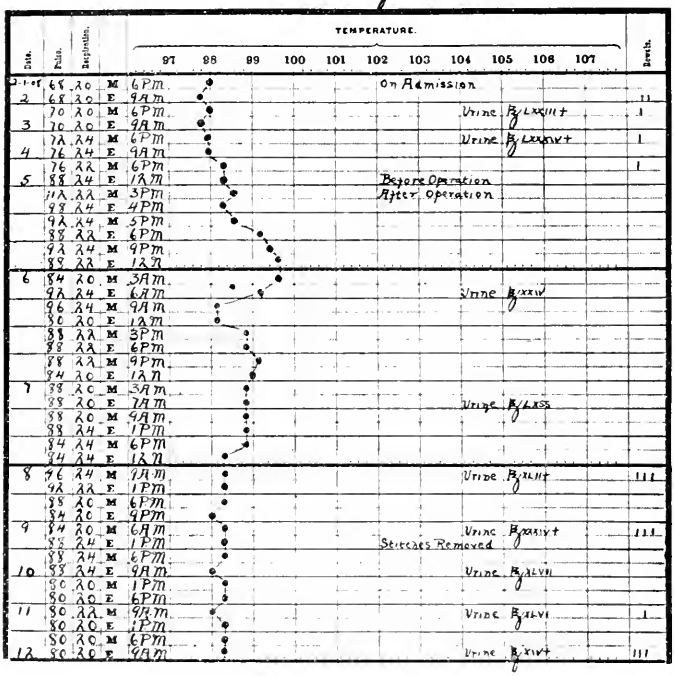

FIG. 8. Temperature chart, showing how slight the effects of the operation were.

open to serious objection, and while the percentage of tumors suitable for radical operation will always be a small fraction of the whole, the reckoning should be made from the statistics of the surgical clinic and not of the pathological laboratory. In the clinic of the Hospital of the University of Pennsylvania we have had all told, nine cases of operable tumors; six of

the cerebrum and three of the cerebellum. These represent approximately 20 per cent. of our series of tumor cases. While the decompressive operation is wonderfully effective in palliating symptoms and prolonging life, and some of the results of the radical procedure appear discouraging, no case should be branded inoperable until so proved by a thorough exploration through a liberal opening. Only by early diagnoses and by a resort without procrastination to well-executed craniotomies, even though there may be some doubt as to the precise location of the tumor, can we determine whether or not we are justified in relapsing into that state of pessimism which characterized the attitude of the profession not many years ago.

Examinations of the patient were made with the view of determining the effects of the operation and removal of the cyst upon her nervous system from time to time during her stay at the hospital. On February 8, three days after the operation, a note was made that the ataxia was fast disappearing and that the patient's mental condition, although never dull to the extent of being seriously affected, was becoming much brighter. On February 12 the record was made that all functions of the arm and leg were restored. The ataxia had disappeared. Testing for astereognosis, the sense of location and position, and cutaneous sensibility the results were negative. Motor functions were normal in both arms. The patient seemed to have a little less voluntary control of the left side of the face, but she stated that this had always been the case. Hemianopsia was much less marked as was shown by various tests. On the fifteenth the affected limbs appeared to be entirely normal, and the patient evidently saw much better. She was able at this time to detect much smaller objects about her room.

The examinations of Dr. de Schweinitz, already detailed, give more explicitly the facts as to her eyes and vision.

The completeness of the removal in this case of a large parieto-occipital cyst, and the entire disappearance of the severe and threatening symptoms produced by it, entitle it to be regarded as one not merely of palliation but of cure. Apparently the cyst was benignant in character. We have seen the patient within two weeks of the presentation of this report. In every particular she was in excellent health, being with the exception of the very partial hemianopsia, free from signs of cerebral disease.

From the standpoint of physiology, especially as regards the functions of the parietal lobe and the possibility, in considering lesions of this lobe, of subdividing it functionally into several parts, the case also is of much interest.

We have had the opportunity of studying at least four classes of cases, giving different symptom complexes, according to the degree of implication of the various portions of the parietal lobe. Possibly in time more than this number of syndromes can be separated for diagnostic purposes. These symptom complexes are: (1) Pronounced hemianopsia and ataxia, combined with pressure symptoms varying in intensity, such as hypesthesia, hypastereognosis, and slight paresis of the face and limbs; (2) astereognosis and ataxia, combined with symptoms showing various degrees of involvement of cutaneous, muscular, and arthroidal sensibility, but without hemianopsia and with no or only slight paresis; (3) hemianopsia and hemiataxia, with hypesthesia and hypastereognosis and pronounced paresis, especially of the face and upper extremity; and (4) astereognosis and ataxia, with hypesthesia and pronounced paralysis, especially in the lower and upper extremities. These symptom complexes, more or less pure at first, become complicated as the tumor or cyst increases, and with it the variety and extent of the symptomatology. From cases already recorded, illustration of each of these symptom groups might be given.

In considering the question of the position and extent of an opening for the removal of a tumor or cyst, the osteoplastic flap should be planned somewhat differently in accordance with these symptom complexes. For the first, in which pronounced hemianopsia and hemiataxia are the dominating symptoms, the incision for an opening three and a half or four inches in length for its superior boundary should be made, about one inch from the mesal edge of the hemisphere and in such manner that about one half of it should be cephalad and the other caudad, of the line of the parieto-occipital fissure. The base line connected by the somewhat converging sides of the opening should be over the upper part of the temporal lobe. With the second symptom complex in view—that in which astereognosis and ataxia, without hemianopsia, are the chief guiding symptoms—the opening should be carried as close as possible to the middle line of the skull, and should be an inch more anterior. The incision for the upper line

of the third opening, for a case giving the syndrome in which hemiataxia, sometimes with hemianopsia and also with hypesthesia, impairment of the muscular sense and hemiparesis or hemiparalysis, especially in the face, are present, should extend about one third in front and two thirds behind the central fissure, and about one inch or one and one half inches from the median line, the base, as in the first opening, being over the temporal lobe. The incision for the upper limit of the fourth opening should be as near as possible to the mid-line of the skull, while its sides should be in about the same relative positions to the central fissure, that is so as to uncover the cerebral surface about one third in front and two thirds behind this fissure. By having the lines of the opening in the manner here indicated, the surgeon will probably find the main portion of the growth near the center of the field of operation.

HERPES OF THE MEMBRANA TYMPANI: DUE TO ZOSTEROID AFFECTION OF THE PETROSAL GANGLION

By Thomas J. Orbison, M.D.,

of Los Angeles, Cal.

The infectious nature of herpes zoster (zona, zosteroid) is accepted by competent observers. That the location of the responsible lesion is in the cells of the spinal ganglion type is likewise accepted.

The various syndromes attached to the zosteroid infection of the different ganglia have been carefully worked out in many instances. One of the most recent is that by J. Ramsay Hunt in an interesting paper presented at the meeting of the American Neurological Association in 1906, and published in the Journal of Nervous and Mental Disease, February, 1907. Hunt's paper dealt with herpetic inflammation of the geniculate ganglion; its basis was a series of sixty cases, all of which had a facial palsy of the peripheral type in addition to the zoster. The deductions as to the zoster-zone of the geniculate included all portions of the auricle. The membrana tympani seemed to be tacitly included in this zone, but the case to be recorded below would seem to warrant the substitution of the petrosal for the geniculate.

The basis for the deductions as to the zone of the geniculate rested upon a mapping-out of the areas of anesthesia subsequent to the extirpation of the Gasserian ganglion for the relief of tic douloureux, in the cases of Spiller and Frazier, and of the second and third cervical ganglia in a case of Harvey Cushing.

The zoster zone of the petrosal ganglion has not, heretofore, been mentioned, as far as I can find. The petrosal ganglion of the glossopharyngeal nerve plays the same role as the geniculate on the seventh. It supplies the membrana tympani by means of the tympanic plexus which sends branches over the promontorium lying just posterior to the membrane. These reach the "stratum mucosum," or posterior layer of the membrane by many filaments that come in from both sides and spread in a fine plexus

throughout it (the anterior layer, "stratum cutaneum," is supplied in part by the auriculo-temporal).

The relation of near-by parts is as follows: The geniculate ganglion lies above, posterior to and separated from the membrana tympani by the bony septum that separates the canal of the facial nerve from the tympanic cavity. It is (comparatively) remote from the tympanic plexus and connected with it by the fine rami anastomotici. Any affection of the geniculate must needs be extensive to reach down to the tympanic plexus—extensive enough to involve the facial, certainly, as was the case in all of Hunt's series. The chorda tympani nerve passes just posterior to the upper edge of the membrana, but does not supply it; it passes, in this location, through the tympanic plexus, but gives no branches to it. The lesser petrosal nerve comes over from the otic ganglion and is intimately connected with the tympanic plexus at a point above the promontorium. The promontorium lies just back of the membrana and is exposed when the membrana is removed.

The Ganglion Petrosum.—Chas. K. Mills in his authoritative work, "The Nervous System and Its Diseases," says on page 103: "Various well-known cranial ganglia correspond morphologically and functionally to the ganglia of the dorsal spinal roots—the petrous and jugular for the glossopharyngeal," etc. The functions of the former have to do (vide supra) with the membrana tympani. The branches that spread over the posterior surface of the membrana come from it. Inflammation of the petrosal ganglion would extend, logically, to the membrana tympani. An herpetic inflammation would exhibit its typical lesion or lesions on and posteriorly to the membrana. Thus we have mapped out for us the herpetic zone of the petrosal ganglion.

Anatomy of the Membrana Tympani.—It is a very thin, smooth membrane stretched out between the external acoustic canal and the cavity of the tympanum. It is fastened by a somewhat thickened margin in the sulcus tympanicus by means of a fibrocartilaginous circular pad and is at that point sharply marked off from its surroundings. It is covered on its lateral surface by the "stratum cutaneum," a continuation of the external skin. Following upon this is the "stratum radiatum"—a connective tissue membrane in which the fibers run radial to the handle of

the malleus; likewise a "stratum circulare" in which the fibers are concentrically arranged. These last two layers are connected to the fibrocartilaginous ring. The inner surface is the "stratum mucosum"—a continuation of the mucous membrane of the tympanic cavity. It is in this layer that the branches (rami tubæ) of the tympanic plexus ramify.

Rarity of Herpetic Inflammation of the Auricle.—Hunt has examined the records of the Manhattan Eye and Ear Dispensary of New York—47,600 cases in the past ten years; the Brooklyn Eye and Ear Hospital—15,000 cases in the last five years; the New York Eye and Ear Infirmary for the last twenty-three years—averaging 10,000 cases yearly; in all of these the diagnosis of herpes of the auricle was made only nine times. In the Massachusetts Eye and Ear Infirmary the diagnosis was made thirty-three times in 65,000 cases. Admitting that the condition may have existed and was not diagnosed as herpes, nevertheless it will be seen at a glance that the affection is a very rare one. As for herpes of the membrana tympani—it is well-nigh unheard of. Pollitzer says of it that Hartmann saw it once. Hunt speaks of it in this wise—the typical eruptions are situated "on the concha, on the lobule, the tragus, the marginal portions of the auricle (helix and antihelix) and within the auditory canal—indeed, as rarely happens, on the *membrana tympani* itself" (the italics are his).

Record of a Case of Zosteroid Herpes of the Membrana Tympani.—Miss C. is of a long-lived, healthy family. A point of great interest in her family history is that her brother had herpes zoster when he was a young man. Another point to be reckoned with is that gouty and rheumatic affections have been the prevailing family ills. As a child she was inclined to be nervous; was never robust but never ill. As a young girl she was healthy. As she grew older she was often troubled by stiffness in various joints; this was especially marked in the mornings and they would be "limbered up" by exercise. As she has advanced in years she has had rheumatic attacks that at times were severe, *e. g.*, her hands have often become incapacitated, due to painful joints.

During the last three or four years she has often had zosteroid eruptions along the course of various nerves. Her brother, who was a well-known physician in New York City before he came to California, kept a careful watch over the condition and

can vouch for it. In addition, she has had gouty attacks that have been controlled by colchicum.

In November, 1906, her present illness began with a painful throat and naso-pharynx—a typical gouty condition. This was at once followed by severe aching pains all over her body and accompanied by anorexia and vomiting (note the infectious nature of the onset). On the third day there was earache, tinnitus and fulness in the ear on the left side; the pain was not superficial but deeply seated. The tinnitus was at times crackling in character. Deafness soon followed and the ear felt as though it were "built away out" from the head.

Dr. W. H. Roberts, of Pasadena, was called in and his notes of the aural examination showed a "bleb directly on the membrana tympani and beginning on the lower left quadrant posteriorly." He noted that the examination gave but little pain as the aural speculum was manipulated. The bleb soon covered the whole drum and the appearance of it was as though it were a rounded crystal. It remained intact three days and was associated with deep tenderness back in the ear. At the end of that time a watery liquid escaped during the night. Since then the deafness has entirely gone. There has been no more herpes of the membrana tympani, but there have been eruptions elsewhere, *e. g.,* in the mouth on the palatine and alveolar regions. During the whole illness there has been a polyneuralgia that has been persistent and most annoying. Other unpleasant symptoms have been mental depression and general neurasthenic condition. I was called in consultation by Dr. Roberts and made the diagnosis of herpes of the membrana tympani due to a zosteroid infection of one of the cranial ganglia; probably the petrous, on the glossopharyngeal—plus neurasthenia and polyneuralgia.

Conclusions.—The petrosal ganglion is of the spinal ganglion type (Mills). Herpes zoster (zona, zosteroid) is accepted as an infectious inflammation of ganglia of the spinal ganglion type. These ganglia have their "zoster zones." The "zoster zone" of the petrosal could be either (*a*) widespread (to include its outermost branches); (*b*) intercalated between similar inflammations of neighboring ganglia of like type (geniculate, jugular, Gasserian, etc.); or (*c*) confined to its own principal branches.

The principal end branches of the petrosal ganglion are those that intersect the membrana tympani after they leave the promontorium, which is just back of the membrana and is exposed when the membrana is removed.

In my case the herpetic exhibition was at first limited to and on the posterior surface of the membrana tympani and was,

therefore, an exhibition of an herpetic inflammation of the main branches of the petrosal ganglion (later, other spinal ganglia were affected). I say it was limited because the evidences were wanting that would have implicated the geniculate; there was no involvement of the facial nerve, chorda tympani or pars intermedia of Wrisberg; nor were any other ganglia convicted of implication, *e. g.*, there was no involvement of the auriculo-temporal nerve; affection of the otic or of the sphenopalatine ganglion would have had to be severe to reach over to the tympanic plexus via the lesser and greater petrosals respectively; such an extensive involvement would have reached the geniculate as well; it was not reached in my case, therefore they may be dismissed.

The condition of herpes zoster (zona, zosteroid) of the membrana tympani is so rare that its rarity alone is of interest. There is an added and much wider basis of interest in its study, inasmuch as cumulative evidence may help to map out the various ganglia zones more clearly, thus adding to knowledge. As to the "zoster zone" of the geniculate ganglion as depicted in Hunt's paper it would seem as though it were hardly sufficiently proven. The fact that the extirpation of the Gasserian ganglion—or severance of the nerve centrally to the ganglion—left intact the sensation in the auricle would not necessarily prove that the geniculate ganglion was the avenue of that sensation. According to Hunt (or, better, accepted by him) the auriculotemporal nerve fibers do not pass through the inferior division of the fifth to the Gasserian ganglion and the question is asked "how do they reach the brain?" His answer is that they must do so via the geniculate ganglion. But is that the only course? He suggests another possibility, *i. e.*, they might pass to the otic ganglion and thence to the geniculate via the lesser petrosal nerve; the lesser petrosal is connected with the geniculate ganglion, but it is even more intimately connected with the petrosal ganglion of the ninth cranial nerve by means of the tympanic plexus.

Another suggestion is that afferent stimuli might return via the auricular branches of the tenth; but would not sensation travel by way of the jugular ganglion of the tenth rather than by the geniculate, which is a more round-about way? In other words, the zoster zone that is ascribed to the geniculate could be explained by intercalating the zones of other ganglia—the petrosal ganglion being responsible for the membrana tympani, etc.

In all of the sixty cases in Hunt's series there was a facial palsy of the peripheral type which was construed to mean that the geniculate was the ganglion at fault and that the herpetic exhibition measured the zone of the geniculate inflammation. Would it not be just as logical (in the absence of positive proofs) to explain them as being due to an extension of inflammation *to* the geniculate *from* other nearby ganglia or by the well-known frequency of the implication of the neighboring ganglia in herpetic inflammations—the geniculate being involved synchronously with others? Likewise, the facial palsy might be a separate affection that came on as a result of the same cause that produced the herpes; "facial palsy is the commonest form of paralysis" (Mills).

In both of the two cases described in Hunt's paper this might seem to be the case. In the first case the exciting cause was an hour's exposure of an elderly woman to a winter storm (a sufficiently severe cause to account for both affections). The second case was a periodic drinker who had a facial palsy following an exhibition of zoster that had attacked the occipito-cervical area. A discussion of the merits or demerits of the various ganglia that are responsible for the several zoster zones in literature is academic rather than scientific in character, inasmuch as the positive proofs of a sufficient number of autopsies are wanting. In the second of Hunt's cases the geniculate ganglion was, unfortunately, not able to be examined; there is no record of nearby ganglia other than the Gasserian, which was normal. The "pars intermedia of Wrisberg" on the affected side was found to be sclerotic. This nerve has its origin in the geniculate ganglion (Mills). A sclerotic condition of the nerve would not, however, prove an herpetic inflammation of the ganglion.

In my case the herpetic blebs that appeared in the mouth—on the palatine and alveolar regions—were explained by an extension along the ninth nerve from its petrosal ganglion (the primary seat of infection) or by an extension to the spheno-palatine ganglion along the vidian via its branches, the greater superficial and deep petrosals. Finally the condition of zosteroid in my case was one that was superimposed in a severe neurasthenic condition that was itself due to a long-continued seige of domestic cares, worries and anxieties. Its course was long. It varied as did

the nervous state of the patient. It has seemed to be of an infectious nature, chronic in its course and dependent upon the lowered tone of the individual; a subject that was primarily gouty and neurotic.

I wish to express my warmest thanks to Dr. Chas. K. Mills, of Philadelphia, for his counsel relative to the above paper.

TABES ASSOCIATED WITH TROPHIC CHANGES SUGGESTING ACROMEGALY.[1]

By F. X. Dercum, M.D.

PROFESSOR OF NERVOUS AND MENTAL DISEASES, JEFFERSON MEDICAL COLLEGE; NEUROLOGIST TO THE PHILADELPHIA HOSPITAL.

The following case, because of the trophic changes present, is of more than usual interest.

H. B., white, age 36, was admitted to the Philadelphia General Hospital, May 29, 1906. He was a stone cutter by occupation.

Family History.—The family history was negative. Father died at the age of 75; mother at the age of 74. One brother and two sisters, living and well.

Personal History.—Had none of the diseases of childhood nor any other affection of moment until twelve years ago when he contracted syphilis. Skin lesions were present subsequently.

Present Illness.—His present illness began about six years ago with shooting pains in both legs and with numbness in both feet. Shortly afterwards he began to stagger when walking and also suffered from weakness in the legs. Three years ago he was obliged to give up his work.

Condition upon Admission.—Station extremely ataxic. Gait ataxic. Is unable to walk with eyes closed. Knee jerks absent. No ankle clonus. No Babinski sign. Sensation is delayed upon both lower extremities, more on the right. There is present also some ataxia in the arms though the reflexes are normal. There is no facial inequality; no trouble in eating or swallowing; the tongue is protruded in the median line and the angles of the mouth are retracted equally well. Both pupils are widely dilated and equal. They react to accommodation but not to light. The extra-ocular muscles are apparently normal. There is no ptosis.

The patient gave his height as five feet, eleven inches. He adds also that while young he grew very fast, and was already very tall at the age of sixteen.

[1] Read by title at the Thirty-fourth Annual Meeting of the American Neurological Association, May 20, 21 and 22, 1908.

Reëxamined, September 6, 1906, he complained of girdle sensations and also of marked constipation. There was also some difficulty in voiding urine. The biceps tendon jerks had disappeared. An ophthalmoscopic examination did not reveal any optic atrophy. Vision was good but he was unable to read for any length of time.

Reëxamined, May 6, 1907, his condition was about the same, save that gastric crises had made their appearance. At this time there were no arthropathies. Paresthesia, numbness and burning in legs and feet were complained of and tactile anesthesia was noted in both lower extremities below the knees. Pinching was felt as touch but not as pain. Further, when the toes were passively flexed or extended, the patient could not tell their position. The sense of pressure was much diminished in both legs; the patient's answers during these tests were very uncertain also as regards sensation in the hands and forearms.

An ophthalmoscopic examination, August 16, 1907, revealed the vessels to be rather small in caliber but the fundus was otherwise clear and normal. At this time also tubercle bacilli were noted in both urine and feces.

Reëxamined, October 17, 1907, symptoms suggesting acromegaly were noted. The countenance of the patient seemed to have become lengthened. The chin was enlarged and protruding, the nose was prominent and large, as were also the zygomatic arches and the occipital protuberance. The enlargement of the latter was especially pronounced.

The hands and wrists were now also noted to be enlarged. In the right wrist, for instance, the ulna was noted as of unusual thickness and breadth. This was also true of the distal portions of the metacarpal bones and the proximal and middle phalanges of the fingers. Similar changes were noted in the left wrist. Both of the feet were flattened and unusually broad, particularly was this true of the right foot. The tarsus was unusually thickened and also the proximal ends of the metatarsal bones. Both heels were much enlarged, projecting backward to a marked degree. The deformity was greater in the right foot. (The patient stated that some time ago he had received an injury of the right foot, but that it had caused him no pain.) The internal condyle of the right knee was greatly enlarged as was also that of the left, though to a less degree. An examination of the knee

joints failed to reveal changes of the joint surfaces such as are commonly found in arthropathy. The joints were apparently normal, no roughness was present nor was there any effusion. The malleoli of both feet were very large, especially those of the right foot. The lower portions of the tibiæ were also much enlarged.

The left arm could not be fully extended, due to a deformity of the elbow. This the patient attributed to a fracture of the arm occurring in childhood. In the right there is marked enlargement of the internal condyle. The acromial end of the clavicle is much enlarged upon both sides, more so upon the right. The patient is much emaciated and the spinal column can be thoroughly examined. The spinous processes appear to be thickened. The ribs also are broad and thickened. There is also some dorsal curvature though the patient states that he was already somewhat bowed when a young man.

The patient states that about the time the pain in his legs began, the teeth in the upper jaw became loose and dropped out one by one. He lost all of the molars and one of the pre-molars in the left lower jaw and two of the molars, one of the pre-molars and one of the incisors in the right lower jaw. He would frequently pick out teeth when they were loose without pain.

There was also present at this examination very marked hypotonia and marked anesthesia of both lower extremities as far as upper thirds of thighs. In the hands and forearms, decided sensory loss was noted as was also sensory retardation. At this examination also dulness is noted over both apices and over the bases of both lungs.

Reëxamination of the eyes, October 22, 1907, revealed the pupils as before markedly dilated. They were as before typically Argyll-Robertson. The optic nerves showed distinct narrowing of arteries and the presence of white lines around their borders. Nerves were of good color but their appearance denoted a beginning atrophy.

December 19, 1907, it is noticed that the patient is very weak and can hardly speak. Swallowing also is quite difficult. His condition did not improve subsequently; he became gradually weaker, lost rapidly in flesh and died December 21, 1907.

Autopsy, December 22, 1907.—The general autopsy revealed tuberculosis of the lungs, chronic myocarditis and interstitial nephritis.

The brain and spinal cord were removed for examination as were also the thyroid gland, the pituitary body and both suprarenal bodies.

Hypophysis.—The hypophysis is fully twice, if not more, as large as normal. The capsule seems thicker than usual and is covered by a layer of endothelial cells. The vascularity seems somewhat increased but the vessels are normal in appearance. In the organ the vessels are normal but here and there some of the smaller ones seem to be surrounded by a thin layer of homogeneous material, probably colloid substance. The capillaries are much dilated and engorged with blood. In some areas the cell-chains are quite long. The cells themselves are quite distinct, on the whole, and respond to the stains. Some are fragmented and apparently degenerated, but these are not numerous. Several tubules are to be seen, and these contain colloid material. Along the periphery the chromatin cells are quite large and stain deeply, at times showing lighter areas in the protoplasm. These cells are both scattered and arranged in groups, but all in all they do not seem to be as numerous as they should. Such changes as appear to be present are noted in the glandular portion.

Adrenal.—Although the adrenals are larger than normal, especially the left, no marked changes are noticed save that the cells of the zona fasciculata seem to have undergone an unusual degree of fatty change, the cells having practically disappeared. The other zones appear normal. The right adrenal shows the same condition.

Thyroid Gland.—The thyroid, save for an apparent increase here and there in the connective stroma reveals no particular change. The acini vary greatly in size, some of them being excessively large. They are lined by the normal cubical epithelium. The walls show no tendency to plication and they are well filled with colloid material.

The Nervous System.—Sections of all three major portions of the cord show marked sclerosis of the posterior columns; some of the fibers along the septum and surface are still intact, while along the posterior cornua and gray commissure quite a number are unaffected.

In the present case, the trophic changes noted can hardly be regarded as having been typical of tabes. There were no arthropathies properly speaking; no changes in the synovial membranes,

ligaments or cartilages, no effusion into the joints. The changes consisted in an enlargement of various prominences, of numerous bony structures and especially of the distal portions of all four extremities. They were, in short, such changes as are commonly noted in acromegaly. In this connection the findings are exceedingly interesting. In the nervous system they are evidently those of ordinary tabes. The changes in the ductless glands, while not pronounced, are nothwithstanding of special interest. The pituitary body was unquestionably enlarged, though this enlargement was far less than that met with in acromegaly ordinarily. However, such changes as were present were hypertrophic in character and were observed in the glandular portion. The changes in the adrenals consist apparently of simple hypertrophy with fatty change in the zone fasciculata. As far as the thyroid is concerned, definite changes can hardly be claimed other than some increase in the connective tissue elements of the organ.

The findings in this case are exceedingly suggestive. It is not improbable that the changes in the pituitary body bear some relation to the bony changes. The thought suggests itself that perhaps in cases of tabes generally, where there are marked trophic changes in the bones, there are also changes of the pituitary body, and it may be wise to examine the pituitary body and the other ductless glands in such cases. The internal secretions are probably destined to play an increasing rôle not only in general pathology but also in nervous pathology. Perhaps we have here a hint also for future study in other affections.

Society Proceedings

THE NEW YORK NEUROLOGICAL SOCIETY

March 3, 1908

The President, Dr. B. Sachs, in the Chair.

A CASE OF TUMOR OF THE BRAIN

By E. G. Zabriskie, M.D.

The patient was a married woman, forty-five years old, who was first seen with Dr. Post, on July 6, 1907. She gave a history of having had several miscarriages of not more than six weeks duration.

Three years ago, while apparently in excellent health and after a day's washing, she went into the dining-room and was looking over the evening paper when she suddenly became dizzy and felt as though she were going to faint. At the same time she experienced a numbness in the left foot, as though it had gone to sleep. On attempting to stand, the leg gave way under her, and she fell backwards. She was quite positive that she did not lose consciousness, and stated that she was in her right mind throughout the attack, which lasted altogether about three minutes. Her son tried to give her some brandy, but she could not swallow it and by this time felt all right and got up. Since then she had repeated attacks of similar nature. A few months afterwards, the exact date not being quite clear, she had had her first convulsive attack, when she first consulted Dr. Post, early in 1907, she was having them about twice a week. They never lasted more than three minutes. Shortly before their onset she had a constant desire to look backwards, as though someone were behind her and she wanted to "shove them away." The attacks always began with a spasm or twitching of the muscles of the left foot as far as the knee, then extending up the thigh and "heart," and then to the throat, giving her a choking sensation. Recently, she had felt the spasm in the muscles of the left arm as well. She had complained of soreness in that arm, and occasionally dropped objects that she carried in that hand. After these attacks, the leg felt numb, this sensation sometimes persisting for two days after a severe attack.

The motor power of the left leg had been impaired for about a year, and for the past five or six months she had noticed weakness of the corresponding arm. Her eyesight had been growing dim for the past fourteen months, and there was a history of transient attacks of diplopia. Under the use of potassium iodide she had been gaining weight for the past six months, but prior to that she had lost. She felt nauseated at times, but never vomited. There was tinnitus of the right ear. She had no trouble in holding her water, but there was frequent micturition and a slight hesitancy of the stream at times. She said she had been subject to headache all her life, which were vertical in character until the onset

of these attacks; since then they had been occipital and bitemporal in character, and consisted of a sensation as though some one were pressing through the head from right to left.

The gait was decidedly limping, indicating a paresis of the left leg. There was no static ataxia. The reflexes were sluggish on both sides; not stronger on the left. There was normal plantar response on the right side; on the left it was difficult to obtain, but when present seemed to be of the Babinski type. The abdominal reflexes were diminished on the left side. There was slight ataxia of the left arm, with some loss of motor power in both the left arm and left leg. There was no qualitative loss of sensation on either side. There was slight spasticity in the left arm. The pupils responded normally to light and accommodation, although they were perhaps slightly sluggish. Ophthalmoscopic records made by Drs. Mittendorf and Skeele indicated a double optic neuritis. There was a slight but positive astereognosis in the left hand, and a well-marked disturbance of position and joint senses in the left arm and leg. Hearing was acute, and equal on both sides. There was no facial paresis.

Dr. John F. Erdmann, who had done the operation in the case shown by Dr. Zabriskie, said that in opening the skull he made rather a large flap, the base of which was three and a half inches in length. Upon turning back the flap, there was no pulsation of the dura, and upon incising this, the tumor was found in the region indicated. It sprang from the falx cerebri, and, contrary to expectations, was readily enucleated with but slight hemorrhage. It weighed four ounces. The woman's convalescence was uneventful, and she left the hospital on the twelfth day. Within four or five days after the removal of the tumor a gradual but marked improvement in the eyesight and in the function of the paralyzed arm was apparent. An interesting feature of the case was the immense thickness of the skull, which the speaker said he had noticed in other cases where he had done the decompression operation.

Dr. Zabriskie said that while the left arm was still somewhat ataxic, the patient had made an almost complete recovery as regards the other sensory disturbances. She was now able to distinguish coins, and the like, perfectly although single objects were occasionally wrongly named, if she were confused or tired. The Babinski sign had disappeared, and there was now no difference in the motor power between the two sides.

Dr. William M. Leszynsky said that Dr. Zabriskie was fortunate in seeing this case before any marked changes had occurred in the motor tract. In a somewhat analogous case that had come under his observation, an endothelial tumor almost as large as the specimen shown in this instance was removed from the brain. The patient made a good recovery and died eight years later from accidental coal-gas poisoning.

Dr. Arthur C. Brush, of Brooklyn, referring to the thickness of the skull in the case operated on by Dr. Erdmann, said he did not think that was a constant factor in these cases. On the contrary, in cortical and subdural tumors he thought rarefaction of the skull was the rule.

A CASE OF DOUBLE HEMIPLEGIA

By Charles E. Atwood, M.D.

The case was presented to show the Jackson hemiplegic sign after a double hemiplegia, and incidentally to call attention to one or two other points of special interest: *e. g.*, the bulbar type of dysarthria causing the limited facial palsy and retention of emotional reflex as in smiling, and

the effects of a similarly placed bilateral lesion, from a symmetrical atheroma.

The patient was a married man, 37 years old, a painter, who applied for treatment at the Vanderbilt Clinic on February 28, 1908. He was ambidextrous, working mostly with the left hand, but writing with the right hand. He had a chancre when he was was 18 years old. His wife had had several miscarriages.

In July, 1907, he had his first stroke of apoplexy, affecting the right face, arm, leg and speech. The onset was slow, after severe headache. This first paralysis cleared up gradually.

The second stroke of paralysis occurred on January 28, 1908, six months after the first one. It affected the left face, arm and leg, and was most marked in the arm. Since this second stroke he has had a pseudo-bulbar type of speech, there being dysarthria and indistinctness of speech, with nasal quality. The lower part of the face only was affected. The patient closed both eyes well, and smiled on both sides. There was greater obliteration of the left labio-facial fold than the right. The examination was conducted with some difficulty, on account of the inability to understand the patient's replies. The reflexes on both sides were increased, left more than right; the skin reflexes were apparently lost. Sensation seemed unimpaired. Oppenheim's sign was not obtained. Babinski's sign was greater on the left than on the right.

The Hughlings Jackson law regarding respiration in hemiplegics held good in this case, despite the fact that there had been a double-sided similarly placed lesion. The intercostal movements on the left or more paralyzed side, during ordinary or automatic respiration, had a greater amplitude than on the sounder side; while in volitional, *i. e.,* cerebrally controlled movements, such as might be induced when the patient was instructed to breathe deeply, the amplitude was greater on the sounder side. It was noted also that the breathing, as would be expected when the cerebral inhibitory control was withdrawn, partook of the Cheyne-Stokes character.

The patient had absolute control of his eyes. The paralysis in the leg was much less than that in the arm. Smell was apparently unaffected. The bladder was not affected. The vocal cords were reddened, thickened and did not quite approximate, but moved equally. The left palatal reflex was greater than the right.

As to whether the second shock increased the old palsy of the opposite side, by affecting the uncrossed pyramidal fibers, Dr. Atwood had been unable to determine. Apparently it did not.

The diagnosis was that of a similarly placed lesion of the two sides, due to a thrombosis of the lenticulo-striate artery, occurring first on the left side and then on the right. A point of great interest is the location of vascular atheroma so symmetrically placed.

UNUSUAL EFFECTS OF AN INJURY OF THE SPINE

By Pearce Bailey, M.D.

The patient was a young man, who, eighteen months ago, was struck in the back by a heavy ice-hook used in loading ice. The instrument was shaped something like a boat-hook, and the injury it inflicted was in the region of the second lumbar vertebra. There was an immediate loss of power in the left leg, and ten minutes later the right leg, which was now the worse of the two, became similarly affected. On the left side there

was an area of anesthesia extending down the back of the leg, and below the knee anteriorly. The paralysis had particularly affected the glutei muscles, which were much atrophied, while the rotators at the hip had apparently escaped. There was complete loss of power of the anal and vesical sphincters, without impairment of the sexual functions, the power of erection and ejaculation having been retained.

The symptoms in this case were suggestive of the Brown-Séquard distribution, although not typically so. Both knee jerks were absent. The disturbance was confined chiefly to the upper sacral segments, and the lesion was probably destructive in character. Retained sexual power, with paralyzed sphincters, was the striking feature.

Dr. Charles L. Dana did not think this patient's condition could be improved by a surgical operation.

Dr. Smith Ely Jelliffe said that if any operation were undertaken, especially with the view of restoring the functions of the bladder and rectum, it might be well to consider the work of Dr. Kilvington, of Melbourne, in connection with the intra-spinal bridging of nerve trunks in the lumbar region, nerve crossing as he has termed it (*British Medical Journal*, April 27, 1907, p. 988).

Dr. L. Pierce Clark said that while the operations that had thus far been undertaken for the betterment of these conditions were still largely theoretical, it was possible that their practical value would be demonstrated in the near future. Two of these operations had already been undertaken under his direction, the results of which were too early to report upon. The operation consisted in anastomosing the extra-dural nerve root from one spinal segment above the break to another below the break by splitting the nerve and then splicing one half of it to a similar section taken from the nerve below, in this way endeavoring to effect a plexus or a cabling to bridge over or around the gap. The same thing had been done by Kilvington in Melbourne, Australia, but he, instead of splitting the nerve, utilized the entire nerve trunk. The operation is not difficult and requires little special skill aside from that used in ordinary operations of peripheral nerve anastomosis elsewhere in the body.

A PATHOLOGICAL REPORT ON MYASTHENIA GRAVIS

By Fred. S. Mandlebaum, M.D., and Herbert L. Celler, M.D.

(By invitation)

In this paper the authors reported a case of myasthenia gravis that had come under their observation, with a detailed pathological report. The conclusions presented were as follows:

1. Neoplasms of thymic origin had been noted too frequently in myasthenia gravis to be ignored as a possible etiological factor in a certain proportion of the cases. In the cases reported by the authors, an unusual type of tumor, hitherto undescribed in this disease, was present.

2. While definite proof was still lacking, it seemed most probable that the disease was the manifestation of a toxemia of indeterminate origin.

3. The action of the toxic agent was not confined to the muscular system, but the organism was generally affected, as evidenced by the widespread presence of lymphocytic infiltrations throughout the body.

4. Although no degenerative changes were demonstrable in either brain or cord, the occurrence of lymphocytic infiltrations in the medulla, observed for the first time in the case reported, indicated the involvement of the central nervous system in the general toxemia.

5. The changes in the muscle fibers were purely degenerative, the result of the toxemia, and not dependent upon a primary myositis. These degenerative lesions bore no relation to the site of the lymphocytic infiltrations.

6. A study of the specimens in the case reported seemed to show that the lymphoid cells, wherever present in the tissues, were derived from the perivascular lymph spaces.

Dr. Harlow Brooks said that at a meeting of this Society about two years ago he had reported a case of what he believed to be myasthenia gravis, which, especially in its pathological appearances closely conformed to the description given by Drs. Mandlebaum and Celler. The lymphocytic infiltration spoken of by the authors of the paper was well marked, and there was one hemorrhage of considerable size in the medulla which was unquestionably the immediate cause of death. The fact that this hemorrhage had occurred, and that definite lesions were found led some of the members present to believe that the case was not one of myasthenia gravis. In his case, Dr. Brooks said, the lymphocytic infiltration was most marked in the medulla.

Dr. Joseph Fraenkel said that if a case of myasthenia gravis were characterized by the myasthenic reactions and the pseudo-paralysis of the muscles, particularly those supplied by the medulla, he thought there was no question about the diagnosis of the case referred to by Dr. Brooks.

The speaker asked Dr. Mandlebaum if any attention were paid to the parathyroids at the post-mortem examination, and whether there were any parathyroid tissue in the thyroid? The possible involvement of the ductless glands in these cases was now being investigated under the stimulus of the work of Lundborg.

Dr. William Hirsch said he had had occasion only a few weeks ago to see the most complete case of myasthenia gravis that had ever come under his observation. The patient was referred to him by a laryngologist, who had consulted him because of the fact that the patient complained mostly of some trouble in his voice. The condition of the vocal cords was suspicious of a paralysis, but not typically so. Upon examination, the case proved to be one of myasthenia gravis. The patient had the characteristic bilateral partial ptosis, an involvement of the muscular apparatus of the entire body, etc.

Dr. Herbert L. Celler said that in quite a number of cases of myasthenia gravis that had been reported, hemorrhages similar to those described by Dr. Brooks had been found in the medulla and elsewhere in the nervous system. These had always been described as of recent origin, and death could hardly be attributed to them. Dr. Cellar said that the parathyroids had not been examined in the case reported by Dr. Mandlebaum and himself. Even were the parathyroids affected, it would not necessarily follow that a lesion would be found there. As Chvostek himself points out the symptoms might be due to a functional hyperactivity of these glands which could not be determined by pathological examination.

The president, Dr. Sachs, who had seen the patient whose case was reported by the authors of the paper, said the man, while apparently in fairly good condition, suddenly developed the symptoms that led to his death. The case in every respect was absolutely typical of myasthenia gravis, a disease the clinical picture of which was now so well defined that we were not likely to make a mistake as regarded its diagnosis.

Dr. Mandlebaum, in closing, said the pathological examination of the

medulla in the case reported hardly showed a sufficient cause for the sudden fatal termination. Perivascular lymphocytic infiltrations were found there, but they were regarded as expressions of a general toxemia, and as they were present in other organs as well, they had probably little or no bearing upon the patient's sudden exitus.

Dr. George W. Jacoby read a paper on "Psychiatric Expert Evidence in Criminal Proceedings; its Imperfections and Remedy."

REPORT ON THE QUALIFICATIONS OF THE MEDICAL EXAMINER IN LUNACY.

L. Pierce Clark, M.D., for the committee, submitted the following report:

Mr. President: Your committee, appointed to inquire into the legal responsibility of medical examiners in lunacy, reported upon the subject at the last meeting of this society. It was shown at that time that the main responsibility of the examiner rested upon the ground of negligence, as in the regular practice of his profession. In the absence of proof of this there could be no real cause of action except on the ground of conspiracy. A consideration of the latter was, however, outside the province of the committee's work. The main issue in the report that warranted further report concerned the fact that examiners are not responsible to the patient for errors in diagnosis. There are abundant reasons why a more exacting requirement should be made for the proper qualification of all examiners of lunacy. Inasmuch as the primary qualification of court experts is their standing in the class of lunacy examiners, it may properly be argued that the committee's work is coincident with the purpose of the preceding paper.

In order that the qualifications of proposed candidates for appointment by the court should be better fitted for their work, it seems desirable to your committee, and it recommends, that the state medical licensing board shall have power to examine such candidates and issue certificates of qualification.

Your committee therefore urges that such certificates be made a part of the requirements of a proposed lunacy examiner's credentials before the court shall finally create such an applicant an examiner.

The committee further suggests that the medical licensing board require that the candidate shall at least have examined mental cases covering the principal forms of insanity, and shall have filled out some half dozen certificates of lunacy at the state hospitals of their residential district.

A certificate covering this experience may be required of the medical superintendent of said state hospital.

The committee furthermore recommend that the president of the state lunacy commission shall be made a member of the medical licensing board in order that he may supervise and regulate this new function of the medical licensing board.

Dr. Frederick Peterson said he found himself heartily in accord with everything that Dr. Jacoby had said in relation to expert testimony, and he expressed the hope that the society would see its way clear to establish a committee in accord with Dr. Jacoby's recommendation.

Dr. Adolf Meyer said it was true that the suggestion made by Dr. Jacoby was based on prolonged experience in Germany. In Switzerland, a similar practice prevailed, although there additional experts could be drawn into the case. In this country, many efforts had been made in

the past to improve upon the present method of introducing expert evidence in criminal proceedings, but the attempts, unfortunately, had always met with failure, largely because the physicians were not sufficiently informed about the fundamental differences of the laws in this country, and because they would not realize that after all it is not merely the law that is at fault. As the case stood at present, the medical expert was placed in the position of giving an opinion in a medico-legal case whether he had enough facts or not upon which to base an opinion that was worth expressing. It was not likely that we could revolutionize our present laws bearing on this question; but the medical profession should take it upon itself to raise the standard of the individual medical expert by reviewing and scrutinizing his testimony more closely than was now done. Then, if it were found that he had exceeded proper bounds, the matter could be taken up by the professional community, as represented in the medical societies. This of course was a very delicate proposition, and required careful consideration. The speaker said he had recently made the suggestion that as many cases as possible of court proceedings, especially of *habeas corpus* proceedings, should be reviewed by properly constituted authorities and placed before the medical and legal public, so as to create standards which might serve under any form of law. That would be a step in the right direction which might assist in quickening the conscience of the individual physician, and eventually it might perhaps lead to a feeling of caste among the members of the medical profession which would prevent gross transgressions in the line of medical expert testimony.

Dr. Charles L. Dana said he was in entire sympathy with the high ideals that Dr. Jacoby had set forth in his paper upon expert evidence. In fact, they approached so close to perfection that it would be very difficult to reach them. Probably, the best we could do at present would be to lay our plans for a campaign which would last for a long time. This campaign should be laid out along definite lines, and with the idea that we should attempt small things at first, but step by step we should finally achieve our object. The suggestion made by Dr. Jacoby to have physicians appointed to the courts was practically prevented by our Constitution, which allowed any man accused of a crime to call in his defense any witness he chose. The first step in the right direction, the speaker thought, was to change the standard of the qualifications of the examiners in lunacy, as suggested in the report of the committee read by Dr. Clark. That would give a higher status to the class of men from whom the experts could be drawn. Another practical suggestion was that made by Dr. Meyer, namely, to have the testimony of medical experts critically reviewed by a properly qualified committee or individuals. Such a procedure might have a salutary effect upon expert witnesses, and serve to some extent to improve the present condition of affairs. Furthermore, the various local neurological societies, or perhaps the American Neurological Association might establish a standard, based on training in years and experience, by which the expertness could be measured. Another step in the right direction would be to assist Dr. Bailey and his colleagues of the Psychiatrical Society, to pass the bill which was introduced into the Legislature of this state last winter, to allow persons who were accused of crime and whose defense was insanity, to be taken to a state hospital and kept under observation for a time long enough to determine their mental condition. He had been assured of its utility in Massachusetts by Boston alienists, and in Vermont by a prominent lawyer of that state.

Dr. Hirsch said the disagreements of medical experts before the court, especially in sensational trials such as the one recently concluded in this city, had always given rise to very serious criticism on the part of the public, while the medical profession was inclined to attribute them to some defect in the law. The laws of the State of New York were no more deficient in this respect than were those of other countries. The German law, for instance, was no better than ours, and a remedy for these disgraceful court proceedings lay in something else than the law. All it required was honesty on the part of the men who were called upon to testify. As long as a man would go and testify for money to any thing that he was asked for, we could expect to see no improvement in the present state of affairs. No law, no matter how perfect, could remedy the disgraceful dishonesty of certain men. If we could raise the moral standard of our medical experts, then no change in the law would be necessary.

Dr. Smith Ely Jelliffe said that in approaching this subject of trying to find a remedy for the imperfections of our present system of expert evidence in criminal proceedings, one of the first things we should seek for was to have more or less cordial unanimity in the medical profession itself. Under present conditions criticism of each other was to him the deplorable feature. Every man's hand was turned against his brother and all willing or anxious to believe scandal about each other; there was an absolute lack of sympathy and understanding of other men's opinions, and a lack of ability to see that the men who were criticised might be right from their point of view. There were too many opinions founded on rubbishy newspaper reports. It ultimately resolved itself into a question of law and the presentation of evidence, and the methods of its presentation. Those who had any legal experience whatever were aware of the fact that the methods of presenting evidence permitted opposing counsel to present hypothetical questions which contained only the facts of their own particular side. As long as that condition existed, we shall have to deal with it as best we can.

In seeking for a betterment of conditions, from a constructive point of view, Dr. Jelliffe said the way to get at it was to begin at the educational standpoint, and he was heartily in favor of the report of the committee which was read by Dr. Clark, and which favored raising the standard of medical examiners in lunacy. One of the greatest deficiencies of our medical curriculum was the lack of attention that was given to the study of psychiatry; four hours a year is devoted to mental diseases by one medical college in this city, as compared with seventeen hours a week for Munich and other institutions abroad. With that fact in mind it was not difficult to understand why some of the medical profession should know so little about psychiatry, nor was it difficult to understand why men with diverse training should differ so widely in their opinions on a subject which was so extremely intricate as the disorders of the mind.

First, then, we should start from the educational standpoint, increasing the knowledge of the general practitioner on these subjects and raising the standard of the lunacy examiners. This would lead naturally to the idea suggested by Dr. Meyer, namely, the establishment of an *esprit de corps* among physicians and a feeling of caste with reference to men of standing. In this way we might purge the present system from one of its greatest defects, if defects they be, rather than the natural outcome of the frailties of human kind.

The suggestions contained in Dr. Jacoby's paper were ideal, but under

present conditions they must be regarded as largely chimerical, and it was only by gradual steps, looking to changes in our present laws and methods of education, that we could hope to effect a change. The defects complained of were as old as law itself, and were much better understood by lawyers than doctors.

Dr. L. Pierce Clark said he was a member of the Psychiatric Society's committee referred to by Dr. Dana, which was appointed to represent the interests of a bill introduced into the state legislature, the object of which was to refer cases of questionable insanity in criminal trials to one of the state hospitals for observation. Years of campaigning would be necessary before such a bill could be passed, and a number of lawyers and judges who were consulted said that such a procedure interfered with the constitutional right of the individual, and when they were informed that such a law was now in existence in the states of Maine, New Hampshire and Massachusetts, they declared that it would undoubtedly be held unconstitutional if seriously questioned by any one in the court. It might be wise to get all the judges to pass upon the constitutionality of a measure before enacting it in a law. Of course the idea would have to be worked out by the several judges acting in a private capacity. Several judges had already assented to this proposed plan.

Dr. Joseph F. Terriberry said that while comparatively little attention was given in this country to teaching psychiatry, yet he wished to take exception to the statement made by a speaker that ignorance was at the bottom of this question, nor was it necessary, or indeed possible, as experience had shown, to look to the legal fraternity for aid. The remedy was in the hands of the medical men, and it lay in simple honesty on their part.

Dr. Joseph Fraenkel said he did not think that medical experts were as a class dishonest, and he thought no object could be gained by calling each other names. If a question is put to a medical expert, and the question is a hypothetical one, the medical expert cannot be accused of dishonesty if he answers it the best he knows how. This did not always necessarily imply the possession of expert knowledge. If an expert's opinion was always necessary, what would they do in the smaller centers, where such an opinion was inaccessible? Many of the questions asked simply required an intelligent answer, and if that answer disagreed with the opinion of the colleagues on the witness stand, it did not necessarily imply dishonesty on the part of the holder of the opinion.

Dr. G. Alfred Lawrence thought there should be greater *esprit de corps* between the legal and the medical professions. If the members of these two professions could get together, instead of being in apparent or real opposition, something undoubtedly could be accomplished. The medical profession certainly could not do anything in this matter without the aid of the lawyers, and the latter could help us a good deal if we but appreciated the fixed forms and due processes that are essential to all legal procedure and formulate our propositions in such a manner as not to be at variance with such methods of legal procedure.

Dr. William Steinach said that in the selection of medical experts, the court should be limited to that particular class who have qualified as experts in mental diseases, instead of choosing them from among genitourinary surgeons and dermatologists, gynecologists, surgeons, etc., as had been done heretofore. The same objections that had been raised to expert medical testimony obtained in other lines, and he recently heard of a case where an expert in automobile construction was called to the

stand and treated very much by judge and lawyers as medical experts usually are. Before we could get the laity to pay much attention to this question, the medical profession should purge itself so that there would be less cause for just criticism, and the primal step in that direction was to raise the standard of examiners in lunacy, as suggested in the report read by Dr. Clark.

Dr. Robert C. Woodman thought that it was a mistake to attempt to establish a single standard of qualification for consultants in medico-legal work and for examiners in lunacy who should make the ordinary commitments of patients to the state hospitals, and that any attempt to do so must seriously limit the usefulness of the state institutions for the insane by making entrance thereto difficult, and so automatically limiting the freedom of discharge. He regards the commitment of the insane as a medical question in most instances and rather than new obstacles to admission he would prefer to see amendments to the law that would allow any physician legally qualified to practice medicine to take his insane patient to the special insane hospital for treatment. Approximately one third of the counties of the state have no town of ten thousand people; such qualifications as the committee proposes for medical examiners are not to be had in such communities, where it would be unfortunate in the extreme to have to send to a distant county for a medical examiner before placing the insane under care. Besides, as a matter of fact the family physicians very often submit excellent commitment papers and by reason of their intimate acquaintance with their patients they are, to a degree, protected from mistakes into which the most expert consultants might fall.

Dr. I. Strauss thought it was unfortunate that the society had seen fit to mingle the discussion of these two subjects, namely, the standard of examiners in lunacy and the question of medical expert testimony. They had no bearing on one another, and should have been kept apart. The crux of the whole question seemed to be that when a poor man was on trial for his life and the point of his insanity was raised, then any ordinary examiner was satisfactory, but in a sensational case, only a renowned expert would answer. The latter class, of course, would not be affected by any change in the lunacy laws, but the former would.

In deciding the mental status of a defendant, there ought to be no question of taking sides. He should be examined by experts, and an attempt should be made to reach an unbiased diagnosis. Any attempt to change the present laws covering this subject would doubtless clash with the constitutional safeguards that now existed, and we then came back to the only other solution, namely, to raise the ethical standard of the individual members of the medical profession.

The president, Dr. Sachs, said that more than fifteen years ago he brought this subject up before the Medico-Legal Society, and he had found then, as now, that it was difficult to get the legal profession to unite with the medical profession in any attempt to improve the existing conditions. In view of that fact, the medical profession should do what it could, and then, in case of failure, the onus rested upon the lawyers. Medical experts should not be blamed too harshly. Doctors, like other men, were only human, and for that reason they should keep out of temptation as far as possible, and the only way to keep out of temptation was not to be tempted, and when they are called to the stand, they should testify to the court and not to either party. Ofttimes, the expert did not get the facts from his lawyer; they were purposely kept from him, and

the opinion of the latter was therefore based on false premises. A majority of the medical experts, Dr. Sachs thought, were perfectly honest, but the lawyers were often bent on deceiving them for their own selfish purposes. In many instances, instead of trying to ascertain the truth, as is done in the courts abroad, an effort is made to keep as much of the truth as possible from the jury.

Dr. H. C. van den Burgh said that as a member of both the medical and the legal profession he could say for the latter that they were always prepared to meet the medical profession half way and more when a tangible proposition upon this subject was presented in a candid and straightforward manner by a representative body of medical men, such as this society. As to the constitutional features of the subject that had been touched upon in the discussion, he wished to say that the medical expert who was testifying for the people in an unprejudiced and truthful manner at the same time represented the defendant, who was one of the people. Medical experts of that class always commanded the respect of the legal profession, and with them they were always ready to work in harmony.

Dr. Jacoby, in closing, said he had tried to present this subject as dispassionately as he could. Even with the inadequate laws that now governed this feature of our legal procedure he thought it was possible to adjust our method of giving medical expert testimony so that it would be on a par with that of other countries, first, by giving the general practitioner a better knowledge of psychiatry, and, second, by establishing a special class of experts. It would perhaps take a quarter of a century to do this, but a beginning had to be made some time.

THE PHILADELPHIA NEUROLOGICAL SOCIETY

February 24, 1908

The President, Dr. J. W. McConnell, in the Chair

A CASE OF PARALYSIS OF ASSOCIATED UPWARD MOVEMENT WITH PRESERVATION OF ISOLATED UPWARD MOVEMENT OF EYEBALLS

By Alfred Reginald Allen, M.D.

Dr. Allen's case was that of a white, Irish-born male about forty-five years of age who had had a chancre some years before. He presented the following points in ophthalmological examination:

He had fair vision in each eye but fixed naturally with left, the right eye swinging out. When the right eye is covered and the patient is told to fix a test object with left eye he does so and is able to follow the test object with the left eye in all directions. When the left eye is covered and the patient is told to fix with the right eye and then follow the test object to the right or left he is unable to do so unless he concentrates his attention on the left eye which is covered and makes that go to the left or right as the case may be. In this case the right eye turns inward or outward in its corresponding coordinate manner.

If he fix with his left eye and test object be moved upward the left eye follows the object upward to the normal limit of superior excursion

in a normal manner. The right eye during the test is only slightly elevated and is at the same time turned and rotated outward, showing the pure action of the inferior oblique minus the superior rectus. In case the right eye fix and follow the test object upward the behavior of the left eye is precisely that of the right eye under the same circumstances, *i. e.*, pure action of inferior oblique minus the action of the superior rectus; while the right eye is elevated in a normal and orderly manner. The pupils react freely and equally to light.

In the test where the patient's left eye is covered and we try to get him to follow the test object inward with the right eye, concentrating altogether on the right eye, he fails but when the test object is well past the line of central fixation on nasal side the patient is able to detect movement of object thereby showing that there is a normal stimulation of the retina to the temporal side of his macula. Therefore it cannot be said that he fails to follow the object because he does not know where the object is.

The explanation of this case is that the upper motor neurones from the left cortex to that part of the right oculomotor nucleus which presides over the innervation of the right internal rectus are interrupted, and the only way that the right internal rectus is brought into play is by an impulse traveling down from the right motor cortex to the left abducens nucleus and from here an impulse passing upward on the posterior longitudinal bundle to that part of the right oculomotor nucleus which innervates the internal rectus. This explains why he must concentrate his attention on his left eye which is covered in order to move his right eye inward.

As to the explanation of the difficulty in associated upward movement it must be remembered that the inferior oblique and superior rectus cell groups of one side are intimately connected by means of an association commissure and by this means do we get associated upward movements. There is, therefore, a break in this association tract and whereas each eye can do its work alone when fixing there is no associated upward movement. Where this association commissure or tract is situated is not definitely known. It may be directly across the raphe by the shortest cut between the two parts of the oculomotor nucleus or again (and this is not improbable) a set of axones may take origin in the inferior oblique and superior rectus portions of the oculomotor nucleus of the right side and pass to the nucleus of Darkschewitsch of the same side, then on the posterior commissure to the nucleus of Darkschewitsch of the left side and from there to the corresponding cell groups of the oculomotor nucleus of the left side. When the relationship between the posterior longitudinal bundle and the nuclei of Darkschewitsch and posterior commissure are remembered such an explanation seems quite plausible.

The nucleus of Darkschewitsch is very likely in no sense a part of the oculomotor nucleus.

Dr. W. G. Spiller referred to two cases of this character, one was reported by Dr. Krall before the Philadelphia Neurological Society, the other was von Monakow's case in which a tumor growing into the nuclei of the oculomotor nerves produced this symptom complex—paralysis of upward associated movement with preservation of the upward movement of each eyeball separately. Dr. Spiller thought the explanation given by Dr. Allen was a proper one.

Dr. T. A. Williams thought it very important not to overlook the psychic factor in the fixation with one eye in front. The man may not

be really making an effort to look at an object and fix it when he does not follow with one eye, but when he really makes an effort he can't help following the object with both eyes. If that is not the case Dr. Williams said he was quite prepared to stand corrected on the case. He said he did not pretend that that was any more than a mere suggestion. If that is not the case, might there not be another factor in the situation, namely, the particular focusing of the lesion upon that portion of the third nucleus which subserves the function of the internal rotation of the right eye, and conservation of that portion of the sixth nucleus which presides over the internal rotation of the right eye; that is to say that the left sixth nucleus is not attacked, nor are any of its tracts involved so that when an effort at bilateral deviation of both eyes is made it may be accomplished through intervention of that part of the left sixth nucleus which sends fibers to the right internal rectus, thus conserving what Grasset calls the "levo-gyratory" function. The automatic levo-gyratory function might in this way derive its mechanism from the nuclei of the third nerve in the floor of the third ventricle.

Dr. T. H. Weisenburg said that the man presented by Dr. Allen came to his clinic at the Medico-Chirurgical Hospital and that with Dr. Ingham he studied the patient a number of times and thought the condition was purely one of muscular disease and did not regard it as a case of associated paralysis at that time, and that he would be surprised to learn that the patient had developed paralysis of upward movement in the space of two to three weeks, that representing about the time elapsed. Dr. Weisenburg stated further, that in examining the patient at the present time he did not seem to have changed very much and he hardly believed it to be a case of paralysis of upward movement and he still regarded it as a case of involvement of the ocular muscles. Dr. Weisenburg referred to another case under his care, that of a young women, about twenty-eight years of age, who has been seeing double all her life.

Dr. Allen in closing, alluded to Dr. Spiller's statements in reference to the nucleus of Darkschewitsch, saying that he was aware that at one time it was thought to be part of the oculomotor nucleus, but now the idea is that it has more to do as the end station, or one of the end stations of the posterior longitudinal bundle, at the same time being the starting point of a large part, if not all, of the posterior commissure. Now there are very likely fibers running from the oculo-motor nucleus to the nucleus of Darkschewitsch. He said he knew that there was no proof that the decussation between the two oculomotor nuclei takes place by means of the posterior commissure, but he mentioned it simply as a very possible way of communication and one thoroughly in keeping with communications which take place all the way from the twelfth nucleus up to the third nucleus by means of the posterior longitudinal bundle, but he was very careful not to state that dogmatically. As far as the sixth nucleus on the left side being in perfect contact with that part of the right oculomotor nucleus, having to do with the rectus internus of the right side, as Dr. Williams has called attention to, that, of course, goes without saying. That is the only possible way the man has for internal rotation of the right eye.

Apropos of what Dr. Weisenburg said, also apropos of what Dr. Lloyd had called attention to, Dr. Allen said there is some swinging up of the covered eye when upward rotation is tested, but no more swinging up than could be caused by a rather fair action of the inferior oblique. And if you measure the orbits carefully and note the base of the cornea

in each eye you will find that the eye which does not fix in upward rotation is on a very perceptibly lower plane than the eye which does fix. In other words, there is lack in that eye of superior rectus action. If it were muscular he could not look up with either singly.

A CASE OF APHASIA

By W. B. Cadwalader, M.D.

Right-handed man, sixty years of age, presented himself for treatment with a history of an apoplectic attack four years previously.

On examination it was found that there was weakness of the right side of the body with slight impairment of sensation of the right upper limb. When objects were placed in his right hand he could recognize them, but more slowly than when placed in his left. This was hardly a true astereognosis. There was very slight ataxia of the right side. The pupils were equal and reacted to light and accommodation. The ocular movements were normal, and there was no evidence of palsy of the extra-ocular muscles.

The eye examination showed 3/6 vision in the left eye, and 2/6 vision in the right eye. There was a slight neuroretinitis but no hemianopsia. On test made with printed and written words and sentences he could not read when made to rely upon vision alone. If allowed to spell out each word letter by letter he could read some of the shorter and easy words slowly and laboriously. When prevented from doing this he could not read except an occasional short easy word. On asking him not to attempt any longer to read the words but to read the letters only, he read them off with apparent fluency, but constantly calling an "a" an "e" and *vice versa*. Sometimes mistakes were made in other letters but this was not constant. His difficulties in reading words and letters were precisely the same with the largest and smallest types. On testing him with figures he could read them rapidly and fluently, not only the individual figures but when combined in groups. He could not write at all, although his weakness did not seem sufficient to explain this. There was slight motor aphasia, sometimes misplacing words, but on the whole this was not marked. There was no apraxia.

His mental condition was apparently good, understanding and appreciating everything that was said to him, and in general his psychic functions were normal.

His family said that he had always been a man of good habits and of moderate education and previously had been able to read and write fluently.

Dr. Calwalader regarded this case as one of partial letter blindness and nearly complete word blindness.

Dr. Charles K. Mills said that the case was particularly interesting in the discussion of word blindness. It would seem to be corroborative of the views of those who believe in the extreme differentiation of centers in various portions of the zone of speech in the cerebrum as against the views of those who contend so strongly for the opposite. This man, he thought, was nearly monoaphasic. He noticed that the patient used practically all parts of speech in the few sentences that he employed. He was evidently not word deaf. He was able to recognize objects quite well but could not recognize words except to a very limited extent. Letters he recognized except one or two. Dr. Mills said he could hardly conceive an explanation

of such a case except upon the doctrines of localization which are supposed to be classical but which have been so seriously attacked quite recently.

Dr. Spiller said that the patient was more word blind than letter blind. This was the result probably of the method in vogue at the time the man was taught reading by the spelling of words. There have not been as yet any cases of alexia in persons who were taught by the new method of learning, viz.: learning by words first and letters later. When alexia occurs in persons taught reading by this method we shall likely find the recognition of letters more affected than that of words, as letters are acquired later.

Dr. Williams inquired whether the case had been examined in regard to his psychic functions, because one of the main points on which Pierre Marie insisted when he attacked the classical doctrines of aphasia was that the aphasic had lost the functions of performing that which he had learned didactically in general. It would be interesting to know whether the patient had lost any of the slightly complicated operations used in cooking or some similar occupation.

A CASE OF BILATERAL INVOLVEMENT OF THE COCHLEAR AND VESTIBULAR BRANCHES OF THE EIGHTH NERVE

By T. H. Weisenburg, M.D.

A young man of eighteen had specific iritis which was treated by mercurial inunctions. About two years after that his right ear began to buzz and hearing became very much diminished to be followed very soon by a similar condition in the left ear. Since this time hearing in both ears has become very much diminished, especially so in the right side. Since the beginning of his trouble he has complained of various oral noises, such as escape of steam, and whistling, buzzing and cracking sounds. This condition persisted for three years and in the meantime he never had dizziness or vertigo and did not stagger.

One night he went to bed and on rising in the morning the buzzing in his ear constantly increased and hearing was almost totally lost and then he began to stagger, especially to the left, and complained of dizziness. Since that time he has felt as if his head was constantly moving around or that objects in front of him were moving in the same direction. This dizziness was not influenced by change of position with the exception that sudden movement would increase it. He has not had headache, nausea, vomiting or disturbance of vision and has never had convulsions.

Examination shows besides the evidence of the old iritis an almost total deafness on both sides. There is on lateral deviation of the eyeballs nystagmoid movements which are equally marked to either side. Power in both upper and lower limbs normal. There is no ataxia with either eyes open or shut and the reflexes are a little prompt. His gait is typically cerebellar. There is no choked disc.

Diagnosis.—The patient evidently had specific disease and a bilateral involvement of both eighth nerves in so far as the cochlear distribution is concerned for he had for three years a gradual increase in deafness with noises in his ears. Suddenly the deafness became total, the noises increased and he first began to have vertigo with typical cerebellar staggering. Evidently there must have been an extension of the process either to the vestibular division of both eighth nerves or into the semicircular canals. The differential diagnosis between these two conditions

is very difficult and it is a question whether it can be made. Should there be, however, an involvement of the semi-circular canals there should be besides the vertigo and staggering some general disturbances, such as vomiting and also in such case there would hardly have been the history that is present in this case, *i. e.,* there would have been an involvement of hearing and equilibration at the same time or very soon after. It is possible then that we have here first of all involvement of the cochlear branch with an extension of the process to the vestibular but in such case it would be difficult to reconcile the fact that there was a sudden extension of the neuritis to the vestibular branch.

Dr. Donaldson stated that the remarks to which Dr. Weisenburg alluded were based on observations made by William James, on a series of deaf mutes, in most of whom destruction of the semi-circular canals had probably occurred. In these cases, three sets of symptoms were observed; first, the inability to judge direction when swimming under water; second, almost complete absence of susceptibility to sea-sickness; and third, freedom from dizziness after rotation.

Dr. Mills asked whether Dr. Weisenburg thought the lesions were in the auditory canal.

Dr. Weisenburg said he thought the man had an involvement of the semi-circular canals.

We have well studied and recorded cases of cochlear and vestibular disease separate and conjoined and separate becoming conjoined. Dr. Mills believed that all these conditions occur as peripheral affections. In his book he has a chapter on diseases of the vestibular nerve, separately considered, giving the clinical phenomena there for the affection of this nerve. In other words, we may have disturbance of equilibrium from vestibular disease entirely independent of disturbance of hearing, from cochlear disease, or the reverse. Uusually affections of both nerves occur at the same time. Sometimes these cases are due to grippe, sometimes to syphilis, occasionally to labyrinthian hemorrhage. Dr. Mills thought Dr. Weisenburg's case was one in which the cochlear nerve was first affected and later the nerve became affected.

The localization of disease in the cerebello-pontile angle has now become a matter of comparative ease in most cases, though we have an occasional case in which labyrinthine disease needs to be very fully considered before making this diagnosis. There was such a case at the University Hospital a year or so ago. Operation was declined at the time. The event proved that it was best not to have operated. The involvement of the facial or fifth is not present in this case of Dr. Weisenburg's. Dr. Mills stated that none of the symptoms presented could be regarded as absolutely cerebellar. Of course the involvement of the seventh or fifth or both, and cerebellar symptoms would separate the cerebello-pontile case from one like the present.

Dr. Wiliams thought it a very interesting case. Given that it is a luetic process, which he understood Dr. Weisenburg to affirm, it seems very strange that such a process should involve the endolymph of the cochlea for four years without spreading to the endolymph of the semi-circular canals. One might, therefore, readily invoke a pathogenesis of radicular cause. In that case it is very strange that after four years, supposing it was a meningitis process, the inflammation should not have extended to the vestibular portion of the eighth nerve. Dr. Williams thought this point could be settled by lumbar puncture. It is certainly not a cerebellar affection nor one of the nucleus of Deiters because no

symptom referable to the nucleus of Deiters or the cerebellum such as hypotonia or asynergia of other parts have manifested themselves.

Dr. Weisenburg stated in closing that he was not at all certain that the differential diagnosis between an involvement of the vestibular branch and the semi-circular canals could be made and his object in presenting this case was to call the attention of the society to such a possibility.

THE PATHOGENESIS OF TABES DORSALIS

By Tom A. Williams, M.D., of Washington, D. C.

Controversy as to the pathogeny of tabes has not ceased since Duchenne surmised its sympathetic origin, and Charcot later supposed it to be a posterior column dystrophy, similar to that of Friedreich's ataxia. His great authority prevented due attention to the researches of Obersteiner and his followers, until the memoir of Redlich appeared in 1897. The discoveries as to the syphilitic etiology of tabes caused greater attention to the work of Nageotte, who in 1894 had indicated the constancy of lesions on the radicular nerve at the point where it receives its meningeal sheaths. These lesions correspond to one or other stage of the granulomatous process, varying as they do from simple round cell infiltration to granuloma, and even breaking down with formation of cavities. They are due to primary chronic meningitis, evidenced by the lymphocytosis found by spinal puncture during life, and postmortem when skillfully looked for; although the tendency of the process to resolution and fibrous-tissue formation leaves only a slight thickening in the membranes, already fibrous by nature.

The changes in the cord are consecutive to this. That this is so, is proved by similar changes of the posterior column after disease or experimental section of individual roots, and by the changes occurring after their mechanical affection of the radicular nerves due to the increased intro-spinal pressure caused by the growth of cerebral tumors.

The noxa falls upon the root fasciculi very disparately, and this corresponds to the disparate nature of the sensory troubles, which do not preponderate so much as formerly supposed upon the fibers which subserve the sense of attitudes and of muscular movement; for it is now definitely shown that cutaneous sensations are always involved more or less, though probably later in the disease.

The superficial lightning pains described by Gowers and the psychometric analysis of the sensibility of tabetics by Vaschide are an index of this; while the researches of Head enable us to explain the modifications in terms of deep, protopathic, and epicritic sensibility. The fibers subserving the life of internal relation may differ morphologically from those subserving external relationship, as contended by Pierre Bonnier with particular reference to the eighth nerve, the principal posterior root, where the cochlear portion, whose function concerns the outside world is affected only rarely, while the vestibular portion is concerned with intrinsic relationships and is involved very commonly indeed in the tabetic process.

However this may be, it is certain that impaired sense of attitudes is always accompanied by impairment of the deep pain senses of perception of the vibrations of the tuning fork by the bones; and as these functions are conveyed in the same peripheral path, while they are separated within the cord, clinical evidence is in entire harmony with the pathogenetic theory advanced by Nageotte. The data furnished by the optic nerve

symptoms are similarly best explicable by a meningeal affection involving in this case not a posterior root but a homologue of an intra-spinal path.

Those tabetic symptoms referable to the sympathetic do not differ from those when produced by experimental section of the spinal roots, nor from those in syringomyelia, which, however, attacks the cell bodies in the intermediolateral columns. Charcot's negation of changes in the sympathetic is effectively disproved by the researches of Roux, who found the medullated fibers markedly decreased in tabetics. The anterior roots are not unaffected; but the relative absence of serious myopathies early in the disease is accounted for by the rapid regeneration of the fibers. This is shown by the "terminaisons en crosissance" exhibited in Nageotte's preparations and by the results of section experiments. The regeneration of the posterior root fibers extends only to Redlich's ring, at which they lose the neurilemma sheath.

Finally lymphocytosis and reflex iridoplegia, the two most characteristic signs of tabes, are found in many cases of syphilis without the tabetic symptoms; indeed both sometimes occur in the secondary stage, the former in as many as forty per cent.

The contention of Babinski and Nageotte is therefore accepted, that a chronic syphilitic meningitis is responsible for what has been called tabes dorsalis; and that it was formerly disregarded on account of a tendency to the occurrence of resolution and fibrosis of the lesions. The practical application of this conclusion is of the greatest importance in the treatment of the disease. Cases taken early may be completely arrested; and in all cases, the active manifestations may be resolved if treated before destruction of the noble elements has occurred, though naturally the residues of former exacerbations cannot be removed.

Dr. W. G. Spiller said the subject was particularly interesting to him because he was in Vienna at the time Obersteiner and Redlich brought forward their views regarding the constriction of the posterior root fibers, and he was in Paris when Nageotte was first proclaiming his theory. Dr. Spiller said a patient had just died in his service at Blockley who had pronounced tabes with preservation of the patellar reflexes. This was tabes of the lower type. Dr. Leopold is now examining the spinal cord. Unfortunately he was unable to get the vestibular nerves; he had traveled forty miles to get the spinal cord. There can hardly be any doubt that the fibers of the column of Clarke in many pronounced cases of tabes are degenerated. Dr. Spiller said he could with the lantern slides in his demonstrations to students show the degeneration of the column of Clarke with low power. This degeneration probably has much to do with the ataxia.

As for meningitis, he thinks it is common in tabes in at least a slight form. Unquestionably syphilis is the great cause of tabes, but Dr. Spiller said he was not yet prepared to state that it is the only cause of tabes.

Dr. Williams, in closing the discussion, said he purposely omitted reference to the well known work of Dr. Spiller in his paper because he felt he had not time to discuss it. He was very glad to hear Dr. Spiller speak of the degeneration of the fibers within the column of Clarke. Nageotte insists that it is exceptional for the long fibers to be affected first, whereas the classical teaching is that the long fibers are those affected first. Dr. Spiller's experience is exceedingly valuable in that connection. Dr. Spiller's skepticism Dr. William thinks is justified. Any skepticism he has as to the theory of Nageotte is welcome. That is the only scientific attitude to take. In regard to the peripheral nerve palsies,

Thomas in 1902 found that the ganglia are often involved. The peripheral terminations of these cells would then degenerate and present peripheral neuritis. In regard to tabetic degenerations, though he does not explain why it begins there, Nageotte yet shows that when a nerve fiber is attacked that the decay tends to begin at the extreme periphery just as it commences there in a decaying tree. Dr. Williams said that any little novelty he brought to the discussion laid in the facts he presented as to the Obersteiner ring. The process itself begins, as Nageotte says, in the root, but in early cases the first indication is often seen at the radicular ring, because there the nerve loses its neurilemma sheath. One might fail to notice the fact that in the root some fibers are missing, whereas the degeneration beyond the radicular ring persists and is manifest to the older methods of examination. Careful examination shows always degeneration of the posterior root, but much more marked degeneration of the spinal side of the radicular ring. The reason for that is that the nerves have lost their neurilemma sheath there. Only careful examination will show that there are in the roots regenerating fibers. The regeneration is such as to give rise to early resumption of activity in the muscles, so that it is often clinically unappreciable except by careful examination. Another point cogent in Dr. Spiller's remarks was that he had so often noted muscular atrophy. He has observed frequency of muscular atrophy in tabetics. He has also observed the frequency of meningitis. The alleged absence of muscular atrophy clinically and meningitis postmortem were two weak points in adhering to the doctrine of Nageotte. Dr. Spiller's cooperation in the removal of these objections is most gratifying to Dr. Williams, who said he was very glad indeed to have had the opportunity to present his paper before the Philadelphia Neurological Society and thanked the society very much for the manner in which his paper had been received.

THE AMERICAN NEUROLOGICAL ASSOCIATION

The Thirty-fourth annual meeting held at the College of Physicians of Philadelphia, May 20, 21 and 22, 1908

The President, Dr. Charles W. Burr, in the Chair.

The President delivered his address, entitled: The Mental State in Chorea and Choreiform Affections. (See this journal, p. 353.)

PROGRESSIVE HEMIPLEGIA DUE TO GLIOSIS AND VASCULAR LESION OF THE RIGHT CENTRUM AND CORTEX

By Frank R. Fry, M.D.

Eight weeks before the necropsy there were slight recurring spasm and paresthesia of the face, and a still slighter spasmodic tendency in the hand on the left side. There was a partial paralysis of the hand and arm which had been gradually advancing for four months. There was no paralysis in the lower extremity. There was a complete absence of objective sensory signs in the paralytic member and elsewhere. There were also absent the general signs of cerebral tumor, *i. e.*, no vertigo,

vomiting, headache, or optic neuritis. The diagnosis was a lesion, probably a neoplasm, in the ascending frontal convolution. The patient later developed general convulsions and a full hemiplegia of the left side.

He died after an operation. A gliosis was found which invaded much of the frontal lobe and backwards slightly beyond the ascending parietal convolution.

The features of the case were: the presence of paresthesia or subjective sensory phenomena for many weeks before objective signs of sensory disturbance appeared in the same areas; the diagnosis of a probable tumor from the presence of an advancing paralysis with symptoms of corresponding cortical irritation, although all the general symptoms of tumor were absent; the presence of data from which an extensive growth of the glosis in seven to eight weeks could be estimated.

A CASE OF APRAXIA WITH AUTOPSY.

By John H. W. Rhein, M.D.

(To be published in this journal.)

Man of fifty-five developed progressive loss of eyesight, together with inability to recognize the position of his body and limbs; loss of the sense of location generally; loss of tactile sense in the left hand, and disturbance of the temperature sense in the left hand. The stereognostic perception was impaired on both sides. Finally, there was a contracture of the left arm and hand, with rigidity of the left leg, some rigidity of the right arm and leg, but not to the point of contracture. Progressive mental failure.

Autopsy revealed an area of softening in the cortex, in the occipito-parietal region on the right side. Sections of the brain showed degeneration in the white matter of both occipital lobes, and part of the parietal lobes.

Discussion of apraxia.

Discussion.

Dr. Joseph Collins said that the case reported had very decided interest for him because the patient had been for a brief time under his observation and because he had utilized him both at the American Neurological Association meeting in Boston two years ago and at the meeting of the New York and Philadelphia Neurological Societies in this city a year or so ago, as an illustration of a clinical type of cerebral arteriosclerosis which he has frequently encountered and to which he had desired to call attention. This patient was considered by some physicians who had observed him to have a brain lesion of the nature of a neoplasm. When Dr. Collins saw him he was receiving antisyphilitic medication. Dr. Collins' diagnosis was made in regard to his lesion, entirely aside from his agnosia, apraxia, and loss of the sense of position which conditions he had then much as Dr. Rhein related them to-day. Dr. Collins maintained then and later that the patient conformed in practically all the clinical manifestations with a type of arteriosclerosis which he had described at the meetings mentioned and he thinks that the autopsy corroborates that view. So far as Dr. Collins could judge from the brief report which was given by Dr. Rhein the morbid condition was in reality a softening, probably dependent upon a widespread arteriosclerosis,

although he did not recall that anything had been said about the evidences to be found on further microscopical examination of the brain. The softening which Dr. Rhein describes is probably an encephalomalacia dependent upon an arteriosclerosis. Dr. Collins called attention a year ago to the patient's face and features, his attitude of fixation, an apparent transformation from an animate being to an inanimate object, a fixation of face and feature that reminded of paralysis agitans and he pointed out at that time that the complex of symptoms could be explained only by positing such an extensive lesion as arteriosclerosis. The focal symptoms which Dr. Rhein had discussed were obviously dependent upon the softening which he described, but the general symptoms he believed could be interpreted only on the supposition that there was some widespread disease such as arteriosclerosis. Surely many of the symptoms which the patient had had could not be ascribed to the softening. Dr. Collins asked whether the autopsy showed arterial lesions, whether serial sections had been made of the brain ganglia and how much degeneration existed.

Dr. A. Gordon said that in cases of hemiplegia of cortical or subcortical origin, it is frequently noticed that there is disturbance of function of the opposite side. He asked whether a systematic histological study of the corpus callosum was made by Dr. Rhein, as it appears that symptoms were present on both sides. Apraxia is not infrequently the result of involvement of the white matter, particularly of the corpus callosum.

In closing the discussion, Dr. Rhein, in answer to Dr. Collins' query, stated that there was general arteriosclerosis present. Microscopic studies had been carefully made. The arteriosclerosis was not very intense, consisting of moderate perivascular distension and infiltration. This was especially noted in the necrotic area. It was also present in the degenerated white matter.

In further answer to Dr. Collins' remarks, Dr. Rhein stated that, in his opinion, the chief importance of this arteriosclerosis was in its localization, rather than the lesion itself. The visual disturbances, the absence of degeneration in the calcarine region, the degeneration of the white matter of the occipital lobe, with partial degeneration of the inferior longitudinal bundle and optic radiations, present a picture which is not in accord with the present views of the pathology of cortical blindness. He had no explanation for this, except that perhaps the calcarine cortex did not govern primary vision independently, but that it operated in conjunction with other portions of the occipital lobe.

Dr. Gordon's point was taken up in detail in Dr. Rhein's paper. A study of the corpus callosum was made, and this phase of the subject was considered in discussion of the cases of Liepmann, Van Vleuten and Mass.

TREATMENT OF FACIAL SPASM BY MEANS OF INJECTIONS OF ALCOHOL. REPORT OF THREE CASES

By Hugh T. Patrick, M.D.

Brief report of three cases of typical facial spasm treated by injections of alcohol into the facial nerve at the stylo-mastoid foramen. Good results in two, failure to reach the nerve in the third. Exhibition of photographs.

Discussion.

Dr. J. J. Putnam asked whether Dr. Patrick considers that the injection of the alcohol induces a special pathological condition of the nerve and how he explains the result as compared, for example, with the failures which characterized his own efforts some years ago to treat facial spasm by nerve stretching.

Dr. Gordon asked whether Dr. Patrick had any experience with his treatment in hemi-spasms following peripheral facial palsy.

Dr. H. M. Thomas said that he had seen a number of cases in which operation was performed for facial spasm, and he thinks the facial palsy is as bad as the original condition. The only case of spasm in which a cure had been effected was one in which Dr. Cushing made an anastomosis of the facial and the spinal accessory nerves. That patient is practically well, although she has not recovered complete voluntary power. Dr. Thomas said the emotional power came back perfectly and came back before the voluntary power in the paralyzed face.

Dr. G. L. Walton emphasized the presence of other obsessive tendencies in the victims of tic. Sufferers from spasm may be neurotic but not necessarily obsessive. He spoke of the importance of carefully correcting refractive error before resorting to direct attack upon the seventh nerve. He mentioned an obstinate case of many years standing completely cured by a final adjustment of glasses and muscular exercises for insufficiency.

Dr. Charles E. Beevor said he was glad Dr. Patrick made the distinction between tic and facial spasm, the spasm is anatomical movement of muscles, whereas tic is functional. An important point in regard to the treatment is in regard to hitting the nerve. He supposed the evidence was that if the nerve is not entered one cannot get any paralysis. Is there any twitching the moment the needle goes into the nerve? The motor nerve if you irritate it will twitch. Dr. Beevor thinks that we ought to get twitching of the nerve the moment the needle enters it. He wished to know how much alcohol Dr. Patrick used, whether one or two injections, and whether he employed absolute alcohol.

Dr. J. Ramsay Hunt said that among the other questions he would like to ask was whether Dr. Patrick had found any etiological factor in these cases which might reflexly have caused the spasm. When Brissaud separated the tic movements of the face from the spasms he spoke of the reflex origin of these spasms; the sensory limb of reflex arc being represented by the fifth nerve. Babinski, in a recent contribution to this subject, regards the facial spasm as due to some lesion, either of the nucleus or of the trunk of the facial nerve, regarding the trunk of the facial nerve as purely motor. Last year before this Association among other sensory functions of the seventh cranial nerve, Dr. Hunt spoke of reflex facial spasm, and he thinks that may have some practical importance in the treatment of these cases. In going over the literature of facial spasms he has been surprised at the number of cases in which there has been associated auditory disturbances, rupture of the tympanum, accumulation of cerumen in the external canal, chronic otitis media or nerve deafness. There is also another group of cases of central origin, notably two cases of aneurism with pressure on the facial nerve at the base of the skull, without any pressure on the pons or on the fifth nerve. Dr. Hunt could not understand how the irritation of the motor nerve

trunk could induce a state of more or less rhythmical and recurrent spasms in the facial nerve. He believes that the sensory mechanism of the facial nerve plays the afferent factor in the production of some types of facial spasm, so that we must not only consider the fifth nerve as an afferent factor, but also the seventh nerve. This sensory mechanism may also help us to a proper understanding of the secondary contractures and twitchings of facial palsy. The sensory mechanism of the seventh cranial nerve consists of the geniculate ganglion, the nerve of Wrisberg, the petrosal branches to the middle ear and sensory fibers passing out into the motor trunk, and distributed to the region of the concha, auditory canal and tympanic membrane.

Hence the importance of the internal, middle and external auditory mechanism as a reflex factor in facial spasms.

Dr. W. G. Spiller remarked that we should not confound facial spasm such as Dr. Patrick had described with the tic occurring after a facial palsy. The latter was never observed unless some return of voluntary power was present but was incomplete. Lipschitz has shown that the tic of the lower part of the face developing after facial palsy is synchronous with winking. When regeneration occurs it is not probable that the same association of nerve fibers and nerve cells in the facial supply exists that was present before the palsy. Nerve cells formerly in connection with fibers of the upper branch of the facial nerve may in the process of regeneration be brought into association with fibers of the lower branch. Dr. Spiller had observed repeatedly that the tic of the lower part of the face in old facial palsy occurs at the same time as winking.

Dr. Putnam said he would like to express his acquiescence in what Dr. Spiller stated and would suggest that strychnia might affect the sensory or motor irritation. Strychnia causes increased motion not by acting on the motor, but the sensory centers.

Dr. Patrick, in closing, stated that he does not know how the alcohol acts. He has an idea that it blocks off something after the injections, just as stretching the nerves in facial neuralgia blocks off something. It is a pathological filter which holds back what we don't want and lets what we do want get through. Dr. Patrick said he does not know anyone who has thoroughly worked out the action of alcohol on the nerve fibers. In years gone by he tried stretching, with the same result as Dr. Putnam. The spasm returned with voluntary motion. He does not put forward the alcohol treatment as a radical cure. If the patients remain cured for a long time he does not see any objection to repeating the injection if necessary. He has not made the injection for Bell's palsy and would not. He had injected 15 or 20 minims of a 75 per cent. solution in the first case; this he thinks is too strong. Afterwards he used 40 to 50 per cent. He uses 15 to 20 minims because he puts in the needle and pulls it out a little way and then puts it in again, and so distributes the amount. During the several punctures he did not get any distinct twitching in the facial muscles. He presumes if the hypodermic needle had hit the nerve it would have caused sufficient movement to be perceptible to the operator. As to the etiology, he has nothing to say. The injections are easily made and the results to date are so gratifying that Dr. Patrick resolved to report the cases. He disclaimed any originality in the idea of alcohol injections in the treatment of facial spasm, stating that the idea was not his.

(*To be continued*)

Periscope

Archiv für Psychiatrie und Nervenkrankheiten

(42. Band. 1. Heft)

1. Concerning Variations in the Course of the Pyramidal Tract. BUMKE.
2. A Rare Disease of the Pyramidal Tract with Spastic Spinal Paralysis and Bulbar Symptoms. KINICHI NAKA.
3. A Comparison of the Frequency and Causes of Suicidal Tendency in the Navy and Army. PODESTA.
4. The Condition of the Reflexes in Paralyzed Parts after Total Division of the Spinal Cord. (Continued.) MICHAEL LAPINSKY.
5. Further Investigations on the Anatomy of the Surface of the Human Brain. RICHARD WEINBERG.
6. Contribution to the Nosography and Histo-pathology of Forms of Amaurotic Paralytic Idiocy. KARL SCHAFFER.
7. Contribution to the Knowledge of Vessel Alterations in the Brain Cortex in Psychoses. ELMIGER.
8. Drunkenness and Responsibility. E. MEYER.
9. Concerning Bulbar Paralysis in Lipomatosis. E. OSANN.
10. Stampede of Horses. H. DEXLER.
11. A Contribution to the Anatomy of the Optic Radiations in Man. U. TSUCHIDA.
12. Suicidal Attempts during the Act of Birth. W. SIGWART.

1. *Variations in Pyramidal Tracts.*—Bumke reports a case anatomically in which in a single nervous system he found united nearly all the main types of aberration from the normal of the cerebro-spinal motor tract. The patient was a woman of thirty-six who died from the results in the heart of an articular rheumatism. The paper confines itself essentially to the anatomical findings, and is excellently illustrated by a series of drawings from the midbrain to the lumbar cord, showing motor degeneration which had followed an extensive cerebral softening some time before death. The investigation, which demands special study, illustrates in striking manner the possible wide distribution of motor fibers in certain cases.

2. *Spastic Spinal Paralysis with Bulbar Symptoms.*—Naka describes an unusual disease of the pyramidal tract with reference to the possible occurrence of primary disease of this tract. The case described presented typical symptoms of spastic spinal paralysis, with which in the late stages were associated bulbar symptoms. Anatomically a degeneration of the corticospinal neurones of the motor tract was demonstrated. Although the degeneration of the lateral tracts was by all means the most conspicuous feature of the pathological findings, there was nevertheless slight alteration in the ventral horn cells. There was likewise found marked alteration and diminution in number of large cortical pyramidal cells. It is inferred that the degeneration of these cells was a later process than that of the pyramidal tract, at least macroscopically. The case is of

interest as indicating the possibility of a primary spastic spinal paralysis of which Erb collected but eleven unquestioned cases.

3. *Suicide in Army and Navy.*—A statistical study of the frequency and causes of suicidal tendency in the German navy and army goes to show that the frequency of suicidal attempts is about half as great in the navy as in the army, but slightly higher than among similar persons in civil life. Suicidal tendency in the army is much more frequent during the first months of service and in the recruiting period. The common soldiers are more often affected than the under officers. In the navy, on the other hand, the danger is greater in the later years of service, and the under officers are more likely to be the victims than the rank and file. In the great majority of cases in both army and navy the cause is mental disturbance.

4. Continued article.

5. *Surface Anatomy of Brain.*—Weinberg returns to the question as to whether individual, sexual and other variations of the external brain form may be due to other factors than those generally accepted. The study, which is an attempt toward the solution of the problem, is based on the examination of fifteen male and ten female brains with due consideration of possible pathological factors. As a result of the investigation the writer details twenty points which he regards as significant in relation to variation in brain form. These points concern themselves with fissures, convolutions, anastomoses and such matters of gross cerebral form. The value of such work in general must depend upon the number of brains studied. If few are used, generalizations are manifestly dangerous.

6. *Amaurotic Idiocy.*—In concluding a detailed paper on the nosography and histo-pathology of amaurotic idiocy Schaffer places the severer form as described by Sachs and the lighter as described by Spielmayer as two branches of a great clinical family which may be named the cyto-pathological family amaurotic idiocy. There is, however, as a case described in detail proves, also a teratological form of amaurotic idiocy, although this has nothing in common with the family type.

7. *Vascular Alterations in Psychoses.*—Elmiger, in a brief communication, reaches the conclusion that cell infiltration of the vessel walls of the brain cortex is characteristic of general paralysis. The conclusion is based on the microscopic examination of twenty-eight brains, twelve of which were from cases of paralytic dementia and the other sixteen from various other forms of mental disorder. In the latter cases no vascular cell infiltration was demonstrable.

8. *Drunkenness and Responsibility.*—In a study of the responsibility of persons intoxicated with alcohol Meyer reaches the following conclusions, among others, that a very large proportion of criminal acts, especially those of violence and of a sexual character, are committed during drunkenness; that drunkenness (Rausch) is an acute alcoholic poisoning leading to temporary psychic disorder, in which physical appearances may be lacking. It is advised that, in a formulation of a new law, drunkenness in relation to responsibility be given the same consideration as other psychical disorders.

9. *Bulbar Paralysis and Lipomatosis.*—Osann describes a case of bulbar paralysis with lipomatosis because of its exceptional complexity and its consequent difficulty of diagnosis. The patient had suffered from childhood from a large tumor of the right leg. She was admitted to the

hospital at the age of thirty-eight with symptoms, referable to the brain and spinal cord, of extensive character, including marked disturbances both of motion and of sensation with reaction of degeneration in certain muscles. Bulbar symptoms were conspicuous. The entire illness lasted five months. The autopsy showed multiple lipomata in the thoracic and abdominal cavities and also in the spinal canal. There were in addition marked degenerations of bulbar nuclei, and a compression myelitis at one point with consequent secondary degenerations.

10. *Stampede of Horses.*—Dexler in a contribution to the knowledge of psychoses among animals discusses particularly the well known stampede of horses. The writer considers various theories which have been advanced to account for the phenomenon. The paper in general is an entertaining and useful contribution to animal psychology with somewhat direct reference to man. One question raised, for example, is whether psychiatrists should consider panic among men as belonging to the psychoses.

11. *Optic Radiation.*—Tsuchida presents a detailed anatomical study of the course of the optic fibers in man. It is not possible in the space of this abstract to give the elaborate results of the investigation. The paper is a further contribution to the knowledge of a complex anatomical region. The study was made on the basis of a single patient with porencephalic defect in the occipital region.

12. *Suicide at Birth.*—On the basis of a case of acute mental disturbance during the act of birth Sigwart draws attention to the rarity of transitory mental disturbance closely associated with the act of birth in contrast with the frequency of post-puerperal psychoses. The patient was septic during convalescence but was finally discharged recovered mentally and physically. The author discusses the relation of such transitory psychoses to infections, to the mental and physical strain associated with childbirth and to the frequency of infant murder under these conditions.

E. W. Taylor (Boston).

Journal de Psychologie, normale et pathologique

(Fourth year. No. 6. November–December, 1907)

1. Criminal Responsibility before the Geneva Congress of Alienists and Neurologists. J. Grasset.
2. The Physiological Explanation of Emotion. G. Revault d'Allonnes.

1. *Criminal Responsibility.*—In the report, prepared by Gilbert Ballet and adopted by the Seventeenth Congress of Alienists and Neurologists held at Geneva, August, 1907, a position was taken upon the question of criminal responsibility somewhat at variance with the well-known views of Grasset. The present article is Grasset's reply to his critics. He points out the false logic, the frequent misstatement, and the generally unscientific attitude of his opponents in a way which seems both masterly and conclusive. He reiterates his former views (Journal of Nervous and Mental Disease, February, 1906, p. 135, and March, 1907, p. 210) with an added emphasis; still holding that the medical expert, contrary to the views of the congress, must express his opinion, when giving testimony upon the criminal's responsibility but an opinion that

must be founded strictly upon a medical basis and not upon a social or philosophical basis. The opinion must be based solely upon his knowledge of the actual condition and functional capacity of the prisoner's psychic neuroses at the time of the committing of the crime. All questions as to the amount of punishment to be inflicted to fit the crime and the criminal's degree of responsibility, or as to the requirements of society, for its own protection and vindication, do not concern him as a medical expert. Furthermore, in this capacity, he is not called upon to voice his opinion as to the relationship of responsibility to the philosophical question of free-will. These questions the court alone must undertake to answer.

Grasset insists that medical science has advanced far enough to-day to point out, upon a pathophysiological basis, the degree of responsibility of any given individual. As he says there are responsible, irresponsible and partially responsible criminals. He urges that it is the prerogative and duty of the medical man alone to determine the degree of this responsibility. The court's special duty extends only to the determination of the appropriate punishment and of the best mode of protecting society. The medical expert concentrates his opinion upon the criminal as a physiological being, whether normal or abnormal. The court takes into consideration only the welfare of society. A mutual recognition of each other's special duties and qualifications and a harmonious application of the special functions of both court and medical adviser ought to result in the best for both society and the criminal. As a corollary to his views Grasset again most scientifically urges the establishment of prisons for the responsible criminals, asylums for the irresponsible and prison-hospitals or hospital-prisons for the partially responsible.

2. *The Physiological Explanation of Emotion.*—This is a reply to and brief criticism of Pieron's opposition to some of d'Allonnes' former physiological explanations of the origin of emotion (*Jour. de Psychologie,* Mars.–April, 1906). D'Allonnes denies that Pagano's experiments warrant the arbitrary deduction of Pieron that the caudate nucleus of the striate body is the psychic center for the emotional feelings. He maintains that there are no *psychic centers* in this restricted sense; there are only *psychic functional cycles* or circuitous mechanisms for the elaboration of what we denominate psychic phenomena. In such a psychic cycle the striate body doubtless plays a rôle. Its injury, therefore, will modify the full expression of the emotion, but this does not warrant the statement that this striate body, when separated from the cortex on the one hand and from the peripheral nervous apparatus on the other is capable by itself of causing any emotional feeling. d'Allonnes declares that the same criticism applies to similar deductions drawn from the well-known experiments of Sherrington.

Psychology does not countenance any such narrow conception of the origin and nature of the affective faculties. The author reiterates his view that there are three sorts or types of feeling. He indicates this in an illustrative way in connection with the disgust which Sherrington's dog manifested when given a piece of dog-meat to eat in its milk. There was first a nauseous disgust, then a decided repugnance, and finally a state of mind which d'Allonnes calls an inemotive disgust. Each of these feelings has a particular physiological basis, representing different stages of elaboration in the development of the nervous system.

METTLER (Chicago.)

Centralblatt für Nervenheilkunde und Psychiatrie

(April 1, 1907–April 15, 1907)

Clinical Disease Pictures Simulating General Paralysis.—Dr. Finckh reports two cases whose clinical pictures simulated that of general paresis.

Case I.—Patient is a male, age 59, butcher by occupation and led rather an irregular life. At 30 contracted lues for which he received the usual treatment. In July, 1892, he was admitted to the psychiatric clinic of Tübingen. He stated that for several years he suffered from fainting spells, experienced peculiar sensation in both arms and legs, and not infrequently his eyes closed involuntarily. He appeared confused, spoke slowly and stammered. (No record of physical status.) In October of 1892 he was admitted to another hospital. While there he was able to give an account of himself; he knew his native place, but could not give his age, and was not able to make simple arithmetical calculations. He was euphoric, laughed in a silly manner when questions were propounded to him. The pupils were of medium size; left was smaller than right; light and convergent reaction were prompt. Internal organs were intact. Another examination on January of 1893 revealed unequal and unresponsive pupils, defective speech, ataxic gait, and exaggerated knee jerks. He was loquacious and gave expression to delusions of grandeur which were only transient. A month later he had an epileptic convulsion and following this he became irritable and reacted to olfactory and visual hallucinations. His general mental attitude remained without important changes. At times he was depressed and irritable, but then again euphoric and grandiose. At all times, however, he showed marked evidence of mental reduction. Physical condition showed some variability. In April, 1895, his pupils reacted to light, but in October, 1897, they were described as round, equal and reacting only sluggishly to light; and knee jerks were unequal (right active and left sluggish); tongue was in the median line and showed no tremor, and speech was much impaired. In 1897 Romberg's signs could not be demonstrated. In December of the same year he had another convulsion. From September, 1906, to January, 1907, his physical and mental status showed no important changes. He was euphoric, expressed ideas of grandeur, spoke of having a great deal of money, was oriented as to time and place, and his grasp on surroundings was good. Physically his pupils were equal, irregular and light reaction was sluggish. Right side of face was elongated and no tremor of facial muscles and no Romberg. Speech was scanning and writing tremulous. Cervical and cubital glands enlarged. No arteriosclerosis.

Case II.—The patient is 55 years of age, military man, contracted lues at 27 and was treated with antisyphilitic remedies. In 1882 the left pupil was dilated. Since 1885 he became forgetful, irritable, inattentive, drowsy and showed no capacity for employment. He was slovenly about his personal appearance and was very hypochondriacal. His left pupil was dilated, left side of face weak, speech slow, writing tremulous, tendon reflexes present (later exaggerated), spastic gait and ankle clonus. At times complained of pain in the head and legs. During the further course of the disease the symptoms were variable. At times he suffered with insomnia, was indifferent and apathetic; yet at other times he was optimistic, elated, showed active interest, and had good grasp. Later ideas of grandeur, exalted mood and marked eroticism developed together with hallucinations and distinct clouding of consciousness of a long duration

and insomnia; whereupon quickly a depressive state with grandiose delusions followed. A characteristic change in the history of the disease occurred in 1887 when patient had an attack of fever (typhoid?) with formation of decubitis and pus. This fever lasted ten months. Following his convalescence he became quiet, orderly and tractable, although quite often he had delusions of grandeur and later became hypochondriacal, depressed and manifested suicidal inclinations. For the ensuing seven years (till 1895) his mood was variable. Frequently he was happy, had numerous plans, maintained an exalted attitude and expressed grandiose delusions; at other times he was hypochondriacal, irritable, melancholy and reacted to hallucinations. Physically the left pupil was still dilated and sluggishly reacted to light, left side of face was weak, gait spastic and knee jerks exaggerated. In 1895 some improvement was noticed and he was discharged from the hospital. For several years he reacted to false perceptions. For past ten years he has held a state position as a teacher. A letter received from him in 1906 showed that his intellectual and emotional faculties were intact.

(Evidently the author did not take in person a final mental status of his patient.)

The author makes an exhaustive differential diagnosis. He distinguishes the above disease pictures from all forms of mental disorder save puerperal psychosis (unfortunately his patients were of the male sex). He excludes general paralysis mainly because the diseases were of long duration, and of the marked improvement in memory, grasp and orientation. He classifies them as syphilitic brain disease. His cases are incomplete in many respects; no reference of cytological examination and antiluetic treatment for the neurological disease was made. Beyond doubt, the diagnosis of paresis cannot be eliminated. It is to be hoped that Dr. Finchk will follow up his interesting cases to their terminations and report the pathological findings which will most probably be the ordinary picture of paresis.

(May 1, 1907)

Apropos of "Jung's Psychology of Dementia Præcox" and the Application of Freud's Methods of Psychopathological Analysis. ISSERLIN.

Isserlin in the first part of his paper gives a brief analysis of Jung's book on the psychology of dementia præcox and in the second part he offers a severe criticism of Jung's theories. The book is reviewed in the JOURNAL and the reader may draw his own conclusions.

(May 15, 1907. XXX)

1. Apropos of Residual Symptom and Its Significance. HEILBRONNER. (Utrecht.)
2. Psychology and Psychiatry. SPECHT.

1. *Residual Symptom and Its Significance.*—Wernicke was the first one to differentiate between the disease process and disease product. The residuals in the sense of Wernicke are defective insight and close adherence to the reality of the false ideas. Such a prototype is common to delirium tremens and Bonhoeffer and Lipman have observed such residuals. Heilbronner reports four cases in which residual symptoms have persisted for a certain period of time. The first two patients were alcoholic and suffered from an acute hallucinatory confusion. With the disappearance of

these symptoms the patients maintained that their former delirious experiences were real. The first patient gained a complete insight five weeks after the onset of the psychosis. The second patient had no profound appreciation for his delirious experiences. The third patient was alcoholic and presented evidence of cerebral lues; he developed a delirium from which he recovered, but had no insight into his false ideas for a number of months, when he finally rectified them. The fourth patient was a demented epileptic; he was subject to delirium with hallucinations. With the subsidence of the acute mental disturbance, patient was totally amnesic for that entire period, and hence offered no intelligible explanation for his experiences. The author states that the residual symptom may be eliminated either by process of reasoning or by mere forgetfulness, or it may remain latent for a definite period of time when it may be brought to the surface by some exciting cause. The residual symptom has a great relation to chronic alcoholic psychosis and he enters into a long discussion regarding the latter's various phases. He deals with highly theoretical subtleties and arrives at no definite conclusions.

2. *Psychology and Psychiatry.*—The author believes that the problem of psychiatry resolves itself into two parts: (1) The grouping of mental symptoms, according to modern clinical views, the proper recognition of the disease, and definite relationship of the disease entity to each other and its proper differential diagnostic significance. (2) The explanation of the symptoms and disease picture in accordance with psychological laws in relation to abnormal states. The progress of modern psychiatry is essentially dependent upon the methods of scientific psychology. The objection raised by many that psychological methods are not applicable to psychiatry is rejected by the author. He maintains that a scientific psychiatrist must be thoroughly versed in psychology.

(June 1, 1907. XXX)

1. The Significance of Sexual Trauma in Adolescence in the Symptomatology of Dementia Præcox. ABRAHAMS.
2. A Few Remarks on the Examination of the Ganglia Cells in Fresh State. DE MONTET.

1. *Significance of Sexual Trauma in Adolescence.*—The author reports three interesting cases of dementia præcox, the clinical picture of which showed a distinct sexual undercurrent. In all of them a definite sexual trauma in adolescence could be demonstrated. He is of the opinion that the sexual experiences in childhood do not cause the disease, but simply modify the clinical phenomena. He is not in a position to state whether or not each case of dementia præcox contains a hidden sexual complex of childhood, and says: "A psychological analysis of dementia præcox must be carried on along the lines of Freud's studies; from them important investigations will be adduced."

2. *Examination of the Ganglia Cells in Fresh State.*—The author offers a method for examining ganglion cells in a fresh state without the aid of heat or fixation. The staining material is prepared by dissolving neutral red in a physiological saline solution and heated to 30–40° C. A thin lamella of the ganglion is cut with sharp double knife and placed on a slide which was previously warmed. A few drops of the warm staining solution is put upon it and one minute later a cover glass is applied. The microscopic picture is distinct; the achromatic substance is of a light rose color; the nucleus is of a darker hue; the nucleolus is intensely red; the protoplasm between the layers shows fine granules.

(June 15, 1907. XXX)

1. Method for Fixation of Cellular Elements of Cerebro-spinal Fluid. ALZHEIMER.
2. Apropos of Death in Katatonic Seizures in Old Dementia Præcox. DREYFUS.

1. *A Method for Fixation of Cellular Elements of Cerebro-spinal Fluid.*—The author offers the following method for fixation of cellular elements of cerebro-spinal fluid: In a centrifugal tube with a conical base is put 10–15 cm. of 96 per cent. of alcohol and then 5 cm. of cerebro-spinal fluid is added. The tube is closed with a plug of cotton. It is allowed to be centrifuged for one half or three quarters of an hour. Then it will be noticed at the bottom of the tube: a distinct coagulum, which is marked in general paralysis, cerebral syphilis and meningitis, but which in normal subjects has the thickness of ordinary paper. The alcohol is poured off and the coagulum is fixed with absolute alcohol, ether and alcohol and ether. The coagulum becomes thick; it is taken out with a fine needle and embedded in celloidin and then sections are made. The following staining material may be used: Unna's polychromes, methylene blue, Unna's modification of Pappenheimer's stain, or methyl alcohol. Without encountering special difficulties any of the following agents may be substituted for alcohol: sublimate following with triacid stain, Flemming's solution and Altman's stain, or Formol-Müller and Zenker's fluid, Giem's stain after Stridde. For the last two fixing materials paraffin is used. The coagulum is passed through xylol. For alcohol fixation the sections are cut from 10 to 15 microns thick, for other fixations 3 to 5 microns thin.

This method offers great advantage for the study of different kind of cells. Karyokinesis can be observed. Plasma cells can be demonstrated. Manifold forms of cells may be met which were heretofore not classified. A better understanding of the significance of the free cells of pia mater can be gained.

2. *Death in Katatonic Seizures in Old Dementia Præcox.*—The author does not agree with Tetzner that katatonia with convulsions belongs to epilepsy or paresis. He reports a case of dementia præcox, katatonic form. The patient was 31 years of age, the clinical phenomena presented the picture of katatonia and physically there were no signs indicating organic brain disease. He had an attack of convulsive seizures which was soon followed by death. Autopsy revealed no pathological conditions of the viscera and the brain showed no paretic process. The cerebro-spinal fluid was scanty, the ventricles were comparatively contracted and relative diminution in volume between the cranial contents and the weight of brain was apparent. Normally the difference between cranial contents and the weight of brain amounts to 10–15 per cent., whereas in this case it was only 1.2 per cent. The cause of death as well as the convulsions is attributed to acute swelling of the brain.

M. J. KARPAS (Ward's Island).

Revue de Psychiatrie et de Psychologie Expérimentale

(March, 1907)

1. Surgery Among the Insane. L. PICQUÉ.
2. Remissions in Dementia Præcox. MLLE. PASCAL.
3. Epileptic Attacks Following Head Injury.

1. *Surgery Among Insane.*—A plea for surgical intervention in suitable cases among the insane confined in asylums. Three quarters of the insane suffer from surgical troubles and they certainly should never be allowed to die without having proper treatment.

2. *Remissions in Dementia Præcox.* (Continued.)

3. *Epilepsy Following Head Injury.*—The author says there is a marked analogy between the pathology of idiopathic epilepsy and traumatic epilepsy. If this is so one ought to encounter more cases of larvated epilepsy among the cases of the ordinary convulsive variety. A case is reported following a traumatism in which there appeared successively convulsive, procursive and psychic epilepsy, all three forms favorably influenced by bromide.

Miscellany

STUDY OF APHASIA. Besta (Morgagni, 1907, January).

Besta reports this interesting case of motor aphasia from the psychiatric institute in Reggio Emilia. It showed a slight spasmodic paresis extending over the entire body musculature, with moderate increase of tendon reflexes and no Babinski's sign, in addition to a subcortical motor aphasia without mental involvement. After death a symmetrical lesion involving the inner segment of the nucleus lenticularis (globus pallidus) of both sides, and a secondary degeneration of the strio-caudate and striothalamic fibers in the lemniscus was found. The pyramidal tract appeared to be not at all involved. Besta concludes that the motor pathway for speech is very probably autonomous; that it goes, either with or without interruption, through the globus pallidus, above all thereupon through the principal lemniscus.

C. E. ATWOOD (New York).

ISOLATED PARALYSIS OF SERRATUS MAGNUS. Henri Claude and Paul Descamps (L'Encephale, 1907).

This is a careful clinical, anatomical and etiological study of the case of a cloth-cutter paid by piece-work, who, being short of money, over-worked for several days. During this period he had a febrile attack. Ensuing upon this, crackings beneath the right shoulder blade occurred, followed by pains which soon extended along the distribution of the radial nerve. They came on at night and were relieved by movement. Suddenly he noticed the lower angle of the scapula project. This alarmed him. He consulted, and after a course of sulphur baths the pains disappeared, but the deformity persisted. The authors attribute the palsy which is accompanied by R. D. to a degeneration of the long thoracic nerve, the origin of which was a rheumatismal affection of the subscapular bursa along which it has an extensive course. They draw attention to the great diagnostic importance of these sub-scapular rubbings.

TOM. A. WILLIAMS (Washington, D. C.).

Book Reviews

PATHOLOGISCH-ANATOMISCHE UNTERSUCHUNGEN ÜBER AKUTE POLIOMYELITIS UND VERWANDTE KRANKHEITEN VON DEN EPIDEMIEN IN NORWEGEN, 1903–1906. Von Francis Harbitz, Professor der pathologische Anatomie an der Universität zu Kristiania, und Olaf Scheel, I Assistent am pathologisch-anatomischen Institut zu Kristiania. Jacob Dybwad, Christiania.

In this comprehensive monograph of some two hundred pages, well illustrated by plates and figures in the text, the authors have made an analysis of the pathological material that has come to the pathological institute in Christiania for the years 1903–1906 during which epidemics of varying intensity have been prevalent in the northern Scandinavian countries.

As the senior author of this paper in 1898 made a complete study of the cord changes in this disorder, and as the appearance of Wickman's study in 1905 has rendered it less necessary to go into a detailed study of the histological changes in the cord the authors have busied themselves particularly with those conditions showing a pathological as well as etiological relationship with poliomyelitis, such as other forms of myelitis, multiple neuritis, bulbar paralyses and encephalitis. Special attention has been paid to the problem of etiology.

The basis for the study consisted of 19 cases, 17 of which were typical acute poliomyelitis, in a number of which the bulbar nuclei being involved.

Speaking of the symptomatology the authors call attention to the general occurrence of exanthematic prodromata in all of their cases, arguing they hold for a general infection. Sore throats were very common, and acute diarrheas equally prevalent. Contagiousness they hold is proven. Over one thousand cases have been under observation in Norway in the past three years.

We cannot well summarize the pathological findings in this place. The authors are not able to posit an organism in the nervous system, although they quote Geirsvold's positive findings at length, but the type of reaction throughout is one induced by the bacterial toxins. It is an acute, interstitial, exudative, and infiltrating inflammation, the specific action on nervous tissues having the character of a definite neurotoxic nature.

The monograph is a thorough and scholarly production and in view of our own recent epidemic in the United States can be read with profit.

JELLIFFE.

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Original Articles

POSTERIOR COLUMN DEGENERATIONS FOLLOWING INJURY TO THE POSTERIOR ROOTS OF THE SEVENTH CERVICAL NERVES

By H. W. Mitchell, M.D., and A. M. Barrett, M.D.,

FROM THE LABORATORY OF THE DANVERS INSANE HOSPITAL

It is always of value to add to the literature of the anatomy of the nervous system those cases of well-defined lesions which can be studied by the method of Marchi, and which bear on the unsettled problems of localization and fiber relations.

The following contribution is a study of the degenerations of the posterior columns of the spinal cord, resulting from an injury to the posterior roots of the seventh cervical nerves.

The material for the study came from a patient of the Danvers Insane Hospital, who was admitted there, into the service of one of us, Mitchell, in November, 1905, and died twelve days later. The patient was a man aged 47, who for ten years previous to his admission had been a hard drinker and, unable to hold any position, had led a vagabond life, and on two occasions had been an inmate of the Foxboro institution. When about 45 years of age he had a convulsion, while intoxicated, and last April had an attack of delirium tremens. During the night of October 26, while sleeping in a hay mow with drunken companions, he fell some distance to the floor and on the following morning was picked up in an unconscious condition by his companions. Later in the day he walked some distance to a friend's place of business, where he spent the day and night. He complained of feeling badly and it was observed that his head was turned to

the side and bent in a peculiar position. On the day following he walked about two miles to a physician's office. His arms were then weak, he was unable to feed himself and it was difficult for him to rise from a chair. He was sent to a hospital and on November 1 was admitted to the Danvers Insane Hospital.

A summary of the physical examination made on the day following showed that he had a temperature of 102, pulse 98, respiration 30. As he lay in bed his head was turned to the right and drooped towards the shoulder. Save for a slight harsh systolic murmur, examination of the chest gave nothing important. The bladder was distended and there was a constant dribbling of urine. The urine had a trace of albumin.

The neurological examination showed that there was marked paralysis of the arms and legs. The right arm was held stiffly in a flexed position and could only be raised a few inches. The left arm was even more helpless. The legs could not be moved voluntarily. There was some twitching of the muscles of the right leg. Attempts to correct the position of his head caused pain which he referred to the back of his neck. Eye movements were not impaired, more than that there was a slight nystagmus on forced movements. Pupils were equal and reacted to light stimuli. Arm and knee jerks were not obtained. Cremaster and plantar reflexes present. No Babinski reflex. There was no control of bladder or bowel. Electrical reactions were not abnormal. Subjectively, he complained of prickly feelings in both arms and legs. His reaction to sensory stimuli was diminished over the entire body. Pressure over nerve trunks was not painful. He appreciated difference in pressure, but differentiated very poorly between sharp and dull points. He made many mistakes in recognizing between heat and cold. Position sense was not impaired. No astereognosis.

At this time his mental attitude was not peculiar except that his memory was uncertain for the events of the previous ten days. On the day following his temperature rose to 103 and he began to have visual hallucinations, imagining that he saw a bottle in his bed. His grasp on questions became unclear, he was dull, and it took stronger stimuli than normal to gain his attention. A few days later he developed bed sores, and the urine gave evidences of a severe degree of cystitis. By November 10 he had failed much. His stupor was more marked and his speech

was difficult and his tones feeble. The right arm could be stirred a little, but otherwise all voluntary movements were absent in arms and legs. Below the level of the second rib, excepting for the outer surface of both arms, pin pricks produced no pain reaction (Fig. 1). Over the arms he recognized light touches and appreciated difference in pressure. With temperature tests he made many mistakes. The tendon reflexes remained absent. Swallowing became more difficult, and on November 12 he died in coma.

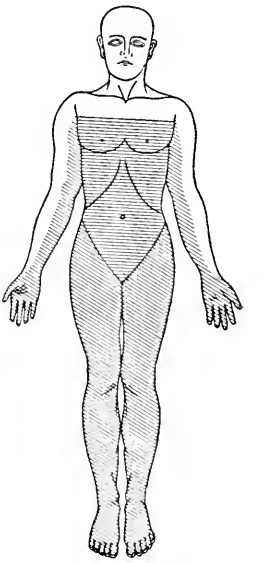

Fig. 1. Shaded Area Shows the Extent of the Sensibility Disturbance.

The autopsy was held within a few hours of death. It was found that both arches of the fifth cervical vertebra were fractured at the point of juncture with the articular processes and that they were easily movable. Around their broken ends was a slight degree of hemorrhagic exudate. All other vertebræ were intact. On opening the spinal canal, there was no narrowing at the point of fracture. The cord was swollen and for a distance of about 15 mm. filled the canal tightly. The dura mater here had a slight transverse fold, evidently marking the point where the cord had been directly injured. On opening the dura, the sixth and seventh cervical segments were a little larger and softer than those adjacent.

The brain showed no gross abnormalities.

The chief findings in the internal organs were: Acute hyperemia of the spleen; multiple small abscesses of both kidneys; pyelitis; cystitis.

The material from which the following descriptions were made was prepared by the method of Marchi (Fig. 2). But, in addition, preparations were made by the method of Weigert, and by methods to demonstrate the cell structure of the horns.

The seventh cervical segment presented the appearance of severe injury. Its contour was somewhat distorted and various areas of blackening showed the presence of degenerated fibers.

On the right side the anterior horn appeared a little crushed and both anterior horns were dotted with black. In the anterior commissure were blackened fibers and on both sides the anterior roots were degenerated and their course was marked by blackened streaks. With nuclear stains, the cells of the anterior horn, except the anterior mesial group, presented the characteristic picture

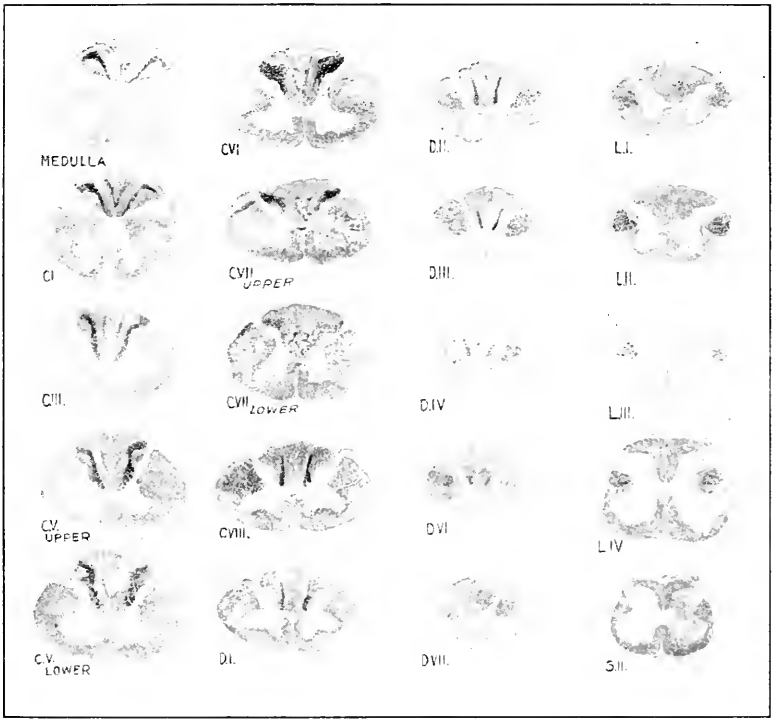

FIG. 2. Posterior column degenerations following injury of the seventh cervical segment. Above the injured segment ascending degeneration of the seventh cervical nerves. Below the injured segment descending degeneration of comma tract.

of the axonal reaction. The posterior horns did not appear injured. In the anterior columns of this segment there were scattered blackened fibers. Nowhere were these grouped in any considerable numbers, but they were more abundant in the zone bordering the anterior horns. In the lateral columns were several clumps of degenerated fibers, best marked in the upper part

of this segment, and both lateral columns were vacuolated and showed severe injuries.

In the posterior column, instead of the common picture seen in crushing of a segment, and where the whole column was more or less filled with blackened fibers, the greatest changes were here present in the entering root fibers. In the upper part of the segment degeneration was better marked than in the lower. Almost all the entering posterior root fibers were blackened. The greater part of these curved around the tip of the posterior horn, while a few turned into the gray substance.

The sixth cervical segment showed no sign of direct crushing. Both anterior horns were sparsely dotted with black, and with nuclear stains, the cells of the horn showed the same changes as were present in the seventh segment. In the anterior commissure were a few blackened fibers. In the anterior and lateral columns the degenerated fibers lay chiefly in the regions bordering the gray substance. The entering zone of the posterior roots was thickly dotted and streaked with blackened fibers, the whole area spreading more mesially than in C VII. While the greater part of these were seen in cross section, some were arranged in streaks and were probably a part of the sixth root. The degenerations in the median part of the column were more compact and had a streak-like form.

In C V the broad club-shaped dorsal end of the degenerated zone was hollowed out on its outer side, marking the region where undegenerated root fibers had come in. The section from the upper part of the segment showed this still more marked and the area had already taken on the ⌊ shape seen in the intraspinal course of posterior root fibers. The median areas of degeneration appeared as two streaks diverging dorsally from the median septum. They lay at about the border between the columns of Goll and Burdach.

In C III the root degenerations had taken a more mesial position. The median areas of degeneration appeared much as in C V.

In C I the root degenerations had a pronounced ⌊ shape, and no longer reached the dorsal margin. The median areas were shorter, each curved a little outward, and ventrally met in the median line. In the lower medulla the root degeneration ap-

peared much as in C I, but the median degeneration was far less marked.

Below the point of injury to the cord the eighth cervical segment showed no sign of direct injury. The anterior horns were dotted and streaked with black flecks in their outer parts, and on the right side the fibers of the anterior root which passed to the outer edge of the mesial angle were blackened. A few blackened fibers lay in the anterior commissure. The posterior horns showed a few blackened fibers extending inward from the inner angle of the horn. In the columns of the cord the antero-lateral ground bundles were sparsely dotted with black. But in the region of the lateral pyramidal tract degenerated fibers were abundant.

In the posterior columns the entering root zone showed no degeneration. Along the outer side of the paramedian septum in each posterior column was a club-shaped area of blackened fibers. Each was widest at its ventral end, where degenerated fibers extended quite up to the curving margin of the gray substance. Dorsally it tapered out a little distance from the dorsal margin. From the position of these areas and from their form they correspond to the comma tracts of Schultze.

In lower levels these tracts became progressively shorter and more tapering, and were traced into the sixth dorsal segment. The degenerations in the lateral pyramidal region were visible as low as the second sacral segment.

The foregoing case presents two interesting anatomical points: First, while the cord at the level of the seventh cervical segment was considerably injured in the anterior, and more especially the lateral regions, the injury to the posterior columns in a peculiar way involved chiefly the entering posterior roots of the seventh cervical nerve, together with a few of the fibers of the sixth, the remainder of the column showing only a small degeneration in the median part. The picture presented in Marchi preparations corresponds with the figure of the degeneration of the intraspinal part of the posterior roots, which has been described after experimental section of the posterior roots, and in a few cases of disease of the roots in their extra-spinal course, and showed the ascending course of the long fibers of the seventh and sixth cervical nerves in the posterior columns. In this course they lay entirely in the column of Burdach in about its

median part, and their progressive approach to the median line in their ascending course bears out the already well-established rule of the median displacement of the posterior roots by those fibers entering in higher levels.

The only explanation which can be here offered for the peculiar and system-like median degeneration seen in Goll's column is that it is an ascending degeneration of those fibers in the middle of the posterior column which were injured at the level of the fracture. It seems strange that such an irregular and slight degeneration as is present at the level of injury should assume such a compact tract-like course as is here the case.

The second and more important feature of this case is the comma tract degeneration, and its relation to the degeneration of the posterior roots. The slight injury to the posterior columns as a whole, and the marked involvement of the entering nerve roots has an important bearing on the origin of the fibers of the comma tract, and their exogenous origin. Three opinions are maintained in the literature as to the origin of the comma tract—first, that they are of endogenous origin, *i. e.*, fiber processes of cells of the posterior horn (1); second, that they are the downward continuation of fibers of the posterior roots (2); third, that they are made of both endogenous and exogenous fibers (3).

The conditions present in this case seem to lend evidence to their exogenous origin, because of first, the marked degeneration of the posterior nerve roots as they enter the cord, and second, a lack of any significant direct injury to the posterior columns or the nerve cells of the posterior horns, none of which show the changes which are characteristic of an injury to the fiber process of a cell.

The comma tract degeneration extends to the sixth dorsal segment, and in this extent agrees with two other cases in the literature, one (1) in which the seventh cervical segment was injured and the other (4) in which the posterior root of this segment was injured, in both of which the comma tract degeneration was traced into the sixth dorsal segment.

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OSSEOUS PLAQUES OF THE PIA-ARACHNOID AND THEIR RELATION TO PAIN IN AKROMEGALY.

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Since Marie in 1886 first named and described the symptom-complex known as akromegaly, many and numerous have been the observations reported. The pathological findings as well as the symptoms have varied. The major clinical features, such as increase in size of the extremities, nose, lips and lower jaw, however, we now consider as distinctive.

Among the lesser symptoms, pain seems to play an important role. It was Sainton and State (1) who first called attention to its prominence. In analyzing the cases in literature, they found it present in 50 per cent. of the cases. Sousa Leita (2), Sternberg (3) and others considered it for the most part as a transient symptom. Osborne (4) believes that almost every case of akromegaly has pain more or less constant in some portion of the body, but he included head pains.

As to the pathology of these pains, Sainton and State believe them to be due to the presence of osseous plaques in the spinal pia and to the changes, mechanical and irritative, caused by them upon the nerve roots and substance of the cord. This I do not believe to be correct, and it is the object of this article to show that there is no definite pathology of the spinal cord in akromegaly, and also to show that these plaques play little or no part in the production of the pain of this disease.

The pains referred to by Sainton and State were distributed sometimes in the extremities, sometimes in the spinal column, and sometimes in the joints. They divided them into five classes—osteo-arthritic, neuralgic, muscular tabetic and akro-paresthetic. The findings in the case they report include the osseous plaques in the spinal cord together with degenerations of Goll's tract and Gowers' tract. The role of these plaques they suggest, is prob-

ably mechanical, and they cite the similar findings in cases reported by Duscheneau, Henriot, Finzi and others.

To observe whether mechanical pressure exerted any influence I examined several nerves lying under these plaques and found no evidence of thickening or any degenerative change. The spinal cord in two cases, examined in the lumbar and cervical regions, showed only a slight increase of the neuroglia, and the presence of arteriosclerosis; this latter accounting for the neuroglia increase. Other cases in literature showed a similar picture. A review of the pathological findings, as collected by Sternberg (5), Hinsdale (6), Brooks (7), Arnold (8) and others shows that changes in the spinal cord are inconstant, the cord being normal in many cases—Packard (9), Cagnetto (10), Strümpell (11), etc.; in other instances showing varying degenerations of the tracts—Sainton and State (12), Adler (13), Cagnetto, Arnold; in others syringomyelia, and in Barrett's (14) case the picture of a severe anemia. In the case I report only arteriosclerosis was to be noted.

Furthermore, plaques are very frequently present in many diseased states and in conditions in which no pain is present. Virchow (15) long ago emphasized this. "For a long time," he says, "they were thought to be the cause of tetanus, chorea and all other forms in which pain was a prominent symptom." They reasoned that the presence of the bone and its processes caused a strong irritation of the nervous system. "This is an error because we frequently find them present in individuals who have not the slightest symptoms."

Zanda (16) was able to collect sixty-three cases showing osseous plaques and found that half occurred in mental disease, that they were frequent in retrogressive changes, as old age, chronic insanities, diseases of the spinal cord, tuberculosis, marasmus and chronic suppuration.

The giant, Wilkins, reported by Bassoe (17), gave no history of pain throughout his life and yet many thickened, partly calcified plaques were found, one even adherent to a nerve root. Microscopically, there was a moderate amount of diffuse degeneration of the spinal cord. Linsmayer's (18) case of akromegaly showed osseous plaques with no definite microscopical changes in the spinal cord or nerve roots save arteriosclerosis and clinically there were no symptoms of pain. Strümpell's (19) and Cagnetto's (20) cases showed a similar picture. In the case reported by

Pearce Bailey (21) pain was a prominent symptom in the hands and feet, radiating to the shoulder and to the hips; yet the spinal cord, grossly, showed no changes, and microscopically showed only thickening of the arteries. In Packard's (22) case, sections of which I have studied, the pains were only present during the period of onset, a duration of three to five years. Since that period the patient had been free from pain. The examination of the spinal cord by Dr. Spiller (23) showed grossly numerous plaques, and microscopically no degenerations.

It seems to me that we must look elsewhere for the explanation of these pains. In reviewing the pathological findings of these cases of akromegaly, the prominent feature of arteriosclerosis should be noted, and the trophic changes incident thereto, in the joints, muscles, viscera, skin and spinal cord could easily explain this symptom. In the arthritic pains the excessive production of lime salts, dependent upon arteriosclerosis and also upon the retention of lime salts and phosphorus in akromegaly, as shown by Moraczewski (24), explains the pathology of pain in the joints.

Arnold (25) and Cagnetto (26) and others have examined the muscle and have shown the presence of connective tissue overgrowth and degeneration of the muscle substance itself. The presence of a chronic myositis here might easily explain the incident of muscle pains in any period of the disease.

The question as to the origin of these plaques is of some interest. Virchow (27) long ago thought them direct transformations of connective tissue into bone. Rokitansky (28), Ziegler (29), Schmaus (30) simply mention their frequency in the pia-arachnoid and describe their gross appearance. Zanda (31) has found that the bone formation takes place from the dura after adhesion with the arachnoid is formed. In the cartilaginous period they have no connection with the dura. In the stage of ossification, blood vessels from the dura grow into them. Their frequent presence in retrogressive conditions, such as senility, uremia, mental disturbances, in fact, in all those conditions in which arteriosclerosis is associated or plays a leading part, indicates clearly to me the natural overgrowth of connective tissue from passive congestion and chronic irritation, and in such areas of connective tissue overgrowth lime salts are frequently deposited. Among the favorable situations for this process are the blood vessels, lymph

nodes, lungs and spinal cord. The associated finding of arteriosclerosis in many cases of akromegaly indicates clearly one of the predisposing factors for the production of osseous plaques.

The first case I wish to report shows the presence of these osseous plaques in uremia and arterio-capillary fibrosis. The clinical history of this case unfortunately is very meagre. The patient, D. B., an old man, was brought to the Philadelphia General Hospital in an unconscious condition. He lived three days. The physical examination by Dr. Potts showed no paralysis of face or extremities. The diagnosis of uremia and chronic interstitial nephritis was made and was corroborated at the necropsy. General atheroma of the cardio-vascular system was also noted, together with chronic endocarditis. Examination of the spinal cord showed numerous plaques covering the posterior surface from the cervical region to the lumbar. These measured from 1 to 3 cm. and were cartilaginous in appearance; a few had the firmness of bone.

Microscopical Examination.—Transverse and longitudinal sections were made of these plaques, and stained with eosin-hematoxylin. Those cut transversely show a central zone of osteoid material; surrounding this are layers of fibrous tissue, hyaline in character. In this are to be noted a few cells, resembling osteoblasts. The upper surface of the section shows only a few connective tissue cells; the lower shows a more active proliferation of connective tissue. In this region the plaque was in contact with the substance of the cord. This proliferation occurs in the pia. No evidence of true bone is to be seen.

The spinal cord was studied with Weigert, hemalum and Nissl stains. A moderate increase of neuroglia in certain parts of the column of Goll of the cervical section and arteriosclerosis are the chief features noted. In the lumbar region the arteriosclerosis is not so marked. There is marked thickening of the spinal pia throughout.

A study of the spinal nerve roots under these plaques showed no evidence of mechanical pressure. No changes were to be noted by the Weigert or hemalum stains.

In the second case, one of akromegaly, the clinical features reported by Dr. Packard in the *American Journal of Medical Sciences*, 1892, and by Dr. Spiller pathologically before the Philadelphia Neurological Society, showed these plaques grossly. Micro-

scopically, by the Weigert, hemalum and Marchi's method no evidences of degeneration were to be noted.

The clinical notes by Dr. Packard, 1892, may be briefly summarized. Patient was first seen in 1885. In 1877 had pains all through his body, which were supposed to be rheumatic. Since that time until 1885 had vague pains through his body with gradually increasing weakness of the legs. In 1892 he was again examined. Aside from headache no other symptoms of pain were noted. Temperature, pain and tactile sensations in the extremities were good. Knee jerks were absent. His general health was farly good. Beside the distinctive features of akromegaly no other symptoms relating to the subject are to be noted.

Examination of the cord in the lumbar and sacral regions failed to show any degeneration.

To summarize briefly:

1. Osseous plaques are frequently present in the pia-arachnoid.

2. They are found in many diseases, such as uremia, tuberculosis, retogressive conditions, etc.

3. Arteriosclerosis seems to be the underlying factor in their causation.

4. The presence of these plaques upon the spinal pia in akromegaly does not explain the production of pain in that disease.

5. There is no definite pathology of the spinal cord in akromegaly.

I wish to thank Dr. Spiller, under whose direction this work was undertaken, for the privilege of reporting these cases, and for his assistance in the examination of the material.

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Society Proceedings

THE AMERICAN NEUROLOGICAL ASSOCIATION

The Thirty-fourth Annual Meeting held at the College of Physicians of Philadelphia, May 20, 21 and 22, 1908

The President, Dr. Charles W. Burr, in the Chair

(*Continued from p. 534.*)

A SPECIAL DIAGNOSTIC PHENOMENON IN THE ATTITUDE OF THE HEAD IN CEREBELLAR DISEASES. REPORT OF SIX CASES, FOUR OF WHICH CAME TO AUTOPSY

By Alfred Gordon, M.D.

In cerebellar diseases the head not infrequently assumes a definite position; in the majority of cases it is inclined towards the side of the lesion; tumor, hemorrhage, etc. This symptom, together with cranial nerve involvement, changes in the reflexes, hemiasynergy, tendency to fall towards the same side, constitutes a symptom group, characteristic of cerebellar diseases. Dr. Gordon has observed that shortly after the beginning of cerebellar disease, or some time later, or else shortly before death, the head changes its position: instead of remaining inclined to the side of the lesion, it turns to the opposite side. Then the least voluntary or involuntary attempt to turn the head to its original position provokes intolerable pain and vertigo which are immediately relieved by permitting the head to return to its newly acquired position. Dr. Gordon observed this phenomenon in six instances. Four cases came to autopsy and the tumors were found on the side toward which the head was originally inclined. Two cases were only clinical, and in them the phenomenon was the same as in the first four. The importance of this manifestation is evident. If for example other symptoms point to a lesion of the left side of the cerebellum, but the head is inclined to the right and an attempt is made to turn the head to the left with the result of increase of pain and vertigo,—it is to be presumed that the lesion is on the left side. This phenomenon will also be useful for diagnosis in cases in which other symptoms are wanting or are very slight.

While four anatomico-clinical cases are not sufficient for generalization, they deserve attention from a diagnostic standpoint.

Discussion

Dr. Joseph Collins asked in what way the position of the head Dr. Gordon described differed from that pointed out by Hughlings Jackson twenty-eight years ago and which has since been corroborated by all writers on disease of the cerebellum: and more than that, how it differed

from the statements of Bruce, who has recently reviewed the works of Jackson, and who says in his last contribution to the subject that this position of the head which Dr. Gordon describes is quite pathognomonic. Wherein should we give position of the head greater diagnostic significance than we have been accustomed to give it?

Dr. William C. Krauss said he had seen cases in which this position of the head was present in a remarkable degree. In one case choked disk came on late in the course of the disease and on the side opposite to the position assumed. In the second case with all the symptoms of cerebellar disease present, an operation was performed and no tumor found in the cerebellum. On autopsy a tumor was discovered pressing on the superior cerebellar peduncles cephalad of the tentorium producing the same symptoms as if situated in the cerebellum itself.

Dr. M. Allen Starr speaking of the staggering as indicative of the situation of the tumor, said some twelve or fourteen years ago he made a collection of forty cases of cerebellar tumor in order to try to determine whether the direction of the staggering gave any indication as to the side of the tumor. In one third of the cases the patient staggered toward the tumor and in two thirds of the cases he staggered away from the tumor. That statement has been repeated in many subsequent articles, and he has taken pains to keep track of cases of cerebellar tumor published since. He has now about 100 cases and has come to the conclusion that absolutely no conclusion can be reached in regard to the direction of the staggering as an indication of the position of the tumor, and he thinks that is borne out by Dr. Gordon's statements, because in two cases the patient staggered to the left and in two to the right.

Dr. Joseph Fraenkel said that forced movements and forced attitudes of the head or extremities are commonly observed in cerebellar disease. The general diagnostic value of these motor phenomena is undisputed and has been frequently described in literature. More debatable, and hardly yet to be used as a guide, is the value of these phenomena as indication of the exact location of a lesion. The opinions concerning this question, to be found in the literature, are still very contradictory. The physiological literature of this question makes statements quite contradictory to those found in the clinical literature. And even the latter is by no means unanimous. To Dr. Fraenkel it appears that the contradictions are explained by the different nature of the lesion—irritative or depressant; by the different location—in the vermis, on the cortex, or subcortical, and finally, by the varied duration of a lesion—whether at the beginning, when compensatory efforts are developing, or when fully established.

Dr. Frank R. Fry, stated that Dr. James Collier in *Brain* in 1904 had made an observation to the effect that often intracranial tumors of large size or of long duration were capable of pushing the cerebellum down and backwards until portions of it and the pons were actually engaged in the foramen magnum, even to the extent of being indented by the margin of the foramen. In this way false localizing signs were produced in some cases. Doctor Fry in 1905 had reported to the association an instance of this kind, where a large frontal-lobe tumor had by pressing on the cerebellum produced very complete symptoms of a cerebellar tumor. (JOURNAL OF NERVOUS AND MENTAL DISEASE, October, 1905.)

Dr. Philip Coombs Knapp said that in a good many cases of tumor

in any part of the brain a change in the position of the head will cause an increase both in dizziness and headache. A patient with tumor of the occipital lobe, for example, had a very peculiar way of rising from the bed, because, unless he moved in a particular way he would have a very severe attack of headache and dizziness. His experience had usually been that the headache and dizziness were increased, when the head was turned to the side opposite the tumor, so as to bring the tumor higher than the rest of the brain, although the opposite rule occasionally obtained. In cerebellar growths, which ordinarily are attended by a greater degree of dizziness than growths in the cerebrum itself, these symptoms might be exaggerated.

Dr. J. Ramsay Hunt stated that he had observed fixation of the head in cerebellar lesions in two cases of softening, in which the patients had recovered. He regarded it as an attempt to fix the head and in this way to compensate for the loss of cerebellar function. He has, however, never been able to determine any fixed position that might be used as a localizing factor in the cerebellum itself. He thinks that the position of the head as a localizing factor in tumors especially should be used with a great deal of care, because such a fixed position of the head may be produced by meningeal irritation alone; which is so common a symptom with all organic affections of the posterior fossa.

Dr. C. E. Beevor thought the position of the head so very uncertain that it is not of very much value for diagnostic purposes. In a very large number of cases there is no characteristic position of the head. Pressure on the medulla oblongata and pons may occur, and in some of the cases Dr. Gordon had recited Dr. Beevor gathered that the Babinski phenomenon was present; that is a phenomenon which he thinks we can never get from pure cases of cerebellar disease, therefore in some of Dr. Gordon's cases the condition present must have been something else.

Dr. John H. W. Rhein stated that Schmidt had described a symptom-complex which he called "Schmidt's Syndrome," consisting of the development of dizziness and vomiting when the patient would lie on the side opposite a cerebellar lesion. These symptoms disappeared if the patient turned over.

Batten, apropos of a case of cerebellar tumor, concluded that special attitudes were not rare in cases of cerebellar lesion. He observed that the ear was drawn to the shoulder opposite the lesion, and that the face was turned to the side of the lesion. He did not believe this was pathognomonic.

Dr. Rhein was convinced that the positions of the head were not pathognomonic of cerebellar lesions. In two patients under his recent observation the head was retracted. In one of the cases there was tumor of the middle fossa with no involvement of the cerebellum, and not sufficient hydrocephalus to cause this symptom; and in the other case there was hydrocephalus with cystic formation at each cerebello-pontile angle.

Dr. T. H. Weisenburg said he had recently collected and published a number of cases of cerebellar tumor. In none was the so-called cerebellar attitude of the head a diagnostic feature. In two of his cases the head was held in the so-called cerebellar position but this was due to diplopia. When it was corrected the attitude promptly disappeared.

Dr. Gordon, in closing the discussion, stated that he wished to emphasize in his paper that in many cases of cerebellar tumors the head is inclined to one side and frequently the lesion is on the side to

which the head is inclined. He observed in two clinical and four anatomico-clinical cases watched for a long time, that at a certain period of the disease the head turned to the opposite side. He also noticed that when he attempted to put the head on the opposite side to which the lesion pointed there was increase of vertigo and headache. The patient instinctively turned his head to the opposite side. The turning of the head to the opposite side Dr. Gordon thought is novel and deserving attention, as it may be of a localizing value. He gave no explanation of the phenomenon.

Dr. B. Sachs read a paper entitled: Two Cases of Aphasia Relieved by Operation and their Bearing on Modern Theories of Aphasia. (To be published in this JOURNAL.)

DISCUSSION

Dr. H. H. Hoppe said that he wished to report a similar traumatic case of aphasia in which the aphasia became progressively worse. The case was the result of a depressed fracture under the temporal muscle which was overlooked at the time of the accident, and two or three months later the man presented a mild form of sensory aphasia. He was a German who had acquired the English language and had lost his power to name things in German, but not in English, and during the next few months he lost progressively his power to name objects in English, therefore it was determined to trephine. Over the seat of the injury there was found a little spicule of bone around which a cyst had developed, not larger in size than a hazel nut. The patient recovered from the operation but never completely regained his use of the German language; there was always some defect in naming objects.

Dr. Spiller presented a brain from a case of motor aphasia in which the lesion implicated Broca's area and the anterior part of the island of Reil without extending into the lenticular nucleus. Only to this extent was Marie's lenticular zone involved. (To be published in this JOURNAL.)

Dr. Joseph Collins said that so far as any evidence was presented by Dr. Sachs that he could comprehend the first case was not a case of aphasia at all, but a case of anarthria. Dr. Collins does not understand that a man can have aphasia who has a full and undisturbed capacity of internal speech, nor does he recall that anyone else holds that view. If this man had anarthria and agraphia he probably had a lesion of that part of his cortex which sends motor neurones down through the internal capsule which go eventully to the periphery of the body, and especially to those peripheral parts that are devoted to externalizing speech. Nor is Dr. Collins at all convinced, even if he thought it were a case of aphasia, that any evidence had been offered to show that the lesion did not affect the lenticular zone of Marie. Given a case which has a hemorrhagic disorder of the cortex which results in the formation of a cyst he sees no reason why there should not be similar hemorrhagic exudation or destruction in the subcortical region which would involve Marie's lenticular region. But he sees no reason for assuming that there was or was not such subcortical lesion, for to his mind it was not a case of aphasia. Dr. Collins said it was evident from the examination of the specimen and of the photograph from Dr. Spiller's case that the lenticular zone was affected.

Dr. Fraenkel said that by courtesy of Dr. Elsberg he had occasion to see the patient spoken of by Dr. Sachs. The patient showed what he

believed to be, undoubtedly, a "dysphasic" and not a "dysarthritic" disturbance of speech, in the sense in which these terms have been used heretofore.

The patient did not formulate "crippled" words; he did not formulate any beyond "olis" and "easy," and these were correctly built and enunciated.

The speaker was not satisfied from the short examination, that there was no disturbance of the understanding of speech.

The case under discussion teaches us that, for the present, and for clinical purposes, the traditional methods of locating disease on the cortex in the speech area are still reliable.

Dr. Adolf Meyer said it would seem very desirable in a discussion of this kind to distinguish theoretical problems of aphasia from the purely practical issues and not to go into the position where false theoretical views might be excused on the ground that they seem to tally in cases which really bring no direct proof one way or another. If we speak of places of maximum vulnerability of certain speech mechanisms we are always on good safe practical ground and that is what has been touched by these cases. If we speak of the aphasia mechanisms in themselves we must have more facts than these cases present. Dr. Meyer thought if we kept these two things apart we could facilitate discussion considerably and especially keep out of mere rehearsal of the claims of Marie and Dejerine, which is always a rather distressing issue at the present time. The case that Dr. Spiller spoke of had a plain involvement of the lenticular zone, although probably not of the lenticular nucleus.

Dr. Sachs, in concluding, said that he did not wish to utilize these cases as a contribution to the difference between Marie's and Dejerine's views and he believed that operative cases cannot be utilized in that way either to support or dispute the theory because we do not know exactly how deep the lesion is and can only determine this on post-mortem table. Fortunately, neither case reached the post-mortem table.

As for Dr. Collins' statement that the first case was not one of aphasia, the speaker remarked that unless we accept everything Dr. Collins says without criticism and disregard everything that other people say, we will have to insist that this is a case of aphasia and not anarthria. This was a case in which speech as such was deficient, the man could not express himself at all, whereas there was every reason to believe that he had an understanding of language. Another point, the only reason why Dr. Sachs believed the lesion did not involve the lenticular zone, or ganglia, was because there was a very brief period of paralysis and every symptom had completely disappeared except the speech disturbance. He could not concede that there can be any serious invasion of the lenticular region without some accompanying paralysis. He thinks when there is speech disturbance pure and simple without paralysis of any kind, the question of involvement of the lenticular region can be eliminated.

Dr. Sachs said in answer to those who are so very positive as to whether the lenticular zone was involved, that Dejerine has showed conclusively that Marie's conception of the lenticular zone is very elastic. Marie has defined it one way and he has gradually enlarged the lenticular zone so that now it practically takes in everything in the vicinity of the ganglia and extending up to the white matter. In support of what Dr. Fraenkel said relative to the man's understanding of spoken speech, Dr.

Sachs stated he did not know at what period Dr. Fraenkel saw the patient. Dr. Fraenkel replied in the beginning of February. Dr. Sachs continued then it must have been at the time he saw him too. At that time Dr. Sachs had very little doubt about his almost complete understanding of speech. He obeyed all commands, did everything he was told, in short, there was no reason to think there was any disturbance on the sensory side, he recognized objects and was able to read silently without being able to read aloud; therefore Dr. Sachs thought the statement he made correct. Dr. Sachs agreed perfectly with what Dr. Meyer said and simply insisted on the practical value of these things, and as to arguing theories of aphasia he thought the cases he reported were not suitable cases in support or opposition to the prevalent views on aphasia.

Dr. Charles E. Beevor made an address entitled: The Cerebral Arterial Distribution. Dr. Beevor illustrated his remarks with lantern slides.

Discussion

Dr. M. Allen Starr thought the members were extremely indebted for the beautiful demonstration, for the pictures and the wonderful specimens Dr. Beevor showed. Dr. Starr said there were a great many points in the paper that bring forward new facts that we are not familiar with. Many of the points, especially the distribution of the vessels to the internal capsule and basal ganglia, are quite new to Dr. Starr, and he thought they would appeal to all as being a very valuable original contribution to this subject.

Dr. Patrick related a bit of gossip in relation to the subject under discussion. He said he was credibly informed that when these wonderful injections had progressed to a very considerable extent Dr. Beevor happened to be in the company of one of the learned professors of the Continent and someone present asked him to tell the professor about his injections and Dr. Beevor, in his exceedingly modest and unassuming way, started to say that he made simultaneous injections of the arteries of the brain with soluble colors. The learned professor interrupted with, "Impossible, impossible, it could not be done!" Whereupon Dr. Beevor modestly subsided, not wishing to take issue with the learned professor of the Continent, who, of course, knew much more about it than Dr. Beevor. Dr. Patrick said that the very brief presentation Dr. Beevor had given of this matter did not really impress one to the proper extent, because one can only get a sort of an idea of the extreme patience and skill, aside from the originality of the idea, which have been exercised by Dr. Beevor, when one has the opportunity to look over the whole lot of brains he has in his collection, so uniformly successful have these injections been. The uniformity of the success of his method demonstrates almost better than anything else its positive value.

Dr. Putnam asked Dr. Beevor if there were any functional significance in regard to the distribution of these different arteries.

Dr. Adolf Meyer asked whether any studies were made in regard to anastomosis between the vessels inasmuch as it is, of course, of greatest importance to know what possibilities of supply there are in case of occlusion. This question is especially suggested by the fact that the dorsal aspect of the entire optic thalamus in these injections uniformly seemed to have been attributed to the posterior cerebral distribution. He thought this relation of the anterior dorsal part of the thalamus appeared

unusual while islands of softening in the posterior optic thalamus do not infrequently occur. Dr. Meyer considered Dr. Beevor's researches extremely interesting and with the addition of statements with regard to the substitutive supply, they will, no doubt, for a long time remain the most important source of pertinent information.

Dr. Joseph Collins said he would like to carry the question one stage further and ask, is there anything in Dr. Beevor's investigations which would tend to indicate the distribution of the terminal blood vessels. In sections that were being passed around Dr. Collins could not quite make that out, and he thought this would be just the matter that would put an affirmative or a negative upon that question.

Dr. Beevor, in closing the discussion, said he thanked the members of the Association for the very kind way in which they had received his paper and he also thanked Dr. Patrick and Dr. Starr for the very kind words they uttered on the subject.

In regard to the function, he did not think the distribution had anything to do with the function; so far as he can see he thinks it is purely anatomical, not functional. The fact that you can show that such a small body as the corpus subthalamicum has a double supply is against the latter idea. The question in regard to the anastomosis, of course, is a most important one. Dr. Beevor said he did not go into it in his paper, but he would be very glad to express his views on the matter as follows: In the first place, the basal arteries as described are certainly end arteries, they have no connection with their fellows; also, the basal arteries have no connection with the cortical. If you inject the basal you will find the cortical uninjected. If you inject the cortical you will find the basal uninjected. With regard to the optic thalamus there is an anastomosis between the posterior cerebral and the anterior choroid arteries. Dr. Beevor found that out in a rather interesting way when he was injecting three arteries. He tried injecting by three arteries and ligaturing the anterior choroid and the posterior communicating arteries, but he found that the injection came around by means of the posterior cerebral into the anterior choroid area, and therefore there is a communication through the choroid plexus between the posterior cerebral and the anterior choroid, so all these particular preparations came to naught, as far as the distribution of the anterior choroid was concerned. In regard to the cortex, Dr. Beevor said he can fully bear out what was shown by Duret, that there is a connection between the different systems at the periphery. If you inject the three arteries and put a clamp on the anterior cerebral you will see the middle cerebral gradually oversteps its boundaries, then if you take the clamp off the anterior cerebral, the anterior cerebral turns out the injection promptly. Each has its proper sphere. In that way, as has been said before by other authors, you can inject the whole cortex of one system. There is another point in which Dr. Beevor agrees with Duret: there is no communication, or very little, between the branches of any one system.

If you inject the middle cerebral artery and ligature one of its branches and inject the three arteries, you find that the artery which is ligatured is not supplied by its fellows of the same artery, middle cerebral, but gets it supply from the anterior cerebral. He agrees with Duret that there is no plexus, so to say, there is no free intercommunication on the cortex, that it is only on the periphery of the systems. Another point he agrees with Duret, as soon as an artery gets inside it is an end artery.

That point would be very useful in the question about aphasia, but he has not yet made any diagrams of the exact distribution. The centrum ovale is supplied from the cortex. It is quite distinct from the basal distribution. It might be useful in determining some of the points that were raised in regard to the supply to the lenticular nucleus and the white matter outside of it.

Dr. C. W. Burr, the president, thanked Dr. Beevor, in the name of the society.

Dr. H. H. Donaldson read a paper entitled: The Weight of the Brain as Influenced by Nutrition or Disease.

Discussion

Dr. Putnam asked whether there were any chemical investigations carried out which would indicate on what portion of the brain substance this loss Dr. Donaldson mentioned mainly falls, whether it is the myelin or nerve element that suffers most.

Dr. B. Sachs asked whether there were any facts known showing there is a greater relative loss of brain weight than weight of other organs, whether the brain does anything more than merely participate in the general deterioration of the nutrition. He said if Dr. Donaldson had any facts of that sort he would be very glad to know about them.

Dr. E. C. Spitzka said that in the case of studies made on the brains of distinguished men by anatomical authorities allowance had been made repeatedly for a decrease in weight, through age or disease, up to as much as 150 grams.

Dr. H. H. Donaldson, in closing, said in answer to Dr. Putnam's question, the only examination that has been made of this material has been the determination of the percentage of water in the diseased or under-nourished specimens, and that shows us a slight decrease in the percentage of water under these conditions, but not enough to be at all significant. It is less than one per cent. below the percentage of water which is normal. We have at present no other data indicating the nature of the change that occurs.

As to Dr. Sachs' inquiry: the point of the investigation seems to be the following; that usually the brain is considered as unaffected by these general disturbances of the body, nevertheless a measurable variation in the weight of the brain has been demonstrated experimentally. The alteration of the brain is, of course, in percentage values much less than that of the muscles, but probably more than that of the skeleton; a comparison with other viscera has not been made. But that 5 per cent. of loss may occur in the brain, and that it should have been previously disregarded is the point.

(*To be continued.*)

PHILADELPHIA NEUROLOGICAL SOCIETY

March 27, 1908

The President, Dr. J. W. McConnell, in the Chair

A CASE OF SYRINGOMYELIA

By S. D. Ingham, M.D.

A.B. 24 years. Peddler. Heredity and previous history are negative. About four years ago he first noticed weakness in right shoulder and inability to raise the arm over the head. This weakness has increased for the most part gradually, involving the other limbs to a less degree. At the present time his condition is as follows: Right shoulder, arm and hand are paralyzed except for slight action of the triceps. Left arm is weak in all the movements, most in those controlled by the deltoid and shoulder girdle. Right leg shows some weakness. Left leg is nearly normal in strength. Eyes show inequality of pupils, and weakness of left internal rectus muscle causing divergent squint.

Facial movements are limited and the muscles are slightly atrophied. Tongue shows no atrophy or fibrillary tremor. Muscle wasting is extreme in right shoulder girdle and supraspinalis and infraspinalis, deltoid, trapezius and pectoralis. Biceps and anterior brachial muscles also are much atrophied. Triceps and forearm muscles are less involved. Some atrophy and fibrillary tremor are seen in the left shoulder region and slight trophic change in the right leg. Reflexes are absent in the right arm, except the triceps jerk. All are absent in the right leg. All present in left side of body.

Sensation.—Diminution of pain and thermic sense is found over the distribution of the lower part of the trifacial nerve of each side, more on the right, and anesthesia to touch, pain and temperature over an area the size of the hand on the right shoulder. Tactile sensation is preserved with loss of pain and temperature sensations on right side down to level of umbilicus. Pain sensation is lost on the right side from the umbilicus down including the leg. Tactile sensation is preserved and temperature sensation is lost over the entire left side of the body, except the upper face and scalp. Some dysuria and sexual weakness have recently developed. The case is unusual in showing such extensive involvement of the spinal cord and medulla.

Dr. F. X. Dercum said that he understood that the patient had recovered damages recently as a result of a trolley accident from which he claimed he was made seriously worse. Dr. Dercum said he would like Dr. Ingham to state what the changes were.

Dr. Ingham said in answer that he did not go into the etiology of the case or the course of the development, but as a matter of fact the patient was in the service of Dr. Pickett at the Medico-Chirurgical dispensary at the time the accident occurred, a year ago last November. At that time he was attending the dispensary, and he was able to flex the right arm to about a right angle or slightly more. His grip was sufficient to carry articles of a few pounds. After the accident, which was not a very serious one from the standpoint of traumatism, he developed a condition of considerable mental irritability, restlessness, etc., which was, no doubt,

due to shock or excitement, and dating from that time his motor disturbances were very noticeably increased, and the course of the disease had been more rapidly progressive. The disease had been practically quiescent for about a year previous to the accident, the time Dr. Ingham had had him under observation, but the year following the accident the weakening and atrophy were decidedly more marked and the progress more rapid. His occupation before the onset of the disease was that of a peddler and he carried quite heavy grips. His first complaint was that the strap over his shoulder seemed to tire his shoulder. The effect of traumatism or of overuse of the muscles may or may not have had an effect in causing the disease. It is often argued that it does have that effect. That was the shoulder on which he carried the heaviest weights and was the point receiving greatest injury from the accident mentioned.

Dr. Dercum said he had the notes of a case he examined many years ago in which a man sustained a severe fall on an icy step. The only symptom Dr. Dercum could find at the time was a dissociated loss of sensation involving one arm. That man passed from under his observation and quite a number of years afterward came under the care of Dr. J. C. Wilson, presenting then the typical symptoms of a syringomyelia, and finally died of the affection.

AN ATYPICAL CASE OF FRIEDREICH'S ATAXIA

By Dr. W. G. Moore

This patient, H. M., age 16 years, was admitted to the Nervous Department of the Philadelphia General Hospital, service of Dr. Spiller, February 29, 1908. His chief complaints were that he felt nervous, was incontinent of urine, and that he staggered when he walked.

His family history shows no other member to have had any condition similar to his. He has one brother and one sister alive and in good health.

At the age of nine months, he fell from a baby coach, and according to the statement of his aunt, he was "all twisted up." A physician examined him and stated that he had received an injury to the spine. He was given internal medicine and massage and in three days was all right again. No paralysis was noticed and it was questionable whether the fall had much importance.

His legs have been weak since birth, and he was unable to walk until he was six years of age, and then his gait was staggering. He has been incontinent of urine since an early age, ever since he can remember.

Dr. Knipe examined the eyes and reported that the pupils are regular but unequal, the right 4 mm. and the left 3.5 mm. The irides react freely to light, and in accommodation and convergence. There is a concomitant convergent strabismus, the right eye being the fixing eye, and the left in 15 degrees, up 15 degrees. Both media are clear and the fundi negative. The right eye shows hypermetropia of two diopters, and the left of three diopters.

The upper extremities show nothing abnormal other than underdevelopment. The lower extremities are not spastic, and present diminished resistance to passive motion. When admitted, the patellar tendon reflexes were decidedly prompter than normal on both sides. Now while they are increased, they are not so prompt as when the boy entered

the hospital. The Achilles tendon reflexes are normal. There is no patellar or ankle clonus on either side. Irritation of the sole of the foot on either side produces flexion of the toes. When the sole of the right foot is irritated, flexion of the left great toe is obtained very distinctly at times, at others it is absent. Heel to knee test shows a slight degree of ataxia on each side.

He has not the characteristic hyperextension of the third phalanx with flexion of the first and second phalanges of the great toes. His gait and station are ataxic and the ataxia is markedly increased by closing the eyes. Pain and temperature sensations are normal in all parts of the face and limbs.

When admitted February 29, he was incontinent of urine and felt no desire to urinate. About March 12 he began to feel the desire to urinate, but could not hold his water. Now he can hold his water for about ten minutes after the desire comes.

Mentality is fair. He answers questions readily, but sometimes contradicts himself. He is rather more mischievous than the average boy.

Dr. W. G. Spiller said he wished to emphasize what is particularly atypical about the case; the loss of vesical control and the exaggeration of the tendon reflexes are unusual in Friedreich's ataxia, and unusual also are the decrease in the exaggeration of reflexes under rest in bed and the regaining of the control of the bladder.

A CASE OF ARTERIOSCLEROSIS

By G. E. Price, M.D.

Patient from the Neurological Dispensary of the Jefferson Hospital.

S. S. Male, age 75 years. The family and previous personal history had no bearing on the case. There was no history of alcoholism or of syphilitic infection.

The patient complained of difficulty in walking, marked general asthenia, dizziness, occasional headache, incontinence of urine, dyspnea, pain in the shoulder and arms, failure of memory.

Examination.—The gait was shuffling or stammering, the station was good; there were no eye symptoms excepting slight nystagmus. The reflexes of upper and lower extremities were somewhat exaggerated. Disappearing ankle clonus was present on the left side, the Oppenheim reflex on the left side. Babinski's sign was absent. The tongue and fingers presented only faint tremor and the speech was mumbling, but not ataxic or scanning. There were no emotional outbursts although the patient was restless and irritable. There was marked immobilization of features and body in general.

Attention was called especially to the gait and general immobilization as characteristic of the cases of cerebral arteriosclerosis reported by Collins.

HEMIPLEGIA WITH PARALYSIS OF THE NECK MUSCLES FROM A SMALL MYELITIC LESION

By William G. Spiller, M.D.

The patient, a woman aged 57 years, was admitted to Dr. Spiller's service at the Philadelphia General Hospital on February 14, 1907. She gave the history of having had two attacks of hemiplegia, one about twelve years previously in which the left side was paralyzed, and one six years previously in which the right side was affected. She had frequently "rheumatic" pain. She had also incontinence of urine. The pupils were unequal, the left being the larger. Iridic reaction was very slow to light and in accommodation, and in the left eye was probably absent. When she came under observation the left upper and lower limbs were very weak. The tendon reflexes were exaggerated. The muscles of the neck were rigid but the head was not retracted. Its voluntary motion was greatly impaired. The woman said she had had pain in the neck about five weeks, but the face was not expressive of pain when the head was at rest.

The paralysis and rigidity of the neck muscles in hemiplegia were remarkable and there was no means of deciding whether they were a part of the hemiplegic symptom-complex or were a complication.

The woman died March 2, 1907.

The right lenticular nucleus was entirely destroyed by an old cyst which extended into the inner capsule and destroyed its anterior limb. The anterior horn of the right lateral ventricle was much enlarged, owing to the destruction of the head of the caudate nucleus. A small cavity was found in the left lenticular nucleus.

In examining the cord with the naked eye what seemed to be a small hemorrhage was found in the left anterior horn at the fourth cervical segment. This area extended through the fourth cervical segment into the third cervical segment but not into the fifth cervical segment. The first and second cervical segments were not obtained.

The microscopical examination gave the following results:

Third Cervical Segment.—The alteration was not confined to any one region, but here and there throughout the section small hemorrhages and swollen axis cylinders were found. The small vessels within the cord had greatly thickened walls. Round cell infiltration was seen within the cord but not within the pia. The nerve cells of the anterior horn were not numerous at this level and those that were present were rounded and had imperfect dendritic processes.

The cord was less severely affected at the fourth cervical segment and was about normal at the fifth cervical segment.

Sections from the middle thoracic region appeared normal. Round cell infiltration was not distinct.

The anterior pyramids in the medulla oblongata and pons stained well by the Weigert method and did not appear to be degenerated, neither was there any round cell infiltration of the pia. The small vessels of the pia had thickened walls.

It was evident from the microscopic examination that the paralysis of the neck muscles was caused by this sharply limited myelitis. A myelitis of so small extent is a very unusual finding.

OSSEOUS PLAQUES OF THE PIA ARCHNOID AND THEIR RELATION TO PAIN IN AKROMEGALY

By S. Leopold, M.D.

The writer discussed the subject of osseous plaques of the pia arachnoid and their relation to pain, especially the pain of akromegaly. He does not believe with Sainton and State that there is a painful form of akromegaly. After reviewing the literature on the subject and discussing the pathology of the spinal cord in akromegaly he concludes with the following summary:

Osseous plaques are frequently present in the pia arachnoid in many diseases, such as uremia, tuberculosis, mental conditions, etc.

Arteriosclerosis is the underlying factor in their causation. Their presence in akromegaly does not explain the production of pain in that disease. There is no definite pathology of the cord in akromegaly.

Dr. Leopold in closing, said that Zander reported about sixty-three cases of osseous plaques and he has found them present in retrogressive conditions such as senility and mental conditions. Dr. Leopold thinks these plaques are nothing more than the result of a chronic inflammation from arteriosclerosis: the presence of fibrous tissue overgrowth and of arteriosclerosis favors the disposition of lime salts in these situations. The spinal cord, as we know, is a frequent place for bony material to be deposited as are the arteries and lymph nodes.

PHILADELPHIA NEUROLOGICAL SOCIETY

April 24, 1908

Dr. F. X. Dercum in the Chair

A CASE OF DESCENDING UNILATERAL PARALYSIS

By Alfred Gordon, M.D.

Patient, girl of 23, began to suffer from pain in the right side of the neck some years ago. The pain radiated to the same shoulder. Shortly afterwards she noticed an awkwardness and heaviness in the right arm and hand. About eighteen months later the same condition was observed in the right leg; she would scrape the floor in walking and had some difficulty in going upstairs. At about the same time she experienced difficulty in swallowing fluids.

At present there is a distinct weakness of the right arm and leg. Atrophy is marked in the hand. The knee jerks are plus on both sides, the paradoxical and Babinski signs are present on the right. Nystagmus is present in both eyes. She also has occasionally difficulty in swallowing fluid. Sensations are not impaired.

The diagnosis is probably multiple sclerosis of an unusual type.

Dr. W. G. Spiller said if the nystagmus and difficulty in swallowing could be explained in some other way he would be inclined to think the case one of tumor of the spinal cord, possibly a fibroma on the cord at the level of the lower and middle cervical roots. He had seen a case very similar to this in all except the dysphagia and the nystagmus, presented

before this society a number of years ago. A tumor was found on the spinal cord. A tumor in the cervical region can irritate the posterior roots and cause severe shooting pain and flaccid palsy in the upper limb of the same side, later by pressure upon the cord it may produce spastic paralysis in the lower limb of the same side, and still later in the lower limb of the opposite side. That is the clinical picture that Dr. Gordon has described. If the dysphagia could be regarded as of hysterical origin Dr. Spiller would be inclined to think the case one of tumor in the cervical cord, but even with due consideration of the nystagmus and dysphagia it seemed probable that some slowly developing lesion was in or on one side of the cervical cord even if higher levels were also involved.

Dr. Gordon, in closing, stated that it was surprising to him to hear the possibility of a tumor discussed in the case. He did not see how in this case any trace of tumor could be found. He examined the patient carefully, the pain is absent at the present time. She had a short period of pain, gradually developing paralysis, and difficulty in swallowing is only in regard to fluids and only at times. At present Dr. Gordon believes, taking into consideration the paralysis, the increased reflexes, symptoms pointing to motor tract involvement, and marked nystagmus, that it is a case of multiple sclerosis.

LOWER ARM TYPE OF BRACHIAL PALSY; KLUMPKE SYMPTOM GROUP

By F. X. Dercum, M.D.

D. K., female, age 67, white, widow, native of Ireland, was admitted to the Jefferson Hospital, January 14, 1908.

The family history is unimportant save that two sisters died of tuberculosis.

Personal History.—The personal history was uneventful. General health had been good for many years past. About nine months before admission she suffered from a cold. The symptoms as far as could be learned were those of an ordinary bronchitis. Subsequently, however, pain made its appearance in the right shoulder and arm. The onset was very gradual and only slowly did the pain become marked. She was treated for rheumatism without avail, while massage and electricity seemed to make the pain worse. For a time there was some swelling of the hand.

On admission it was noted that the patient was a fairly well developed woman, though somewhat emaciated. She carefully protected the right shoulder and right arm against movement. The right arm was very sensitive to touch, the slightest movement causing great pain. There was also wasting very moderate in degree of the muscles of the right shoulder, right shoulder blade, of the arm and forearm and a somewhat more marked wasting of the muscles of the hand. A careful examination showed that the tenderness was most marked over the median and ulnar distributions. Spontaneous pain, very severe in character, was also present. The patient could flex the forearm upon the arm, though she could not extend it. The flexors of the wrist and the flexors and extensors of the fingers, the pronators and the muscles of the hand were all of them notably weak. The thenar and hypothenar eminences were decidedly flattened.

There was a well marked ulnar anesthesia, quite typical in distribution. Over the rest of the extremity, as well as over the shoulder, hyperesthesia was present.

In addition it was noted that the pupils were unequal, the left being much the larger. Both pupils reacted to light and convergence. The right palpebral fissure was decidedly smaller than the left and the right eyeball was distinctly less prominent. In addition it was noted that the skin of the right side of the neck and right side of the face was quite dry, differing often markedly in this respect from the skin over the left side of the face and other portions of the body. The patient volunteered the information that she did not sweat upon the right side of the face.

The general examination elicited nothing abnormal, that is, there were no anomalies of motion, sensation or of the reflexes of the lower extremities or of the left upper extremity. The sphincters also were under good control. A careful visceral examination also proved negative. This was especially true of the lungs. The urine was normal.

A careful examination of the root of the neck by palpation and percussion failed to reveal any information. This was also true of studies made with the X-ray.

The patient was placed in bed. Salicylates, iodides, mercurials were at various times given internally and various local methods were applied, all of them without avail.

Recently a distinct dullness to percussion began to be noted above the clavicle, as well as distinct resistance to palpation in the root of the neck. A skiagraph was again made and this time revealing a distinct shadow in the same region.

It would appear, therefore, that the symptoms owe their presence to a slowly growing neoplasm, the nature of which must remain a matter of conjecture. However, it evidently presses upon the cervical roots which enter into the formation of the lower and posterior cords of the brachial plexus. The case would appear to differ, however, from the average case of the Klumpke syndrome as shown in the vasomotor involvement, namely the suppression of sweating. In order that the oculo-pupillary phenomena should be present, there must be involvement of the roots of the first dorsal segment. If, however, there also be present vasomotor and secretory symptoms as in the present case, they would be referred to the fifth and sixth cervical segments of the cord. While it is true that some of the symptoms would suggest rather a combined or diffuse brachial palsy than one limited to the inferior and posterior cords, still the motor involvement is such as is commonly found in the lower arm type. The thought occurs that the vasomotor and secretory symptoms are in the present instance to be explained by the involvement of the trunk of the sympathetic rather than by the involvement of the roots of the fifth and sixth segments. Of late also pains slight in character, yet unmistakably present, have begun to make their appearance in the *left* arm. Could it be possible that we have here a lesion which is beginning also to involve the roots upon the left side or perhaps that there is also a lesion within the cord and that the sympathetic symptoms on the right side are due to intermedio-lateral tract involvement?

The evidence is on the whole distinctly in favor of a neoplasm at the root of the neck on the right side, external to the vertebral column and the patient has given her consent to an exploratory operation.

Dr. McCarthy asked whether the reflexes were changed.

Dr. Dercum replied there was no change in the reflexes.

A CASE OF MULTIPLE SCLEROSIS

By S. D. Ingham, M.D.

A. H. Age 35 years. Saleswoman. This patient was shown before the Philadelphia Neurological Society about three years ago by Dr. Peter and is again brought before the Society to show the marked improvement which has taken place since that time.

To go briefly into the history, the patient was in robust health until eighteen years of age when she sustained an accident, receiving quite a severe cut in the left gluteal region. After this she always complained of a weak back and had frequent and prolonged attacks of diarrhea. A few years later began to have "fainting attacks" which came on during gastro-intestinal disturbances and which occurred every few weeks. During these attacks in which she was unconscious from two to five minutes, she would lie rigid with eyes open and staring.

Several years later the patient had what she thinks was nervous prostration following the taking of an anesthetic for gynecological examination. Soon afterward extreme weakness in the back and legs developed, and when she first came to the Medico-Chirurgical Dispensary four years ago she was in a condition of spastic, ataxic paraplegia, all tendon reflexes increased, those of the lower limbs spastic, ankle clonus and Babinski. Walked with feet wide apart dragging the toes.

Vision normal with exception of contraction of visual field and reversal of colors. No pupillary changes. Eye grounds normal.

During the past three years the symptoms have improved with the exception of bladder and rectal weakness which have appeared and increased during that period.

Present Condition.—Gait is slightly stiff and ataxic but the toes are not dragged and movements are more sure and stronger than three years ago.

Knee jerks and Achilles reflexes are increased but there is no ankle clonus. Babinski sign is present on both sides. Sensory changes slight over back and legs, more marked hyperesthesia over abdomen. Abdominal reflex absent.

Vision is normal except very slight concentric contraction of visual fields and reversal of colors.

No pupillary nor eyeground changes.

The fainting attacks have occurred at intervals of a few weeks during entire period and always in connection with gastro-intestinal disturbances and their character is the same as when they first appeared.

There have been no signs of organic disease above the dorsal cord and the contracted visual fields, fainting attacks, and emotional disposition suggest the probability of hysteria complicating the disseminated sclerosis.

Dr. Dercum said he personally recalled this case. There was some discussion at the time she was presented before the Neurological Society three years ago as to the presence of hysteria.

CASE OF CONVULSIVE MOVEMENTS OF RIGHT ARM, HYSTERICAL IN NATURE

By M. D. Bloomfield, M.D.

Case of convulsive movements of right arm, hysterical in nature in a patient whose history of onset of the seizure and objective findings, such as endomyocarditis and arterio-sclerotic vessels, might easily throw one off the right track in diagnosis, especially since no stigmata of hysteria are present. So far these movements have lasted five years and despite every effort at treatment by some of our most eminent men, these movements are progressing for the worst. As soon as the patient's mind is directed from himself the movements cease.

A CASE OF PREFRONTAL TUMOR, WITH EXHIBITION OF THE BRAIN

By C. W. Burr, M.D.

Dr. Burr exhibited the brain from a patient with a tumor of the prefrontal lobe. He said that the interesting points in the case were—the suddenness of onset, the shortness of the course and fewness of symptoms. The history is as follows:

A woman, 39 years old, who had had slight headache at times for two years, but no other symptoms, was picked up unconscious in the street. It was not known whether she had had an epileptiform convulsion or not. She remained in bed for several weeks suffering from severe, diffuse, continuous headache. Her eyesight rapidly failed. She then came to the hospital.

Physical examination. She was somewhat dull in thought but explained this by saying "that her head hurt her so much that she couldn't think." She answered all questions intelligently and, indeed, the only mental symptom present was slight slowness of thought. Gait and station were good but she stated that after standing or walking for a time she became dizzy. There was no paralysis of the arms or legs but the left side of the mouth drooped a little and on voluntary movement of the face the left side lagged somewhat. No ataxia or tremor. The knee jerks were slightly increased but not spastic. There was no ankle-clonus and no Babinski jerk. She could not see to read but could distinguish people. The irides reacted to light and with accommodation and convergence. There was no oculo-motor palsy. Both discs were choked and there were small retinal hemorrhages.

Nine weeks after the attack of unconsciousness she was found dead in bed. At necropsy there was found an endothelioma in the right prefrontal lobe about as large as a hen's egg, imbedding itself within the brain but having pushed the cortex ahead of it so that it lay in a socket formed of brain tissue.

A CASE OF BRAIN TUMOR

By J. Hendrie Lloyd, M.D.

Dr. James Hendrie Lloyd showed a brain with a tumor growing in the motor area of the right cerebrum. The patient was a very old

woman, a Polish emigrant, unable to speak English, who had died recently in the Philadelphia Hospital. The true nature of the lesion had been overlooked and the case had been regarded as one of ordinary hemiplegia. This was doubtless due to the difficulty of obtaining a history and of interpreting symptoms, and to the close resemblance which the case bore to ordinary cerebral softening in the aged.

The tumor was located exactly in the fissure of Rolando at about its middle third. The arm centers must have been first involved. The ascending frontal convolution was compressed in front, and the ascending parietal convolution was compressed behind the growth. The tumor measured about five centimeters in diameter and was flattened by contact with the skull. It was entirely meningeal, compressing the brain substance and could easily have been removed by surgical operation. From its location it may have caused interesting sensory as well as motor symptoms, but unfortunately these had been overlooked. The hemiplegia was of the spastic type, with greatly exaggerted reflexes.

Dr. Lloyd called attention to the ease with which brain tumors may be overlooked or confused with other lesions, especially cerebral hemorrhage and softening. The present case had been under the care of at least three visiting chiefs at Blockley, but its real nature had not been suspected.

A CASE OF PARTIAL CROSSED HEMIASYNERGIA

By Tom A. Williams, M.D., of Washington, D. C.

The possibility of its aiding in the future solution of some hitherto unsolved problem regarding the course of impulses coming to and leading from the cerebellum had induced Dr. Williams to present this remarkable case of cerebellar hemi-asynergia in which the opposite side of the face is also implicated.

It is unfortunately, however, impossible as yet to infer legitimately regarding the actual lesion; for the patient is in all probability syphilitic and both cerebellar lobes may be implicated.

Dr. Williams was obliged to Dr. Elliot of the Emergency Hospital, Washington, for the privilege of reporting the case.

It is that of a man aged 31 years who six months ago began to feel weak and dizzy and had severe pains in the loins and occiput which kept him awake at night. For three weeks he worked and then went to bed for two months by medical orders. About two months ago he noticed that the left hand was clumsy and that silly thoughts occupied his mind while he was perambulating the streets without occupation. These were chiefly about money. He also had noticed some difficulty about swallowing.

Physical Examination.—On the facial movements being tested they are seen to be accompanied on the right side by a very decided irregular, coarse tremor of the cheek, jaw, lips and tongue, the left side remaining in equilibrium except in so far as it is shaken by the communicated movements of the right side. The eyes are also unsteady when turned to the right. On protrusion of the tongue, its right side is agitated by a similar tremor which almost ceases in about ten seconds, after which interval there is also more control over the facial trembling.

In the left arm there is slight asthenia, considerable impairment of

the attitude sense, decided clumsiness and marked impairment of the diadocokinesis. There is also an intention tremor, finer than that of the face, and not maintained more than ten seconds. The general sensibility is very acute and there is no true muscular weakness.

Rotation tests show no anomaly of vestibular, and the pupils react well to light and accommodation.

If the lesion is a single one it would seem that the contra-lateral ponto-olivary fibers which traverse the raphe are implicated after doing so and lead to the facial intention tremor.

ON THE HYPERESTHETIC AREAS (HEAD'S ZONES) IN VISCERAL DISEASE

By M. D. Bloomfield, M.D.

The speaker said he had reviewed the entire literature on the subject, the experience of those who have tried the method, their results, etc. He cited five cases: (1) Gastric cancer, (2) aneurysm of ascending arch of aorta, (3) tumor of ovary, (4) inoperable sarcoma of pelvis, (5) sarcoma of testicle in which the hyperesthetic areas, over zone corresponding to the anatomical division of the abdominal wall, were the chief diagnostic symptoms in these cases. Twenty-five cases of chronic visceral involvement, tumors, ulcers, parenchymatous nephritis, gangrenous appendicitis in which the sign was most conspicuous. He compared the laboratory report of twenty cases studied by him in which that department had not more than in eight cases given the proper light as to the nature of the illness, while the clinical symptoms predominated. However, the laboratory analysis was always most helpful.

One case in particular was a patient, 48 years of age, who was treated for chronic constipation by some of our most distinguished men for over five years, who presented the Head's zone over the epigastrium. After studying the case operation was advised and disclosed a beginning carcinoma of the pylorus. Patient was operated on by Dr. Deaver and has gained forty-five pounds since operation, three months ago.

Dr. Bloomfield spoke of the various theories advanced explaining this phenomenon, reviewing especially Head's article on the epicritic and protopathic sensory pathways.

Dr. Price said it was interesting to him in showing the practical value of Head's zones. He recalled Dr. Dana's paper in which he recorded his studies of a case with complete section of the trifacial nerve, also cases of cerebral hemorrhage which involved part or all of the sensory portion of the capsule. In these cases he found no sensory disturbances as described by Head and concludes that the value of this method is limited to the distribution of the spinal nerves and not to higher pathways or to the cranial nerves.

Dr. McCarthy said that as Dr. Bloomfield had called attention, one must be careful as to the question of suggestion in outlining the areas. The question of reflexes is important. Working out the question of Head's zones in connection with the lungs at Phipps Institute Dr. McCarthy concludes that we shall have to depend more upon the objective sensory test to the patient than anything else. He is inclined to think the work he has done in testing will be of practically no value at all in so far as the test concerns tuberculosis. These patients with tuberculosis are

extremely suggestible and in the final analysis of the records he has made he fears he will have to throw out the work because of this. A case he saw recently he thought might have some bearing upon this subject. A case with several medical men in attendance, in which a certain amount of attention was paid to Head's zones, one set of the medical men thought the condition one of appendicitis, while the other set advocated a possible pneumonia. On more careful analysis though abdominal reflexes were increased, unless the patient's attention was markedly distracted while the zone was tested, a well marked hyperesthetic area was present. This, however, varied greatly and at times was absent when other areas were irritated at the same time. The case finally turned out to be one of pneumonia and terminated by crisis. Dr. McCarthy's experience with the method showed that by countersensory stimulation you could vary the sensory limitation.

What is more important to Dr. McCarthy in Dr. Head's work is the study of the exit points of the spinal roots. He has been in some communication with Head recently concerning this matter and he is of the opinion that the corresponding areas of the scalp, the spinal column and certain hallucinations connected with visceral disease have a distinct value. More recently Head has paid more attention to this phase of the subject than the hyperesthetic zones.

Dr. McCarthy said his experience would not corroborate Dr. Bloomfield's as to the great value of Head's test. It had a reactive diagnostic value, not an absolute one by itself. In connection with diseases of the lungs and heart it has been disappointing.

Dr. Ludlum thinks that in addition to the hyperesthetic areas we get in the skin in following back to the vertebræ we will find that the vertebra is tender. He has studied one hundred cases.

Dr. McCarthy asked in what percentage of tuberculous cases Dr. Ludlum found the distinct Head sign.

Dr. Ludlum replied that he could not remember the exact number.

Dr. Dercum thought that we did not pay attention to Head's work sufficiently. He thinks these hyperesthetic zones probably have some value. The fact that we now and then have pain in pneumonia referable to some region other than the thorax, *e. g.*, the abdomen, does not mean we have here a disturbing factor in regard to Head's zones. Pneumonia is a general infection and there is no reason why there should not be pains elsewhere than in the thoracic cavity. That is true also of other infections.

In regard to hysterical areas Dr. Dercum believes that with proper care those areas can be eliminated. Dr. Dercum said he felt much admiration of Head's work. He thinks Head's enthusiasm was very great when he cut various cutaneous nerves of his own arm in order to study the difference between the epicritic and protopathic sensory sensibility, knowing that such study would have to be made upon a person who was also a trained observer.

Dr. McCarthy referred to the fact that Sollier years before had brought out his work. He wrote very extensively on hysteria and allied conditions, about 1896 or 1897, in this work he called attention to some of the facts mentioned. He contended that all cases of hyperesthesia indicated a subjacent anesthesia and the reverse. Anesthesia over stomach indicated hyperesthesia over the mucous membrane. This work of Sollier's has been entirely lost sight of in the investigation of Head's work.

Personally, at that time Dr. McCarthy thought it was rather ridiculous. More careful investigation into the reflex disturbances leads him into a position which is not so positive as to the fact that Sollier's work should not be taken more seriously.

Dr. Bloomfield, in closing, said that Head in his original article states that Head's lines cannot be elicited in chronic cases. Dr. Bloomfield has had the opportunity to examine many of them and to-night he presented one whose history pointed to infection five years previously, who presented Head's lines very prominently. Another gentleman he had with him Dr. Bloomfield did not show owing to the lateness of the hour. A gangrenous appendix was removed from him by Dr. Deaver and that patient still shows Head's lines. Dr. Bloomfield stated he had a case operated on by Dr. Deaver for appendectomy who developed thrombosis and showed Head's lines for five months after the operation.

A CASE EXHIBITING THE SYMPTOMS OF ATAXIA OF THE LEFT ARM, WITH INABILITY TO RECOGNIZE OBJECTS IN THE LEFT HAND, WHILE ALL FORMS OF SENSATION WERE PRESERVED

By J. H. W. Rhein, M.D.

The patient, an Italian woman of 60, three years previously noticed she used the left hand and arm less skillfully than formerly. This symptom was of gradual onset. She presented besides a slight ataxia in the left arm, and inability to recognize objects in the left hand with the eyes closed. This was especially interesting in view of the fact that all forms of sensation were intact, including the sense of pain, localization, touch and muscle senses, the recognition of three dimensions and the physical qualities of the object held in the hand with the eyes opened or closed. When the eyes were closed, however, she could not name correctly any object placed in the left hand. The knee jerks and arm jerks were slightly increased and equally so. There was no paralysis —in fact, the examination was in other respects entirely negative, except a contraction of the visual fields, which was reported by Dr. William Campbell Posey.

The case is interesting in relation to the discussion of astereognosis, recently introduced by Prince of Boston, Raymond and Egger, and Dejerine and Claparède, and more recently by Kutner, in a paper entitled "Transcortical Psychic Paralysis (Transcortical Tastlähmung)."

Dr. Rhein believes that this case was probably a pure example of the condition described by Kutner. The ataxia was probably cortical in origin, and a lesion in the right parietal region would explain both of these symptoms.

Dr. Spiller said it was a great pity Dr. Rhein's case could not be shown before the society as it was extremely rare. He knows of no case on record of uncomplicated complete tactile asymbolia without the slightest sensory disturbance. The woman could tell the form of an object, but could not recognize its use. Dr. Spiller said in Vienna as far back as 1893 the term astereognosis was commonly used. Its meaning was the inability to recognize objects by contact without vision. The distinction we are to give now to stereognosis refers merely to the recognizing of an object in its three dimensions: it has nothing to do with recognizing the nature

of the object. In the case Dr. Rhein reports the woman did not have astereognosis in the old sense of the term. That is she could recognize the dimensions of an object and still was utterly unable to give the use of that object. That is to say she could distinguish it as a sphere, a cube, etc. There was no disturbance of sense of position, according to Dr. Rhein, which appeared noteworthy to Dr. Spiller.

THE PATHOLOGY OF ACUTE ANTERIOR POLIOMYELITIS

By W. B. Cadwalader, M.D

The cases which the speaker reported are typical examples of acute anterior poliomyelitis, one occurring in a child and two in adults, dying of bulbar involvement on the seventh, fourth and fifth day of the disease respectively.

Intense round cell infiltration was found throughout the gray matter of the anterior horns of the spinal cord. Cellular infiltration of the pia was very marked, but the infiltrating cells were rarely found on the outer surface of this membrane. The ganglion cells were much degenerated and many destroyed. Changes were found in the medulla and pons and in two cases, the cerebral peduncles, basal ganglia and cerebral cortex were also affected. The white columns of the spinal cord were slightly infiltrated with round cells. In one case neuronophagia was only marked in the lumbar enlargment of the cord. The ganglion cells of the posterior spinal ganglia of one case were severely degenerated.

Conclusions.—1. Acute anterior poliomyelitis is essentially an acute poiloencephalomeningomyelitis.

2. The process is the same during infancy and adult life.

3. The process is most marked in the lumbar and cervical enlargements of the cord and frequently may extend upward as far as the cerebral cortex.

4. Interstitial changes predominate and occur together with parenchymatous changes. Parenchymatous changes never occur without interstitial changes.

5. The localization and intensity of cellular infiltrations depend upon the distribution and vascularity of the area affected.

6. Neuronophagia is an important factor in the destruction of ganglion cells.

Dr. McCarthy said he found Dr. Cadwalader's paper very interesting and very instructive from several standpoints. He took exception to one statement made in the preliminary part in regard to the meningeal changes not being altogether pathologically correct. Dr. McCarthy thought the question of the exudate to a certain extent dependent upon the acuteness of the process. In the usual forms of spinal meningitis, the exudate may be mainly internal, in the tuberculous and septic-tuberculous types the exudate is mainly external.

As to the other septic types of meningitis Dr. McCarthy did not remember whether this exudation is internal or external.

In connection with the subject of neuronophagia Dr. McCarthy said he had a certain amount of interesting material. One is a case Dr. Dercum made a study of at the Philadelphia Hospital and later was admitted to the Phipps Institute, exhibiting a condition in which both the lower and upper extremities, the pectoral muscles, were affected by some

atrophic process beginning before birth. Dr. McCarthy went to the Philadelphia Hospital to see the mother of the man in the out wards who stated that the condition was congenital. He also found that Dr. Dercum had made the same diagnosis of congenital poliomyelitis. The examination showed a most extensive disappearance of the anterior horn cells throughout the spinal cord from top to bottom. There were a few cells scattered, just enough to keep up function in a way. There was very little evidence of vascular or scar tissue. This might have been expected since the man was 29 or 30 years old and the condition must have existed since childhood. The mother stated the condition was present from birth. The only explanation for the disappearance of these cells is by diffuse poliomyelitis. The meninges of this case did not show any changes.

Dr. McCarthy also alluded to another case of congenital amputation of the arm, which belongs to the same group of cases under consideration. The amputation occurred about the middle of the arm and microscopic examination of the spinal cord, the patient being a man somewhat over 30, showed marked disappearance of the anterior horn cells. Microscopic examination showed cells much like those he had observed in the adrenal gland of a chicken; that is a retrogressive type of degeneration somewhat different from any other type seen. His conclusion in this case was that the cells were partially destroyed at some prenatal period and that a certain amount of restitution of cell life had taken place, but not enough to bring the cells to the function power of ordinary ganglion cells.

Dr. Spiller thought the rapidity with which nerve cells disappear under inflammatory conditions remarkable. He had reported with Dr. Sherman a case of poliomyelitis where the gray matter was involved and the white matter also, but less intensely. The man was able to walk 38 hours before he died. When Dr. Spiller examined the spinal cord it was extremely difficult to find any nerve cells in certain portions.

The view Dr. Cadwalader expressed is the view Dr. Spiller has held for a number of years, viz., that there is not a strict limitation of the process to the gray matter. Dr. Spiller said he had studied a case in which poliomyelitis followed small-pox and the gray matter alone was involved in the lumbar region, but in the thoracic region the white columns did not escape. Another explanation why the lumbar region is so often involved is that it is very vascular.

Strümpell has advanced the view that there is a process similar to poliomyelitis occurring in the brain in polioencephalitis. The existence of this condition has been doubted. Dr. Spiller said he had had the opportunity in the last few years to examine three cases of encephalitis in which the inflammation was almost confined to the gray matter, although there was some slight involvement of the white matter immediately adjacent. He believes a process occurs in the brain which is not diffuse encephalitis, but is really polioencephalitis, the gray matter being far more involved, the adjacent white matter being only slightly affected.

Dr. Cadwalader, in closing, said that when he spoke of a meningitis occurring in poliomyelitis in contrast with a meningitis of other conditions, he did not intend to convey the idea that the infiltrating cells in the septic conditions were necessarily always confined to the outer surface of the pia. In his poliomyelitis cases and he thinks in all the instances he has been able to find in literature inflammatory cells on the outer side were very rarely found. In fact they were so few as to be

very questionable. Therefore, it is assumed by many writers that the infection might possibly come from the lymph channels between the cord itself and the pia. But that seems to be very doubtful at the present time, so far as any anatomical explanation is concerned. As to the neuronophagia, he thinks there is a possible explanation for the varieties of the cells found in the infiltration. Numerous writers have claimed to be able to demonstrate inflammatory cells which have burrowed into the substance of a ganglion cell and the ganglion cell itself having gradually disappeared, then the lymphocyte or lymphocyte-like cells increased in their protoplasm at the expense of the ganglion cell and the nuclei stained a deeper blue color with various stains. In other words a deeper staining reaction of the nucleus. Here may be a possible explanation for the many different varieties of cells seen in this form of inflammation which has puzzled everyone so far. No one seems to have arrived at any definite conclusion as to where they come from.

NEW YORK NEUROLOGICAL SOCIETY

April 7, 1908

The President, Dr. B. Sachs, in the Chair

A CASE OF REFRIGERATION BELL'S PALSY ACCOMPANIED BY LATERAL OSCILLATION OF THE EYEBALL

By L. Pierce Clark, M.D.

The patient was a boy with complete refrigeration Bell's palsy of the right side of the face of ten months' duration. The case was of special interest because of a peculiar lateral oscillation of the right eyeball during a forceful attempt to close the eyes. This movement of the eyeball appeared to be largely induced by the internal rectus at the beginning of the act in closing the eyes. At the beginning the right eyeball was moved inward and upward, as was usual in these cases. The movement was no sooner begun than the lateral see-saw nystagmoid movement began. The left eye also exhibited the same movement but in a much less degree, and was partly under the control of the will. It might be inhibited at the beginning or end of the attempt on closure, but not while the strongest effort was being put forth. The lateral oscillation occurred only on attempt to close the eyes; no other facial movement brought it out. Schlesinger recently reported a similar phenomenon in a medullary lesion of the seventh and twelfth nuclei, which gradually passed away as the primary lesion improved. Dr. Clark said that so far as he was aware, this was the first report of a case of the occurrence of this form of eye movement in a typical case of Bell's palsy. The explanation of this eye symptom was that a voluntary impulse attempted transmission over the seventh nerve, but being blocked by the palsy lesion, it overflowed by way of the posterior longitudinal bundle into the third nucleus. It was known that the movements of all extrinsic muscles of the eyes were so closely related, that some anatomists claimed a common nuclear origin for all muscles of the eye; that is, that the orbicularis palpebrarum partly originated in the hinder portion of the third nucleus. The internal rectus being the strongest intrinsic eye muscle whose movement was most inti-

mately associated with the action of the orbicular was the explanation for the kind of eye movement shown in this case.

Dr. I. Abrahamson after examining the case shown by Dr. Clark, called attention to the fact that the right external rectus was also apparently affected. There was decided nystagmus as the eye was swung to the right.

Dr. Henry H. Tyson said he considered Dr. Abrahamson's observation incorrect, inasmuch as what Dr. Abrahamson thought was a paralysis of the right external rectus muscle was not a true paralysis; but only a limitation of the conjugate movement to the right.

Dr. Tyson thought that Dr. Clark's explanation was partly correct; but it did not account for the excessive nystagmic movement to the left.

Dr. Tyson had examined the eyes in this case for Dr. Clark and had reported the following condition and theory. Vision in right eye varied from 20/40 to 20/20, due to spasm of accommodation. Left eye, 20/20. Sensibility of cornea, right diminished, left normal. Motility of each eye tested separately apparently normal. Conjugate movements to left exceed those to right by fifteen degrees. Orthophoria for distance, very slight exophoria (0.5°) for near. Fusion power of muscles tested by prisms for distance—adduct. 12°, abduct. 4°, sursumduct. 3°, deorsumduct. 3°. Candle test shows no muscle palsies.

Upon forced movements of eyes to extreme right or left a slight psuedo-nystagmus was produced, sometimes horizontal, other times mixed. Upon attempting to forcibly close right eyelids, the eyes move up and to the left, and horizontal oscillatory movements ensue with the eyes moving through an area of about 35°, extending from a point five degrees from the right of the median line to a point about 30° to the left of the median line. This movement seemed to be due to overflow stimuli from the facial nuclei to the third nerve nuclei through the posterior longitudinal fasciculi, and also to the centers for associate movements. The congugate movement to the left in this case being the stronger, we thus obtain the nystagmic movement in that direction. H. Schlesinger, who reported a somewhat similar case (*Neurol. Centralbl.*, No. 26, p. 242), stated that in all probability the horizontal movements of the eyeballs were due to central disturbances of innervation. An analysis of this case would show that it was due to overflow stimuli, as heretofore stated, and an insufficiency in the congugate movements to the right. The pupils in this case were at times equal, at other times unequal, the right being the smaller, due to spasm of the sphincter pupillæ. Light and convergence reflex normal. Orbicularis lid reaction positive. Sensory reflex positive, more marked on left side. Psychic positive. Color field test gave the red field largest, then green and white being the smallest, indicating a hysterical element as a complication.

Dr. R. A. Diffendorf, of New Haven, Conn. said he had observed identical nystagmus-like movements in cases of catatonic stupor whenever the closed eyelids were forcibly opened. This symptom had been encountered so uniformly that it had come to be regarded as a characteristic symptom of catatonic stupor. This symptom had been discovered during an extensive study of eye movements in the insane carried on during the past year by Dodge and himself. Thus far they had been unable to explain its origin. It is possible that Dr. Clark's case may throw some additional light on this matter.

Dr. M. Allen Starr recalled one case of typical Bell's palsy in which

these oscillatory movements of the eyeball were present on attempting to close the eye. The phenomenon gradually disappeared.

A CASE OF NEURALGIA LIMITED TO THE SENSORY FILAMENT OF THE SEVENTH NERVE

By M. Allen Starr, M.D.

The patient was a young woman, the wife of a physician, who was suffering from an intense type of neuralgia, the pain being absolutely limited to the sensory filament of the seventh nerve, which supplied the anterior surface of the auditory canal and a part of the external ear. The observation was interesting on account of the strict limitation of the pain, corresponding as it did with the area which Dr. J. Ramsay Hunt had described in connection with his cases of facial palsy following herpes auricularis. In his case, Dr. Starr said, there was a noticeable flattening of the face on the affected side, which rather strengthened the diagnosis of a disease of the facial nerve.

Dr. J. Ramsay Hunt said that while he had not seen a case of idiopathic neuralgia of the sensory mechanism of the seventh cranial nerve, these cases have been collected and described in his article in the *Archives of Otology* for December, 1907, "Otalgia Considered as an Affection of the Seventh Cranial Nerve."

There was, in his opinion, no question but that Dr. Starr's case was one of primary tic douloureux of the seventh cranial nerve.

Dr. Hunt has observed reflex otalgias in the same distribution as well as post-herpetic otalgias following herpetic inflammation of the geniculate ganglion of the facial nerve.

In these cases the pain was likewise circumscribed as in Dr. Starr's case, to the depths of the ear, the auditory canal and the interior of the auricle.

At the present time he has a case under observation of a young woman, who has suffered from recurrent lancinating pains in the region of the left auricle and auditory canal for the past five months. These pains are gradually diminishing in intensity, and are typically neuralgic in character. The auditory mechanism is entirely free from any evidences of organic disease.

This otalgia followed an acute febrile disturbance and a small crop of vesicles on the posterior surface of the left auricle. This case was evidently post-herpetic in its origin and is interesting because of its long duration.

Dr. William M. Leszynsky, who had seen the patient whose history was reported by Dr. Starr, said he could confirm what had been said in regard to the character of the pain. It was apparently a very severe but an atypical form of trigeminal neuralgia.

A CASE OF THROMBOSIS OF THE POSTERIOR INFERIOR CEREBELLAR ARTERY

By I. Abrahamson, M.D.

The patient was a man, 44 years old, a native of Russia and a tailor by occupation. His previous history was unimportant. He had been

moderately alcoholic; denied venereal disease. About two years ago he began to complain of morning headaches, with transient attacks of vertigo and scotoma, and tinnitus of the right ear. His vision also gradually became poorer. About four weeks before coming under observation he had left-sided headaches, with evening vomiting and dizziness. A week later he noticed that he staggered to the left and suffered from absent-mindedness and mental confusion. He became unable to walk or stand, falling to the left. There were also dysphagia and numbness of the left face.

When Dr. Abrahamson first saw the patient the speech was difficult and raucous, the throat being filled with frothy mucus. Speech was nasal in character; the facial expression was blank. There was unilateral sweating of the right face, and apparent weakness of facial innervation. Taste and smell were normal. The heart's action was irregular and intermittent, with a slight systolic murmur. The pulse was intermittent; tension was increased and the arterial walls thickened. The urine contained albumin and casts. Briefly, the case was one of nephritis with secondary cardiac and arterial changes (with prodroma, left-sided headaches, vertigo, vomiting, mental confusion, etc.; gradual development of ataxia, falling to left, dysphagia, aphonia, numbness of left face; examination showing dissociated sensory disturbances over left trigeminal and crossed right rest of body, left sympathetic exophthalmia, ataxia, paralysis and anesthesia of left palate and larynx, irregular heart action—symptoms leading to the diagnosis of thrombosis of the left posterior inferior cerebellar artery, and areas of softening in the lateral section of the medulla).

Dr. Starr said the case presented by Dr. Abrahamson corresponded to one reported by him in the *New York Medical Record* on February 11, 1893. Four similar cases had been reported in the *Johns Hopkins Bulletin* by Dr. H. M. Thomas. In each instance a wedge-shaped area of softening was found in the side of the medulla, extending towards the median line. In one instance where the interolivary tract was involved, there was a history of ataxia.

The President, Dr. Sachs, who had seen the case reported by Dr. Abrahamson, said that one very striking symptom was the tremendous amount of cerebellar titubation, the patient almost falling over. The irregularity of the heart and respiration was also very pronounced, but after a few days in the hospital his breathing became regular and his improvement was very marked. The speaker thought there was no doubt that the case was one of thrombosis of the inferior cerebellar artery, and he called attention to the fact without attempting to explain it that thrombotic lesions affecting arteries or blood vessels in unusual locations, such as this one, had, in his experience, been associated with nephritis.

CRANIOTOMY FOR TUMOR OF THE ACOUSTIC NERVE

By Willy Meyer, M.D.

The patient was a woman, 23 years old, who was referred to Dr. Meyer by Dr. George W. Jacoby. She had a slight facial palsy on the left side, with drooping of the left eyelid and the corresponding angle of the mouth. She complained chiefly of dizziness and staggering while walking, and swayed on standing. Hearing on the left side was much impaired. There was slight headache. She vomited at rare intervals.

There was choked disc, with atrophy. After careful observation, the case was regarded as one of tumor of the ponto-cerebellar angle, involving the left auditory and facial nerves, and an operation for its removal was undertaken on January 29, 1908. The occiput was exposed through a large horse-shoe incision, extending from one mastoid to the other, and reaching about two fingers' width above the occipital protuberance. The operation was described in detail, and resulted in the finding of a tumor, bluish-white in color and about the size of a cherry, near the meatus auditorius internus; it was hard to the touch and comparatively easily shelled out in three pieces. The surrounding brain tissue was soft to the touch. Subsequent to the operation there was a good deal of oozing of cerebro-spinal fluid, and, for a few days, considerable edema of the face. Otherwise, the patient's recovery was uninterrupted. Gradual improvement in her eyesight and other symptoms had taken place since the operation, which was done eight weeks ago. Microscopically, the growth proved to be a fibrosarcoma.

Dr. George W. Jacoby said the case presented by Dr. Willy Meyer was clinically clearly one of tumor of the ponto-cerebellar angle. The patient's ataxia, prior to operation, was so marked that she could scarcely take two or three steps. There was also marked dizziness, with progressive deafness and inceasing facial paralysis. The corneal reflex, which had been lost, had reappeared since the operation. Since the operation, the patient had developed a peculiarity of speech, which was best described as explosive or screaming in character, not unlike that of some deaf-mutes. Her gait had improved, and the choked disc had to a great extent retrograded.

Dr. Ramsay Hunt congratulated Dr. Meyer on the very excellent result which he had obtained in this case of acoustic tumor, and said that it was only a few years ago that this group of tumors was regarded as inoperable.

During the past few years Dr. Hunt said that he had had four cases of acoustic tumor operated, and all succumbed to the operation within forty-eight hours. He had also seen a case in coöperation with Dr. Fraenkel which had the same termination, so that the technical difficulties of this procedure were very great indeed. This is the one tumor of the brain in which, in addition to localization, the nature, approximate size and its relation to the surrounding parts may be determined with accuracy before the operation. These facts, with its ready enucleability, increase its surgical importance.

The case reported by him in conjunction with Dr. Woolsey was, as far as appears in the literature, the first case in which a definite operation had been attempted to remove an acoustic tumor in which the complete diagnosis had been made. Dr. Woolsey chose the basilar occipital route, enlarging the opening to near the margin of the foramen magnum. The tumor was enucleated without difficulty; the patient died within twenty-four hours. Post-mortem examination showed that the pons had been lacerated and that death was due to hemorrhage.

Dr. Joseph Fraenkel said this was the first case of acoustic tumor that he had seen alive after the operation. In one case which was diagnosticated by Dr. Abrahamson and operated on by Dr. Charles A. Elsberg the patient survived for a longer period than this, and died from an additional tumor involving the fifth nerve. The speaker emphasized the importance of distinctly recognizing beforehand the side on which the

tumor was located. If that were done, so large a flap as the one made by Dr. Meyer would probably be unnecessary. In tamponing during the course of these operations for the purpose of stopping hemorrhages, too much force was apt to be employed by the surgeon's assistant, thus injuring the delicate brain tissue.

Dr. Charles A. Elsberg said he had explored the ponto-cerebellar angle five times, and he had found that with each case one became more adept in doing the operation with greater ease, and causing a less amount of surgical damage. He did not think such a large incision as that made by Dr. Meyer was necessary, excepting in those rare instances where the exact location of the tumor had not been made out. If the bone was opened on one side, with free invasion of the corresponding mastoid, a good view of the cerebellum and the ponto-cerebellar angle could be obtained by the use of retractors. He used retractors of different sizes, carefully inserted, to draw the cerebellum towards the median line. The speaker said the patient he operated on for Dr. Abrahamson died fourteen weeks after the operation of secondary tumor and meningitis.

CERVICAL RIB AND ITS RELATION TO THE NEUROPATHIES; WITH REPORT OF A CASE

By S. P. Goodhart, M.D.

The speaker gave a general review of the subject. He also showed a woman, 28 years old, with bilateral cervical rib. The salient points of this case were as follows: (1) Onset of the symptoms at the unusually early age of seven years. (2) Severity of the symptoms, which were practically limited to the nervous system. (3) Hypesthesia in the region supplied by the inner cord of the brachial plexus (ulnar distribution). (4) Progressive atrophy of the small muscles of the hand, including the thenar and hypothenar eminences. (5) Secondary cervico-dorsal scoliosis. (6) Stationary character of the symptoms referable to the cervical rib, in absence of surgical intervention. (7) Possibility of demonstrating the exact location of the roots of the brachial plexus in their relation to the supernumerary rib on the left side: digital pressure upon each producing numbness and tingling in corresponding area of arm. (8) Diminution in size of third normal rib on left side, well shown in radiograph. (9) Beginning symptoms due to pressure on the opposite side.

Dr. Charles E. Atwood said cases like the one shown by Dr. Goodhart were not so uncommon, but were not always recognized. The speaker recalled two similar cases that he had seen in London. One of these was for a time regarded as a case of syringomyelia. There was atrophy of the abductor pollicis and interossei of the left hand and *main en griffe*. With the X-ray, bilateral cervical ribs were found, and the left one was removed. Great improvement followed. In the second case there was atrophy of the right thumb of a year's duration, and pain in the corresponding thumb for ten years. Also, for one year, pain in the forearm and near the clavicle. Numbness and anesthesia of some of the fingers were also complained of. In this case, improvement also followed operation. The first patient was a man, aged 37 years; the second, a woman aged 40 years. It is said that some cases recover entirely from operation, and that all are benefited by it.

Dr. L. Pierce Clark called attention to the similarity of the symptoms in certain of these cases to those of progressive muscular atrophy, which would be in favor of the peripheral origin of the latter disease. Quite a number of the cases presented a scoliosis and atrophy of the smaller muscles of the hand, warranting a diagnosis of beginning syringomyelia. The possibility of extra-cervical rib, therefore, should not be lost sight of in doubtful cases of syringomyelia.

Dr. Alfred S. Taylor said he had never removed a cervical rib, but he had made a great many dissections in that region and he thought it was not at all impossible to remove such a rib without damaging the brachial plexus, which, with careful manipulation, could be displaced quite materially from its ordinary position without interfering with the motor function of the nerves. The operation would require a sufficiently wide dissection and careful work.

Dr. Goodhart, in reply to a question as to whether the sympathetic was ever involved in cases of cervical rib, said that it occurred only in association with syringomyelia.

LANTERN-SLIDE EXHIBITION OF PATHOLOGICAL SPECIMENS OF RARE BRAIN LESIONS

By M. Allen Starr, M.D.

This demonstration included a number of rare brain lesions that came under the observation of Dr. Larkin in his capacity as coroner's physician. They were the post-mortem findings in cases of sudden death, and a preceding history was usually unobtainable. In one of the cases there was an enormous aneurism at the base of the brain, which had produced sudden death, although the man from whom the specimen had been obtained had apparently enjoyed excellent health up to that time. Another specimen showed a series of capillary hemorrhages all over the brain, such as were seen occasionally in alcoholic brains. Another showed a hemorrhage into the floor of the fourth ventricle, and involving the pons Varolii. Another specimen showed an enormous cyst lying at the base of the brain, and resulting from an aneurism from the anterior communicating artery. Another showed a hemorrhage into the ventricle of the brain; this occurred in a leukemic patient, and it was not associated with the rupture of any large blood vessel. Another showed an abscess of the frontal lobe of the brain following an operation on the nose. Another showed an abscess of the brain following middle ear disease. Another showed unilateral absence of the cerebellum.

In the case of aneurism of the base of the brain, Dr. Starr said, the patient was a longshoreman, apparently in his usual health, who sat down at the end of the pier to eat his midday meal. Two days later he was found still sitting there, dead.

Dr. Larkin, who had made the autopsies in these cases in his capacity of coroner's physician, said that in the case described by Dr. Starr in which there had been a hemorrhage into the ventricle of the brain, without apparent rupture of any large blood vessel, the patient was a boy of sixteen years, who while walking along the street suddenly fell to the ground. He was taken to Roosevelt Hospital, and died fifteen minutes later. The post-mortem examination showed that the left ventricle was distended by a clot, but the source of the bleeding could not

be made out, excepting small punctate spots in the wall of the ventricle. The patient had an enlarged spleen, and a blood examination made just prior to his death showed lymphatic leukemia.

Dr. G. L. Walton, of Boston, said it would have been interesting to know the histories of the hemorrhagic cases shown by Dr. Starr. The clinical study of such cases had not kept pace with that of intracranial growths, but these hemorrhages at the surface of the brain offered equal opportunity for brilliant diagnosis, and far greater opportunity for successful operation. In a case recently seen in consultation with Drs. Paul and Brewster, a subdural hemorrhage in the right middle fossa was successfully removed as the result of a diagnosis which was based on intense and increasing frontal headache, photophobia, slow pulse and an increase in the right wrist jerk, these symptoms following a blow in the left occipital region. Dr. Paul's conclusion that the hemorrhage was on the right side, based partly upon the location of the blow and partly upon the supposition that the hemorrhage caused pressure of the brain against the skull on the opposite side proved to be well founded.

Dr. Joseph Collins and Dr. Harrison H. Martland read a paper on "The Toxic Effects of Cyanide of Potassium Upon the Peripheral Motor Neurones: A Clinical and Experimental Study." (See this JOURNAL, p. 417.)

Dr. Joseph Collins said he knew of no other case of cyanide of potassium poisoning in which the toxic manifestations were similar to those recorded in this paper. The clinical and experimental studies in connection with this case showed that cyanide of potassium did not produce a neuritis or a poliomyelitis, in the strict sense of these terms, but a degeneration of the entire peripheral motor neurones. The case was interesting, because it corroborated the observations that had been made regarding the destructive effects of lead on the peripheral motor neurones, confirming and paralleling the experiments of Stieglitz made about fifteen years ago. The early symptoms in this case were remarkable and unusual, not at all like those of lead poisoning. They indicated an overwhelming intoxication or infection, and before any knowledge was obtained of the man's occupation, the case was supposed to be one of anterior poliomyelitis.

Dr. J. Ramsay Hunt said that he had observed the case in the acute stage in the New York Hospital. He had regarded it as an acute toxic or infectious process and the question at that time had been raised as to whether the cyanide of potash might have played a rôle. The onset was acute with fever, delirium and retraction of the neck. In addition there were violent, shooting pains in the upper and lower extremities, with retention of urine. No objective sensory disturbances accompanied the motor paralyses. His interpretation of the case had been that of an acute poliomyelitis or disseminated myelitis involving chiefly the gray matter.

Dr. Maitland's experiment was most interesting and conclusive, in showing the effects of cyanide of potassium on the peripheral motor neurones—but that alone would hardly account for the patient's sensory symptoms.

Dr. I. Strauss said that if the case reported by Drs. Collins and Martland had come into their hands last summer while we were experiencing an epidemic of anterior poliomyelitis instead of in 1906, they would probably have classified it under this head. Dr. Strauss had observed a very similar case in the service of Dr. B. Sachs at the Mt. Sinai

Hospital in August of last year. The man was an Irishman of about twenty and he was admitted because of what appeared to be some intestinal intoxication. There were nausea, vomiting and diarrhea, high fever and marked prostration. The day after admission his body was covered with a very profuse maculo-papular eruption and there began to be weakness in the muscles of the legs. This weakness increased to complete flaccid paralysis of the lower extremities and the paresis advanced until arms were affected. The deep reflexes became lost, there was some hyperesthesia along the anterior surfaces of the tibiæ and very marked at the base of the skull. There was rigidity of the neck and retention of urine so that catheterization was necessary. The reaction to faradism was very much diminished in all the muscles of the extremities and the reaction of degeneration followed in the shoulder and upper arm group of muscles including the pectorals. Later a very marked atrophy of the muscles of the limbs especially of the upper followed and has persisted to the present. The patient is recovering although probably will never fully regain normal power in some muscles. This was a typical case of anterior poliomyelitis as it was more frequently observed in children last summer, and as has been described by Wickman in the Swedish epidemic of 1905. It is probably one of the cases complicated with nerve involvement which he classifies as the polyneuritic form.

Dr. Hunt's description of the onset of the disease in Dr. Collins' case as he observed it at the New York Hospital tallies exactly with the one seen at Mt. Sinai. There was fever-paralysis, ascending in type and marked hyperesthesia.

The animal experiments do not explain the symptoms in the case. All that they show is that when cyanide of potash is given to an animal there is a degeneration of the peripheral neurone. There is nothing specific in this action, numerous toxic substances; both organic and inorganic have produced the same lesion in animals. This lesion does not explain the hyperesthesia and other irritative sensory symptoms the case presented in the early stages. The cases of anterior poliomyelitis which Dr. Strauss had so far studied show hemorrhages and a marked leucocytic infiltration of the meninges and posterior spinal ganglia as well, which lesions can account for the symptoms possessed by both Dr. Collins' case and the one Dr. Strauss had described. Another fact which must be borne in mind in discussing this case is that if the use of KCn by waiters is as common as stated by Drs. Collins and Martland it is curious that this should be the first case noted.

In conclusion, therefore, Dr. Strauss thought that neither the clinical symptoms nor the animal experiments were convincing proof that the case was one of poisoning, but he was much more inclined to regard it as a type of anterior poliomyelitis with which we had become familiar through the recent epidemic.

Dr. Sachs said the inference he drew from the findings of Drs. Collins and Martland was that we could no longer consider that the various poisons, both chemical and organic, confined their effects upon the peripheral or spinal neurones, but might affect both the sensory and the motor neurones. Clinically, the case might be regarded as one of multiple neuritis or anterior poliomyelitis. In the case at Mt. Sinai Hospital, to which Dr. Strauss referred, it was impossible to state positively whether it was one of multiple neuritis or poliomyelitis.

Dr. H. C. Van Den Burgh said that in certain mining camps he had

seen cyanide of potassium extensively used in connection with the extraction of gold and silver. Cases of poisoning, so far as he knew, were rare in those camps, and in those that he had observed, death was almost instantaneous.

Dr. Larkin said he had seen fourteen cases of cyanide of potassium poisoning during the past year. In most of these, death was almost instantaneous. There was one case of a druggist who was poisoned by dilute hydrocyanic acid, and the morning after he was brought to the hospital there was motor paralysis of both legs. The symptoms resembled those of an ascending myelitis, and death occurred in the course of four or five days. The microscopic examination of the cord in that case showed complete degeneration of the anterior motor cells, not at all unlike what Dr. Martland had described in his experimental work on rabbits. In those cases where instantaneous death had occurred after the use of cyanide of potassium, the microscopic studies had not yet been completed.

CHICAGO NEUROLOGICAL SOCIETY

April 23, 1908

PRESENTATION OF A CASE OF LEONTIASIS OSSEA

By Dr. Charles Louis Mix

The patient is a woman thirty-two years of age. When she was very young she was troubled with nasal catarrh and was treated for it when she was about five years of age. So far as her general appearance is concerned she showed nothing abnormal until about fourteen or fifteen years ago during her seventeenth year, at which time she began to notice a swelling at the inner corner of her left eye involving the nasal process of the superior maxilla. Subsequently other swellings began to appear in the bones of the upper part of the face, especially in the orbital plates of the frontal bone and in the region of the superciliary ridges. As time went on the right nasal process of the superior maxillary bone also began to enlarge, the enlargement continuing until the present time. Not only are the frontal, nasal and superior maxillary bones involved, but the malar bones and the parietals are also concerned in the process. The bi-parietal diameter is very much increased above what should be normal for her head. When her hair is raised from her forehead it will be noticed that the appearance of the face is such that it resembles a triangle, the chin marking the apex and the frontal bone the base. It will be noticed that the distance between the eyes is almost twice the normal amount by reason of the great increase in the ethmoid bone. The eyes are seen to be extremely prominent being pushed forward at least half way out of their sockets, and it will be observed that there is some lack of conjugation in the use of the eyeballs.

The growth has been extremely slow in rate but fairly uniform in increase in size, and although the patient states that she believes the bone enlargement involving the nasal process of the right superior maxillary bone is smaller than it was two years ago, it seems to be about as large as it was when first seen.

She shows no visual change of any sort. There is not a symptom in

her case either subjective or objective pointing to any disturbance of the pituitary body. Her hand is well formed and small, showing not a trace of acromegaly. There is also no enlargement of the lower jaw. On the contrary owing to the great enlargement of the upper part of the face and of the anterior half of the cranium, the lower jaw by comparison seems abnormally small.

In cases of this sort such subjective signs as appear are chiefly concerned with three structures, the nose, the eyes and the fifth nerve. Her nose shows a great thickening in the region of the nasal bones and the nasal processes of the superior maxillæ. The interiors of the nasal fossæ are largely blocked so that it is difficult for her to breathe through her nostrils. The sense of smell was absent for years but of late it has gradually returned so that she declares that she smells better than she did four or five years ago. Lately she has been able to sneeze, especially after using pepper. Formerly nothing seemed to be capable of exciting this reflex. Very recently she blew some blood from her nose, something which never happened before as far back as she can remember. It would seem from these facts that possibly there is an increase of the opening through the nares, possibly due to a diminution of congestion in the mucous membrane rather than to any actual decrease in the enlargement of the bones.

The eyes have never given her trouble. Subjectively there has never been a symptom of disturbance. Though she does not properly conjugate she declares she has never seen double, doubtless because of the very gradual development of her conjugate deviation. The lachrymal papillæ do not properly fit against the eyeball so that she is troubled with epiphora. When she goes out into a wind the tendency to flowing of the tears is increased. There is no hemianopia, either homonymous or heteronymous. Bi-temporal and bi-nasal vision are equally good. Examination of the fundus shows nothing abnormal. The absence of ocular symptoms and of acromegaly would seem to demonstrate that in her case there is no involvement at all of the hypophysis cerebri.

As to the fifth nerve, most cases of leontiasis ossea show some trigeminal neuralgia. In most of the cases of leontiasis ossea there is some contraction of the foramina of exit of the cranial nerves, especially of those coming forward. In the case of this young woman there has been a great deal of pain referable to the superior maxillary division of the fifth nerve. The ophthalmic division which comes through the sphenoidal fissure has three separate branches, namely, the frontal, lachrymal and nasal nerves and is not involved, possibly because of the fact that the sphenoidal fissure is not encroached upon. On the other hand the superior maxillary division of the fifth nerve which comes through the floor of the orbit and emerges at the infraorbital foramen is apt to be pinched in the orbital floor by the enlarging bone. Her pain formerly was very severe. For a year or two it was kept in abeyance by small doses of iodothyrine. Of late she has been taking one twentieth of a grain of calomel three times a day, she believes with much benefit to herself. At all events pain is now absent.

A few cases of leontiasis ossea have shown increased intracranial pressure, either in the form of persistent and severe headache or of motor manifestations, as of epilepsy. This patient has had some headache but only in amount and kind similar to that suffered by everybody. There has been no headache of a sort indicating any intracranial

pressure. There has never been in her case any sign of cortical irritation either of the sensory or motor cortex. A few cases have been reported in which there are thickenings of the soft tissues accompanying the bony changes in leontiasis ossea. This patient shows no change in the soft tissues at all.

CASE I. TABES DORSALIS (AMAUROTIC TYPE)

By D'Orsay Hecht, M.D.

Dr. Hecht said the patient was a member of the City Fire Department in active service on the hook and ladder. He was 37 years of age; married at the early age of 20, and was the father of several very healthy children. He had exposed himself to venereal disease in his youth and acknowledged to lapses of fidelity even during his married life, but failed to recall a single instance of gonorrhea, or any other venereal sore. It may be said that there is some somatic evidence of past lues in a general adenopathy and bony thicknesses of the tibia. Confining oneself to the conspicuous tabetic symptoms: The pupils are widely dilated and of the Argyll Robertson type; the fundus of the right eye is chalky white, that of the left disc gray white. The optic atrophy is complete. Vision is very seriously impaired. The patient can distinguish fingers held closely before his eyes; he can at best in a gray light appreciate a moving figure, that is all. The knee jerks and Achilles jerks are absent, the cremaster is present, the sexual power is good. Trunk anesthesia is manifest in a border about eight inches in width encircling the thorax. He presents little or no ataxia of gait; has no Romberg and reveals imbalance only when he stands on the right foot alone. Slight bladder involvement in the form of incontinence. The onset and advance of the tabes has been so insidious and attended with so little discomfort except for a few lumbo-sacral lancinating pains that only within the past few months when his vision began to fail appreciably, has he realized the presence of the disease. It is important to note that even though his vision was very defective in February, 1908, he nevertheless attended fires, setting up ladders against high downtown buildings, and doing all that was part of his work with the hook and ladder company, and doing it as well as he ever did. In February, however, he began to realize the dangers that could arise from his poor eye-sight and asked to be put on lighter duty. He is at the present time doing fire-watch service for the Ringling Bros. circus at the Colliseum. He was referred to Dr. Pardee and only by close inspection and observation could it be noticed that he had any impairment of his vision. Of ataxia there was none. It was Dr. Hecht's comment of the case that in the amaurotic type of tabes incoördination symptoms are known to remain in abeyance. This is the common experience and has been satisfactorily explained. The case is unique, however, because of the fact that despite the presence of all the classic symptoms of frank and advanced tabes including the highest degree of optic atrophy the patient has been able to follow his occupation and is even now not embarrassed by the service required of him.

CASE II. INFANTILE CEREBRAL PARALYSIS DUE TO POLIOENCEPHALITIS

The second case presented by Dr. Hecht was one of infantile cerebral monoplegia presumably of polioencephalitic origin. Dr. Hecht said: "This boy now seven years old experienced at the age of five years an acute illness which the mother says ran a course something as follows: On a Saturday afternoon the boy was out playing and felt well. In the evening the mother noticed he was warm and concluded there was some fever. She gave the boy a cooling bath. While in the bath the boy chilled. It was impossible to determine whether this was a genuine chill or merely a sense of chilliness. That night the boy slept very restlessly, starting up in his sleep and displaying a good deal of nervousness and irritability. The following morning he seemed well enough to go to Sunday-school. On his return from Sunday-school the mother for the first time noticed a slight limping and dragging of the right limb. This persisted for three or four days and then occurred a rise in temperature. A physician was called and noticed that the right leg was swollen as far as the knee, edematous and glossy. On the left leg there appeared large red, circumscribed patches of quarter-dollar size. The physician called a consultant and it was their joint opinion that the condition was one of rheumatic purpura. The disability in the right leg seemed to improve, but of the exact time there is no certainty because the child was in bed for three weeks. However, at the end of a month the boy got up and the legs swelled a second time. He was returned to bed for another period of three weeks and the disability in the right leg again became apparent. There had been neither delirium, nor clouding of the sensorium. At the end of this second three weeks the child got up only to be returned to bed with a third recurrence. With this last attack the spots began to fade out, the febrile symptoms disappeared, and there remained only the incomplete monoplegia affecting the right leg.

Dr. Hecht saw the boy for the first time at the Northwestern University Neurologic Clinic about a week ago. The child has limped these past two years to such an extent that raising the shoe one half inch to accommodate the limp has seemed necessary. The sloping of the right shoulder is owing to posture only. The arms and shoulders are normal. There is no atrophy. The disability is so slight as to be hardly noticeable in the gait, but on physical examination the knee jerk on the affected side is distinctly increased as compared with the left as is also the Achilles, and in addition there is a typical Babinski sign. The abdominal skin reflexes are normal.

Dr. Hecht demonstrated the disparity in these contralateral reflexes. He mentioned that there are no sensory changes or trophic disturbances. With this history, mode of onset, and the remnant of paralytic involvement of only a single extremity the diagnosis of cerebral monoplegia of subcortical origin might be ventured with some degree of positiveness and Dr. Hecht thought the lesion could be brought into direct causal relation with the purpura rheumatica. It would hardly be tenable to suppose a pyramidal tract involvement. Dr. Hecht's conclusion was that the case rightfully belonged in the rubric of those cases described and designated as polioencephalitis.

REPORT OF A CASE OF MELANOSIS OF THE BRAIN, CORD AND MENINGES.

By Ralph C. Hamill, M.D.

Family history negative. Patient denies venereal disease, had a head injury at fifteen years of age and typhoid fever at twenty-two, since which his legs have always felt cold. At thirty-one he was examined and found to have a Romberg sign. Ten months before death he had to give up work and developed perforating ulcer of the foot, lost sphincter control and became rapidly emaciated. He also developed bedsores, his hearing became very poor, he had a tendency to fall toward the left side and died of a terminal dysentery at the age of forty years.

The autopsy was held twelve hours after death and revealed reddish brown discoloration of the meninges, of the cord, cerebellum and base of cerebrum. The superior vermis was found practically destroyed by a pigmented growth and there was almost complete destruction of the cord in the region of the eighth and ninth thoracic segments. In the cervical region there was a peripheral pigmented zone, varying in diameter from one half to one millimeter, with a diameter of two millimeters in the upper thoracic region. The central canal was of pin-point size in the upper thoracic region and measured three millimeters at the sixth thoracic segment. Marked cavity formation from the seventh thoracic segment downward forming a large central cavity surrounded by a pigmented zone in the lumbar and sacral regions. There are pigmented, heavy granulations on the ependymal surfaces and pigmented granulations of pin-head size on the choroid plexus. The thickened soft meninges form an adherent ring around the lower thoracic cord. Teased preparations present the following features:

1. Large round nodular cells heavily pigmented with short protoplasmic processes.

2. Thread-like cells with small central nuclei poorly or not at all pigmented. Pigment in fine granules.

3. Cells resembling large glia spider cells. Pigment in large and small granules surrounding nuclei or lying in processes. Cells of types one and three invade nerve substance beneath pigmented meninges and show strong tendency to follow course of blood vessels.

4. Destruction of normal elements wherever invasior is marked. In the cord the posterior columns are much more involved than the anterior.

Periscope

Allgemeine Zeitschrift für Psychiatrie

(Vol. 64. 1907. Heft 2, 3)

1. Left-Sided Motor Apraxia. C. F. v. Vleuten.
2. Alcoholic Psychoses. Kurt Koldstein.
3. Mental Diseases in Russian Army during Japanese War. P. M. Awtokratow.
4. Psychiatry in North America. Fritz Hoppe.
5. Heredity in Dementia Præcox. Ryssia Wolfsohn.
6. Phantastic Degenerates. Karl Birnbaum.

Left-Sided Motor Apraxia.—A 55-year-old coachman, formerly in good health, began to show some general brain symptoms, had an attack of vertigo, after which it was noticed that though he showed an increased desire for activity, he did things in the wrong way. Losing confidence in himself on this account, he was, at his own request, sent to a hospital where, after a short stay, a diagnosis of dementia paralytica was made and he was transferred to Dalldorf. The course of the disease there is described by the author in three stages. In the first, while his intelligence was good, it was difficult to get him to fix his attention for any length of time, hence he could accomplish little mental work. Understanding for speech, ability to repeat words, and to read were intact. In spontaneous speech, he showed a gradually increasing difficulty in finding the correct word. Literal or syllabic paraphasia was never observed. Amnesic aphasia with verbal paraphasia. Paralysis or paresis, except for a slight weakening of the innervation of the right seventh nerve was not present, nor were there alterations of reflex contractures, etc. There were however, motor troubles, which were partly irritative disturbances of unusual and peculiar character, and partly apractic. The irritative disturbances were confined to the right side and were of the following character, first the right arm while quiet showed a moderate tremor which upon innervation increased enormously, second it showed a symptom which has been described by Liepmann under the name of "tonic perseveration"; namely when the right hand had grasped anything the patient was unable to let go for a long time, contraction of the fingers persisting involuntarily. The same symptoms, but in a less degree, were present in the right leg. Outside of the disturbance mentioned the movements of the right arm and leg were well performed "eupractic." The left arm and hand, on the contrary, when the patient was ordered to do something with them sometimes executed a number of movements entirely different from those required, at times remained still, and the patient complained of his inability to carry out the movement ordered. It also showed a slight tremor. That the order given was understood was shown by the patient generally repeating it, and sometimes by his carrying it out with the right hand? He was unable to perform movements of the tongue as required, otherwise mimic movements were good.

In the second stage of the disease a certain amount of dyspraxia in the right hand became evident, though this disturbance remained most marked on the left. The patient now showed a tendency to repeat questions and orders (echolalia), and at the same time there was developed an alteration of speech corresponding in character to the tremor of the hand, and at times the speech muscles seemed to pass into a condition of clonus, so that the patient could only stutter "bu, bu, bu." The mimic movements became altered. Towards the end of this time considerable mental hebetude was manifest, and to the motor-apractic reactions ideo-apratic were added, for instance the patient transposed the stages of complex acts, etc. In the third stage the patient passed into a condition of stupor though when aroused his reactions were similar to those observed in the previous stage, apraxia showed itself in ordinary movements, and the echolalia was more marked. Dementia now became complete, he slept a great deal, the tremor of the right arm had disappeared and in the place of the previously noted movements of the right arm there appeared a flaccid paresis of this member. The left arm continued apractic and showed now a distinct tremor. The patient gradually sank and died in a comatose condition. It is to be remarked that at no time was there choked-disc or other usual tumor symptoms.

The autopsy showed atrophy of the heart muscle, bronchopneumonia, and a large cerebral tumor proved by histological examination to be a sarcoma. The author illustrates the position of this tumor by seven cuts prepared after frontal sections stained by the Weigert-Pal method. Recapitulated, the tumor situated in the left hemisphere had destroyed (1). All of the white matter of the limbic lobe including the cingulum; (2), all of the left side of the corpus callosum to the most posterior portion of the splenium and a small portion of the genu, while on the right side a large part of the genu and about the posterior fifth of the body of the corpus callosum were unaffected; (3), anterior to the genu the centrum ovale of the frontal lobe was affected while posteriorly to this it was compressed but not destroyed. Entirely free were the third left frontal convolution, the central convolutions, the corona radiata belonging to these and the internal capsule. The great basal ganglia showed only minor lesions. The ventricles were moderately enlarged, more in the posterior lobes and to a greater extent on the right than on the left. The tumor had grown over from the median surface of the left hemisphere toward the right and was separated from the right lateral ventricle by only a thin strip of corpus callosum and the septum lucidum, but nowhere impinged directly on the corpus striatum or the optic thalamus of the right side. As the apractic symptoms observed in this case would seem best explained by the severing of the commissural conducting paths in the corpus callosum, the author at this point proceeds to compare these symptoms with those observed in the cases of corpus callosum lesions reported by others.

Summarizing he observes that we have here a tumor, the destruction caused by which was nearly limited to the corpus callosum. The absence of pressure symptoms, he thinks, is readily explained by the fact that the growth took chiefly the direction into the left ventricle. The clinical symptoms must, he thinks, be brought into connection with the callosal lesion alone since the destruction elsewhere was not sufficient to explain them.

Seeking an explanation which will connect clinical symptoms with pathological findings, he decides that the hypothesis of Liepmann that the motor memories of complicated movements being in the main stored up

in a center on the left side, this center through commissural fibres passing in the corpus callosum exerts an influence upon the center on the right side of the brain for the muscular movements of the left arm and hand. This influence being removed through the severance of these fibres in the corpus callosum, we have left-sided apraxia, while since the projection fibres for both sides are intact there is no true paralysis of either side. It is impossible in a review of this kind to do justice to the author's very interesting and instructive exposition of this case. It emphasizes the neeessity for thoroughly analyzing the symptoms in every case of mental disease real or supposed. Doubtless many equally interesting conditions have been unrecognized through too hastily concluding that the patient was "demented."

The Alcoholic Psychoses.—The author would in general consider the alcohol psychoses under the heads of acute alcoholic paranoia (acute hallucinosis) and chronic paranoiac psychosis, while acknowledging that the disease often occurs in a mixed or atypical form. As a contribution to the casuistics of the subject he selects a certain number of cases from the Konigsberg Psychiatric Clinic, giving their histories and analyzing their symptoms. Beginning with the acute cases, he emphasizes the fact that the two conditions most frequently met with are delirium with visual hallucinations, and hallucinosis in which there is little or no clouding of consciousness, and the hallucinations are commonly of the auditory sphere. In some cases there has been observed a distinct alteration of these two conditions. Hallucinations of taste and smell he finds infrequent, and when present they indicate deep involvement, hence a less favorable outcome. Hallucinations of common sensibility are more common, hypochrondriac sensations are about as rare as taste and smell hallucinations and of unfavorable import, pointing to a chronic course.

Primary psychomotor symptoms are never present in a true alcoholic psychosis. Where peculiar attitudes or motions suggesting stereotypy are present they are always of secondary origin due usually to the delusional ideas present. This point the author thinks of the greatest importance as distinguishing these cases from the paranoid and katatonic forms of dementia præcox. That the particular connection between optic hallucinations and conditions of clouded consciousness, and between auditory hallucinations and comparatively well preserved orientation is a fact, the author thinks is indubitable. When it comes to explaining why this is so, there are differences of opinion. He himself thinks that it is not so much due to a difference in the activity in different individuals of on the one hand the visual, and on the other auditory sphere, as to difference in the depth of the physical disturbance, the production of false visual images requiring greater clouding of consciousness than is necessary for false auditory perceptions. This, because, on the one hand the delirant receives more optic impressions, and on the other because the natural tendency on the part of all men to phantasy production runs more in the optic than in the auditory sphere. Taking up the subject of chronic alcohol psychoses the author gives the histories of six cases illustrative of the more prominent symptoms. These presented themselves partly as protracted hallucinoses, partly as residuary conditions with a specially characterized psychical weakness. In his discussion of them the author confesses his inability to bring out anything specially new.

3. *Mental Diseases in Russian Army and Japanese War.*—The author, who was sent by the Red Cross Society to look after the officers and

soldiers who became insane, and to superintend their return to Russia, gives an interesting account of his experiences, his methods of gathering up those who were taken ill at the front, of transporting them to his base at Harbin, where a small psychiatric hospital of fifty beds was established, their management there, and the arrangements for transporting them over the long stretch of railway to European Russia. He also adds some clinical notes on the forms of mental disease observed with tables. Among the officers chronic alcoholism took readily the first place, comprising more than a third of the cases.

Next in order came dementia paralytica and neurasthenic insanity. These three forms together made up 75 per cent. of all the cases.

Confusional forms and hallucinatory paranoia occurred in about two per cent. of the cases. Among the soldiers the epileptic psychoses took the first place, making up together with the alcohol psychoses and confusion 50 per cent. of all the cases. In the time of peace general paresis has occupied among the officers the first place, the alcohol psychoses the second; among the soldiers the acute psychoses came first, alcohol psychoses were uncommon, and the epileptic forms comprised not more than one quarter of all mental troubles.

The large number of cases of chronic mental diseases met with, the author attributes to the fact that during the mobilization insufficient attention was paid to the mental and physical condition of the troops. He mentions in illustration of this an experience of his own. Of four colonels who travelled with him in the same railway carriage one suffered from chronic brain lesion, a second presented plain symptoms of chronic alcoholism, a third had marked arteriosclerosis of the brain arteries and only one appeared healthy. Three weeks later he met one of his late fellow passengers who was being invalided home with a hemiplegia, having been stricken with apoplexy at the head of his command. The author explains the great increase in number of alcohol psychoses by the fact that combination of overexertion and underfeeding and the psychical shock of battle, with the influence of the alcoholic poison, made an influence too powerful to be resisted. In the case of those already chronic drinkers this precipitated an attack, but even in those unaccustomed to alcohol, who under the stress and horrors of the front commenced to drink, even in moderation, the deleterious influence made itself felt. He mentions in this connection the case of a young and previously healthy and strictly abstinent officer who at the front began to take two small glasses of brandy a day, and who at the end of three months was seized with acute hallucinosis which under treatment promptly cleared up.

The confusional cases were characterized especially by depression, the activity and flight of ideas common in amentia being wanting. The author was able to observe a number of cases of severe neurasthenia and neurasthenic psychoses. These showed mainly the picture of extreme depression and irritable weakness of the nervous system. Imperative conceptions and hallucinations were present in the majority of the cases, the latter having usually more or less to do with the horrors of the battlefield. The outcome in these cases was in the main favorable. A number of post-typhoid psychoses were also observed. These in the main ran a course like the exhaustion psychoses, but most of them the author was unable to follow out as they were sent home as soon as able to travel. The number of cases recorded he found multiplied by the fact that in many cases men who invalided home, improved on the way, or even in case of the chronic

troubles had a lucid interval, were through some official stupidity returned to the front, some of them several times.

4. *Psychiatry in America.*—The author give his impressions as obtained during a visit of a few weeks to the United States. He was able to see only the institutions about New York and Philadelphia.

He finds the medical profession in general still suffering to some extent from the former deficiencies in medical education, but of the system in vogue at the Manhattan State Hospital at which he seems to have spent most of his time, he has little but praise. The pathological institute he thinks the crowning glory of this, the largest hospital for the insane in the world. The system of committing lunatics in New York and the arrangements for the care of the acute cases at Bellevue, he finds, however, far from satisfactory, and in this those more immediately interested will possibly agree with him.

5. *Heredity in dementia præcox.*—Among 2,215 cases of mental disease admitted to the Burghoelzi Asylum from 1898 to 1905, there were 647 cases of dementia præcox. Upon these the authoress bases a statistical study of the influence of heredity in this disease. She draws the following conclusions: (1), about 90 per cent. of all cases of dementia præcox have a hereditary tare; (2), of the four factors in heredity, the most frequent is insanity in the ascendants, 64 per cent. Next follow in order nervous diseases, alcoholism and peculiar character; (3), heredity was in 34 per cent. of her cases combined. Most frequently it was insanity with alcoholism or insanity with nervous disease; (4), a distinct influence of heredity upon the form of dementia præcox could scarcely be recognized in the case of the heredity of alcoholism, nervous disease or peculiar character.

Mental disease in the ascendents seemed to make itself slightly more felt in the katatonic and paranoid forms, especially in the former; (5) the influence of heredity cannot be utilized in prognosticating the outcome in the first onset of dementia præcox.

6. *Phantastic Degenerates.*—The author sketches the characteristics of this class of cases which he thinks has a right to a special place among the psychopathic personalities. It is characterized by an inequality in the formation of the conceptional elements, a preponderance in the play of phantasy with an unevenness in the affective sphere, whereby the individual has an undeveloped sense of proportion, magnifying enormously trivial incidents or qualities of things as tallies with his affective condition at the time and minimizing other and more important ones, until he builds up phantastic edifices mountains high. That the ideas so developed may agree in many respects with paranoid delusions the author admits, but points out that in these cases there is never the development of a fixed and permanent delusional fabric as in true paranoia, though the conditions have points in common. Although it is found in the hereditary defectives, he thinks that these people are not to be classed with either the mental or moral imbeciles, nor yet are they hysterics. That a true psychosis may, however, develop upon the basis of this trouble he does not deny.

C. L. Allen (Los Angeles).

Miscellany

THE INFLUENCE OF SMALL DOSES OF ALCOHOL ON THE CAPACITY FOR MANUAL WORK. W. H. R. Rivers and H. N. Webber (The British Journal of Psychology, January, 1908.)

The results obtained by investigation of this problem are very contradictory. Thus Lombard, Rossi, Frey, and in some cases, Jotey ko have found the amount of work decidedly increased after doses of from 5 to 20 c.c. of absolute alcohol. Destrée and in some cases Jotey ko have found a decided decrease with the use of the same doses; and Schaffer and Féré found an initial increase by a fall below normal. Schumberg and Schnyder obtained positive results which, however, varied according to the conditions of the experiment.

The authors, for their work, endeavored to have each experiment under, as far as possible, exactly similar conditions. The experiments were undertaken at the same hour each day, and at the same interval after a meal which was of the same kind and amount. The other conditions of life were also regulated. The same amount of work was done. The same amount of exercise taken at the same times. Disturbing factors were avoided and all articles of food containing caffeine or alcohol were prohibited.

The most important factor to regulate, however, was believed to be the mental state and it is in this particular that these experiments are unique. The method employed to accomplish this result was the use of control substances. The most successful control was a mixture of capsicum, cardamon, chloroform and peppermint. This mixture so disguised the taste of alcohol that the subject could not tell when alcohol was administered and when the mixture was free from alcohol. By this method the authors believe they have done away with the disturbing factors of suggestion, sensory stimulation, and interest. Further control experiments were made with mixtures not so decidedly unpleasant but on the contrary pleasant, to determine whether this factor entered into the results. Of course, in both cases the alcoholic mixture had the same taste as the control. A further control experiment was made by giving the alcohol with the full knowledge of the subject. The instrument used for determining the effect on muscular work was Kraepelin's modification of Mosso's ergograph.

As a result of the experiments carried on along these lines the authors conclude that "Doses varying from 5 to 20 c.c. of pure alcohol are without effect on the capacity for muscular work when the factors of interest, sensory stimulation, and suggestion are excluded."

This conclusion is very interesting and seems to be more in harmony with what we might naturally expect than many of the results of experimental work that has been done. In view of the well-known depressing and paralyzing effects of alcohol this conclusion would make it doubtful if it was under any circumstances even for the first half hour or so after ingestion, as has been claimed, a stimulant. This probability is strengthened by the fact that increase in muscular work was found only once after taking alcohol and that then the subject's knowledge and the immediacy of its action lead the authors to the conclusion that the result was largely psychical in origin. They believe "that the results of most of those who have found the capacity for muscular work increased under the influence of such doses as 5 and 10 c.c. have been due, not to the physiological action of the substance, but to the interest aroused by

taking the alcohol and the sensory stimulation involved in swallowing it, while those who have found a diminution in the amount of work must be open to the charge of having been influenced by suggestion."

WHITE.

THE RELATION OF THE MEDULLA OBLONGATA TO THE PUPIL. L. Bach (Münch. med Woch., 54, 1907, June 18).

The author ascertained through experiments on monkeys, cats and rabbits that decapitation does not destroy the light reflex of the pupil unless there is an injury to the medulla extending to the fourth ventricle, when the light reflex disappears at once. Ruge obtained the same results from his investigations. To further explain these results H. Meyer and the author have made sections through the medulla of a cat, near the spinal end of the fourth ventricle and found that these caused the light reflex to disappear and a dilation of the pupils, while sections above or below that point never produced these phenomena. Exposure of the medulla or application of irritants also influenced the light reaction and produced myosis. As not all sections made at the spinal end of the fourth ventricle caused the light reflex to disappear, Meyer and the writer concluded that only a very circumscribed area was concerned with the light reflex, and are of the opinion that this small area is the pupillary center in the spinal end of the fourth ventricle. Burke and Trendelenburg in their eight experiments on cats were unable to obtain the same results as Bach. These authors are of the opinion that Bach's hypothesis of an inhibitory center must be abandoned.

FRED J. CONZELMANN (U. S. Army).

PARANOIA AND THE WORKINGMEN'S AID FUND. Dr. Tintemann (Münch. med. Woch., 54, 1907, July 23).

The author reports two cases in which two cases of physical injuries of a trivial nature were the exciting causes of the psychosis. Case No. 1, admitted January 1903: A man of marked neurotic and vesanic taint received contusions of the soft parts of the left leg in 1895 which incapacitated him for work for a long time. He received full support for one and one half years from the workingmen's aid fund, when he was declared to be able to earn his own living and all aid was withdrawn. Since then he has been striving, employing legal measures, to obtain the allowance of the aid fund. In addition to the complaint of the injured leg he now has general nervous affections. He felt that he was ill treated by everybody; many wrongs were done to him; he was fully convinced of his incapacity to work; wrote threatening letters to higher authorities. He was weak-minded and showed a marked judgment defect. In the court the question was asked whether the mental disturbance was a direct or an indirect result of the injury. The author expresses the opinion that the injury was the exciting cause in an individual with marked hereditary predispositions, and the patient was therefore entitled to at least part of the support of the aid fund. The second case was that of a laborer, who on account of rheumatism resigned his position and demanded that the annual rental allowance be allowed him for disability. When this was refused he began legal measures, had law suit after law suit which never terminated in his favor. He came in conflict with city officials. Refused to send his child to school; would not buy any shoes for the child in order to show his poverty; was brought to court and fined, refused to pay his fine; was imprisoned; when

relieved, blamed his wife for his imprisonment; it is on account of her he does not get his rental allowance. He was fully convinced that he had won in the legal proceedings but the mayor of the city refused to give out the verdict in order not to pay him a large sum for damages. The mayor owes him now 385,000 marks. The patient had a marked hereditary history, was feeble-minded and showed a marked judgment defect. Tintemann considers these cases closely allied to the real paranoics in whom a trivial physical injury and the desire to obtain a certain pecuniary allowance forms the exciting cause of the psychosis, otherwise they might have passed through life as normal individuals.

FRED J. CONZELMANN (U. S. Army).

TREATMENT OF TETANUS BY INTRASPINAL INJECTIONS OF MAGNESIUM SULPHATE. G. Canby Robinson (Journal A. M. A., August 10).

The author discusses the experiments of Meltzer and Auer in the treatment with intraspinal injections of magnesium sulphate and reviews briefly the reported cases in which this method of treatment was used. He reports a case of tetanus occurring in a boy, aged 11, with no history of traumatism or abrasion, other than a small swelling on the right side of head, in whom the illness set in eleven days before his admission to the Pennsylvania Hospital. The first symptoms had been slight pain and stiffness in the jaws, diagnosed as muscular by the attending physician. On the sixth day, after playing ball, there was profuse sweating followed by stiffness of the right side, especially the leg. On the following day—the seventh—there was general rigidity, which increased until his admission to the hospital, at which time abdominal pain was the chief symptom complained of. A diagnosis of tetanus was made and a lumbar puncture was made; about 8 c.c. of cerebrospinal fluid were withdrawn and 3 c.c. of a 25 per cent. solution of magnesium sulphate was rapidly injected. Five minutes later the neck was less rigid; in an hour there was distinct relaxation of the muscles. In two and one-half hours the boy was drowsy, his neck could be freely moved and his jaws separated; the abdomen was soft, the legs and arms relaxed; no knee jerk could be obtained. Six hours after the injection the rigidity began to return and 18 hours after it was almost as bad as on admission. On the evening of the second day another injection was given; 3.5 c.c of the solution was injected. The small swelling on the scalp—which was first described by the boy's mother on this day—was excised. The tissue contained a subacute abscess, but no tetanus bacilli could be found in either smears or sections. From this time the patient improved slowly till the twenty-third day, when the temperature reached normal not to rise again; after this the improvement was marked, and the boy was discharged on the fifty-ninth day. Besides magnesium sulphate in the treatment, sodium bromid, 1 dram, and chloral hydrate, 30 grains, in twenty-four hours, were used during the first two weeks. Bromids were given throughout the illness, and a few doses of morphin, 1/8 of a grain, were employed hypodermically. The boy weighed 65½ pounds on the forty-sixth day, but weighed 70 pounds when discharged two weeks later. Robinson concludes with a plea for more extensive use of this method of treatment in tetanus, and urges that all cases, whether successful or not, be reported.

Epidemic Acute Poliomyelitis in Norway. F. Harbitz and O. Scheel (Journal A. M. A., October 26).

The authors give an account of an epidemic of acute poliomyelitis occurring in Norway in the year 1903–6, with special reference to the nature and pathology of the disorder. 1905–6 a total of 1,053 cases were reported, with 145 deaths, or 13.8 per cent. Both adults and children were affected, the latter chiefly. The mortality was high in some districts, very slight in others. While most of the cases were followed by the usual paralysis, some were very mild and otherwise atypical, but the morbid process was probably the same. There was some evidence pointing to contagion in some cases, and the disease seemed to leave a certain immunity. The authors have had opportunity to examine necropsy material from 19 cases. Aside from the central nervous system, the findings were in all essentials negative. As regards the nervous system, they summarize their conclusions as follows: "In a severe or fatal case of acute poliomyelitis we, as a rule, have before us a diffuse inflammation of the entire cord with its pia mater, of the entire medulla oblongata and pons, of the basal ganglia and often also of the cortex of the brain to a greater or less extent, always in connection with a similar inflammation in the pia mater." They examined also four milder cases in which the patients had died of complications during the stage of repair; and in these cases by the use of special methods they were able to demonstrate the existence of degenerated conditions in the central nervous system greater than they had expected to find. The common character of the pathologic process in cases clinically resembling Landry's paralysis, bulbar paralysis and localized myelitis was also made evident in their examinations. As regards multiple neuritis in these cases their findings were negative, only what must be regarded as secondary changes being found in the peripheral nerves. A case of what was clinically meningoencephalitis occurring with this epidemic showed similar pathologico-anatomic conditions. Their investigations furnished no evidence of any relationship between this disease and cerebrospinal meningitis. They consider that there is no question but that the acute poliomyelitis is an infectious disease dependent on a specific micro-organism. This is true at least of the epidemic cases. The diplococcus described by Giersvold was found in the cerebrospinal fluid in three of their cases, but was lacking in the majority. While they cannot speak definitely as to its specificity, they think it demands further investigation. It is evidently an organism difficult to demonstrate; it seems to die rapidly, and experience shows that micro-organisms soon disappear in the nervous system. It is their belief that poliomyelitis is due to the direct action of some germ, and many facts favor the assumption that the atrium of infection is in the digestive tract, the nervous system becoming involved through the lymph stream along vessels and nerve trunks or through the blood.

Dementia Præcox. Smith Ely Jelliffe (Journal A. M. A., January 18).

The author defines the conception of dementia præcox, which he considers a fairly reasonable entity, as based on the following principal features: (1) The occurrence of the condition for the most part, in the years about puberty, adolescence and early manhood, *i. e.*, between the ages of 18 and 28. (2) The gradual development of a psychasthenic state deepening into a sense of incapacity which is the beginning of a general process of mental deterioration. (3) Thus a gradual emotional deterioration becomes apparent. This may be an indifference, or emotional stupidity, or

may be characterized, as Stransky has so well shown, as an incongruity between the content of ideas and their natural emotional sequence. This incongruity has been compared to an ataxia by Stransky, and there may be more complete dissociation between ideas and their usual emotional association. This emotional adjustment is one of the marked features. (4) Modifications in the so-called intellectual sphere are apparent, involving lucidity of consciousness and leading sometimes to complete confusion; variation in power of attention; some modification of the faculty of orientation, often not marked; delusion development, often on an hallucinatory basis; loss of perceptive power and the development of the sense of unreality, of inadequacy, ideas, of influence of reference and compulsory ideation. (5) As a consequence of these and other reactions, there results a fairly constant and perhaps distinctive alteration in the character of the individual. Jelliffe believes that the structural defects that later permit the crumbling of the intellectual structure are detectable in many cases before the breaking down has begun. Among the more constant of the characteristic anomalies are affectations or eccentricities of manner; a tendency to seek out striking combinations, neologisms in speech, bizarreness in writing, in artistic production and inapproachability. The finally developed childish, foolish manner so frequently seen as an end product is also perhaps best considered here. (6) Finally, these patients show marked modifications in their general muscular reactions, both in the striped and unstriped muscles. Physical negativisms, stereotypies, affectations of posture and bearing, catalepsies, automatisms and a host of sympathetic phenomena are here included. Jelliffe points out briefly the different characteristics of the different types, the cases of simple dementia or hebephrenic form, the catatonic and the paranoid groups, and goes at some detail into the psychology of the condition. Most of these patients, he says, are not hopeful cases, but some are worth working for, particularly in the predementia stage. When the diagnosis is patent the opportunities for repair have usually been neglected. He believes in training the eccentric and ego-centric children, who recruit this class of the insane, with special reference to their capacities, combining outdoor pursuits with education and utilizing the small social conventions that lessen the opportunities for unregulated affective action.

Book Reviews

Les Accidents Nerveux du mal de Pott chez les Adultes. Par L. Alquier.

This review points out that tuberculosis frequently affects the spinal meninges without producing any deformity. The author out of fifteen autopsies has found eight where involvement of the spine could not be ascertained. He excludes from his description the sub-occipital form. The statistics show that the process most often gives rise to nervous symptoms when it occurs in the dorsal region.

Pure bony compression occurs only in 20 per cent. and even then is generally complicated by other factors. In any case the epidural space is quickly invaded and inflammatory exudates outside it are the chief cause of the compression of the spinal cord. It is a tubercular peripachymeningitis.

The author points out the difficulty of appreciating the rôle of radicular involvement, on account of the contradictory findings of different authors, and on account of the fact that roots are often found surrounded by exudate, although no symptoms have been referred to that distribution, and in a case of anterior roots it is difficult to know how much of the muscular degeneration is due to involvement of the root and how much to lesions of the anterior horn.

The lesion of the cord is generally localized at the level of the peridural inflammation, with secondary degenerations; yet there are exceptions to this. Leptomeningitis is exceptional and may be due to toxic infiltration along the roots. Myelo-malacia may be the sequel, and at a more advanced stage a sclerosis through which, however, many more fibres functionate than the size of the structure might lead one to infer. These two varieties of lesions are generally intermingled in the same case. The former is apparently due to edema, and this explains the relative cure of the form of paraplegia thus caused.

The pathogeny is explained in two ways, (1) Mechanical action upon the cord, roots, vessels and lymphatics, and (2) the action of tuberculous toxins. While the agency of the former is shown by the rapid relief of symptoms upon the formation of a cold abscess or by surgical operation, yet there are cases where no deformation of the cord occurs and where the circulation is not mechanically hindered, as is shown by the rarity of small areas of softening of vascular distribution; and it is necessary to invoke the agency of toxins absorbed via the roots. Of the symptoms, the most important is the early root pains with their diffusion, intermittence and shooting character. Sometimes they take the form of double sciatica, sometimes with hyperesthesia leading one to suspect polyneuritis. They have been mistaken for intercostal neuralgia, pelvic disease, peritonitis, neuralgia of the brachial plexus, pleurisy, nephritic colic, rheumatism, frequently hysteria and even neurasthenia.

But the fact that the patient seeks immobility presumes against the diagnosis of a pure neuralgia. This prodromal phase is followed by less equivocal signs. If these occur suddenly they indicate a giving way of

the spine or the rupture of an abscess. More usually the onset is gradual. A flaccid monoplegia or a single band of anesthesia naturally indicates root involvement, while spastic paraplegia with Babinski's sign or anesthesia of the whole region below the lesion indicates spinal compression. The motor troubles generally precede, and indicate a better prognosis when unaccompanied by general sensory changes.

The author then indicates the four types into which he divides the manifestations. He points out the evil of flaccidity ensuing upon spasmodicity. As to the troubles of object sensibility, they do not always correspond with the level of the lesion. A syringomyelic or contrary dissociation indicates a destructive lesion of the interior of the cord, and it is prognostically bad as are bed sores and sometimes cystitis, which favor secondary infections. The author cites Dejerine's case, and adds two others where lepto-meningitis followed bed sores probably by infection along the nerves.

The diagnosis is only difficult when the root signs are equivocal; but these even when present, may be due to other causes. In its later stages Pott's disease may be mistaken for acute myelitis, which, however, is distinguished by lumbar puncture; but if syphilis complicates, great difficulty is presented. The author shows the difficulty of diagnosis in cases of polyneuritis, amyotrophy, radicular sciaticas of Lortat Jacob and Sabreanu, of myelomalacia, of pachymeningitis hypertrophica, of sypringomyelia, and of spinal paraplegias of the old, as well as with the classical tumor, aneurysm and other spinal compressions.

The most important element in the diagnosis is the root signs; but even their presence does not always make diagnosis possible.

The prognosis is not always bad; but the cure requires several years. It consists of immobilization and general hygienic measures.

The author follows Chipault in his disapproval of surgical intervention. A bibliography of recent work is given.

TOM A. WILLIAMS (Washington, D. C.).

BEITRÄGE ZUR DIAGNOSTIK UND THERAPIE DER GESCHWÜLSTE IM BEREICH DES ZENTRALEN NERVENSYSTEMS. Von Prof. Dr. H. Oppenheim, Berlin. S. Karger, Berlin.

This short contribution to the study of tumors of the brain and spinal cord is a report of several cases occurring in Oppenheim's practice. Most of the cases have been reported in the current medical literature. They reflect the skilled neurological technic of the author. The surgical procedures are extensively considered. As case histories alone are considered, we cannot go further into this contribution than to commend it to working neurologists.

JELLIFFE.

THE DANCING MOUSE. By Robert M. Yerkes, Ph.D. The Macmillan Company, New York.

The author tells us that this book about that species of rodents more properly called the dancing mouse was written for three purposes. First to present accurately and briefly the results of experiments made by the author, secondly, to give as complete a history of the dancer as a thorough study of all the observations made by others on the subject would permit; and thirdly to provide a text-book for reference in the study of animal behavior.

The dancing mouse is supposed to have come from Japan. C. Schumberger once came across an old Japanese wood carving which represents a group of dancing mice. In this carving one half the mice were white and black as were both the father and the mother; of the others two were a pure white and the other two entirely black. From this Schumberger infers that the dancer is the result of the crossing of two other species, namely the black mouse and the albino.

In experiments made by W. Haacke and G. von Guita, by crossing dancers with common white mice they both obtained a gray or black mouse which behaved very much like the wild house mouse; in them the dancing movement disappeared. When the race of waltzing mice is crossed with the race of albino mice, the waltzing habit partially if not entirely disappears. When hybrids produced by the cross just mentioned are paired, they produce dancers in the proportion of about one to five.

The movements which gives the mouse its name is a "giddy whirl" in which the performer merely goes around and around in circles, sometimes to the right and sometimes to the left, as fast as possible, and on all fours, the head pointing inward.

There are three kinds of dancers: those which whirl almost entirely towards the right, those which whirl towards the left, and those which whirl in either direction. The female mice are observed to dance more than the males.

The most important result of the study of the dance of these mice is that there are individual tendencies to turn in a particular direction, and the fact that the whirling increases in amount from morning to evening.

The power of equilibration in the dancing mouse is in general very complete; the seeming reduction which appears under certain conditions should be attributed not to visual dizziness, but in part to excitability and restlessness, and in part to reduced muscular power. The dancers, however, possess less power of grasping with their toes than do common mice, and are also greatly hindered in climbing and moving on narrow planes, owing to their tendency to move in circles. The adult mouse, as has been seen by Yerkes shows more timidity in climbing, etc., than the younger mice, but the younger are the more apt to fall.

The dancing mice are said to be deaf, the reason found by Rawitz being that the utriculus opens broadly into the scala tympani and the nervous element of the cochlea are degenerated. Yerkes says that the cause of the deafness is "secondary nature." The primary change is the broad opening between the utriculus and the scala tympani from which results the streaming of the endolymph from the semicircular canals into the cochlea. When as a consequence of the rapid whirling movements a large quantity of endolymph is hurled into the scala tympani, the organ of Corti in the scala vestibuli is fixed and its parts are rendered incapable of vibration.

In summing up the chapter on structural peculiarities and behavior, Dr. Yerkes says: "All the facts of behavior and physiology which have been established lead us to expect certain marked structural differences between the dancer and the common mouse. The bizarre movements, lack of equilibrational ability, and the nervous shaking of the head suggests the presence of peculiar conditions in the semicircular canals, or their sense organs; and the lack of sensitiveness to sound indicates defects in the cochlea. Yet, strange as it may seem to those who are

not familiar with the difficulties of the study of the minute structure of these organs, no structural conditions have been discovered, which account satisfactorily for the dancer's peculiarity of behavior."

The work makes an interesting introduction to the series and offers more than the usual information in a narrow field of research.

SYLVIA JELLIFFE.

ZUR PSYCHOLOGIE UND THERAPIE NEUROTISCHER SYMPTOME. Eine Studie auf Grund der Neurosenlehre Freuds. Von Dr. Arthur Muthmann. Verlag von Carl Marhold, Halle a. S. 1907.

This monograph is written with the avowed purpose of substantiating the hypotheses of Freud with reference particularly to the nature, origin, and treatment of hysteria and psychoneuroses.

The first part of the work is a very excellent setting forth of Freud's position, particularly his sexual theory of the origin of hysteria. The author accepts this theory in its entirety and in the analysis of a number of cases shows how it is possible to trace the sexual origin even to the early years of infancy and childhood. It would seem that he carries the theory to needless extremes when he endeavors to explain the constipation of women by voluntary retention of fecal matter so that they may experience the voluptuous sensations accompanying evacuation of the rectum, or the frequency with which young men choose the occupations of coachmen and conductors by the pleasing sensations resulting from the constant jarring incident to their work.

Three cases are given at considerable length to illustrate the sexual origin of the symptoms and the value of the psycho-analytic method of their treatment. Particular stress is laid upon the frequent associations of anxiety with a sexual complex, particularly with unfulfilled sexual longings. The method of treatment in all these cases was satisfactory and the author hopes may have spared them the exquisitely chronic and almost hopeless course that is not infrequently seen in the severe forms of hysteria.

In the closing pages the author quotes approvingly Bleuler's remarks to the effect that it makes no difference whether the therapeutic results Freud obtains can be reached in other ways, whether it is good to talk with old or young women about their sexual life, whether conversion, substitution, or defensive reactions in the sense of Freud exist, whether all or only part of that enormous complex of diseases which we call neuroses are dependent upon the sexual sphere. Answer these special questions as we will the significance of the discovery is in no way lessened.

It would seem that this temperate and conservative position of Bleuler leaves little to desire and that it is worth while harking back to when one is tempted to criticise rather harshly some detail of the theory.

Muthmann's work is well worth reading and constitutes a distinct contribution to the much disputed and illy understood subject of the psychoneuroses, and it is no small advantage that its style is pleasing and less ponderous than that of the master.

WHITE.

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Original Articles

CONFUSIONAL INSANITY AND DEMENTIA PRÆCOX.[1]

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The history of the differentiation of acute confusional insanity from other forms of mental disease follows the ordinary course. First established as an independent affection by Delasiauve and Westphal, it might fairly have been supposed to have won definite recognition with the appearance of Meynert's masterly essay in 1889 and Chaslin's monograph a few years later, even though English and American writers, always slow at that time in assimilating the psychiatrical work done upon the continent, made little mention of it.

The subsequent history of confusional insanity, however, has been peculiar. With a number of writers amentia, the term suggested by Meynert, was substituted for confusional insanity, but the development of Kraepelin's doctrine has forced amentia decidedly into the background and established dementia præcox as the chief mental disease. Amentia, according to Kraepelin, occurs in only one half of one per cent. of admissions to his clinique, while dementia præcox occurs in about fifteen per cent. and forms the great bulk of the permanent chronic inmates of the asylums. Stransky reports cases of amentia as a rarity and Jahrmarker believes that many cases of this rare disease are really dementia

[1] Read at the thirty-fourth annual meeting of the American Neurological Association in Philadelphia, May 22, 1908.

præcox or maniacal-depressive insanity. There has been a tendency, however, to include amentia with the toxic and infectious psychoses and to admit some connection if not an actual identity of the affections.

In the differentiation between the two affections Kraepelin, as is well known, stated that in dementia præcox the onset is gradual and there is not a previous history of exhausting influences. Among the characteristic symptoms are negativism, verbigeration, mutism, stereotyped attitudes and katatonic states. The patient is not influenced by emotions, and his attention is defective, but he has good perception and orientation. He has a fair memory for recent events, understands his environment, has correct ideas of time and recognizes persons. Hallucinations and delusions are less frequent. In amentia, on the other hand, consciousness and memory are more impaired, perception and orientation are much affected, the patient has no knowedge of persons or of recent events, and he is often emotional and has hallucinations and delusions. Negativism, verbigeration and stereotyped attitudes are rare. Amentia is of sudden onset, and often follows some exhaustion. Recovery is not uncommon, while in dementia præcox the tendency is to mental deterioration and recovery is rare and apt to be incomplete or followed by a recurrence of the disease with increasing dementia.

Many of the symptoms which Kraepelin attributes to dementia præcox, however, were described by Meynert as characteristic of amentia. The confusion of amentia, for example, is regarded as due to a disturbance of association, the projection system being unaffected. Perception is, therefore, not disturbed, but when the process advances further the projection system also becomes involved, and a state of stupor develops in which perception is also affected. Confusion and stupor, with Meynert, are thus different stages of the disease, the disturbance of perception marking a greater involvement of the brain. Kraepelin, however, assumes that the disturbance of perception is one of several symptoms which serve to differentiate amentia from dementia præcox. There can be no doubt that the cases reported by Meynert and his description of amentia correspond very closely to the cases and descriptions given by Kraepelin and his followers of dementia præcox. The distinction between the two is admittedly difficult at times (Paton). It is therefore not surprising that Bianchi

frankly admits that amentia, acute dementia, dementia præcox, katatonia, stupor and mental confusion are merely syndromes representing certain phases of a complex psychosis, to which he gives the name of sensory phrenosis, or that Régis and many other French writers regard dementia præcox simply as a more advanced stage of acute confusion.

Pathological anatomy, unfortunately, can offer us no aid in deciding whether we have to do with amentia or dementia præcox. The data are still too uncertain, even in the most intense form of the acute psychosis, delirium grave, for us to be able accurately to correlate symptoms and lesions, and in the less severe psychoses the pathological lesions are still more indefinite and inconstant. It is probable that in amentia and dementia præcox there are various alterations in the cortical neurones of the nature of central chromatolysis, dislocation of the nuclei, etc., but they are not constant and show no special differences, when they do exist, in the two affections.

The study of the pathogenesis of the two affections, furthermore, has not as yet reached the point where it can afford any definite help. While exhaustion or intoxication often plays an important part in the genesis of amentia, other cases arise where such a factor can not be discovered. Hereditary taint may exist in either affection, and its actual etiological significance is seldom carefully weighed; in amentia and dementia præcox, as in most nervous or mental affections, the existence of one or more cases of nervous or mental disease in the antecedents, no matter what their nature, is sufficient to prove the importance of heredity as a cause. Jung and Freud have of late sought to demonstrate the influence of various chains of associations or thought complexes in the genesis of dementia præcox, yet Jung is forced to introduce toxines as a further factor, and Weygandt and others of the Heidelberg school, opposing Jung's hypotheses, emphasize the significance of toxic factors in the genesis of the disease.

The attempt to differentiate diseases by the presence or absence of certain clinical symptoms is often necessary, especially in the early stages of investigation, but it is not always successful. Few symptoms are pathognomonic and most of them occur in a variety of affections. True as this is in regard to disease in general it is still more true with regard to diseases of the brain and especially to the more complex affections without such simple

symptoms of excitation or deficit as convulsions, paralysis or anesthesia. Such simple symptoms may point definitely to the seat of the trouble, but even these may give little help as to the nature of the affection. It is an axiom which can not be too often insisted upon, that the symptom in brain disease is dependent not upon the nature of the lesion but upon its location.

Now, whether we consider merely the various symptoms already cited, upon which Kraepelin has based his symptomatology of the two affections, or whether we study the more elaborate psychical phenomena with Sommer, Neisser, Stransky, Gross, Freud and Jung,—the blocking of thought, the narrowing of the field of consciousness, the capricious inability to recall facts, the enfeeblement of apperception, the negative suggestibility, the automatic compliance, the poverty of the emotional attitude, the incongruity between the emotional reaction and the content of thought, the dissociation of mental activity into several simultaneous trends of thought, and the like—certain things must be borne in mind.

In the first place these complex psychical phenomena just referred to, and even the simpler psychical symptoms, such as orientation, negativism, attention and the like, have not as yet been studied with sufficient accuracy and in a sufficiently large number of individuals in health and in diseases of varying kinds. The methods of investigation of these phenomena are not yet generally familiar and the technique is not yet definitely established. We do not yet know how often such phenomena may occur in normal subjects, or, if they do so occur, what are the conditions which may determine them, although some attempts have been made to investigate these points, notably by Stransky. There can be no doubt, however, that some of the psychical phenomena which have been noted in cases of mental disease are not the direct products of the disease itself but the normal reactions of an undeveloped mind reacting perhaps to diseased conditions. Mental depression, explanatory delusions, certain actions of the insane are but normal manifestations brought about by diseased mental states. Other conditions, slowness of thought, resistance to commands, attempts at would-be wit, even Ganser's syndrome itself, may be merely the mental characteristics of a stupid, pert young person who ordinarily has no restraint or good manners. Much of the affectation, posing, mannerisms and romantic dreaminess of the pre-

cocious dement, if an adolescent, is but an exaggeration of the normal characteristics of adolescence so well described by Marro.

As an example of the results sometimes obtained in making psychical tests in supposedly normal persons let me refer for a moment to the very simple test proposed by Marie for the examination of aphasic patients,—the test of the three pieces of paper of different sizes, with specific instructions as to what should be done with each piece. Whether it be from inattention or inability to grasp more than one simple idea at a time, a certain percentage of out-patients without any brain disease will fail to comprehend so simple a command, even when twice repeated. Even people supposed to be better educated, when a command involving two or more simple ideas is given to them, will half comprehend the first portion of the command and pay no attention to the subsequent modifications. The simple command "Lie on your back" is misunderstood by a considerable number of hospital patients; they catch the word "back," pay little attention to the rest of the sentence, especially if the word back be made emphatic, get the idea that the back is the important thing to be examined, and promptly roll over on the belly.

We lack, furthermore, sufficient information as to the modifications in these various psychical phenomena brought about by disease in different forms, or by physiological changes in the economy. We have certain studies, it is true, of the psychical symptoms of a few diseases which affect the mind predominantly, but we still lack psychical studies in cases of ordinary disease, or psychical studies made under varying physiological conditions. Jung, for example, has shown certain resemblances between hysteria and dementia præcox, but much remains still to be investigated.

Admitting, however, that we can determine these complex psychical symptoms as accurately and interpret them as definitely as we can the simpler symptoms of brain disease, the fact remains that neither the symptoms as given by Kraepelin as characteristic of dementia præcox, nor the more complex psychical states since investigated are pathognomonic of that disease. Verbigeration, negativism, katatonia, stereotyped positions, etc., may occur in amentia and the exhaustion and toxic psychoses, perception and orientation may become impaired in dementia præcox. Furthermore certain symptoms may be present at one

period in the history of the individual patient and not in another.

It is furthermore admitted that both diseases may pursue a similar course, although cases of amentia are more apt to recover and less frequently become demented. The outcome of dementia præcox, however, varies. Some cases recover completely, even after the disease has lasted fifteen years (Schaefer); some remain in a paranoid state for years, showing some mental deterioration but no marked dementia; others, especially the more youthful patients, rapidly become demented, and a few succumb in the early stages of the disease. Cases of amentia may result in the same way.

Although the distinctions are not absolute between the two affections, whether we consider the pathology, the pathogeny, the symptoms, or the outcome, it may be claimed that, nevertheless, there is enough to warrant differentiating the two diseases, even though we cannot draw a sharp line of division between them. There is certainly a difference between a case of simple hallucinatory confusion which makes a complete recovery in three months and a case of hallucinatory confusion which rapidly goes on to a stuporous dementia with negativism and stereotyped attitudes, or a case which dies in a few days with intense febrile delirium; yet the first and third types are classed together, and the second is classed under a different heading. There is, however, as much resemblance between the average case of amentia and the average case of dementia præcox as there is between Kraepelin's three types of dementia præcox, and as many transitional forms may occur. The variation in the symptoms is not always due to a difference in the disease itself, but to variations in the extent and severity of the morbid process or to differences in the subjects attacked by the disease. We see this most strikingly, of course, in the various types of mental disturbance due to chronic alcoholism. It is not probable that alcohol causes several different forms of mental disease, but the different mental syndromes that are produced by alcohol are due rather to the intensity of the toxic effect or the differences in the brains that are affected. It is hardly possible at present to prove absolutely by the study of individual cases whether they can be demonstrated to be amentia or dementia præcox. I can merely admit that the distinction is often difficult, and incline, like Bianchi and Régis, to the doctrine that the two affections are identical.

It may be said, however, that all this is a mere question of nomenclature, hardly worth spending so much time upon. Amentia is rarely spoken of to-day by American alienists and still more rarely diagnosticated. It is an ill phrase, any way, for amentia in its true sense, absence of mind, was long ago applied to idiocy. The French term of acute confusion or Bianchi's sensory phrenosis is much better; but it is the fashion to-day to call everything dementia præcox that we do not call by that most vile phrase, neither German nor English, "manic-depressive," so why should we not be content?

The objection to the term dementia præcox is obvious and is similar to that used by Bœrne against the name of the Holy Roman Empire—"weder heilig, noch römisch, noch Reich." It is admitted by Kraepelin himself that the disease sometimes begins after the age of fifty, and some cases do not become demented. The main objection, however, is the emphasis which is laid, by the acceptance of the term, upon the idea of dementia.

As Blumer has recently shown, the definition of the word dementia is still uncertain, but, after being originally applied to any form of madness, it gradually acquired, in the usage of earlier English alienists (Copland, Davis, Reynolds, Maudsley), the significance of "a failure or loss of the mental powers, usually consequent on other forms of insanity"; a terminal state of incurable mental decay.

That many of the victims of "dementia præcox" pass somewhat rapidly into a state of profound mental deterioration, and that many others show sooner or later considerable mental defect is only too true. In other cases, however, there is either complete recovery or healing with defect. In the latter case the patient may show merely a lack of insight into his previous condition, a few explanatory delusions, a deficiency in mental application and the like, perhaps so slight in character as to permit him to resume the ordinary duties of life, or at least to live in the world under a certain surveillance. In other cases there may be fairly complete recovery. I recall a case that seemed fairly typical of dementia præcox, with profound mental failure and most of the characteristic features of the disease, where the patient recovered so as to resume his profession and to lead an active professional life for years, with no mental change or deterioration in twenty years time, unless a lack of memory as to

certain features of his mental illness be regarded as evidence of dementia.

It seems hardly legitimate, however, to cite such slight mental disturbances, such as failure to recollect certain episodes in the past, a diminished capacity for protracted mental effort, a few eccentricities or even some explanatory delusions as a proof that mental deterioration is the inevitable outcome of the disease. Every form of brain disease shows during its progress some mental defect, and after recovery from any serious form of such disease there is usually some permanent defect left behind, although possibly slight in degree. Few persons regain their former state absolutely after severe neurasthenia. It is, however, certainly stretching the definition of dementia beyond its somewhat uncertain limits to pronounce such defects manifestations of dementia.

The character of the mental defect, moreover, is unlike that noted in either paralytic or senile dementia. Kraepelin lays special stress on the sudden flashes of intelligence and the understanding of his surroundings which the "precocious dement" sometimes shows. Only the other day such a patient, whose only response to me for years had been an unintelligible grunt, called me distinctly by name—a striking contrast to the impossibility of getting any spark of intelligence from an advanced paralytic or senile dement. It gives the impression that there are some cerebral neurones still capable of function, but inhibited by some unknown influence, whereas with the paralytic dement the conviction is only too strong that these same neurones are forever destroyed.

The old stuporous states and the term stupor have fallen into a more or less innocuous desuetude since the hegemony of dementia præcox, but the term stupor should not be abandoned and it is often more suitable for some of these conditions than the term dementia. It is often difficult to differentiate between the two or to decide when acute curable stupor passes into incurable terminal dementia, but the effort to distinguish between them should always be made.

Another noteworthy difference between dementia præcox and the other forms of dementia is shown by the course of the mental deterioration. Kraepelin and his followers have emphasized the "dementing" character of the various psychoses which

Kraepelin has grouped under the one heading of "dementia præcox," the tendency which they almost invariably show to mental deterioration. This is, however, only partially correct. In the majority of cases the mental failure develops more or less rapidly up to a certain stage and then becomes quiescent. This mental failure is usually more rapid and more complete the earlier the disease begins, the "hebephrenic" type occurring several years earlier than the paranoid type. This is to be expected, since the earlier in life that any disease attacks the brain the more likely it is to give rise to impairment of the intellectual faculties. The juvenile and adolescent brains are more unstable and more readily damaged irretrievably by disease. We note this especially in cases of hemiplegia: the adult hemiplegic often shows some slight mental impairment, but the juvenile hemiplegic is often an idiot as well.

After the establishment of his "dementia" the precocious dement may live to an advanced age, showing comparatively little change in his mental condition. I remember a case in an asylum, years ago, when the only record for several years was "Same old Sally"—a proof, of course, of the laziness of the physician in charge of the records, yet not wholly incorrect as depicting the unchanging nature of the trouble in one of the typical asylum dements who, like St. Simeon Stylites, keeps a fixed attitude for years. In paretic or senile dementia, on the other hand, the mental failure is steadily progressive, and is associated with progressive physical weakness. In the one case we have plainly to do with a progressive degeneration of the cortical neurones, in the other with a more acute morbid process which does its work of destruction, and then ceases to act, leaving behind a defect. It is true that the mental deterioration may seem to progress somewhat as time goes on, but this is often due to other causes. External stimuli have less effect upon the crippled brain and the deadening influence of asylum life produces "asylum dementia" which follows a life without interest, stimulus or occupation. The dullest of these "dements" are reeducable to some degree, as every asylum attendant demonstrates when his working patients are transferred to another ward, but systematic scientific effort by the physician for such reëducation is unfortunately too often neglected, although Colucci a few years ago showed that it might be attended with good results.

The "dementing" character of these psychoses, therefore, is not progressive, and the "dementia" can often be arrested and sometimes can be decidedly improved. Such a contrast to the true dementia of senility or general paralysis is certainly a reason for criticizing the appropriateness of the term as applied to these conditions.

The fact that a term is inappropriate or etymologically incorrect is not a sufficient reason for discarding it, if, like hysteria, it has the sanction of long usage. Dementia præcox has not that sanction, and, what is still worse, it is a term prejudicial from the start. To the physician, the family and even the patient it emphasizes the feature of dementia as the inevitable outcome of the disease, which to ordinary minds, in spite of the term "acute curable dementia," only too often convokes an incurable terminal state. The teaching of Kraepelin emphasizes the element of mental deterioration if not of actual dementia, which is not an inevitable result even if we accept the extreme doctrines of the Heidelberg school. Bianchi's term of sensory phrenosis is not open to this objection, and, if we accept with him the probable identity of confusional insanity (amentia) and dementia præcox, we can extend a larger hope to the patient and his friends by recognizing that complete recovery is often possible and that the patient is not inevitably doomed to "dementia præcox."

A CASE OF APRAXIA, WITH AUTOPSY[1]

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Until comparatively recently, by apraxia was meant the inability to use an object because its nature, or its use, was not recognized (Küssmahl and Starr (48)). Since Liepmann's exhaustive studies on the subject, however, apraxia takes on a different signification. It is a condition entirely independent of agnosia, or the failure to recognize an object.

According to Liepmann's (23, 24) quite recent opinion, the apraxic recognizes the object and its use, is able to move the limbs readily, indicating that the innervation of each limb is intact; but purposeful movements by the affected limbs are impossible. This, at least, is the definition for pure motor apraxia.

The description of apraxia by Pick (40), Van der Vloet (44), Margulies (28), and von Monakow (34), and the views held by Marie (29), especially, are not entirely in accord with this definition. Psychical disturbances in apraxics; the appearance of apraxia in certain forms of insanity; the element of inattention and lack of concentration so often observed in the apraxic subject, all suggest the possibility of the presence of other elements in the phenomena of apraxia, than are included in Liepmann's definition.

The whole subject is a difficult and complicated one, and the following case, while exhibiting certain symptoms in common with those of some of the reported cases, is generally unlike any case in the literature at my disposal.

The patient, J. C. G., aged 55 years, married; by occupation a railroad builder, and one-time member of the Canadian

[1] Read at the thirty-fourth annual meeting of the American Neurological Association, May 20, 21 and 22, 1908.

Parliament, was admitted to the Philadelphia Home for Incurables, July, 1906, complaining of loss of vision.

The family history was entirely negative. There was no previous history of illness, excepting the ordinary diseases of childhood. He had always worked hard; had been exposed to the elements as a young man, frequently sleeping out at night; and had always been a moderate, and at times excessive, user of alcohol. There was no history of specific disease.

His present disease began with failing vision, about three years before his admission to the Home. At the end of two years it had progressed so that he was unable to read.

Dr. David Webster and Dr. J. F. Terriberry (52) reported this case before the New York Ophthalmological Society in 1906.

Dr. Webster stated that the patient came under his observation August 28, 1905, at which time there was little more than perception of light. When the hand was held before the eyes he knew there was something there, but did not recognize it. He was unable to count fingers at any distance, or in any position, but he did not run into objects when walking about the ward. Dr. Webster believed that he actually saw objects, but could not distinguish their form; in other words, that he was suffering from "visual astereognosis." At that time the ophthalmological examination revealed no cause for the blindness.

Dr. Terriberry reported upon the neurological conditions at the same meeting. At this time the patient was unable to locate touch, which was, however, perfectly perceived. His sense of motion was also disturbed, and he was unable to execute any movement correctly. The muscular sense was absent in all the extremities. There was no disturbance of the sense of heat and cold; and taste, smell and hearing were normal. There was no evidence of palsy.

The vision continued to fail until, at the time of his admission to the Home, he was unable to distinguish objects, and the blindness appeared to be absolute, as he denied seeing the hand, or a flame, when it was held before his eyes.

He had had no difficulty in using his arms apparently, as he always dressed and fed himself, until some time in 1904 (over a year before admission) when his wife noticed that, if she took his left hand to lead him, he did not recognize that she held it. He gradually gave up the use of the left hand on the balusters in going up and down stairs, and in eating and dressing.

Present Condition: *Vision.*—Dr. William Campbell Posey examined his eyes shortly after his admission, with the following results:

"Eyes appear fixed and expressionless, the patient rotating them but rarely, in marked contrast to the searching, rolling movements so frequently observed in the blind. Winking is performed rather less frequently than is normal (Stelwag sign). When re-

quested to regard an object, the patient stares straight ahead, even though the object toward which he is told to direct his gaze is at the side, and the width of the palpebral fissures is somewhat increased on both sides (Dalrymple sign).

"There are no palsies of the extraocular muscles, although it is difficult to make the patient turn his eyes in the direction required. Both corneas are clear, and the eyes are free from any traces of recent or old inflammation. Both pupils are 5 mm. in size, and are quite active to direct and consensual light stimuli. This reaction is obtainable in diffuse sunlight, and to a much less degree from the light of an ophthalmoscopic mirror. The Wernicke pupillary inaction sign was searched for, but was not present, although the pupils reacted more readily to stimulation of certain parts of the retina than others; but these reactions were not from symmetrical portions of the retina, and were probably dependent upon a varying degree of atrophy of the optic nerves, and also upon the impossibility of always confining the light stimuli to definite and circumscribed retinal regions.

"The media are clear, and the optic nerves exhibit the early changes of a simple atrophy. As yet, however, there is no true atrophy, the nerves being dull red-gray, and possessing the appearance so frequently seen in men in middle life who have abused tobacco and alcohol, or who have cerebrospinal sclerosis. The blood currents are also well maintained, and the retina seems well nourished, and there is not sufficient ophthalmoscopic cause for the total blindness which is apparently present.

"Loss of vision appears absolute, as the patient denies seeing the hand when it is held close before his eyes, or even a flame, and that the blindness is not feigned is evidenced by the fact that it is not possible to obtain compensatory movements of the eyeballs by the use of prisms. On account of the absence of total atrophy, the apparent preservation of the functioning power of the retina, and the undoubted active reaction of the pupils to light stimulus, it would appear that the cause of the blindness is central, and not peripheral, and that the lesion producing it must be posterior to the center which regulates pupillary movements."

Sensory Changes: *Sense of Position.*—The patient was unable to tell whether he was standing or sitting, although occasionally, when this symptom was tested, he answered correctly. He failed to recognize the position of any of his limbs, or that they were being moved.

Location.—He was incapable of locating, anywhere about his body, the prick of a pinpoint, light or deep pressure, or heat or cold.

Sensation.—Tactile sensation appeared to be lost in the left hand, but was present in the right hand, and elsewhere about the body.

Pain Sense.—While examinations of the pain sense were rather

unsatisfactory, it was probably preserved. Pricks with the sharp points of the esthesiometer generally were followed by an expression of irritation. The right hand was withdrawn, but the left hand made no movement whatever.

Temperature Sense.—He confused heat and cold on the left hand, but elsewhere about the body the temperature sense seemed to be unimpaired.

Pressure Sense.—He was unable to distinguish between light and deep pressure anywhere.

Stereognostic Sense.—He was totally unable to recognize objects by the sense of touch. He recognized no object placed in either hand.

Motor Symptoms: The general muscular power seemed to be fairly good. The dynamometer registered 40 on the right, and 30 on the left. (He was right-handed.) There was no palsy of any of the limbs, and he was able to walk, although his gait was peculiar and will be described later.

Apraxia: He presented a few motor manifestations which, in all probability, were the result of apraxia, although this conclusion must be somewhat guarded, as the complete loss of the sense of location of the limbs and the body generally, the complete failure to recognize objects by the sense of touch, and the loss of the sense of movement, complicated the study of these symptoms. Another symptom which still further complicated the study of the case, was the behavior of the left arm. It was held in a slightly flexed position, the fingers semi-flexed most of the time, and rarely or never was it moved voluntarily. This was not due to paralysis, as the grasp was fairly good, and at other times the arm was observed to move freely, apparently without any sign of paralysis (Seelenlähmung).

Left Hand.—When asked to place to his ear a watch held in the left hand, no movement whatever of the left arm followed, but instead the right arm was stretched forward, and the right hand grasped either his knee or his coat, while at the same time he believed that he was placing his left hand to his ear.

When asked to squeeze the hand of the examiner with his left hand he was successful at times, but more often distorted, or irregular movements of the right arm followed, or he grasped some portion of his body with the right hand, and squeezed violently.

When a fork was placed in his left hand and he was requested to feed himself, the left arm remained motionless, or moved slightly only.

When told to touch his right hand with the left hand, only a slight movement in the left hand resulted, while the right hand groped aimlessly, as before described. Or, he would rise to his feet, without moving either hand, and believed he was making an effort to obey the command.

On the other hand, on several occasions he was able to take from his trousers pocket a handkerchief and wipe his nose with it voluntarily or when requested to do so, with either hand (reflex movement).

Right Hand.—The right hand was also the seat of disturbances of volitional acts. When a watch was placed in his right hand, and he was told to carry it to his ear, while at times this was successfully accomplished, he more often carried it to his mouth, and went through the motions of chewing; and this, in spite of the fact that he recognized perfectly that it was a watch that he was to place to his ear.

In feeding himself a banana he invariably put the banana to his chin instead of his mouth, if unassisted. In the absence of ataxia this is interpreted as being an apraxic movement.

He was unable to correctly use the hand for the purpose of dressing or eating.

In masticating his food the bolus was not moved around the buccal cavity in the usual manner, the jaw being moved rapidly up and down, and the food was often retained in the mouth a long time before he swallowed it.

When asked to grasp the hand of the examiner with his left hand, he occasionally succeeded, and at such times he would not relax his hold and apparently involuntarily, in fact, grasping more and more tightly, as long as the hand of the examiner remained within his grasp. This was undoubtedly a manifestation of perseveration, of the tonic variety described by Liepmann.

Gait.—The gait was peculiar. Short steps were taken, usually to one or the other side, rather than forward. From this he never varied, even when every precaution was taken to show him that there was no danger of his bumping into anything; in other words, to eliminate the influence of the loss of vision.

Writing.—When a pencil was placed in his right hand, and he was asked to write his name he moved the pencil up and down, from right to left, without forming any letters. He was entirely unable to make any movements when the pencil was placed in his left hand.

Mental Condition.—His intellect seemed to be fairly good. His memory was excellent and he discussed general matters cleverly and intelligently, apparently understanding everything that was said to him. He was, however, impatient, irritable and unreasonable, so that, unfortunately, the examination could not be carried out in the desired detail.

Reflexes.—The tendon reflexes of the arms were present on both sides. There was no Babinski sign or ankle clonus. The plantar reflex was exaggerated on both sides. The patellar reflexes were present, and probably exaggerated, but the rigidity of the limbs prevented much movement of the legs on the thighs.

Station.—With the feet together he stood perfectly.

Contractures.—The left hand was held in a slightly contracted position, as above indicated; that is to say, partial flexion of the forearm on the arm, and semi-flexion of the fingers. This could be passively overcome almost entirely, but there developed at the same time a tremor of both arms, especially of the left. There was also a slight but distinct rigidity of both legs and the right arm, but to a less extent than in the left arm.

Examination of the ears, made by the late Dr. W. G. B. Harland, gave the following results:

"Cerumen in both ears, but causing him no discomfort, and I doubt that removal of the masses would change the findings much. A more important source of error is found in the unreliability of his statement. We can say positively that he hears plainly the ordinary voice, but cannot hear higher tones, this defect being more noticeable on the left side. Deafness for low tones is also worse in the left ear, as shown by the tuning forks.

"He does not know that he is deaf, nor does he suffer from tinnitus. The results of the examination are what we might expect in an old man with a mild degree of middle ear disease, with secondary nerve involvement."

The tongue was protruded straight on command, and in the median line. It was tremulous and tooth-indented.

The left hand was swollen, and the left forearm was generally wasted slightly.

A diagnosis of bilateral cysts in the occipito-parietal region was made, an exploratory operation advised, and on February 1, 1907, Dr. Edward Martin opened the skull in the right occipito-parietal region. The pia was much thickened, and what appeared to be an area of softening was found. The patient made a good recovery from the operation, but the symptoms remained unchanged.

On April 4, 1907, efforts to overcome the contracture of the left arm and hand appeared to give pain. The left arm was very tremulous, and the rigidity generally seemed to have increased. His mentality appeared to have suffered, and it was impossible to make any satisfactory tests of his condition on account of his extreme irritability.

On May 10, 1907, the contractures of the left hand had increased, and the fingers were pressed forcibly into the palm of the hand. There was a Babinski sign on the left side at this date. There was no facial asymmetry. He was confused and violent, and his passages were involuntary.

There was at no time any vomiting, headache, or pain elsewhere in his body.

At this examination his sense of taste was markedly altered. He could not tell salty foods from sweet, and could not tell the consistency of food when it was placed in his mouth.

He gradually failed mentally, became at times excited, refused food, and died of exhaustion.

Autopsy.—At the autopsy there was an intense internal pachymeningitis on the right side, which was not observed at the time of the operation. The inner surface of the dura was covered with a thick plastic exudate, extending over the entire surfare of the brain on the right side. The dura was everywhere adherent to the skull, but was readily dissected away, with slightly more difficulty over the site of the operation. The bony plate which had been removed at the time of the operation and replaced was depressed about 3 millimeters below the level of the skull. Bony union seemed to have occurred in places, but there were two areas at the site of trephining where no closure by bone had taken place.

A necrotic area was observed on the right side of the brain, involving the cortex in the region of the occipito-parietal fissure, extending 3 centimeters in front of, and 15 millimeters behind this fissure, and laterally 45 millimeters from the longitudinal fissure. A cross-section in this area showed that the cortical layer was very narrow—about one millimeter in thickness. In the right occipital lobe the cortex was not half the normal thickness. On the left side it was macroscopically apparently normal.

The brain was first placed in formalin, and then in Müller's fluid. It was divided horizontally into six approximately equal portions, and these were blocked and cut in serial sections, and many of them stained by the Weigert hematoxylin method.

Right Side.—The uppermost levels showed degeneration of

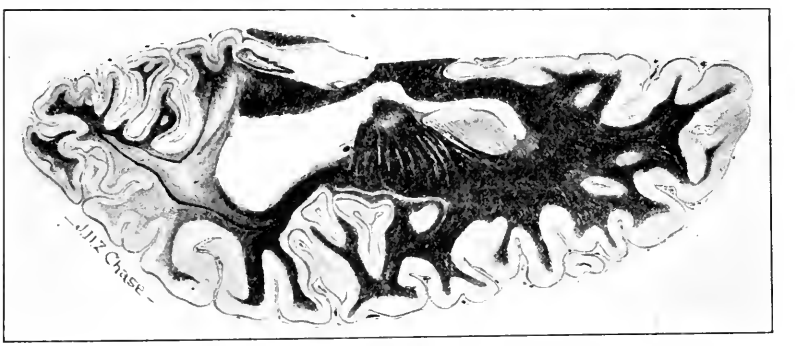

FIG. 1. Showing degeneration of white matter in occipital lobe and of the posterior part of the corpus callosum (right side).

the fibers from the cortex anteriorly and posteriorly to the occipito-parietal fissure. In this region there was some necrosis of the cortex and subcortical substance, the section showing slight loss of tissue, particularly just subcortical.

In sections from a slightly lower level the degeneration was more apparent, and extended further posteriorly, although not entirely to the extreme posterior pole. There was no degeneration of the white matter on the median aspect. With a two

thirds objective there were seen much perivascular distension, and some round-cell infiltration, in the degenerated area, and these changes were less where the degeneration of the fibers was less intense.

At a still lower level the degeneration extended forward into the ascending parietal convolution, and also to the posterior limit of the convexity.

Still lower, the ascending parietal convolution was slightly involved in its posterior aspect. The degeneration began just posterior to the interparietal fissure, and extended to the occipital pole. The median portion of the occipital lobe was not involved.

In sections from block 4 the white matter just posterior to the ventricle was degenerated, as well as the external surface of the occipital region, extending to the occipital pole, although

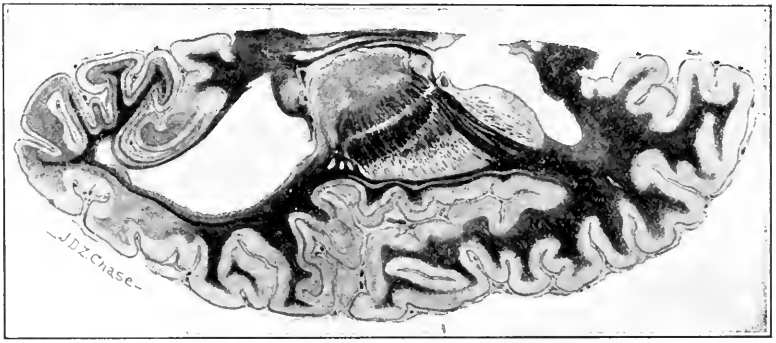

FIG. 2. Showing degeneration of the white matter of the occipital and second temporal convolutions of the optic radiations and the inferior longitudinal bundle (right side).

here it was less intense. The fibers coming from the calcarine region at this level stained well, as did also the inferior longitudinal fasciculus, although it was smaller than in the normal brain. At this level the posterior part of the corpus callosum stained less distinctly than normally. The posterior horn of the lateral ventricle at this level was markedly dilated.

In sections still lower from this block the same condition existed, except that the degeneration extended to the inferior parietal lobe. The inferior longitudinal fasciculus, which was much smaller than normal, was degenerated at this level. The optic radiations and the tapetum were also degenerated. The median surface stained normally.

In sections from block 5 there was, in addition to the condition just described, involvement of the second temporal convolution to a slight extent, in its posterior portion. The splenium stained poorly, while the tapetum and inferior longitudinal fasciculus appeared to be smaller than normal. The forceps was

smaller than normal, and the optic radiations were degenerated. The median surface was normal.

In sections from block 6 there was a small area of degeneration in front of the anterior occipital fissure of Wernicke. The optic radiations of Gratiolet, the tapetum, and the splenium were all degenerated.

In the lowest sections that were cut and stained the degeneration of the white matter did not extend in front of the inter-

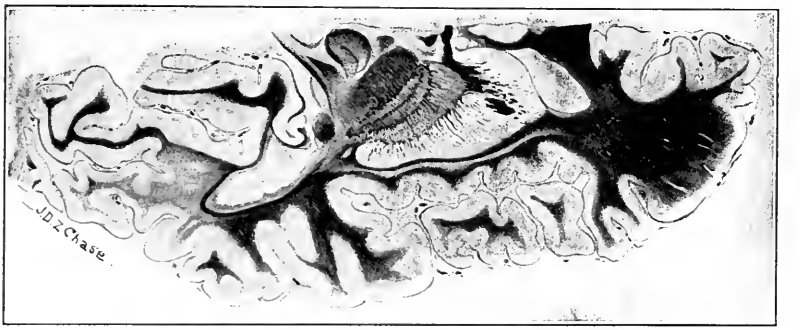

FIG. 3. Showing degeneration of the white matter in the occipital lobe posterior to the interoccipital fissure (right side).

occipital fissure. The inferior longitudinal fasciculus was normal. The internal capsule and basal ganglia showed no abnormality, and the pons, at the level of the red nucleus, which was included in the lower sections, stained normally. The foot of the peduncle showed no degeneration. The zone of Wernicke was not degenerated and the pulvinar and geniculate bodies appeared to be normal.

Left Side.—Sections from the uppermost levels (blocks 1

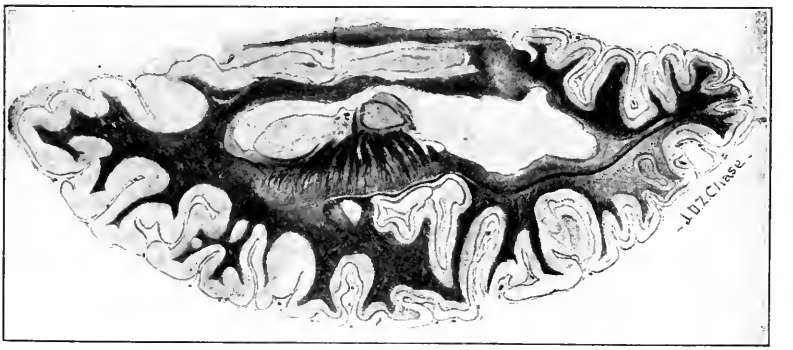

FIG. 4. Showing degeneration in the posterior ventricular region, external portion of occipital lobe, inferior longitudinal fasciculus, corpus callosum, and optic radiations (left side).

and 2) showed only a very slight degeneration in the occipital region. There was also some slight perivascular distension, and infiltration of round cells.

In sections from block 3 the degeneration was very slight, and situated in the external portion of the occipital region. Degenerated fibers could be traced from this region towards the corpus callosum. The inferior longitudinal fasciculus stained normally.

Sections from block 4 were degenerated in the posterior ventricular region. The degeneration in the occipital lobe at the convexity, while apparent, was slight. The median surface was normal. The degeneration did not extend into the parietal region. At this level the optic radiations, and the inferior longitudinal fasciculus, were slightly degenerated, and the posterior portion of the corpus callosum markedly so.

In sections from block 5 the degeneration was still apparent

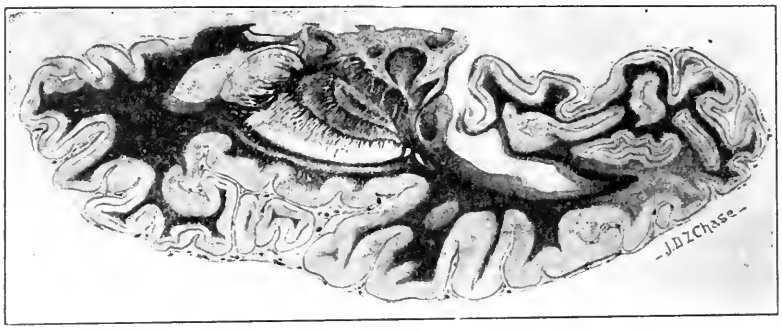

FIG. 5. Showing degeneration limited to the external portion of the occipital lobe (left side).

in the external portion of the occipital lobe. The tapetum and inferior longitudinal fasciculus were smaller than normal, but stained well, and the posterior horn of the ventricle was dilated.

A little lower the sections showed degeneration limited to the external occipital region. The external capsule, the inferior longitudinal fasciculus, and the tapetum were normal at this level, excepting that the latter two were smaller than in the normal brain. The lower temporal lobe was slightly implicated in its posterior portion.

In sections from block 6 the degeneration was still apparent in the external occipital region, but otherwise the sections appeared to be normal, including the basal ganglia, the foot of the peduncle, the pons, and the zone of Wernicke.

The optic chiasm stained normally (Weigert method), as did also the right and left optic nerves.

Microscopically the cells were unchanged in both parietal regions (thionin stain). There was a moderate degree of peri-

vascular distension. The blood vessels were thickened within the brain substance, and a few compound granular cells were found around the blood vessels. The pia was much thickened on both sides, but less so on the left. On the right side hemosiderin was found in the pia.

In the paracentral regions the pia was thickened slightly, and a mild degree of perivascular distension was observed. The cells stained normally.

In the calcarine region there was slight perivascular distension, but the cells stained well. The pia was slightly thickened, the more so on the right.

Sections from the necrotic area in the right occipito-parietal regions showed the presence of a few compound granular cells, a number of spider cells, marked perivascular distension, thickened blood vessels, and some perivascular infiltration. A few of the perivascular spaces contained blood pigment, probably hemosiderin.

Sections from the pons and medulla oblongata and the spinal cord, stained by the Weigert method, showed no degeneration.

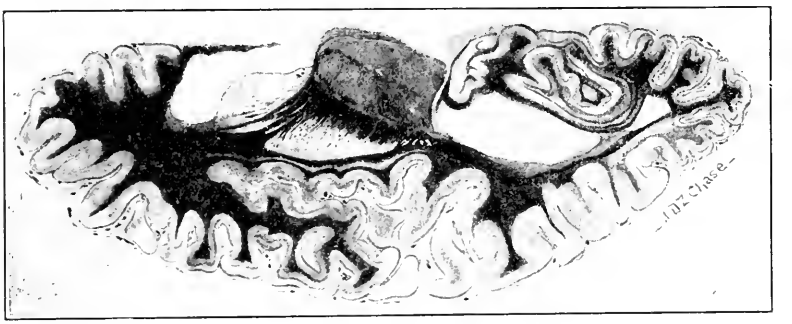

FIG. 6. Showing slight degeneration of external part of the occipital lobe (left side).

Cells in the anterior horns of the cervical and lumbar regions were slightly diseased. The Nissl bodies were atrophied, and the yellow pigment was increased. Some had lost their prolongations, but there were many normal cells.

SUMMARY: A man of 55, at the time of his admission to the home, was blind; was totally unable to designate the position of the limbs; could not locate touch anywhere; could not recognize objects by the sense of touch; and his touch and temperature senses were imperfect in the left hand. The left hand, although capable of some reflex acts, could not be moved voluntarily. The right hand was apraxic, and apraxic phenomena were present in chewing and walking.

The autopsy revealed the presence of degeneration of the white matter of the right occipital and parietal regions on the convexity, and the posterior portion of the temporal lobe, the calcarine region remaining intact. The inferior longitudinal fasciculus and the optic radiations were degenerated on the right, and probably though less markedly on the left. On the left side there was degeneration in the occipital and temporal regions to a much less degree, leaving the median surface intact. The corpus callosum, in its posterior portion, was degenerated. Elsewhere the brain was apparently normal.

The process was probably the result of arteriosclerosis which caused degeneration in the white matter from deficient nutrition. The necrosis of the right convexity was probably of the same origin.

The difficulties of explaining the symptoms presented in this case are very great, on account of the absolute loss of the sense of position and movement, the loss of the ability to locate tactile impressions, and the total blindness.

Was his utter lack of knowledge of the position of his body—for he could not tell whether or not his limbs were moved, or whether he was standing or sitting—due to a loss of muscle sense, or was it related in some way to the blindness?

Pathologically the general loss of muscle sense was not explained. The lesion in the right parietal region would account for the sensory change on the left side, but absence of involvement of the left parietal region leaves this symptom on the right side unexplained, for, according to von Monakow, the parietal region is the center for the muscle sense and the sense of location.

The absence of lesions sufficient to explain in the usual way the general loss of the sense of position of the entire body leads us to suspect that the destruction of the fibers going to the visual centers may play some part in causing it.

A possible analogy is seen in those cases of mind or psychic paralysis (Seelenlähmung) in which, with the eyes open, a limb is moved freely, but when the eyes are closed the limb acts as if dead; that is to say, when the optic element is cut off, or poor, the movement is disturbed, and the location of the limb is not recognized. An example of this was present in Liepmann's patient who, with the eyes closed, lost all knowledge of the position of the right arm. This is not unlike the facts presented by my patient, whose entire body, on account of his blindness, was

constantly in the same condition as was the right arm of Liepmann's patient, when he closed his eyes.

Or, was the loss of the sense of position of the body a form of disorientation, analogous to that which is observed in mind- or psychic blindness (Seelenblindheit)? Against the acceptation of this latter view, however (in spite of the fact that the post-mortem lesion was that which is commonly found in mind-blindness), is the absence of the characteristic symptoms of mind-blindness. The mind-blind patient sees objects, and has light impressions, but is unable to recognize the nature of the objects which he sees.

In my case, as the examination of Dr. Posey and myself clearly demonstrated, the man was totally blind. The report of Drs. Webster and Terriberry does not convince me that he was suffering, at that time, from mind-blindness. All the symptoms which he is reported to have presented could be readily accounted for by pure blindness with slight preservation of light perception.

It is possible that there might have been some light perception preserved after his admission to the home, in view of the fact that when the hand was suddenly placed before his eyes, while he was walking, at times he hesitated or stopped, according to the observation made by the orderly; but this observation was never confirmed by myself, although frequently tested for, and he was often seen to run into things and had injured himself in that way. In mind-blindness there is a loss of the recognition of the nature of the objects, with preserved sensation, and this is not true in my case.

Apraxia.—The movements which I believe to be probably due to apraxia are as follows.

1. The curious disturbance of the gait. The patient took short, rhythmical steps, usually to one side. That this distorted gait was not due to his blindness is probable, as he did not seem to have any fear of bumping into things, and the peculiarity of the gait continued in spite of his being assured that every precaution was taken to prevent his bumping into objects.

2. The false movements. In eating, for example, in an effort to place to his mouth a banana, he would invariably place it first upon his chin. This was not due to ataxia, as there was not the slightest appearance of this symptom in any of his movements.

3. Or, if a pen were placed in his hand, and paper before

him, and he was asked to write, only hacking movements from right to left resulted (apraxic agraphia?).

4. A watch was placed in his right hand, and he was requested to put it to his ear. He recognized that it was a watch, though not by touch, but instead of putting it to his ear (although he sometimes succeeded) he usually placed it to his mouth, believing that he was holding it to his ear, at the same time going through the movements of chewing.

5. When asked to touch his ear with the right hand, he made groping movements in the air, or grasped his knee, or his coat, and pressed tightly, believing that he was touching his ear. Or, he would stand up and make one or two steps, still believing that he was touching his ear.

6. Apraxic movements of the muscles of mastication.

Some reserve is necessary in deciding whether or not these movements were apraxic; as the studies could not be carried beyond a certain point, or in great detail, on account of the loss of the sense of position, the blindness, the loss of perception of the nature of objects by touch, and the perseveration present in the left hand. These symptoms, therefore, do not entirely accord, in all respects with the definition of apraxia given by Liepmann, for these reasons.

According to Liepmann's definition of apraxia, an object is recognized, its use known, the limbs themselves are readily moved, so that the innervation of each limb is intact, but purposeful movements (Zweckbewegungen) of the affected limb are lost.

The amorphous movements of G. in walking and attempting to touch the nose, however, correspond to similar movements in Liepmann's case.

The false movements in eating, in the case of G., which were present in Kleist's (16) case, and possibly the amorphous movements of the hand, in the effort to write (if this was not pure agraphia) may be classified under the head of "False Movements."

Agnosia.—Can the element of agnosia be eliminated in explaining the nature of these movements?

When my patient was eating a banana he recognized the object, although not by touch, but by a psychical process. He understood that, in order to eat it, he must put it to his mouth; but when he made the effort to place the hand to his mouth he never succeeded, the banana invariably landed on the chin.

In attempting to place a watch to his ear he recognized that it was a watch, not from hearing the tick, or from the sense of touch, but from the fact that it was a watch that the examiner would naturally place to his ear for him to listen to. In other words, he recognized the nature of the objects which he attempted to use. Moreover, he could use his right hand for reflex acts. He therefore knew the nature of the object, its use, and was able to move the right arm, but he could not use the object properly. It seems to me, therefore, that the false movements that occurred under these circumstances were apraxic.

The explanation of the symptoms in my case is scarcely to be found on the basis of agnosia; at least my conception of agnosia. As I understand it, agnosia is a loss of the understanding or the perception of the nature of things, while sensation is preserved, an illustration of which may be seen in the loss of sensory memory forms (Wernicke), or a loss of the connections between sensations and memories in mind-blindness, mind-deafness, and mind-paralysis, which, according to Hartman (12), are expressions of agnosia.

Nodet (36) defines agnosia as a disturbance of secondary identification, with persistence of the primary identification, and Liepmann, Hartman and others also looked upon agnosia as a failure of recognition (Erkennen) with preserved sensation. The identification of the fresh impressions with the memory forms does not occur, either on account of the loss of the latter, or on account of a hindered connection between both of them (Liepmann).

In other words, agnosia is a difficulty of recognizing cortical sensory impressions, and a difficulty of connecting these impressions with a mental picture. For example: One takes an object in the hand in which all forms of sensation are preserved; recognizes its shape, physical, and other properties, and can describe them fully; but is unable to recognize the use of the object, or its name. This failure of recognition is due to an interruption between the memories for objects, and the sensory centers in the cortex.

These are not the conditions present in my case. There were, on the left side, distinct sensory changes manifested in the disturbance of the temperature sense, the sense of location, the sense of movement, and the muscular sense; while on the right side

the sense of location, the sense of movement, and the muscular sense were altered.

Felix Rose (43), who believed that my case was one of agnosia, stated that in agnosia the confusion of movements is based upon the fact that the patient takes one object for another; for example, a toothbrush is used as a pencil. He admits that in motor and ideatory apraxia the substituted movements are observed, but the association between the logical act, and the executed act is evident; for example, the patient brushes the beard with the toothbrush. Rose believed that agnosia produces especially the substituted movements, while this is the exception in motor apraxia.

In the case of G. there is no analogy with these statements of Rose.

Substituted movements occurred when my patient made an effort to touch the ear, for example, when the right arm was used in a groping manner, as illustrated in Liepmann's case. While he placed the watch to his mouth instead of to his ear, it was not because he did not recognize what the watch was. Moreover, in the case of G. the primary identification was at fault, a condition the opposite to that described by Nodet and others, who state that in agnosia primary identification is preserved.

Apraxic Movements.—While the apraxic movements could not be studied in detail, for the reasons above enumerated, those which I claim for my patient are similar to those described in at least some of the cases in the literature.

In Liepmann's (21) case the apraxic movements consisted, among others, of amorphous movements which were represented as follows: If Liepmann's apraxic was asked to point to his nose, the request was followed by a strained position of the arm on the right side, nodding of the head, or repeated bowing, the patient thinking all the time that he was touching his nose. This was analogous to the movements observed in my case, in which the patient, in an effort to touch his ear, groped in the air aimlessly with his right hand, or grasped his knee, or stood up and made one or two steps.

When Liepmann's patient was asked to point to the left hand with the right, he nodded "yes," and lifted an ink well in front of him. He could not make a fist with his right hand, distorted movements following instead. He could not dress or undress him-

self (which was true in my case), and many special acts, as well as imitation movements, were unsuccessful.

The similarity of the movements in my case to those of Hartman's patients is striking. In his cases the direction of the movements was missed. When the patient attempted to touch the right extremity with the left he failed, inappropriate, distorted movements occurring as to direction and form. Taking nourishment by the mouth was performed very slowly, improperly, or done in stages.

In Pick's (40) cases the disturbances of movement were somewhat different, and may be looked upon as expresions of idiomotor apraxia. For example, a patient brings a lighted match near a candle correctly, lights it, and then finally blows it out.

Again, the patient handles a pistol as if it were a musket; or, if given a cigar and a matchbox, the patient evidently recognizes the cigar, but opens the matchbox, sticks the cigar into the open end, and presses upon it as if it were a cigar-cutter. He then rubs the cigar on the side of the matchbox as if it were a match; but finally the movement is made correctly.

Or, a watch is shown to the patient, and he is asked what it is. After many questions, he takes it in his hand and raises it to his lips. This resembles the movements in my case.

Bonhoffer's (4) patient struck the cigar on the matchbox.

D'Hollander's (14) patient, when requested to strike a match, grasped the matchbox in his left hand, took out a match with the right, hesitated, placed the match in his mouth, then finally replaced it in the matchbox again. His case was one of paralytic dementia in which the autopsy revealed the characteristic lesion of this disease without focal lesions.

In Kleist's (16) case of cortical, or innervatory, apraxia, the patient was requested to sharpen a pencil with a penknife. He took the pencil in the left hand, grasped the knife with the right, sometimes with the back of the blade, and sometimes with the cutting edge uppermost, and supinated the forearm so extremely that the knife was held with the edge, instead of out and under, in and down. When he had succeeded in pronating the arm sufficiently in the effort to sharpen the pencil, the necessary extension and flexion of the arms brought the position back to supination. He then pressed and scraped the pencil, then shook his head with an expression of distress. At another time the patient made an effort to cut a piece of paper with a pair of scissors. Instead

of placing the thumb and forefinger in the openings of the scissors, he held the scissors between the thumb and forefinger, and pressed these against each other. Later the thumb and finger were placed in the openings, but in the effort to open them they slipped out, and he held the scissors as before. Instead of opening them he pressed the handles together. Once or twice he succeeded in making the cutting movements, but then lapsed again into the movements just described (innervatory apraxia).

In Kleist's cortical, or innervatory apraxia, the disturbance is manifested in all forms of movement. There is an incapacity to perform those movements which arise from special memories; in other words, memories acquired by practice. Antagonistic, or useless movements appear. Partial acts are undisturbed, but the preparation of the act is implicated in its innervation. The motor memories are involved (auto-kinetism), not alone the sensomotorium—differing from Liepmann's apraxia in which the sensomotor "Eigenleistungen" is intact—and therefore not due to any disturbance of the movement formula.

In Strohmayer's (46) case the patient put his hand, instead of the spoon, into the soup, or cut with his fork or the back of his knife, while recognizing the objects and their use.

Bonhoffer's patient, in making an effort to shut the door, moved the hand in an apraxic manner. He held the key in his hand, making thrusting movements with it instead of the proper ones. He was unable to write certain letters, although he knew them, but produced figures without character. The latter Bonhoffer believed was an apraxic agraphia.

In Lewandowski's (17) case of progressive paralysis, the patient was able to make only three movements with the left arm, *i. e.*, to the back of the ear, to the mouth, and rubbing movements. These, however, were probably not true apraxic movements.

In a second case reported by Lewandowski (18) there was apraxia of the eyelids. The patient was unable to close the eyes on command, neither one at a time, nor both together. This was, according to this observer, independent of a slight left facial paralysis.

It will be seen that there is a great variety of abnormal manipulations ("Handlungs," Liepmann; "Agierns," Abraham (2)) of objects, which are described under the head of apraxia. These may be divided into (1) manifestations of ideomotor apraxia (ideokinetic, of Liepmann, formerly motor apraxia); (2) ideatory

apraxia (ideomotor apraxia—Pick); (3) innervatory apraxia (Kleist).

Von Monakow (34) classifies apraxia as follows: (1) Bilateral apraxia associated with right-sided hemiplegia; (2) agnosia, or sensory apraxia; (3) amnesic apraxia, as in cases of progressive paralysis; (4) unilateral apraxia, as in Liepmann's case; (5) ideatory apraxia (Pick). He states that in apraxia movements remain undisturbed, or are only slightly involved, in (1) breathing, swallowing and eating; (2) in elementary movements of orientation, such as turning the eyes and head in the direction of an irritant, when there is not a concomitant central optic, or other trouble; (3) in gross reflex acts, as in movements of defense; (4) locomotor movements, as sitting up, or walking; (5) in simple acts (moderately free from involvement); and in (6) simple automatic movements, such as unbuttoning a button.

According to Liepmann, motor apraxia affects single limbs, and is rarely bilateral (cases of Hertzog (10), Liepmann, Strohmayer, and possibly my own case), in contrast to ideatory apraxia in which both sides may be affected. Motor apraxia appears in simple acts, such as putting out the tongue, or making a fist. This is not true of ideatory apraxia, which manifests itself in complicated acts especially. In motor apraxia the limbs do not obey the psychical wish. In ideatory apraxia there is a failure of the psychical conditions for the correct completion of the act, but the limbs respond properly.

In motor apraxia the motor memories for the extremities are preserved, but they are insufficiently connected with the other cortical fields. The ideatory process and the motor memories are separated. In ideatory apraxia the motor memories are intact, but the ideatory scheme is at fault.

Resorting to explanation by formulas, Liepmann (19) employs Wernicke's scheme.

According to Wernicke, the sensory centers (S) perceive sensory impressions. SA represents the psychosensorial pathway, so that at A there develop end memories (Ausgangvorstellung), which are discharged at Z. AZ is the intrapsychic pathway. Z represents the end memories (Zielvorstellung). These excite the motorium through ZM. ZM is the psychomotor pathway.

Liepmann modifies this formula so that Z represents the chief end memories (Hauptzielvorstellung; Erfolgsvorstellung). From

Z there go a number of paths to z^1, z^2, z^3, z^4, and from each of these there goes a path to M, the motorium. He then substitutes for the M, J the innervation. M = the motorium which becomes excited to new activity through the action of J, the innervation.

The path Z to z must be reckoned as the intrapsychic region, so that the horizontal line G (see diagram A) would represent a division of the intrapsychic and psychomotor regions, not the vertical line G in diagram B.

Diagram A.

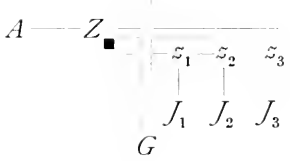

Diagram. B.

Each memory for a partial act (z^1, z^2, z^3, z^4, z^5) is called a "Zwischenzielvorstellung." From each z there goes out an innervation J which results in a movement or action. Failure of reaction in inattention, and the early disturbance of purposeful movements (Handlung) in brain diseases, show that the Z's give off false z's, but the proper connection between the z's and the J's is maintained. The J's correspond constantly to the z's, and this is the condition that Liepmann believes is present in the Apraxia of Pick.

Summarizing the whole matter, Liepmann states that purposeful movements (Handlung) in a normal individual, are the result

$$\left(\begin{matrix} W_K^0 \\ K \\ | \\ J \\ | \\ B \end{matrix} \right).$$

Diagram C. Memory Complex.

of a memory complex made up of partial memories (see diagram C); W, the direction memory (Richtungsvorstellung); o, optic

memories; K, general kinesthetic memories; k, limb kinesthetic memories; J, the innervation; B, the external movement. Purposeful movements depend upon a number of these complexes which replace the z's in the formula.

The same disturbance can be referred to the lapse of these complexes as to the lapse of the memories themselves. The subjective result would be that instead of a proper $W^0_{K_k}$ a false $W^0_{K_k}$ would appear, or in a false place. The objective results would be that a false J and B would appear, but always corresponding to the $W^0_{K_k}$. The innervation fails proportionately to the ideation, resulting in ideatory apraxia.

But, if there is a separation within each complex, so that everywhere the J's, or that J with the immediately adjoining k, do not any more correspond with the rest of the memory complex, then there results a disturbance of movement which is motor apraxia.[1]

In other words, Liepman believes that, in motor apraxia, there is a dissociation of the kinetic memories by reason of an organic focal lesion. The psychic element and its associations are preserved.

Kleist (15) modifies Liepmann's formula so that each direction memory contains a general kinesthetic element (K), as well as optic element (O), claiming that in Liepmann's formula the K is psychologically incorrect, being properly only a component of each direction memory, and should be written W^0_K. He believed that the partial memories are not a part of the chief end-memories, but a part of the complex of the total movement memories. His formula is as given on next page:

Lesions of Apraxia.—In Liepmann's (23) case, which is the foundation for the newer conception of apraxia, numerous connections between the left central convolutions and the cortex of the frontal region were severed by a subcortical focus in the frontal brain. A subcortical focus in the left parietal region implicated the connections from the occipital and temporal regions with the central convolutions, and destruction of the corpus callosum, as far as the splenium, separated the central convolu-

[1] This is an incomplete résumé of this phase of the subject, and reference should be made to Liepmann's "Ueber Storungen des Handelns, etc.," by any one wishing to study the subject more fully. Some of the matter relating to the elucidation of the subject by formula is translated freely from the original.

tions from the entire right hemisphere. In the right hemisphere there was also a focus where the gyrus supramarginalis joined

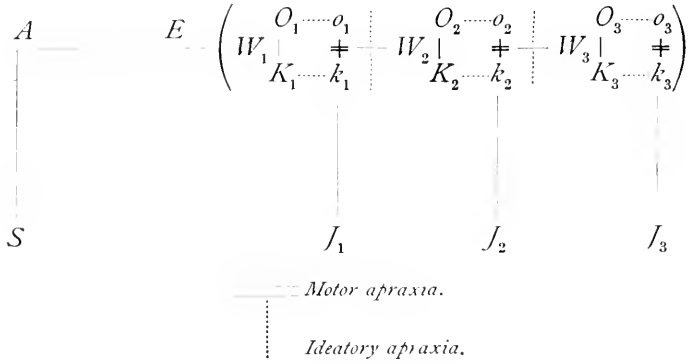

Diagram D. Kleist's modification of Liepmann's formula.

the gyrus supra-angularis in the right hemisphere; and a second focus that destroyed most of the fibers from the left face, arm and leg centers.

Hartman (12) reported three cases of apraxia, with post-mortem examination. In the first there was a tumor in the left frontal region, which spared Broca's region and the neighboring white matter, and extended to the anterior thalamic levels in the left corpus callosum. In the second case there was a tumor which destroyed the corpus callosum from the level of the anterior commissure to the posterior end, and which did not extend to any part of the brain. In the third case there was a hemorrhage in the second right frontal convolution the size of a walnut.

In Van Vleuten's (52) case of left-sided motor apraxia there was a cylindrical tumor involving the entire corpus callosum, the left gyrus fornicatus, and part of the frontal brain. The cortical white matter was not involved.

More recently a case was reported by Liepmann (27) and Mass, with left-sided agraphia and apraxia, in which there was a cyst that began in the left upper frontal region and in the gyrus fornicatus, and extended posteriorly in the latter to the paracentral region. The left half of the corpus callosum was entirely implicated in the left hemisphere. There was also a focus the size of a pea that interrupted the pyramidal tracts. In the

right cerebellar peduncle there was a focus the size of a lentil, and one in the pons outside of the lemniscus, and one in the right thalamus.

In Von Bechterew's (5) case there was a circumscribed lesion in the left hemisphere in the middle part of the posterior central region, and in the posterior part of the gyrus supramarginalis.

Strohmayer (46) found in his case of apraxia a lesion in the lower parietal region, where there was an extensive loss of substance which reached forward to the posterior central convolution. It extended below to the fissure of Sylvius, and above as high as the anterior parietal fissure. The vertical section at the posterior end of the splenium showed that the focus spared the white matter of the optic region, and that parts of the forceps major and the superior longitudinal fasciculus were involved. The striking similarity of Liepmann's case is commented upon.

In Pick's (42) case of senile dementia with apraxia there was atrophy of the brain accentuated in both frontal lobes, and the left inferior parietal lobe. It was less severe in the right inferior parietal lobe, and both temporo-occipital lobes. Nothing abnormal was observed in the central convolutions, the superior parietal lobe, the cuneus and precuneus. This case is believed to have been the result of disturbance of the association centers of Flechsig.

Westphal (54) recently reported a case of motor apraxia in which the autopsy showed the presence of internal hydrocephalus, and several foci of softening in the right frontal and occipital regions, and in the left optic thalamus.

Abraham (2) described a case of total aphasia, mind-deafness, apraxia, agraphia and alexia, with autopsy. The brain was atrophic, especially in the post-central regions. The occipital lobes were relatively preserved. The lateral ventricles were distended.

The usual lesion of paralytic dementia was present in D'Hollander's (14) case of aphasic apraxia, but no focal lesion was found. There was some hydrocephalus.

Von Monakow (34) has recently reported two cases of apraxia with autopsy. In one apraxia was a partial symptom in a case of progressive sensory and motor aphasia. A tumor was found in the corpus striatum compressing the entire region sup-

plied by art. foss. Sylvii. The cortical speech centers, central convolutions, and the corpus callosum were intact. He looked upon the case as an example of apraxia due to a subcortical lesion in the central ganglia.

In a second case the apraxia was a transient symptom, occurring in a case of sensory aphasia, and the autopsy revealed the presence of a lesion of the right central ganglia, and an infarct of the posterior third of the temporal lobe, implicating the gyrus supramarginalis.

A study of these facts shows that a variety of lesions have been described in cases exhibiting apraxic symptoms, and the lack of uniformity in the lesions described is striking.

What seems fairly well demonstrated in all this data is that the destruction of the corpus callosum plays a distinct part in the causation of left-sided apraxia, probably by cutting off from the right frontal brain the influence of the left frontal brain. The predominance of the left frontal brain over the right side has been demonstrated by Liepmann in cases of left-sided apraxia in right-sided hemiplegias, and dyspraxia of the left hand in right-sided apraxia and aphasia.

Manfred Fraenkel (8) described a case of mirror-writing and apraxia of the left hand with right hemiplegia, and came to the conclusion that the brain centers on the right side were, to a certain extent, under the control of the left side of the brain; that the left cerebral hemisphere directed not only speech, but "Handeln"; that the right cerebral hemisphere did not contain an independent center for movement memories; and, finally, that it was therefore closely related to the lower centers.

Rothman (45) has made similar observations in right-sided hemiplegia. He believed that the precedence of the left side of the brain for manipulations was demonstrated.

Liepmann and Mass, in analyzing their recent case, agreed pathologically with Hartman's second case, and Van Vleuten's case, *i. e.*, that the left hemisphere exercises a directing influence upon purposeful movements of the left hand; and that destruction of the corpus callosum causes a localizing symptom, *i. e.*, dyspraxia of the left hand. Liepmann and Mass claim that it amounts to the same thing whether the interruption of the corpus callosum separates the left hand from the memory centers of the left hemisphere, or, if the corpus callosum is a direct pathway

for impulses to the sensomotorium of the right brain. Whether the chief rôle is the separation of the left hand center (Liepmann) or of the left frontal brain (Hartman), Liepmann believes the differences in opinion disappear if, as is probable, the kinetic memories of the sensomotorium of the upper extremity are in the middle frontal convolution.

Van Vleuten's case showed an intense apraxia of the left, and relative eupraxia of the right, hand. In this case the corpus callosum was destroyed, excluding part of the splenium.

In Hartman's case there was grave apraxia in the left hand, and a suggestion of apraxia in the right hand. The corpus callosum in this case was also destroyed, including the splenium.

In Liepmann and Mass's case the corpus callosum was destroyed, excluding the splenium. These observers believed that this demonstrated that the preservation of the posterior fourth and fifth of the corpus callosum does not guarantee eupraxia of the left hand.

Liepmann and Mass do not doubt that the splenium plays a rôle in the conduction of impulses from the left to the right hemisphere. Van Vleuten's case showed this, and Liepmann and Mass's case also showed that the posterior portion of the corpus callosum is of less significance for eupraxia in the left hand than the middle portion.

Hartman believes, with Liepmann, that there is a center in the frontal lobes for the mechanism of motor cerebral activity, analogous to Broca's region in aphasic troubles, and that destruction of the left frontal lobe causes apraxia. The right frontal lobe needs the coöperation of the left brain for the outflow of purposeful movements. Defect in the right frontal region (right mid-frontal) causes partial conduction-apraxia of the left side, with preserved movement memories (Hartman's third case).

Felix Rose (44) has recently reported a case of transient bilateral apraxia of the muscles of the inferior distribution of the facial nerve, of movements of the tongue and the jaw, and of the left hand, in a patient suffering from Jacksonian crises of the face and right arm. He believed that there was a diffuse meningo-encephalitis (no autopsy) which caused these symptoms, and concluded that it must be deduced that while the right hemisphere exercises no influence on the limbs of the right side for unilateral movement, and certain bilateral manipulations, for cer-

tain bilateral habitual motor functions, particularly of the muscles of the jaw, tongue and face, the integrity of the two hemispheres and their association pathways is necessary.

In discussing the significance of the corpus callosum in relation to apraxia, von Monakow (34) stated that for the preservation of praxic movements the integrity of the corpus callosum was not necessary, and in support of this opinion cited a case of intense hydrocephalus, in which the corpus callosum was only rudimentary and the majority of the fibers of which were degenerated. The patient could not only use both hands alternatingly in housework, but could also write. There was no apraxia, in spite of the fact that the white matter of the left parietal lobe and of the left temporal lobe, was defective to a great extent.

Van Vleuten claims that his case supported the view held by Marie, that apraxia is due not only to a destruction of a movement idea, but to a separation of the corpus callosum connections, between the material basis of the movement idea in the left hemisphere and in the right hemisphere. The special teaching of his case was that dyspraxia could be caused by a lesion destroying simply corpus callosal fibers. In van Vleuten's case the right hand was not apraxic, and he believed, therefore, that the left-sided apraxic center has not any influence over the sensomotorium of the right side.

Von Bechterew (5) believed that apraxia was localized in the parietal lobe, and was due to a loss of movement memories.

Pick's view in his case, with autopsy, was that there was a disturbance of the association centers of Flechsig.

Liepmann's opinion is that apraxia results from a dissociation of the motor centers from many regions of the brain. And further, that the entire memory of complicated acts is not confined to certain places in the cortex, either in the central or parietal convolutions. Optic, tactile, kinesthetic, as well as acoustic, elements, he states, figure in the general memory, and a circumscribed focus could scarcely cause the loss of the entire movement memory. The movement memories are collected, he believes, in the left sensomotorium, and there associated with the other territories of both hemispheres.

Margulies (28) held, on the contrary, that apraxia could be localized, for, he states, if inability to accomplish simple acts is present, localization at once becomes evident. In disturbance of

complicated acts the physiologic point of view indicates a primary localization. Bonhoffer believed the apraxia in his case was an expression of a focal lesion.

Kleist, in his recent case, claimed that the trouble was in the innervation, and was a fault of the so-called sensomotor "Eigenleistungen," thus differing from Liepmann's case in which the latter was intact.

Von Monakow (33), in an early opinion, did not subscribe to the view that apraxia could be a permanent symptom of lesion of the parietal lobe, and he believed that it was related to cortical ataxia, as motor aphasia is related to disturbance of articulation. It is due, he thought, to a series of variable and general factors, such as is seen in aphasia, and is the result of "diachisis."

In a more recent opinion von Monakow (34) stated that, in the pathology of apraxia, the region supplied by the left art. foss. Sylvii is the locality of predilection. He believed that the left gyrus supramarginalis plays an important part in the genesis of apraxia, and that in unilateral apraxia of long duration, though not necessarily persistent, there is local or diffuse involvement of the P_2, T_1, and probably also of the corpus callosum.

Intellectual Element in Apraxia.—Marie (29) believes that apraxia is due to an intellectual deficit, and that it really is an aphasia of feeble intensity. He claims that the patient does not understand the orders given, nor has he the exact notion of the gesture corresponding to the order given.

While Liepmann (26) acknowledged that focal disease causes weakening of the intelligence, he looked upon this as a result of the lesion causing apraxia, and not the cause of the apraxia itself.

Van der Vloet (50) does not subscribe to Marie's opinion, and, as a result of the study of 14 cases, believes that dementia and apraxia are independent of each other.

In this connection the recent views held by Abraham (2), apropos of a case reported by him, are interesting. He believed that the psychic element plays an important part in apraxic phenomena, and that this component has not received the attention it deserves in the study of this subject. The symptoms which, in his case, he believed proved this were the following. His patient, in putting on his coat, dropped it accidentally. He then attempted to put the right hand into the sleeve, but could not. Finally, he made a fold in the coat, put his hand through it, and was satisfied.

In another instance he put his trousers on wrong side about, and when his attention was called to it he turned around, believing that this corrected the mistake. The last act represents a psychic deficiency. Abraham questions whether all cases of motor apraxia have not more or less psychical disturbances.

The significance of the psychical element in apraxia has also been emphasized by Margulies (28), who believed that psychical symptoms are manifested in ideatory apraxia, if an incomplete, partial motor apraxia or agnosia is present, apropos of Pick's cases, and of cases of amnesic aphasia in localized senile atrophy.

My own view is that there is always present either a disturbance of intelligence, or agnosia; for example, the apraxic believes he is performing an act properly, when in reality it is being performed in an apraxic manner. He does not recognize the incorrect result, even when he sees it. There is here more than a suggestion of a loss of perception connected with false interpretation of optic and movement impressions. Whether this is psychic, or a manifestation of agnosia, is a question. It is possibly both, and, as Liepmann claims, is the result of the lesion causing apraxia, rather than the cause. The fact that apraxic symptoms have been observed in cases of senile and paralytic dementia (Pick, d'Hollander (14), Abraham (3), Lewandowski (17), Soutzo and Marbe (47), and Marcuse (31)), and in post-epileptic conditions (Pick (40) and Oppenheim (38)) is suggestive in this relation.

Psychic, or Mind Paralysis (*Seelenlähmung*).—The behavior of the left hand in my case resembles, in some respects, the "Seelenlähmung" described by Nothnagel and Bruns. My patient could not move the left arm in voluntary acts demanded of him, and it was held in the same position all day long, with the exception of occasional movements of a voluntary reflex character. For example, he could perfectly well put his left hand in his hip-pocket, take out his handkerchief, wipe his nose, and return the handkerchief to his pocket, and did so on several occasions.

In Bruns's (6) case of sensory aphasia, alexia, agraphia, slight paraphasia, and right hemianopsia, with lowered touch, pain, temperature and position senses on the right side, the right arm was incapable of spontaneous involuntary movements, but could be used in pure reflex or unconscious voluntary movements. Moreover, by practice other movements were possible.

It made no difference if these movements were controlled by the eye, ear or hand. My case differs from this in that the arm was not helped by practice, or assistance from the other hand, or the hand of the examiner. Bruns believed that in his case the symptoms were due to softening of the left parietal and temporal regions, as well as the gyrus angularis, and the posterior part of the internal capsule. The rest of the hemisphere, especially the central convolutions, was intact. He stated that disturbance of the sensory centers, and the subcortical fibers whereby the destruction of the fibers from these to special motor centers is occasioned causes an incapability to use the limb in voluntary movements. If, by reason of the breaking of these fibers, the psychical center has no longer an influence over movements, there exists a mind-paralysis for voluntary movements, although these may be unhindered reflexly. Therefore, it is the result of an unilateral destruction of the sensory centers and their association fibers.

Somewhat similar cases have been observed by Anton (1) and Bleuler (7).

Bruns concludes that this condition is a disturbance that arises alone from the falling out of the memories for movement.

Nothnagel, who was the first to use the term psychic paralysis (Seelenlähmung), believed it was due to a separation of the cortical fields for motor memories in the parietal lobe from the motor centers.

Liepmann subscribed to the view held by Monk and Nothnagel. Monk defined "Seelenlähmung" as the complete loss of sensory memories of a portion of the body, which, as Liepmann states, corresponds to the more recent view of the loss of kinesthetic memories, and to Meynert's motor asymboly, Nothnagel's "Seelenlähmung," Heilbronner's cortical apraxia, and Liepmann's loss of limb kinetic memories (Gliedkinetischenvorstellung), and therefore not a manifestation of apraxia. In Liepmann's case of "Seelenlähmung" the limbs could only be used skillfully in speaking and gestures.

Perseveration.—When, after some effort, my patient succeeded in grasping the hand of the examiner, he continued to grasp it with increasing pressure, which would persist as long as the hand of the examiner remained within his grasp. This symptom, without doubt, was an example of tonic perseveration.

Perseveration has been classified by Liepmann (19) as tonic, when contractions of the muscles persist; clonic, when there is an alternation of contraction and relaxation, without apparent cause; and intentional, when an act becomes repeated instead of the intended new one. Cases of this character have been reported by Veschidi (49), Vurpas, Kleist and Liepmann, and have been met with in verbal deafness, mind-blindness and cases of agnosia. According to Liepmann the rare cases of tonic perseveration are due to disturbance of the motorium itself. This opinion seems to be somewhat confirmed by the pathological findings in my case, in which the right ascending parietal convolution was in part degenerated; that is, providing we believe that the motor centers are not entirely confined to the precentral region.

Tactile Paralysis (*Tastlähmung*).—The failure to recognize the nature of objects by touch is difficult to explain in my case. The impairment of sensation on the left side, outside of the loss of the sense of position, and movement sense, sufficiently explains its presence on this side, but on the right side all forms of sensation were intact, except the sense of position, movement and localization.

There was no ataxia, in spite of the presence of sensory symptoms which usually cause this phenomenon. In the absence of lesions causing tactile paralysis, it is difficult to explain its presence unless we attribute some significance to the optic memories, in other words, it may be the result of a separation of the sensomotorium from the optic centers.

In Nicolauer's (35) case of apraxia and tactile paralysis (Tastlähmung) in the same limb, the question arose whether both symptoms were not due to a focal trouble in the right hemisphere, which involved the central convolutions, causing disturbance of touch and other sensations on the one hand, and which interfered with motor acts by injury to the conduction of the nerve impulses required for their proper performance, on the other hand.

The pathologic cause of the apraxia in my case must remain problematical, unless apraxia may be caused by lesions of the occipital and temporal lobes in which, perhaps, are lodged, in part at least, the memories for purposeful movements.

It occurs to me that perhaps the memories for purposeful movement are not centered in any one locality, though there may

be a region where all the sensory impressions are congregated, but this is only a meeting-place, so to speak, for these impressions which have their cortical localization in the primary centers.

If I am correct in my opinion that my patient was not mind-blind, but actually cortically blind, the lesions of the occipital lobe, confined to the convexity and leaving the medial areas intact (including the calcarine region), do not coincide with the present-day view of the central localization of vision.

If it is true that vision is centered in the calcarine region, which was not degenerated in my case, why did he not see?

I am not prepared to make the statement that vision depends not only on the function of the calcarine cortex, but needs also the coöperation of the cortex of the entire occipital lobe, but I am willing to admit that this is a possibility which seems to be suggested by the postmortem findings in my case, for the inferior longitudinal bundle and the optic radiation were not entirely, or completely, degenerated.

In conclusion, it is worthy of note that in a number of the cases of apraxia with autopsy a certain degree of hydrocephalus has been found. What significance this may have is conjectural, and I shall at present go no further than to call attention to the possibility that the association of hydrocephalus and apraxia may be more than coincidental.

It is with much pleasure that I gratefully acknowledge the kindness of Professors H. Leipmann and A. Pick, and Dr. Felix Rose for so kindly sending me reprints of their papers on this subject.

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Society Proceedings

THE AMERICAN NEUROLOGICAL ASSOCIATION

The Thirty-fourth Annual Meeting held at the College of Physicians of Philadelphia, May 20, 21 and 22, 1908

The President, Dr. Charles W. Burr, in the Chair

(*Continued from p. 565.*)

Dr. Charles K. Mills and Dr. Charles H. Frazier, read a paper entitled: A Brain Tumor Localized and Completely Removed, with Some Discussion of the Symptomatology of Lesions Variously Distributed in the Parietal Lobe. (See this journal, p. 481.)

Discussion

Dr. B. Sachs said one remark was made that he would like to say a word about; Dr. Mills referred to this being the era in which exploratory operations were being done rather to excess. Granted that this is perfectly true, he wished to put on record some fortunate experiences in exploratory operations with reference to cure of optic neuritis. At the Mt. Sinai Hospital there have been four distinct cases in which they could not locate the tumor because there were no localizing symptoms, but as the result of exploratory operation in these cases the optic neuritis has improved very much, and the sight has been preserved where vision was threatened; therefore Dr. Sachs would like to say a word in favor of exploratory operations, particularly in cases in which the site of the tumor cannot be accurately recognized.

Dr. Beevor congratulated Drs. Mills and Frazier on their splendid results. He said the case interested him particularly owing to the fact that he had had one like it, and Sir Victor Horsley had operated. The man had similar symptoms, but in addition had fits. Dr. Beevor's patient also had marked hemianopsia. The difference was that in Dr. Mills and Frazier's case the hemianopsia disappeared and in Dr. Beevor's patient it did not. It is a question of the depth to which the tumor extends, if the tumor extends deeply and destroys the optic radiations improvement of hemianopsia does not occur. He thinks hemianopsia is really due to pressure on the optic radiations. In Drs. Mills and Frazier's case it was a cyst which caused pressure.

Dr. Putnam said he could not forbear to place on record the fact that all patients with tumor who are operated on do not die. He had a patient from whom a cerebellar tumor was removed, an endothelioma, and she has continued to do well, so that at the end of four or five years she is in a very good condition. She was almost blind and the eyesight has slowly and steadily improved.

Also, in relation to the decompression operations, he has had excellent results in one case of hemianopsia, obviously a basal tumor involving the

fifth nerve on both sides. It seemed impracticable to remove the growth. The decompression operation has given excellent results, except in regard to hemianopsia which was obviously due to serious injury of the optic tract. The Wernicke reaction was present in a very marked degree.

Dr. M. Allen Starr said he would like, also, to say a word in favor of decompressive operations. He has had two or three remarkable experiences of this kind in the last six months, and while he is not prepared to give the details he would say that in these cases there have been rather large swelling and hernia without external opening, the scalp remaining intact or healing up sufficiently to give perfectly firm appearance on the surface. The operation has had a remarkable effect in the improvement of the patients. One young man of 22 who was practically helpless and in an almost moribund condition, and hemiplegic before the operation, four months after the operation was able to walk four or five miles and to use his hands well and was intelligent. His eyesight had been obliterated by the atrophy following neuritis before the operation was undertaken. The operation was done by Dr. McCosh when the condition was apparently hopeless. We, of course, meet with cases where the results are immense hernias and death, but if we can save even one in ten it seems to Dr. Starr that the operation should be undertaken.

In regard to Dr. Frazier's remark that he preferred operation in one stage, Dr. Starr agreed thoroughly with him. He believes every possible means of hastening the operation is to be sought for by the surgeon so that every possible means of shock is to be avoided. He thinks statistics of only 6 per cent. of tumors being operable surprising; from his own statistics he would think 10 per cent. operable; those of Chipault, who is even more optimistic, show that 12 per cent. are operable.

Dr. Theodore Diller said he would like to add his testimony to those who had spoken in favor of the decompressive operation. He had seen it bring great relief on several occasions. Only very recently, about six weeks ago, he saw a patient one afternoon who presented the usual signs of brain tumor. He had severe optic neuritis, the swelling of the disc being 7 diopters. An operation was done very promptly the next day; and 10 days later the patient was completely relieved of his headaches; and his eyesight which had been 5/200 at the time of the operation was very greatly improved, so that he was then able to read an ordinary typewritten page, and his mental condition had also greatly improved. A very interesting point about this case was the fact that although there was the great improvement in the vision, ten days after the operation examination of the eye-ground showed no reduction whatever in the swelling of the optic nerve; although subsequently (three weeks later) it did subside to about 4½ or 5 diopters. How this great improvement in vision could occur without a reduction in the swelling is a thing Dr. Diller was unable to explain.

Dr. Mills, in closing, said he thought he had been slightly misunderstood as indicated in what had been said about exploratory operations. However, the misunderstanding led to the introduction of some discussion on decompressive operations. He was in favor of decompressive operations which are not necessarily exploratory. At the University of Pennsylvania the surgeons and neurologists have been very active in this work of cerebral decompression. What he intended to say was that the case was important as being one of cure, whereas so many of the cases were simply palliative and in not a few instances simply exploratory. The whole question of the effect of decompression would be taken up at Chicago by Dr.

Spiller and others at a combined meeting of the Neurological and Ophthalmological Sections of the American Medical Association, where Philadelphia and especially the University of Pennsylvania, experience would be given.

Dr. Charles H. Frazier of Philadelphia said, as to the operability of tumors, he had quoted the statistics of Chipault, not because he believed them to be accurate, but because he thought they represented the views prevailing among medical men. In his own experience he has seen nine operable tumors, three in the subtentorial region and six in the pretentorial region. This represents between fifteen and sixteen per cent. of tumor cases which have been under his care. With reference to the subject of decompressive operations, upon which the discussion has turned, he thought there was a great tendency to be content with this operation in cases of doubtful localization, without attempting to search for the tumor. The decompressive operation is so much easier of execution, requires so much less knowledge of the finer points in the technic of cranial surgery, and entails so much less responsibility on the part of the surgeon that there is a danger of this being resorted to in many instances in which an exploration should have preceded it. If surgeons would explore for tumor more frequently, even when the evidence is not very positive, they would discover more of them in the early stage, and thereby increase the percentage of operable tumors. Failing to find the tumor, one can always resort to a decompression. It matters little what part of the brain is explored, the osteoplastic flap may be replaced and a decompression effected in the temporal region. As the base of one's flap, in the great majority of cases is in the temporal region, one can remove from this region enough of the skull cap for decompressive purposes without the necessity of making another incision. This proceedure he has carried out in a number of instances.

Dr. Patrick inquired how Dr. Frazier explored when the brain is exposed and the tumor is not in sight.

Dr. Frazier, replying to Dr. Patrick's query, stated that the simple decompressive operation implied a relief of intracranial tension without exploration and without relation to the situation of the growth. He would urge neurologists to advise and insist upon exploration more frequently than they do, falling back upon decompression only as a last resort.

Dr. Theodore Diller and Dr. George J. Wright read a paper entitled: A Study of Hysterical Insanity with an Especial Consideration of Ganser's Symptom-Complex. Report of Eleven Cases. (To be published in this journal.)

Discussion

Dr. Charles L. Dana, New York City, said he would like to ask Dr. Diller in his concluding remarks to give a definition of hysterical insanity; to tell what it is. Dr. Dana said he had read a good many articles on this subject but each one who writes seems to have a different conception of hysteria and the total result made an interesting puzzle. Dr. Dana thought it might be better to drop the name hysteria altogether from our nosology. He did not think we would ever get a definite and clear-cut psychosis as long as we try to drag an uncertain element into the descriptive phenomena. He did not say this in any criticism of Dr. Diller's paper, which he thought very timely, and one which he hoped would lead to discussion which will settle the matter one way or another.

Dr. Smith Ely Jelliffe said he thought all of the members who had

any experience not only with the normal mind but with the abnormal mind, generally speaking, have come into relation with this particular form of reaction, *i. e.,* the Ganser syndrome, and he said he was in accord with Dr. Diller when he held that the Ganser symptom-complex must not by any manner of means be considered as pathognomonic of any mental disorder, especially not of hysteria. Ganser's symptom-complex is an interesting reaction, found in a number of different conditions: its presence always signified something, and offers a great clue to the type of mind under observation. In medico-legal work, especially in the detection of malingering, the Ganser symptom is of much importance, but certain modifications which have been well defined by Ganser himself, as well as by his followers, as pathognomonic of certain states which do not hold must be made. There are many so-called alienists who hold that the type of reaction which has been so well studied by Ganser and named after him is a form of nonsense, and is an evidence of malingering. Such, in their ignorance, claim it has no significance whatever, whereas it seems to him from a fairly extensive experience in medico-legal work, and in observations made in reformatories, asylums and jails, that the relationship of the Ganser reaction to certain psychological traits was remarkably illuminating on the subject of personality.

Dr. Alfred Gordon thought that when we use the term hysterical insanity we must imply that insanity is caused by hysteria. When we examine closely such cases, when we follow a sufficiently long time the mental manifestation of hysteria, we cannot accept the term psychosis, that is the psychosis with which we are familiar, the typical psychoses, such as mania, paranoia, melancholia. The question of Ganser's symptom as being pathognomonic of hysteria cannot be held. He has examined a large number of cases of all forms of insanity in which he has found the Ganser symptom. The so-called delusions of hysteria are not true delusions, they are only disconnected dreamy beliefs, which change from day to day from one examination to another, and these cases, even in the pronounced forms of hysteria, could not be accepted by his observations, at least, as cases of genuine insanity.

Dr. Henry R. Stedman said that in considering the predominant manifestations of dementia præcox and the fact that hysterical manifestations are so exceedingly common in the hebephrenic form, it struck him as going rather far to consider these comparatively minor symptoms, the Ganser syndrome, as a sure diagnostic sign of an hysterical psychosis, and he agreed with Dr. Gordon that if there is such a thing as a hysterical psychosis it must be based on physical stigmata. Dr. Stedman has met very few cases of hysterical insanity of that type, mental disease combined with pronounced physical stigmata of hysteria, and he thought that the term hysterical insanity was disappearing from our nomenclature.

Dr. Joseph Fraenkel spoke of the vague and indefinite character that the word and concept "hysteria" still has to most of us.

Therefore it is very unsatisfactory to discuss such questions *en masse* when we do not all agree upon the condition we are speaking about.

So long as we have not adopted a uniform clinical or other distinguishing feature for the condition we are speaking about, such discussion is not very illuminating.

Dr. Adolf Meyer said the problem would he thought get on dangerous ground if we tried to make our criterion of hysterical insanity and hysteria on the traditional lines, especially if we made the diagnosis depend chiefly on physical stigmata, whatever that means. As far as his attitude is con-

cerned, hysteria, as we understand it at the present time is a psychopathological entity (in contrast to the *neurological* entities in the proper sense of the word as well-defined tissue diseases). Dr. Meyer thinks by all means we ought to do what Dr. Dana and Dr. Fraenkel recommend. We also ought to be sure not to enter on a discussion on hysteria without having some definite case or facts to discuss concerning which a definite chain of evolution can be claimed or demonstrated. Then we can add the discussion as to what the Ganser symptom-complex may mean or this or that stigma. The main thing is in the first place that we should have a clear idea of a development of a definite type of reaction. When, then, we can agree on whether we consider that as a specific well-circumscribed something that we all interpret in the same way—a series of events of definite origin, evolution and termination—we can then begin to say whether the Ganser symptom-complex or any other symptom-complex plays a specific or pathognomonic role in it. As soon as we start on that ground we are bound to make progress, no matter how glaringly different our theoretical views may be.

Dr. Diller, in closing, said he was not able to define what hysteria is. He is not able to define what insanity is, unfortunately. He realized that this problem was a difficult one. He had certain ideas in his own mind as to what constituted hysteria and as to what constituted insanity. These things are very hard to express; and he supposed every gentleman present had the same feeling; and with this preface he would say that hysterical insanity is hysteria which presents such profound, prolonged, exaggerated mental symptoms as to constitute the patient an insane person. Some gentlemen had had a little misunderstanding of the authors' views of the Ganser symptom-complex. Dr. Diller said that of 11 clinical cases they found it in typical form in only one. They had been careful to state that they did not regard it as symptomatic of an hysterical state or condition. In the majority of the cases of hysterical insanity it is absent. As in other diseases they were more sure of their diagnosis when they had a considerable number of lines converging and pointing to it,—hysterical conduct, the various analgesias, contracted visual fields, and so on, and so on. They did not wish to give the impression that the diagnosis was an easy one, nor did they wish to appear as special pleaders for it. They had freely admitted the difficulties of diagnosis in reporting their series of eleven cases by relating four or five doubtful ones. They had seen the Ganser symptom-complex in cases which they did not diagnose as hysterical.

Another point emphasized by Dr. Diller he regarded as of considerable value (but like all other points it has its value only in connection with other points), that is the fact that we have a psychosis which appears to evolute from an hysterical basis and one which is different in a great many ways from any other psychosis with which we deal—a negative evidence.

The recognition of a psychosis as hysterical in character is a matter of practical importance since the prognosis and treatment will both be greatly influenced by it.

(*To be continued.*)

NEW YORK NEUROLOGICAL SOCIETY

May 5, 1908

The President, Dr. B. Sachs, in the Chair

A CASE OF MYOCLONIA OF THE TRUNK MUSCLES IMPROVED BY PSYCHOPHYSICAL THERAPEUTICS

By Dr. J. Ramsay Hunt.

The patient was a boy, 15 years old, who commenced to show clonic muscular contractions of the trunk muscles in January, 1903. At that time he showed a tendency to stoop and bend forwards; this gradually became more marked, and finally his body became fixed a large part of the time in this bent position while sitting, standing or walking. Various methods of treatment were tried without benefit, and the patient was finally subjected to a long-continued course of psychophysical therapeutics by Dr. R. B. Kruna, with decided improvement, as he was now able to get about in a fairly comfortable manner. It was because of this very considerable improvement and the method of treatment employed that Dr. Hunt brought this case before the society. He regarded it as an unusual type of myoclonia, a form of tic convulsif involving the muscles of the trunk. There were no evidences of any organic diseases of the nervous system, and at no time were there any stigmata or crises suggesting an hysterical origin. Mentally the boy was bright and intelligent and there were no impulsions, obsessions or phobias, such as not infrequently accompany the generalized forms of tic convulsif in early life.

Dr. Richard B. Kruna in demonstrating his method of treatment in the case shown by Dr. Hunt, said that when the boy first came under his observation the body was bent forward to an angle of about 80 degrees to the legs; at the same time the spine showed lateral flexion and rotation, as well as the neck. The tic spasms from which he suffered had developed during six months, and had gradually increased in force. The boy had no pain, but in walking the spasms pulled his trunk and head downwards until he was practically doubled up, with his head near his knees. In this position he would brace himself, with one hand upon his knee and the other upon some object near him, such as a table, chair, etc., or upon the ground. With these disadvantages, his gait was of course irregular and jerky, yet surprisingly fast ("monkey gait"). A careful examination of the patient showed nothing abnormal organically, excepting an increase of indican in the urine and ankle-clonus of the left foot.

To find relief from his distressing condition, which had forced him out of school and rendered him an object of ridicule and even fright, he spent over four years at various orthopedic and general hospitals. Every resource of orthopedic surgery was tried, including a variety of plaster-of-Paris jackets, steel braces, extension under anesthesia, and finally an operation, consisting in division of the rectus and ileo-psoas muscles. All these were without effect, as was also an injection of magnesium sulphate solution into the spinal canal. The latter caused temporary paralysis, but did not cure him.

The patient was referred to Dr. Kruna at Dr. J. Fraenkel's suggestion over two years ago for psychophysical treatment: this had been

faithfully carried out, and the patient was now able to stand and walk erect, and in the course of another year, the speaker said, his condition would probably be entirely normal. At present he was able, by voluntary muscle action, to antagonize every tic contraction, but this balancing activity had not yet been developed to the point of being automatic, as it normally should be. The speaker then explained briefly the psychical regime which was part of the psychophysical therapeutics, which included the development of the patient's intellectual and moral strength, the training of his powers of initiative and inhibition, regularity and system in habits, etc.

The physical training consisted in medical gymnastics, starting from an analysis of the tic-attitude into its components, the elementary muscle-spasms. Each of these tic-elements had to receive a remedying antagonistic muscle-exercise. At the outset, the patient was not able to make the antagonizer of a tic-muscle contract by itself. This required a series of assisted exercises. In these, the patient was helped by an assisting force, outside of his own muscular effort, to overcome the tic-spasm. The percentage of efficiency of the patient's own efforts would show increase according to their repetition, and the assisting force was correspondingly diminished. Finally, the assisted exercises became unnecessary and were replaced by unassisted ones. The first land mark of progress was the ability to overcome every tic element by its antagonistic action, and every tic attitude by its counter attitude; the second, identical with re-establishment of the normal condition, would be reached when these voluntary antagonistic actions, by untiring exercise, had become automatic.

Dr. William M. Leszynsky, who had presented the same patient at a meeting of this society several years ago, said that Dr. Kruna was certainly to be congratulated upon the brilliant result he had obtained from his psychophysical method of treatment. When Dr. Leszynsky had him under his care there was some improvement after a similar method of treatment in the hospital, but within five months he relapsed into his original condition. He was then sent to the Hospital for the Ruptured and Crippled, where he was fitted with various kinds of apparatus, and several muscles tenotomized without relief. He was finally transferred to the Montefiore Home. The diagnosis of the case at that time was hysteria, and his physical condition was associated with a peculiar mental attitude which seemed to point to some form of degeneracy.

TWO CASES OF CEREBRAL APOPLEXY, WITH UNUSUAL DISTRIBUTION OF THE SYMPTOMS

By Dr. I. Abrahamson

The first patient was a man, 64 years old, a native of Russia, who was admitted to Mt. Sinai Hospital on April 6, 1908. He had long been addicted to the use of alcohol and cigarettes, both to excess. For three or four weeks prior to admission he had complained of transient attacks of paresthesia of the left hand and wrist, with numbness and weakness. These attacks, which occurred about once a week, were of a few minutes duration, and upon one occasion were associated with mental confusion.

Without further warning, he had a rather sudden attack of paralysis involving the left face and arm; this was transient in character, lasting

only a few minutes, and following this, he had a number of similar attacks. In all of these the left face was involved and his speech was thick, but not lost; these persisted for 24 hours and the last left patient paralyzed. There was never a loss of consciousness; no sphincteric trouble nor vomiting. He had never had severe headaches. Examination showed a facio-brachial partial hemiplegia; leg not at all involved; cause, thrombosis of the second branch of the Sylvian artery; arteriosclerotic in origin.

The second patient shown by Dr. Abrahamson was a woman, 49 years old, a native of Scotland, who was admitted to the Mt. Sinai Hospital on April 19, 1908. The history relating to her present illness dated back three days prior to her admission, when her husband struck her with his fist at the nape of the neck. She fell from the force of the blow, but arose at once and walked to the bed, where she fell again, but succeeded in getting into bed. Shortly afterwards, the fingers of her left hand began to feel numb and she could not move the left arm. The paralysis gradually extended to the face and leg, and her speech became thick. Denies she suffered from unconsciousness. There was no history of vomiting; no loss of control of the sphincters. At the present time there was flattening of the left side of the face, and the tongue was deviated slightly to the left: motor weakness left upper extremity with exaggeration of the deep reflexes; only slight involvement of left lower extremity, etc., *i. e.*, a facio-brachial cerebral palsy. Here the cause was a hemorrhage, rather small, in the neighborhood of the internal capsule.

Both patients showed an unusual amount of implication of the upper facial musculature.

Incomplete hemiplegias are always of interest, especially traumatic hemorrhages into the brain substance.

The President, Dr. B. Sachs, who had both of these patients under his observation at Mt. Sinai Hospital, said they were interesting examples of facio-brachial paralysis, both occurring in the manner of an apoplexy. The woman developed this peculiar form of hemiplegia after receiving a blow behind the ear, and the disability was limited to the face and arm.

Dr. Hunt said he had seen several cases of this type, and had one under his observation at the present time, with almost complete monoplegia of the left arm, only slight weakness of the left face and no involvement of the leg at all. The onset in this case pointed to a cerebral lesion, in all probability in the neighborhood of the cortex. This patient had had a number of convulsive seizures of the Jacksonian type. There were no sensory disturbances and the lesion had not progressed.

During the recent epidemic of poliomyelitis, Dr. Hunt said he saw an old woman, very much emaciated, with a complete flaccid paralysis of the left upper extremity. There was a total absence of any signs pointing to a hemiplegia and he was inclined to regard it either as a spinal hemorrhage localized in the anterior horn, or as an unusual form of poliomyelitis. In the course of a few weeks the flaccidity gave way to a mild increase in the reflexes with slight rigidity. In this case there was no involvement of the face or leg, nor were there any sensory disturbances. She gave no history of headaches, although she had suffered from transient attacks of vertigo during the last year. The case was apparently one of cerebral monoplegia with no trace of involvement of the face or leg, and without irritative symptoms pointing to the cerebral cortex.

Dr. Smith Ely Jelliffe said he had seen a number of cases of facio-brachial paralysis—perhaps half a dozen during the past year. In most

of the cases the histories were similar to those given by Dr. Abrahamson, the onset being mild and the symptoms chiefly limited to the face and upper extremity, with slight impairment, in some instances, of the lower extremity. Through the courtesy of Dr. Janeway, the speaker said, he recently saw a case presenting this, with other features on the oposite side, at the City Hospital, in which the attack was extremely severe. The patient was comatose for a week, but the residual effects of the apoplectic seizure were very slight, being limited on the right side to a paralysis of the arm and lower face. There was no increase in the knee or ankle reflexes; no clonus; no Babinski on the right side. On the opposite side, however, there was a persistent chorea-like tremor, including the face, arm, and leg, with Babinski, clonus, and increased knee jerks, but no paralysis. The tremor was of the semi-intentional type, averaging 180 to the minute. There were marked sensory disturbances on the right side, especially to pain and to temperature. Deep sensibility was affected, but not light touch. Sensory disturbances were absent on the left side.

Dr. William Hirsch thought the main reason that cerebral monoplegia was regarded as unusual or rare was that we always had in mind that the most frequent hemorrhages occurred in the internal capsule, and were apt to overook the fact that those forms of paralysis due to arteriosclerosis, syphilis or thrombosis were more likely to give rise to a diplegia or a monoplegia rather than a complete hemiplegia. In a case of cerebral arteriosclerosis seen comparatively recently the only manifestations were an aphonia, with very slight paralysis of the face. In another case, the only symptom was alexia. A clear distinction should be drawn between these two classes of cases; in one, complete hemiplegia was the rule, while in the other, where we had to deal with vascular changes, a complete hemiplegia was the exception, rather than the rule.

Dr. Leszynsky said he had seen a number of cases of cerebral hemorrhage in which only the face and arm were involved, but, as a rule, upon investigation one usually learned that the entire side was affected at first and that the leg recovered within a few hours or days. Such cases were not at all uncommon in his experience.

Dr. Sachs said that in the internal capsule the fibers were so close together that it was almost inconceivable that a hemorrhage there would involve the arm fibers and leave those of the leg exempt. It would perhaps become necessary at some future date to change our present views regarding the arrangement of fibers in the internal capsule.

DISCUSSION ON THE PRESENT-DAY LIMITATION OF THE CONCEPTION OF PARANOIA

By Dr. M. S. Gregory

In opening this discussion, the speaker said that he would not attempt a complete historical résumé of the paranoia problem, interesting and instructive though it might be, and would consider only those later day conceptions which were still subjects of controversy. He would make no pretense of trying to solve the paranoia problem. It was well known that the origins of paranoid delusion formation were extremely complicated, and it was not his intention to attempt a complete psychological analysis, notwithstanding its great importance, and in spite of the fact that it was

probably only through such analyses that we might ultimately come to understand some of the more subtle distinctions in this field of psychiatry.

After summing up the position assumed by Kraepelin in connection with paranoia Dr. Gregory said we were reduced to the consideration of two groups of cases: the one a comparatively large assemblage of so-called secondary paranoias or paranoid forms of dementia præcox, showing the typical intellectual deterioration, the disorder of attention and blunting of the emotional tone, with more or less systematized delusional formation; and a still narrower and numerically smaller group, in which the delusional formation was unattended by the other attributes of intellectual impairment. Under both of these groups, certain sub-divisions stood out fairly clear. After mentioning these, and illustrating them with detailed clinical cases, the speaker summarized his views as follows:

1. That the grouping of paranoia from only a symptomatic point of view was unscientific and untenable.

2. That when measured by complete clinical criteria, the paranoia group was reduced to about ten per cent. of all insanities which seemed to have some fundamental characteristics in common.

3. That this restricted group was composed of two essentially different types namely, deteriorating and non-deteriorating or degenerative, which should not be confounded.

4. That these two general groupings, owing to our present limited knowledge might, only empirically and arbitrarily, be divided into other sub-groups: (*a*) For clinical and therapeutic convenience. (*b*) To bring about an understanding among the alienists in order to avoid confusion in medico-legal questions.

(*To be continued.*)

Periscope

Revue de Psychiatrie

(April, 1907)

1. The Biological Theory of Sleep. N. VASCHIDE.
2. Remissions in Dementia Præcox. MLLE. PASCAL.

1. *Theory of Sleep.*—The theories of sleep are very numerous and ingenious but serve only to encumber the literature because of the extreme paucity of facts on which they are based. The author proceeds in this article to a critical examination of the work of M. Claparède, "Esquisse d'une théorie biologique du sommeil."

Claparède in this work takes up the different theories of sleep—the circulatory, neuro-dynamic, biochemic and toxic—and rejects them all. The author concludes this subject by saying that it would seem to him that a great distinction should be made between artificial sleep produced by chemical agents, sleep due to the elaboration of organic matter in the body (coma, sleeping sickness), on the one hand, and normal sleep and hypnotic sleep on the other. For Claparède sleep is a *positive* function as opposed to the classical theories which regard it as *negative*. "They regard sleep not as a function but as a cessation of organic activity." This is not true, it is not a purely negative state, not a simple arrest of function but it is a positive function. Sleep is an act of the reflex order, an instinct which has as its aim this arrest of function. We do not sleep because we are intoxicated or exhausted; we sleep in order not to be. In other terms, sleep is a function of defense. It makes itself felt before real fatigue. Prevent the animal from sleeping and it becomes exhausted. The author takes issue with these views at some length. He cannot understand why sleep should be assimilated to the instincts nor how this can explain the phenomenon of awaking. He leans towards psychological explanations and believes that there is psychological activity in the mechanism of sleep more important to consider than biological doctrines. M. Claparède considers that the mechanism of sleep consists in a lack of interest in the present. The author does not see how this supports the biologic theory. If sleep is an instinct such as M. Claparède thinks it, it should be possible to find a sleep center in the brain. The last chapter treats of the relations of sleep and hysteria. By assimilating the reaction of lack of interest with the reaction of defense of the hysterical he tries to bring together the functional psychologic theory of Breuer, Freud and Janet and the physiological theory defended principally by M. Sollier.

2. *Remissions in Dementia Præcox.*—The study of the remissions of dementia præcox is intimately related to its evolution and to the question of prognosis. In relation to this question of prognosis it is interesting to note that its history recalls the history of the same question with reference to general paresis. Prognosis in the two diseases has excited the same debates, the same objections, the same criticisms. Kraepelin's cases are the best studied. He reports cures in 13 per cent. of catatonics and 8 per cent. of hebephrenics. His remarks on these cases, however, indicate that

a searching examination would disclose some mild degree of defect. Aschaffenburg in a study of forty-six cases comes to the conclusion that no case gets perfectly well in the sense of a restitutio ad integram, but that cures are in reality recoveries with defect. The cases will always show some traces of disease; blunting of the emotions, indifference toward the members of the family; little interest in the events of the outside world; absence of initiative, etc. Such symptoms occurring in persons of limited intelligence are necessarily obscure. Weygandt takes the same position. He calls attention to the fact that many cases which he considered cured have a relapse, and that these relapses may occur after many years—in one case he cites after a remission of eighteen years. Pfersdorff in a study of 150 cases found 16 who were able to work for a long time. Hecht cautions against optimism in the prognosis. Bleuler says the cases of cure are for the most part followed by relapses. Christian does not mention remissions but speaks of periods of calm. Meus believes the prognosis grave. Deny and Roy admit incomplete remissions. Masselon mentions remissions followed by relapses and remissions of long duration equivalent to cure.

The author gives the histories of several cases and concludes that there is no cure for dementia præcox in the rigorous scientific sense of the word. All pretended cures are accompanied by vestiges of dementia more or less obvious and are often followed by relapses. The remissions are of many kinds. One group is characterized by the disappearance of the episodic troubles (delirious states, hallucinations) but with the persistence of the phenomena of dementia. A second group is characterized by the arrest, attenuation and disappearance of the phenomena of dementia. These are of two orders: essential (apathy, aboulia, intellectual defect) and secondary (tics, impulsions, stereotypies, laughing, neologisms, verbigeration, disorders of conduct). It is only the secondary phenomena that are arrested, attenuated or disappear. As Kraepelin says: It is above all in the affective sphere that traces of dementia præcox are to be found. In a discussion of the symptoms of the disease the author says the dementia of dementia præcox can not be compared to other dementias, either paralytic or senile. The true long remissions are found in the phase corresponding to affective regression in which the essential elements of mental life are not destroyed. In the amnesic phase the destruction is much more serious and irrecoverable. So far as known remissions are spontaneous—without known cause. As to the frequency of remissions, all are agreed that the cases of simple dementia, hebephrenia and catatonia are most favorable while there is greater chance for remission early in the history of the case before grave lesions have occurred. The author believes in abortive forms of dementia præcox. She does not think that they should be denominated pseudo-dementia for they show by their evolution the same signs as franker cases. The same problem of diagnosis occurs in other diseases, as, for example, typhoid fever without fever, scarlet fever without eruption. Kraeplin, Bleuler, Aschaffenburg, Weygandt, Crocq, etc., have demonstrated that these benign forms suffer relapses. They should therefore be considered as forms of dementia præcox with incomplete and temporary remissions. All these cases show loss of power of adaptation and the predominance of psychic defect in the affective sphere. The author has designated the early symptoms in these slowly evolving cases as pseudo-neurasthenia. In many of these aborted cases the damage is mostly to the higher—the moral faculties. Hence we have a form of acquired moral insanity. Kahlbaum recognized this and

called it heboidophrenia. Under this name Hecker described certain attenuated forms of hebephrenia. Schüle called them professional idlers. Fink has described a mild form of hebephrenia characterized by defects in the moral sphere. Ilberg speaks of these brilliant persons who later become liars and vagabonds. Trömner insists on the ultimate vagabondage of these mild cases. Wernicke describes them under the name moral auto-psychoses of puberty. Ziehen recognizes them as the heboïdes of Kahlbaum. Cramer, Sommer and Kern recognize them in certain criminals and vagabonds. Wilmanns in a study of 127 vagabonds found sixty-six cases of dementia præcox.

The duration of the remissions is very variable, often lasting many years. The tendency is to recurrent attacks of excitement—the circular form of dementia præcox. This recurrent tendency has been especially described by Kraepelin. It often coincides with the monthly periods in women and also often with the conditions incident to pregnancy and parturition. He gives 9 per cent. of hebephrenics and 24 per cent. of catatonics as the result of pregnancy or rather coming on following confinement. The original center of the disease may cicatrize, as it were, but the disease is apt to be started afresh by accessions of auto-intoxication, as well of the menopause as of pregnancy. This intermittent evolution of the disease, especially when melancholic or maniacal symptoms occupy the foreground, makes it necessary to differentiate it from circular insanity. Kraepelin calls especial attention to this difficulty. The author adds that it is significant that these cases when over the attack attach little importance to it and are indifferent regarding it.

WHITE.

Deutsche Zeitschrift für Nervenheilkunde

(Vol. XXI. Nos. 1–2)

1. Contribution to the Physiology and Pathology of the Contralateral Conjoined Movements. CURSCHMANN.
2. The Clinical Manifestations of Paralysis of the Abdominal Muscles, Upon the Basis of a Case of Isolated Partial Paralysis After Anterior Acute Poliomyelitis. STRASBURGER.
3. Contribution to the Clinical Study and Operative Treatment of Tumors of the Spinal Cord. BREGMAN.
4. Metastatic Abscess of the Pons. BREGMAN.
5. The Sense of Vibration. HERZOG.
6. Contribution to the Question of the Route of Ascending Myelitis. SALLE.
7. Congenital Myatonia. ROSENBERG.
8. The Changes in the Skeleton as a result of the Early Contractures of Progressive Muscular Distrophy. DREYER.
9. Myopathic Muscular Hypertrophy. V. BECHTEREW.

1. *Contralateral Conjoined Movements.*—The author has carried out a series of examinations in a number of young individuals, in order to determine at what age symmetrical contralateral conjoined movements of the extremities cease, and in what group of muscles they are most pronounced. The experiments were performed first by having individuals carry out a number of simple voluntary movements, and second, by applying weights to the organs carrying out the voluntary movements, which acted as

reënforcement for the conjoined movements. These movements were present in practically all cases under 10 or 15 years. In younger children they appeared simultaneously with the first voluntary movement, and Curschmann therefore calls this phenomenon "the infantile type." The groups of muscles most constantly involved are the extensors and adductors of the thumb and fingers. Other muscles, if they manifest these movements at all, usually cease to do so after there is inhibition from fatigue or other cause. When the weight reënforcement was employed the conjoined movements were found in all cases up to 22 years, and in older persons there is reason to believe that there is at least some indication of the phenomena at all ages. Involuntary movements are more apt to occur in the right than in the left hand, excepting in left-handed persons. Occupations that involve the independent use of the two hands, as in piano-playing, have an inhibitory effect. Reflex conjoined movements occur in early infancy, particularly the plantar reflex. As soon, however, as the development of the pyramidal tract causes the appearance of the normal plantar flexion of the toe the conjoined movement ceases. Passive movements never produce conjoined movements at any age. In pathologic conditions Curschmann has noted these conjoined movements in cases of arthritis. In disturbances of the peripheral motor neurones they are frequently almost constant. In persons suffering from amputation the attempt to innervate the amputated extremity caused the conjoined movements in the other. If, however, the extremity had been amputated for many years the movements were not present. In twenty cases of infantile cerebral paralysis the contralateral conjoined movements were present seventeen times; five times as a result of either arm or leg; twelve times only when movements of the spastic paretic extremity were attempted. In five cases they were more intense when the paretic extremity was moved. In the three negative cases there were extreme contractures or flaccid paralysis. Certain interesting aspects of the reflex movements are also described. In tabes, the movements are absent. In chorea, they are present even in advanced age. In paralysis agitans, they are either diminished or absent. In two cases of myotonia they were present in an intense form even as a result of passive movements. In flaccid paralysis due to hysteria they were absent if either limb was moved.

2. *Paralysis of the Abdominal Muscles.*—The author reports the case of a boy of 14 who, after an attack resembling acute anterior poliomyelitis, had a flaccid paralysis of a portion of the muscles of the abdomen. The symptoms of this paralysis may be grouped under the functions of abdominal compression, which may be tested in a variety of ways, or the ability to regulate the relative positions of the pelvis and thorax. In Strasburger's case the abdominal compression by the muscles was fairly good. There was however, paralysis of the recti, and as a result, sinking of the lower portion of the pelvis, and inability to rise from the supine position without the aid of the hands. As a result Strasburger concludes that the transversalis was less affected than the rectus.

3. *Tumors of the Spinal Cord.*—A man of 44, for more than a year, had suffered attacks of pain in the left lumbar region. Later there were paralytic phenomena in the left and then in the right lower extremity. Progressive paralysis of the left leg of spastic character, slight paresis of the right leg, an area of anesthesia in the left lumbar region in the area of the distribution of the first to the third lumbar roots, dissociated sensation of the right lower leg, difficulty in urination, increase in the tendon reflexes of the legs, especially on the left side, and the Babinski reflex in

the left foot were present. The diagnosis rested between syphilis and tumor of the cord. The specific cure being without result an operation was performed, and a fibromyxoma removed from the region of the eighth, ninth and tenth dorsal vertebræ. The patient died of meningitis. The second case, a girl of 14, gradually developed spastic paralysis of both lower legs, with disturbances in urination, and pain in the side and legs. A diagnosis of a localized disease in the region of the dorsal cord was made. The condition not yielding to counter-irritation or other measures, operation was performed, and an irregular tumor found beneath the arch of the second dorsal vertebra, involving the spinal cord. No improvement followed, and the patient died three months later. The tumor proved to be a round-celled sarcoma arising from the pia, invading the spinal cord.

4. *Metastatic Abscess of the Pons.*—A man of 38 suddenly developed headache, vertigo, tinnitus, vomiting, paresthesia in the right half of the body and face, weakness of the extremities on the right side, difficulty in speech, deglutition and coughing, diplopia, and disturbed micturition. There was slight paresis of the right side of the face, slight difference in the pupils, and associated paralysis of the ocular movements toward either side, with preservation of the movements up and down. There was also paralysis of the soft palate, bulbar speech, some loss of hearing, especially on the left side, paresis of the extremities on the right, and disturbance of sensation. The patient rapidly grew worse, there was total blindness, convergent strabismus, and death five weeks after admission. The diagnosis was comparatively easy, especially in view of the disturbance in the movements of the eye, and the crossed paralysis. At the autopsy a large abscess of the pons varolii was found, secondary to suppuration in the right kidney.

5. *Sense of Vibration.*—The author gives a careful analysis of the literature of the sense of vibration. He reaches the conclusion that no case has been observed that proves that this a peculiar form of sensibility, but that many observations are on record which strongly indicate that it is due to the stimulation of the nerves of touch, and the sensory nerves of the deeper parts, and that it is not transmitted by the pain or temperature tracts.

6. *Route of Ascending Myelitis.*—The author has made a series of experiments on rabbits which had for their principal object the determination of the rôle played by the central canal in the distribution of toxic substances introduced into the spinal cord, particularly with reference to the ascension of the destructive action. He employed various antiseptic irritative substances such as turpentine, diphtheria toxin, and bacteria. These were introduced by means of a hypodermic syringe which was so inserted that as nearly as possible the material was injected close to the canal. He reports a number of illustrative experiments, the chief results of which were that, although the central canal often contained inflammatory exudate and detritus, the main channel of diffusion was by means of the perivascular lymph spaces. The changes could often be traced from the lumbar to the cervical cord. It was noted that diphtheria toxin caused very pronounced changes in the cells.

7. *Congenital Myatonia.*—In 1900 Oppenheim described a disease characterized by hypotonia of the muscles, extreme weakness of the muscles and loss of the tendon reflexes. Rosenberg collects all the cases of this disease hitherto reported, including one of his own, occurring in a boy 2½ years old. It is interesting that during pregnancy fetal movements

were barely felt. The disturbances are most pronounced in the legs. The electrical reactions of the muscles and nerves were greatly reduced or absent, but when present were of a normal lightning-like character. Treatment with the faradic current on alternate days produced considerable improvement. Rosenberg concludes after a study of the lesions found in Spiller's case, that the disease is primarily one of the muscles. He discusses the differential diagnosis of somewhat similar conditions.

8. *Changes in Skeleton from Early Contractures of Progressive Dystrophy.*—Dreyer reports two cases of progressive muscular dystrophy occurring in brothers. The first had an attack of fever at the age of two years and severe diphtheria at three. He was weak, could run only with difficulty, and fell easily. At the age of six contractures commenced in the right foot which ultimately assumed the equino varus position. At the age of sixteen an operation was performed for this with success, but at the same time the diagnosis of progressive muscular dystrophy was made. An X-ray showed a thinning of the diaphyses of both humeri. The second case, a brother nine years of age, at the age of 1½ years had had an attack of fever with vomiting and diarrhea. In childhood his body was weak, and progressive muscular distrophy was readily recognized. In this case the diaphyses of the femurs were usually thin. Dreyer discusses the theories which have been suggested to explain the occurrence of club-foot in progressive muscular dystrophy, particularly the theory of antagonistic muscular action, suggested by Hoffer. Regarding the atrophy of the bone, he agrees with the views of Schlippes.

9. *Myopathic Muscular Hypertrophy.*—The author reports three cases of myopathic muscular hypertrophy. The first, a man of 21, developed cramps in the legs at the age of 15. There was a marked hypertrophy of the tensor fascia lata muscle on both sides. Otherwise the muscles were normal, showing no myopathic changes. He had been exposed to cold, and apparently developed thrombosis of the veins in the left leg which was considerably larger than the right. Von Bechterew regards this enlargement as secondary to the phlebitis. There were also some disturbances of sensation.

J. Sailer (Philadelphia).

Book Reviews

PATHOLOGISCHE PHYSIOLOGIE, EIN LEHRBUCH FÜR STUDIERENDE UND ÄRZTE. Von Dr. Ludolf Krehl. Fünfte neu bearbeitete Auflage. F. C. W. Vogel, Leipzig; Paul B. Hoeber, New York. 15 Marks.

We have had occasion to call attention to the third and fourth editions of this excellent text-book. There is no work of its kind that so aptly puts the modern biological aspect of disease in so clear and concise a manner. The new fifth edition reflects the advances that various workers have made and keep it abreast of the requirements of modern research. While essentially a work for the internist, it is invaluable for the neurologist.

JELLIFFE.

THE EYE AND THE NERVOUS SYSTEM: THEIR DIAGNOSTIC RELATIONS. By various authors, edited by Wm. Campbell Posey, A.B., M.D., and William G. Spiller, M.D. Illustrated. Philadelphia and London, J. B. Lippencott & Company. Pp. 988. $6.00.

This work is not encyclopedic in character like the Neurologie des Auges of Willbrand and Saenger, and should not be compared with it. It covers a rather different and broader ground for it gives a great deal of attention to subjects which are neurological and to which the function of vision is incidentally but not always closely related. It will give, we think, to the general reader a great deal of sound neurology as well as all the opthalmology necessary to the neurologist.

The first three chapters cover the subjects of the anatomy, physiology and psychology of vision. Dr. Mills writes of the last topic and describes also the focal diseases of the visual cortex. Dr. Mills gives expression to his well-known views as to the subdivision of the visual cortex. He gives us centers for form, light, color, ocular movement, and in the visuo-psychic field for concrete memories of words, letters, numbers, persons, places, etc. The author admits that this is largely provisional, but he cites a good many cases bearing on these special localizations. He seems inclined to a belief in the existence of a crossed amblyopia from organic disease and his discussion of the topic is a candid if not convincing one. The presentation of the subject of the various types of visual aphasia is illustrated with many important cases.

Dr. Mills's positive views about concrete concept centers, with naming-centers, etc., have a finality about them which in the present state of the subject of aphasia is perhaps not justified; however, it is likely that for the most part the judgments will not be found wrong and we may only have for them a different psychological interpretation.

Dr. Wood writes a chapter on methods of examining the exterior of the eye which is clearly written and well-illustrated. His discussion of amblyopia is rather too brief, though it is referred to further on in the book. We miss any full description of sudden amaurosis.

The extra-ocular muscles are dealt with by Dr. Duane in a most elaborate and thorough manner.

All that really need be said about tumors of the brain in relation to the eye would be perhaps to discuss the effect of these growths upon the visual organs. Dr. Spiller's methods are too thorough for this, and we are glad it is so, for he has written a chapter which forms an excellent monograph on the whole subject. He has, however, been very brief in discussing tumors not connected with the visual system. The author lays especial emphasis on astereognosis as a symptom of parietal lobe tumors, but he thinks it possible this symptom may be caused to some extent by lesion of the post-central gyrus. He does not make out a very definite thalamic syndrome for thalamus tumors and it would seem as if in nearly all the cases with localizing symptoms, the cause was extra-thalamic irritation. His discussion of lesions of the mid-brain is well illustrated with figures and cases. A very careful analysis is made of the subject of the paralysis of associated movements, Dr. Spiller has gone over the situation and he has presented some definite conclusions as a result, assigning the permanent lateral palsies to lesions of the posterior longitudinal bundle and those of vertical movement to lesions in the neighborhood of the oculo-motor nucleus. We agree with Dr. Spiller that lesions of the corpora quadrigemina are often accompanied with drowsiness, and have suggested the possibility even of something like a sleep center in those regions in which quiescent lid-closing and upward rolling of the eyes occur. The symptom-complex of Benedict is described and it should be better known, as it is not so very rare. Cerebellar tumors are among those dealt with very briefly; the author makes the observation, which we can confirm, that in the nystagmus of those cases the movements are more active toward the side of the lesion.

The articles on bulbar and pseudo-bulbar palsy and on myasthenia gravis are carefully prepared presentations of these subjects. Dr. E. W. Taylor, the author, refers to lesion of the thymus and a lymphoid sarcomatous infiltration of the muscles as a possible cause of the disease. This view has received some confirmation and some opposition (Booth's case) since the article was written.

We have gone over about half the book in an analytical way. It seems unnecessary to pursue the method to the end. We have shown that the different authors have treated their subjects in general thoroughly and completely. The only criticism, if it is one, would be that the editors have tried to cover too much ground sometimes in discussing systematically subjects like degeneracy, reflexes, gaits, tic douloureux, etc. We have read with care Dr. Posey's chapters on the eye in its relation to neuroses and psychoses, and it seems to us that he goes rather too far in his views of the importance of eye-strain. The book is well-printed and indexed and copiously ilustrated. We have detected no typographical errors, though we look upon such things with a sympathetic eye for proofreaders are mortal and editors sometimes are tired, but in this case the Homeric Dr. Spiller never seems to nod; and he and Dr. Posey have furnished a volume which redounds to the credit of American medicine.

C. L. Dana.

Krankenhauswesen und Heilstättenbewegung im Lichte der Socialen Hygiene. Von Dr. med. Alfred Grotjahn, Leipzig. Verlag von F. C. W. Vogel.

In the middle of the nineteenth century Germany lagged far behind England, France, Scandanavia and Italy in all that pertains to hospital

construction and management; but the hospitals of Germany have rapidly risen in number and importance, and to-day they compare favorably with those of England or of any of the continental countries. What are the chief factors that have contributed to this development? Have the results justified the labor and expense of building up the costly and complex system of hospitals, sanatoria and asylums which exists in Germany to-day? What is the influence upon social hygiene of institutions for the care of the sick and of defectives of every class? These are questions which the author undertakes to answer. His purpose is to outline the present state of hospitals and asylums in Germany, to study the developmental tendencies exhibited by these institutions, and to ascertain how far these tendencies are deserving of encouragement and acceleration. He shows how specialization in medicine has furnished a strong stimulus to the multiplication of hospitals, and traces the effects of private philanthropic motives in this field of social activity; he proceeds next to analyze and classify so much of German legislation as it is proper to consider in this connection,—the workingmen's insurance laws above all. From 1888 to 1905 the number of persons treated in institutions maintained by insurance funds in Germany, increased from 13,758 to 56,420, and the cost of maintenance of these patients grew from 2,700,000 marks to more than 14 million marks. The greater part of this money was devoted to the treatment of patients suffering from incipient tuberculosis, and if the official records are to be believed, most of the patients who entered sanatoria under the auspices of the insurance funds were, if not actually cured, at least restored to industrial efficiency. Year by year the percentage of the population which comes under the care of hospitals and sanatoria increases, and social hygiene must be the gainer inasmuch as a very large proportion of those who are thus removed for a time from their normal social surroundings, are sufferers from communicable diseases. Dr. Grotjahn concedes that Germany is still behind England in its methods of caring for contagious cases, since the rule in Germany is to place such patients in separate wards or pavilions of general hospitals, while England provides isolation hospitals rather than isolation wards. The separation of hospital patients into social classes by the establishment of varying rates of payment for hospital accommodations more or less comfortable, is roundly condemned; the author regards this system as one which engenders class hatred and bitterness. The volume is rich in statistical material gathered from official and private sources, and it contains many suggestions of a practical nature bearing upon hospital economy; for example, the suggestion that the hospitals of a given district be banded together for the purpose of obtaining supplies in bulk at wholesale rates. Separate chapters are devoted to convalescent homes, to hospitals for nervous cases, to institutions for the treatment of venereal patients, to maternity hospitals, to asylums for the insane, the epileptic, the blind, and other groups of defectives

S. S. GOLDWATER (New York).

ELEMENTS DE MEDECINE MENTALE APPLIQUE'S À L'ÉTUDE DU DROIT. Docteur Legrain. Medicin en Chef des Asiles d'Alienes de la Seine. Preface de M. Garcon, Professeur de Droit Criminel à la Faculté de Droit del' Universite de Paris. Arthur Rousseau, Paris.

With the steadily advancing integration of social forces, and the hoped for supplanting of might by right, of force by law, no one question obtrudes itself with greater demands for a partial solution at least than that of the relation of mental disorder to human responsibility.

Forms of property have changed, the methods of the production of wealth have been modified; the uplift in the working classes makes them more keenly alive to their rights and force. Family relations are modifying and throughout all the avenues of mental operations new combinations are being sought which demand intelligent observation in order to determine their ethical and moral status. To this steady modification of social aims and performance the science and practice of law has endeavored to respond, and the knowledge of the psychiatrist, in his field equally active, becomes more and more important in these newer social integrations.

The work before us represents an intelligent attempt to correlate the fields of law and medicine wherein they have mutual interests, namely, in the sphere of mental responsibility. In the opening lessons the position that law occupies with reference to crime is discussed, a full criticism of the various periods being included, beginning with the period when theological dogmas reigned supreme in determining guilt or innocence; then coming through the idealistic period to the scientific period of Lombroso, his followers and opponents.

In his second and third lectures the Constitution of the Personality is discussed; heredity, the world of sensations and clinical proofs are the subheads, illuminated by a rare analytical and practical faculty. In chapters four, five and six, the evolution of the personality is taken up and in four further chapters the functions of personality gone into. Later chapters discuss dislocation of the personality under the influence of poisons, of mental disorders, hypnotism, etc., while the final chapter concerns itself with dispersion and regression of the personality, with full considerations of the duties of magistrates and physicians, and the subject of attenuated responsibility.

The whole mode of discussion must be read to be appreciated, but it seems to be the most rational and considerate discussion which has come before us.

JELLIFFE.

DIE PROGRESSIVE ALLGEMEINE PARALYSE. Zweite Auflage, Auf Grund der Darstellung von Weiland Professor Dr. R. v. Krafft-Ebing. Neubearbeitet von Professor H. Obersteiner. Alfred Hoelder. Wien und Leipzig, 1908. M. 5.20.

It is highly gratifying to see Professor Obersteiner's revision of Krafft-Ebing book "Die Progressive Allgemeine Paralyse." The entire book has been rewritten, new subject matter added, and its scope much widened. The book, which formerly consisted of 104 pages, is now increased to 194 pages. The whole subject of paresis is exhaustively treated, and is presented in the light of modern neuropsychic researches. The author considers paresis originating mainly from a leutic foundation in a predisposed individual. He puts his views in the following forcible language: "The contents of the cerebro-spinal fluid show the great significance of lues in the etiology of paresis; not only the nearly constant presence of lymphocytosis speaks for this view, but the recent investigations of Wasserman and Plaut make the presence of syphilitic antibodies very probable, although in a great number of these patients other evidences of leutic infection have been wanting."

Again the *rationale* of the modern method of treating paresis lies in the prophylaxis which can be effected by preventing syphilitic infection.

Paresis in the juvenile and senile periods, in women, and Lissauer's

form, is thoughtfully considered. He lays great stress on the cytological examination and extols its diagnostic value. "A negative finding by repeated punctures speaks, at all events, against paresis."

He recognizes eight forms of the disease: (1) Demented form; (2) manic form; (3) expansive form; (4) depressive form; (5) ascending (tabo-paresis) and descending form; (6) circular form; (7) atypical form; (8) acute galloping form.

The chapter on pathology and pathogenesis is brought up-to-date in every respect. Considering the immense wealth of material utilized in his treatise, the clear presentation of facts, and the exhaustive bibliography of the literature (549 references) one feels that such a volume becomes almost indispensable to every student of neurology and psychiatry. It is lamentable that the author did not take the trouble to index it, but this surely will be remedied in the next edition. It may be hoped that this important book will soon be translated into English, for, beyond doubt, there is no treatise in Anglo-Saxon which can replace that of Professor Obersteiner's.

M. J. KARPAS (Ward's Island, N. Y.).

BIER'S HYPEREMIC TREATMENT IN SURGERY, MEDICINE, AND THE SPECIALTIES: A Manual of its Practical Application. By Willy Meyer, M.D., and Professor Dr. Victor Schmieden. Philadelphia and London: W. B. Saunders Company.

Although Bier's treatment by "Stauungs-Hyperæmie" was first published by him in Germany, about fifteen years ago, it has not attracted general attention in this country until recently. Last year Dr. Schmieden read a paper at Washington on "The treatment of bone and joint tuberculosis by hyperæmia," before the National Association for the Study and Prevention of Tuberculosis. He found then a comparative lack of familiarity with Bier's methods among our surgeons, and determined upon the writing of the present manual, with Dr. Meyer (who has been their ardent supporter for some years), in order to disseminate the new teachings. The book contains brief yet fair and comprehensive descriptions of claimed advantages of the hyperæmic treatment, the methods of inducing hyperæmia, and its application in various surgical and medical diseases. It comprises experiences gathered at Bier's own clinic and experiences in this country. The aim of the treatment is to "increase the beneficient inflammatory hyperæmia resulting from the fight of the living body against invasion." The first principle underlying it is that "the blood must continue to circulate, there must never be a stasis of blood." Means formerly employed to subdue an inflammation are discarded. Some of our fondly cherished ideas respecting inflammation, to conform to the theory, must be abandoned. But the authors claim to prove their case and find wide usefulness and many advantages for the Bier methods. For instance, their enthusiasm has led them to employ and highly recommend the methods in the several specialties, even in psychiatry. The facts set forth, however, are rather meager, except in the treatment of surgical conditions. The book is attractively gotten up and the illustrations helpful. Without advocating the wide use of the principles laid down, we recommend the book to those who desire a fairly comprehensive knowledge of the subject.

ATWOOD (New York).

Notes and News

Dr. William P. Spratling, the builder and for nearly fourteen years the Medical Superintendent of the Craig Colony for Epileptics, has been elected to the Chair of Nervous Diseases and Physiology in the College of Physicians and Surgeons, Baltimore, and will move to that city in the fall.

The chair in question carries with it several other positions, including that of visiting neurologist to the City Hospital and to Bay View Asylum, the former having 300 beds for acute cases, the latter more than 2,000.

Psychiatric Extension Course in Munich.—This year the special course in psychiatry which was inaugurated by Professor Kraepelin will be given from the ninth to the twenty-eighth of November. The chief features of the course will be as follows: Psychiatry—(1) Kraepelin, Forensic Clinics, 24 hours; (2) Alzheimer, Normal and Pathological Histology of the Cortex, 20 hours; (3) Gudden, Brain Anatomy, 6 hours; (4) Specht, Clinical Experimental Psychology, 8 hours; (5) Specht, Criminal Psychology, 8 hours; (6) Kattwinkel, Neurological Clinic, 9 hours; (7) Weiler, Physico-Clinical Methods, 7 hours; (8) Plaut, Cyto- and Sero-Diagnosis, 6 hours; (9) Rüdin, Facts and Problems of Degeneration, 6 hours; (10) Isserlin, Psychotherapy and Psychodiagnosis, 6 hours; (11) Liepmann, Apraxia, Aphasia, etc., 10 hours. Visits to institutions, lectures and clinics from 9–12 mornings, 3–6 afternoons. Fee, $15. Address, D. A. Alzheimer, Rückertstrasse 1, Munich.

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Original Articles

OCCUPATION NEURITIS OF THE DEEP PALMAR BRANCH OF THE ULNAR NERVE

A Well Defined Clinical Type of Professional Palsy of the Hand[1]

By J. Ramsay Hunt, M.D.

of New York

Chief of clinic and instructor in nervous diseases in the cornell university medical school; neurologist to the city hospital, babies' hospital, and new york hospital for nervous diseases

Under the heading "Occupation Neuritis of the Deep Palmar Branch of the Ulnar Nerve," I shall describe a group of cases, which present the following clinical characteristics: An atrophic paralysis of all the intrinsic muscles of the hand innervated by the ulnar nerve; the electrical reactions of degeneration; no objective sensory disturbances in the ulnar nerve distribution. The absence of sensory symptoms and the sharp limitation of the paralysis, atrophy and electrical changes to the muscles of the hand supplied by the ulnar nerve, distinguish this type from all other occupation palsies of the hand hitherto described. The clinical importance of these cases is very much augmented by reason of the absence of objective sensory disturbances, and the consequent resemblance to the Aran-Duchenne type of muscular atrophies of spinal origin. A resemblance which may be still further accentuated by the not infrequent association of progressive muscular atrophy with occupations requiring exces-

[1] Read at the thirty-fourth annual meeting of the American Neurological Association, May 20, 21 and 22, 1908.

sive use of the hands. Before proceeding to a detailed description of the syndrome, I shall refer briefly to various types of occupation affections of the upper extremities, and more especially of the hand, which are already recognized by systematic writers.

1. A neuritic form, due to stretching or compression of the nerve trunks, or over-activity in a neuro-muscular distribution. The ulnar and median nerves are especially liable and one or both nerves may be involved in some part of their course. The presence of toxic substances in the body, such as alcohol, may act as predisposing factors, thus rendering the nerve elements more vulnerable (toxico-professional palsies). This type is characterized by the neural distribution of the atrophy, and by the presence of subjective and objective sensory disturbances; as cramps, pains, paresthesias, and anesthesias.

2. A pure myopathic form, due to myositis or to pressure atrophy of the muscles.

3. A combination of the neural and myopathic forms.

4. A spinal form may also be mentioned. This is an early localization of progressive muscular atrophy in the intrinsic muscles of the hand, and is characterized by a progressive tendency, fibrillary twitchings, and the absence of sensory disturbances.

5. A special type of occupation atrophy of the hand has been described by Gessler. In this form the terminals of the intermuscular nerves and their motor end-plates are thought to be involved; frequent muscular contractions without sufficient relaxation inducing an anemia of the parts with consequent nutritional disturbances in the motor nerve endings.

In Gessler's type, as in the one which I describe, there are no objective disturbances of sensation; but his cases according to his description are unlike mine, in that the atrophies and paralyses of the intrinsic muscles of the hand are not limited to the ulnar nerve distribution. The etiological factor in the group of cases which I desire to isolate is a compression neuritis of the deep volar branch of the ulnar nerve, as it passes between the tendinous origins of two muscles of the hypothenar eminence, the adductor and the flexor brevis minimi digiti (Fig. 1).

Anatomical considerations.—I would here briefly refer to the distribution and the relations of the ulnar nerve in the hand.

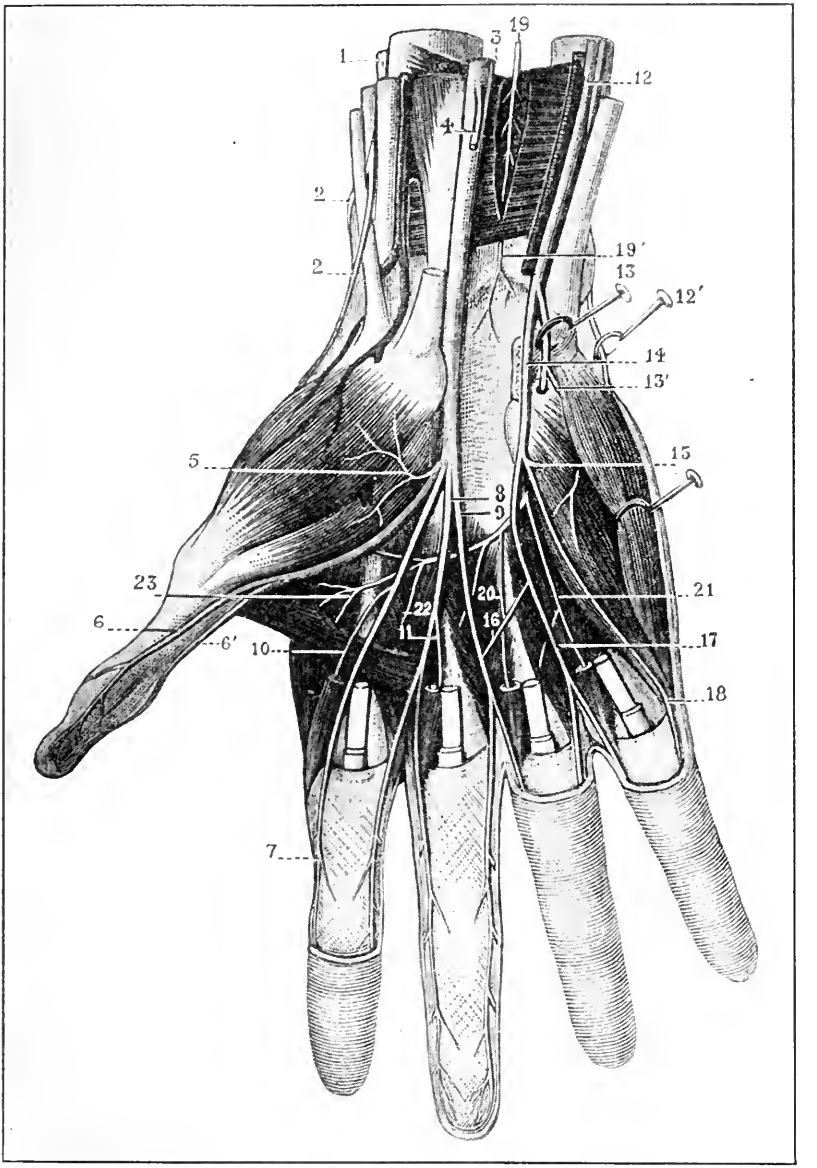

FIG. 1. (Testut's Anatomy.) Showing the superficial and deep palmar branches of the ulnar nerve. 12, ulnar nerve; 12′, dorsal cutaneous branch; 13, deep palmar branch; 13′, a branch to the hypothenar; 14, superficial palmar branch; 15, nerve of the palmaris brevis; 16, anastomosis with the median; 17 digital branch; 18, digital branch; 20, nerve to the third lumbricalis; 21, nerve to the fourth lumbricoids; 22, branch to the interossei; 23, branch to the adductor of the thumb.

The ulnar, at the level of the pisiform bone, divides into two terminal brances—the superficial and the deep palmar (see Fig. 1). The superficial palmar branch is sensory, supplying the skin of the palmar aspect of the little finger and the adjacent half of the ring finger. It also carries a small motor filament for the innervation of the palmaris brevis muscle. *The deep palmar branch is purely motor*, and innervates the following intrinsic muscles of the hand: those forming the hypothenar eminence—the abductor minimi digiti, the opponens minimi digiti, and the flexor brevis minimi digiti. It also supplies certain muscles of the thenar region, namely, the abductor pollicis and the inner head of the flexor brevis pollicis; as well as the palmar and dorsal inter-ossei, and the two inner lumbricales.

The other intrinsic muscles of the hand are supplied by the median nerve. These are the abductor pollicis, opponens pollicis, the outer head of the flexor brevis pollicis, and the first and second lumbricales.

In the upper forearm the ulnar sends muscular branches to the flexor carpi ulnaris and the inner half of the flexor profundis digitorum. The dorsal cutaneous branch of the ulnar, which innervates the skin of the posterior surface of the little finger and inner half of the ring finger, is given off about two inches above the wrist.

The clinical type of occupation neuritis which I desire to isolate is characterized by the absence of objective sensory disturbances, so that the nerve compression must take place below the point where the superficial palmar branch (sensory) is given off. It is further characterized by atrophic paralysis and reactions of degeneration in all the intrinsic muscles of the hand supplied by the ulnar, with preservation of those supplied by the median nerve. Therefore the compression must take place before the deep branch of the ulnar nerve breaks up into its numerous muscular branches, which begins immediately the nerve trunk has passed between the tendons of origin of the abductor and the flexor brevis minimi digiti (see Fig. 2). This short section of the deep palmar branch which intervenes between the giving off of the sensory branch and its breaking up into muscular branches, represents the seat of the compression.

This short section of nerve lies on the outer side of the pisiform bone and passes downward, backward, and outward, wind-

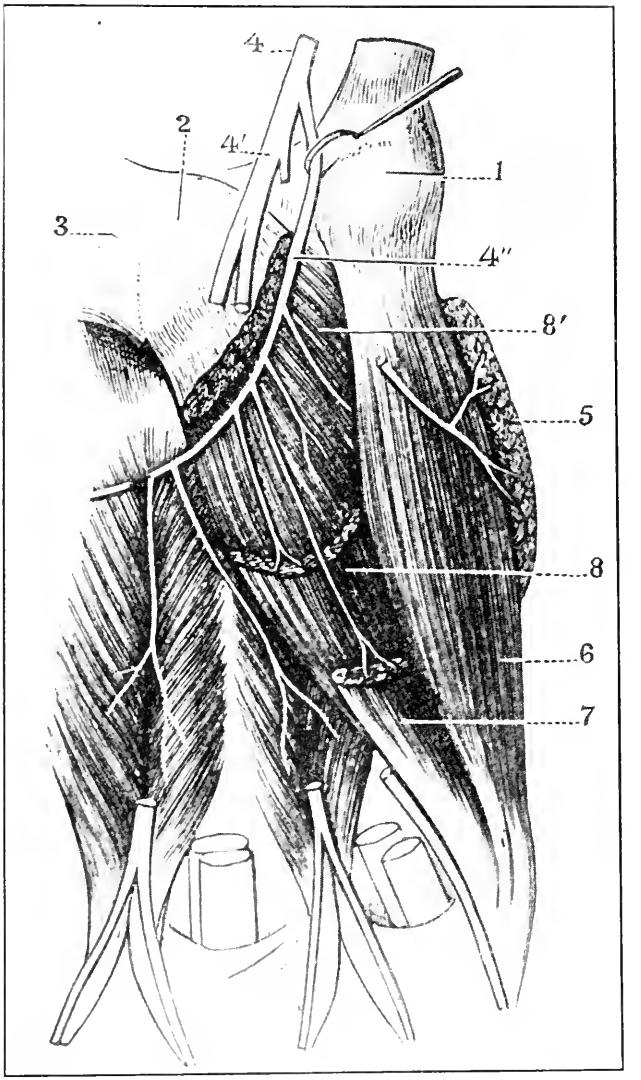

FIG. 2. (Testut's Anatomy.) The deep palmar branch of the ulnar and its relations to the abductor and flexor brevis minimi digiti. 1, pisiform bone; 2, unciform process of the unciform; 3, anterior annular ligament; 4, ulnar nerve; 4′, superficial branch; 4″ deep palmar branch; 5, palmaris brevis muscle; 6, abductor minimi digiti; 7, Flexor brevis minimi digiti; 8, 8′, Opponens minimi digiti.

ing beneath the hook of the unciform bone and thus reaching the deeper structures of the palm.

Reports of Cases

Case I.—Admitted to the Cornell Clinic for Nervous Diseases, service of Dr. C. L. Dana, March 2, 1906. A. R., Hebrew, born in Russia, unmarried. Age 24. Has been a jeweler by occupation for several years.

He is not addicted to the use of alcohol, and enjoys good general health. Lues is denied. In his work as jeweler, the object upon which he is working is held firmly in the left hand, and is grasped chiefly by the thumb and fingers. The filing process is performed by the right hand. He works from eight to ten hours a day, and experienced no inconvenience until about four months before he came under observation. At that time he observed a weakness and awkwardness in the movements of the fingers and thumb of the left hand, which was soon followed by distinct evidences of muscular wasting in the interosseous spaces. The atrophy was especially noticeable in the first interosseous space. He had no pain in the hand at the time of onset and has had none since, nor have paresthesias been present at any time. The weakness and consequent disability were of such a character that he was forced to abandon his occupation, although retaining a good grasping power in the hand.

Status Præsens.—On attempting to straighten out the left hand, it is found that the fingers cannot be fully extended, but remain slightly flexed. This tendency to flexion is more apparent in the little and ring fingers, although slightly present in the middle finger. The little finger is held somewhat abducted. On testing the various movements of the fingers, it is found that the basal phalanges cannot be flexed upon the metacarpal bones while the fingers are held extended. Abduction and adduction of the fingers are practically abolished. The movements of abduction and apposition of the little finger, and the adduction of the thumb are also abolished. In contrast to the paralysis of the adductor pollicis, the interossei, and the muscles of the hypothenar eminence, the abduction, flexion, and apposition of the thumb are well preserved. There is present a very distinct atrophy in the interosseous spaces, especially the first (see Fig. 3). The hypothenar eminence is also diminished in volume. There is no diminution in the volume of the thenar eminence. No fibrillary twitchings. The movements of the wrist are well preserved, the ulnar flexion of the wrist showing no impairment. The common extensors of the thumb and fingers are also well preserved, as are the deep and superficial flexors. The dynamometer on the right registers 50; on the left 40. There is no tenderness along the nerve trunks of the upper extremity, and

no vaso-motor disturbances. *The touch, pain and thermic sensibility of the left hand and forearm is perfectly normal.* The general neurological examination was negative.

Electrical Reactions.—Strong faradic and galvanic currents applied to the ulnar nerve at the elbow failed to produce any muscular response in the intrinsic muscles of the hand supplied by this nerve; a good contraction, however, is elicited in the flexor carpi ulnaris and the ulnar portion of the flexor profundus

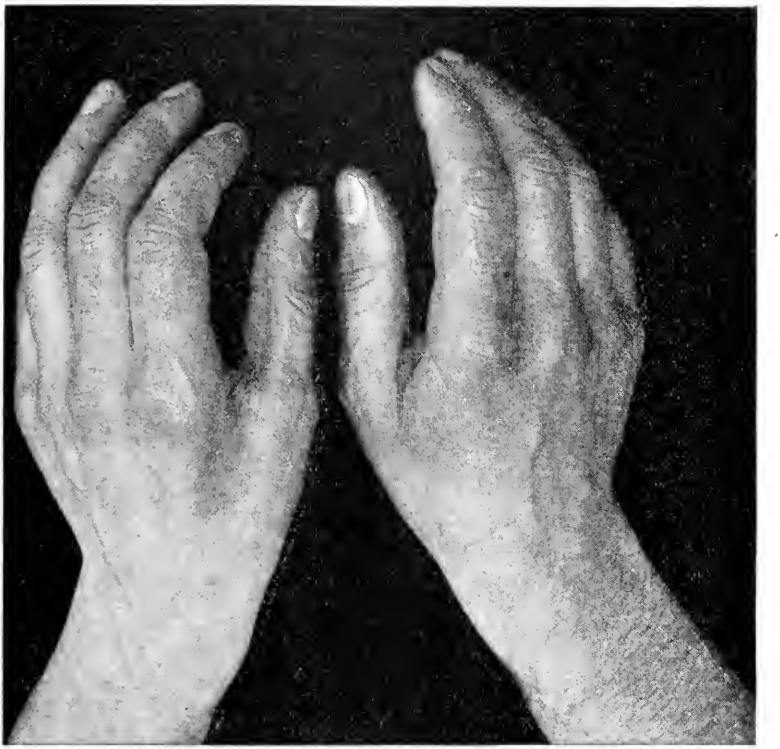

FIG. 3. Case I. Occupation neuritis of the deep palmar branch of the ulnar nerve, showing atrophy of the intrinsic muscles of the left hand.

digitorum. The faradic current directly applied to the intrinsic muscles of the hand, produces a slow and sluggish contraction in the muscles of the hypothenar eminence and the interosseous muscles. The direct galvanic current produces a slow vermicular response with reversal of the normal electrical formula in the muscles of the hypothenar eminence and the interossei. (Note: Qualitative changes were also present in the

muscles of the thenar eminence. These changes, however, were referable to the deeper muscles of this region supplied by the ulnar nerve, the abductor pollicis, and the inner head of the flexor brevis pollicis.) Faradic and galvanic currents, when applied to the median nerve at the elbow and at the wrist produce good contraction in the muscles of the thenar eminence corresponding to the distribution of this nerve.

June 11, 1906. The hand is free from pain or paresthesias, no fibrillations. Strong galvanic and faradic currents to the ulnar nerve at the elbow still fail to elicit any contraction in the intrinsic muscles of the hand supplied by this nerve. Strong faradic currents applied directly to the intrinsic muscles of the hand, produce only a sluggish response, and the galvanic response is still vermicular, with the CACLC = ACLC.

December 31, 1906. The hand is much stronger, the atrophy is less marked, and the inter-osseous spaces have filled out; at no time since the last note has he had any pain or paresthesias in the hand. The grip is practically as strong on the left as on the right side. He says that the hand now feels strong and well, and he has resumed his old occupation.

Electrical Reactions.—The intrinsic muscles of the left hand now respond both to the faradic and galvanic currents when applied to the ulnar nerve at the groove of the elbow. Strong faradic and galvanic currents are required to produce good contractions in the intrinsic muscles of the hand, but apart from this the response is normal in character and shows no qualitative changes.

Case II.—Admitted to the Cornell Clinic for Nervous Diseases, August, 1901, service of Dr. C. L. Dana. H. St. P. An Italian aged 24, married, and a machinist by occupation. Denies lues and is moderate in the use of alcohol. Had typhoid fever in March, 1901. Is of a nervous and excitable temperament, but enjoys good general health. On August 1, 1901, he commenced work with a machine, which necessitated his grasping a handle firmly with the right hand, and at the same time making some pressure against it. This occupation not only required constant flexion and relaxation of the hand and fingers, but there was also associated a pressure against the deeper structures of the palm. He worked nine hours on the first day. At the end of the second day he noticed a weakness of the right hand, but he has continued his occupation up to the present time, although with difficulty. There has been during this period, occasional sensation of pins and needles in the tips of all the fingers, and toward the end of the day, a dull aching pain in the whole right arm.

Status Præsens, August 30, 1901.—On holding out the right hand, a slight tremor in the hand and fingers becomes apparent. In the interosseous spaces there are distinct signs of wasting,

which is more conspicuous in the first interosseous space. The muscles of the forearm and upper arm are normal in appearance and functions. There is a complete paralysis of the muscles of the hand supplied by the ulnar nerve, *i. e.*, the hypothenar, the interossei, and the adductor pollicis. The functions of the muscles of the thenar eminence, which are supplied by the median nerve are well preserved. The dynamometer on the right registers 40, on the left 50. There is no tenderness of the nerve trunks or of the muscles, and *the sensations of touch, pain and temperature of the hand are perfectly normal.* The general neurological examination was negative.

Electrical Examination.—Faradic and galvanic stimulation of the ulnar nerve at the elbow induces a good contraction in the flexor carpi ulnaris, and the ulnar portion of the flexor profundus digitorum, but no response in the intrinsic muscles of the hand supplied by this nerve. Faradic and galvanic stimulation of the median nerve at the bend of the elbow produces a good contraction in the flexors of the wrist and fingers, as well as in the muscles of the thenar eminence supplied by this nerve. The direct faradic excitability of the hypothenar, the interossei, and the adductor pollicis is completely abolished, and the galvanic current in the same muscles produces a slow vermicular response with reversal of the poles. (Note: The patient did not return to the clinic and the subsequent course of the case is unknown.)

CASE III.—Admitted to the Cornell Clinic for Nervous Disease, April 17, 1902. Service of Dr. C. L. Dana. J. M., single, 38 years of age, a brass polisher by occupation. Denies lues; is fairly moderate in the use of alcohol, with occasional excesses. Three weeks before he came under observation, the right hand commenced to feel weak and awkward, and at times numb. It was found impossible to straighten out the little and ring fingers; there was no history of pain, and no tenderness of nerve trunks or muscles; *the sensation of the hand is undisturbed.* As in the previous cases, there was present a paralysis of the intrinsic muscles of the hand supplied by the ulnar nerve, with complete reactions of degeneration. (Note: This case also passed from observation, and the subsequent course is unknown.)

Remarks.—The symptoms in all three cases just described are practically the same. All have a common etiological factor, an occupation requiring flexion or grasping movements of the hand. One hand only was affected, and the weakness made its apearance without pain or cramps. In cases I and III the onset was apparently insidious, while in case II the symptoms made their appearance on the second day. Case I terminated in recovery; cases II and III passed from observation, and the eventual outcome is unknown. Careful tests were made of the objective sensibility of the hand in all of the cases, and it was found perfectly normal. The paralysis was sharply limited to

the intrinsic muscles of the hand supplied by the ulnar nerve. In these muscles the typical electrical reactions of degeneration were present. The flexor and extensor muscles of the forearm were normal, both in their voluntary innervation and electrical responses, as were also the muscles of the thenar eminence supplied by the median nerve. A characteristic attitude of the hand resulted when the attempt was made to extend the hand and fingers. This consisted in a persistence of slight flexion of all the fingers, but more especially of the little and ring fingers, the little finger assuming the position of abduction. The atrophy in case I was quite marked, and was also evident in a lesser degree in cases II and III which were more recent.

The clinical picture just described must be attributed to a lesion of the ulnar nerve; and from the complete absence of objective sensory symptoms the site of lesion must be placed below the giving-off of the superficial palmar branch, which conveys sensory fibers to the palmar surface of the fingers. While it is true that in pressure lesions of the peripheral nerves, motor fibers are more vulnerable than sensory, it is inconceivable that motor fibers alone should suffer without any objective evidences of sensory involvement. I would also emphasize the fact that not one muscle or one group of muscles was paralyzed, but that all the muscles supplied by the deep palmar branch of the ulnar were involved. Therefore the lesion of the deep palmar branch must have taken place before this nerve breaks up into its various muscular branches which begins immediately after this nerve has passed between the muscles of the hypothenar eminence.

These muscles of the hypothenar which I believe are chiefly concerned in the compression of the nerve trunk and consequently in the production of the lesion are: the flexor brevis minimi digiti, and the abductor minimi digiti. The short flexor takes its origin at the tip of the unciform process of the unciform bone, and from the anterior surface of the annular ligament. It is inserted into the base of the first phalanx. The abductor minimi digiti takes its origin from the pisiform bone and is inserted into the outer side of the base of the first phalanx. The deep volar or palmar branch of the ulnar nerve, according to some authorities, passes between the origins of these two muscles; according to others, it traverses the flexor brevis minimi digiti muscle just below its point of origin. Testut describes the course of this branch of the ulnar nerve as follows: "Aris-

ing at the external surface of the pisiform bone, sometimes a little higher, it is directed obliquely downward, backward and outward, traversing the insertion of the flexor brevis minimi digiti; passing beneath the unciform process and then reaching the deep palmar region." It would seem to me probable that the essential factor in the production of this form of neuritis, is one of muscular contraction or pinching as the nerve passes between the abductor and short flexor muscles of the hypothenar eminence near their origin. It cannot be denied that direct pressure may also play a role as well as traction of the nerve, as it passes beneath the hook-like process of the unciform bone.

Diagnosis.—The affections with which this condition may be confused are: the Aran-Duchenne type of progressive muscular atrophy and Gessler's type of occupation atrophy of the hand. Both conditions may give rise to atrophic paralyses in the intrinsic muscles of the hand without the objective disturbances of sensation. The separation of progressive muscular atrophy, beginning in the hand should not present great difficulty. The early involvement of the muscles in the thenar eminence (corresponding to the median nerve distribution) the presence of fibrillary twitchings, the tendency to progression, and the character of the electrical reactions should make the differentiation comparatively easy. The Gessler type of occupation atrophy, which he attributes to an involvement of motor nerve terminals and motor nerve endplates, would present greater difficulties; in fact, it seems to me not improbable that the two cases described in his paper should be classed with the group of cases which I have described rather than be utilized as the basis of a new pathological entity. All other neuritic forms of occupation atrophy of the hands are characterized by definite subjective and objective sensory disturbances.

From the diagnostic standpoint I would emphasize this fact, indeed the most important of all, in attempting to establish a diagnosis. That is to demonstrate the *limitation* of the paralysis to the intrinsic muscles of the hand supplied by the ulnar nerve. In order to do this it is necessary to show the preservation of functions of the muscles of the thenar eminence which are supplied by the median nerve. Usually these are the abductor pollicis, the opponens pollicis, the outer head of the flexor brevis pollicis, and the two outer lumbricales muscles. It must be borne in

mind that variations in this innervation may occur. Both the adductor pollicis and the third lumbricale are occasionally innervated by the median. Furthermore, in palsies in which the intrinsic muscles of the hand are alone concerned, the separation of these various movements of the fingers is by no means easy, because of the preservation of the long flexors and extensors of the thumb and fingers. These in themselves give a very considerable power and variety of movements to the hand and fingers.

Care must also be exercised in the interpretation of the electrical reactions in the thenar region. In an ulnar neuritis, the presence of the reaction of degeneration in the adductor pollicis and inner head of the flexor brevis pollicis which fill in the deeper portions of the thenar region, may mask the otherwise normal reaction in the more superficial muscles of the thenar eminence of median nerve innervation, from the well-known tendency to diffusion of the electrical currents. So that in case of doubt a more crucial test would be the response of these muscles by the indirect current through the ulnar and median nerves respectively.

It is interesting to note in relation to this group of occupation cases, that the deep palmar branch of the ulnar nerve may be severed by the injury. In one case recorded by Gortz, the prong of a pitchfork passed between the third and fourth metacarpal bones, entering the dorsal aspect of the hand; and in another case recorded by Bregmann, the nerve was injured by a spicule of glass which entered the palmar aspect of the hand near the pisiform bone. In both cases with paralysis and atrophy of typical distribution, there was an entire absence of sensory involvement.

Remarks on the Gessler Type of Occupation Atrophy of the Hand, and Its Relation to the Occupation Neuritis of the Deep Palmar Branch of the Ulnar Nerve

Hermann Gessler[2] in 1896 described a type of muscular atrophy of the hand, which he termed "a peculiar form of progressive muscular atrophy in gold polishers." The atrophy was stationary and there were no objective sensory disturbances. The

[2] "Eine Eigenartige Form von progressiver Muskelatrophie bei Goldpoliririnnen." Hermann Gessler. Medecinisches Correspondenz-blatt des Würtembergschen Arzlichen Landes Vereins, Bd. LXVI, No. 36.

non-progressive nature of the affection was sufficient to exclude a spinal type of atrophy. As sensory disturbances were absent, it could not be a neuritic atrophy, in the usual acceptation of the term.

It was also clearly not a myopathy. The distribution and limitation of the paralysis to the intrinsic muscles of the hand, the very considerable degree of atrophy and the accompanying reactions of degeneration, suggested very strongly a neuritic origin. The neuritis must however be limited to the motor nerves.

In order to meet the requirements of this clinical picture, Gessler evolved a fourth form of muscular atrophy. This new form of muscular atrophy was to have as its pathological basis, a degeneration of the terminations of the intermuscular nerves, inclusive of their end plates.

Such degenerative changes in the motor nerve terminals he attributed to the effects of persistent and long-continued muscular contractions, with insufficient relaxation. An anemia of the parts was induced with nutritional disturbances, and as a consequence, motor nerve end degeneration.

The delicacy and fragility of the motor nerve endings were supposed to render them peculiarly liable to such changes.

He refers to experimental work on the motor end plates,[3] carried out on warm and cold blooded animals; and as a result of which he became convinced that such changes must also furnish the pathological basis for some of the muscular atrophies in man.

It may be remarked that no pathological studies have been reported on cases of this character, which might throw light on this important subject. So much for the pathological evidence, on which Gessler's theory was based.

His clinical evidence consists of two cases which were practically identical. The following is, as far as possible, a literal translation of his case report.

I would emphasize their resemblance to the group which has been described as "occupation neuritis of the deep palmar branch of the ulnar nerve."

Gessler's Case Report.—"As the clinical picture is the same in both cases, I will confine myself to the description of the latter.

[3] Gessler, "Die motorische Endplatte und ihre Bedeutung für die periphere Lähmung." Habilitationsschrift, Munschen, 1885.

"Gertrude B., 21 years old, a gold polisher. Has worked for the past 8 years in the Pforzheimer gold ware fabrik.

"She is well developed and in good general health. Up to one and one half years ago, she had experienced no inconvenience from her occupation. At that time there appeared a numbness in the little and ring fingers of the right hand, and she experienced a difficulty in the use of these fingers, and a difficulty in extending them completely.

"Later, she found it more and more difficult to approximate the fingers, spreading movements were better preserved, except in the index finger, which lay in constant apposition to the middle finger. The little finger was fixed in the position of abduction.

"The right hand was colder to the touch and showed a tendency to become cyanotic on slight exposure to cold.

"During the past few months she has found it almost impossible to hold the object which was to be polished, and the hand became almost powerless. About the same time, there developed a curious flexion position of the ring finger, and a marked atrophy of the intrinsic muscles of the hand.

"The patient was forced to abandon her occupation, and after five weeks treatment by the family physician, she was admitted to Ludwig's Spital for treatment.

"*Status Præsens, Feb. 18, 1896.*—Large, well developed girl of healthy appearance. Internal organs are normal. No signs of any affection of the central nervous system. Left upper and both lower extremities are normal.

"RIGHT UPPER EXTREMITY.—The upper arm and shoulder are normal in motility and sensibility, occasionally there are shooting pains in the shoulder.

"The muscles on the extensor surface of the forearm are slightly atrophic; those on the flexor surface are normal in volume.

"*Hand at Rest.*—On the dorsal aspect of the hand the tendons of the extensor communis digitorum stand out prominently, and between them are well-marked depressions in the interosseous spaces. The slightly abducted thumb shows no atrophy, its phalanges are extended and the end phalanx is hyper-extended. The index finger lies in close apposition to the middle finger and is diverted towards the ulnar side, so that it forms an angle with the first metacarpal bone. There is a marked atrophy of the first interosseous muscle. The terminal phalanges of the index and middle fingers are slightly flexed, the basal phalanges are extended. The ring finger is abducted and presents a high degree of flexion, and the little finger is strongly abducted and flexed.

"On the palmar surface of the hand there is apparent, in addition to the flexion deformities of the fingers, *a marked atrophy*

of the hypothenar eminence and a slight atrophy of the thenar eminence.

"The sensibility of the right hand *shows no objective disturbance;* the right hand is colder than the left.

"MOVEMENTS OF THE HAND.—*The movements of the thumb may be carried out normally in all directions,* with the exception of flexion of the distal phalanx.

"The index finger has neither abduction nor adduction, which is also true of the middle finger. The ring finger may be slightly abducted but not adducted. The little finger on an attempted adduction becomes still more abducted. Flexion of the end phalanges is preserved and is accompanied by hyper-extension of the basal phalanges.

"The holding of objects is very difficult and the grip is very much diminished. Extension of the basal phalanges is normal; but is absent in the middle and distal phalanges. Flexion of the basal phalanges of the fingers is impossible.

"The *muscles of the hypothenar show no movement* and the little finger is held permanently abducted. Flexion and extension of the wrist joint, as well as abduction and adduction, are unaltered; although the extensor movement of the wrist seems weaker than normal.

"ELECTRICAL REACTIONS.—The electrical reactions of the muscles of the forearm are normal.

"Electrical stimulation of the ulnar and median nerves produces no contractions in the hand muscles.

"The interossei and lumbricales show complete reactions of degeneration.

"The muscles of the hypothenar show an increase of the galvanic excitability with reversal of the poles, and a diminished faradic excitability.

"*The muscles of the thenar eminence show a diminution to both currents.*"

Remarks.—In the report of Gessler's case just cited, I would emphasize the following points of resemblance to the group of cases which I have described.

1. At the onset there were paresthesias, definitely limited to the distribution of the ulnar nerve; no objective sensory disturbances however.

2. While the muscles of the hypothenar eminence and the interossei, all of which are innervated by the ulnar nerve, were paralyzed, the movements of the thumb were undisturbed.

3. The atrophy, which had reached a high degree in the interosseous spaces and the hypothenar eminence, was only slightly present in the muscles of the thenar eminence.

4. In the description of the electrical reactions, while the muscles of the hypothenar eminence and the interossei show the typical reactions of degeneration with polar changes, the muscles of the thenar eminence show only a diminished faradic and galvanic response.

It is therefore apparent that the distribution of the paralysis, the atrophy and reactions of degeneration, are all suggestive of ulnar nerve involvement.

It will also be noted that the paresthesias noted were in the distribution of the ulnar nerve alone. The median nerve in the hand innervates the abductor pollicis, the opponens pollicis and the outer head of the flexor brevis pollicis; and as it is stated that the movements of the thumb were normal, the muscles of the thenar region only slightly atrophic, as compared with the hypothenar, and the electrical changes consisted of only a diminution in the response, not a degenerative reaction, one would be justified in assuming that the thenar muscles of median innervation were not involved.

This being true, the remaining paralysis and atrophy must be referred to the ulnar nerve alone. The occupation was one requiring frequent, almost constant flexion and grasping movements of the fingers and thumb, and assuming the lesion to be a degeneration of motor terminals from anemia, there is no reason why the paralysis should be limited to the ulnar nerve distribution, nor why the small muscles of the thumb should escape.

For the reasons just detailed, I feel justified in regarding the cases described by Gessler as belonging in all probability to the syndrome which I have outlined; and resulting from compression of the deep palmar branch of the ulner nerve.

Furthermore an attitude of skepticism towards the pathological type of muscular atrophy introduced by Gessler is more than justified. The clinical facts on which it was based are insufficient and pathological confirmation is wanting.

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REPORT OF A CASE OF MYASTHENIA GRAVIS PSEUDO-PARALYTICA WITH NEGATIVE PATHOLOGICAL FINDINGS[1]

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Since the original contributions of Erb, Jolly, and Oppenheim, concerning the symptom complex of myasthenia gravis pseudo-paralytica, the study of this mysterious disease has been stimulated by the investigations of numerous observers on the functions of the ductless glands. Moreover, as a result of this study much that was formerly obscure in other peculiar disorders of the nervous system has been made more clear, and also certain theories more worthy of support. It is now interesting to note in connection with the disease under consideration that there are at the present time a number of reports, showing involvement of the thymus gland or pituitary body, with lymphorrhages in different organs and lymphocytic infiltration in the muscles. The most recent and important of these being that of Mandlebaum and Celler, who found all the above changes to a marked degree. The parathyroids are also receiving their share of attention, and Chvostek in a recent review presents evidence in favor of the assumption that the symptom complex of the disease is best explained as the result of defective functioning on the part of these glands. Yet with all the advantages derived from improved methods in the preparation of specimens and histological study, cases of the disease are still recorded in which no lesions are found to satisfactorily account for the symptoms. Believing such contributions are not only of interest, but also of importance, the writer takes this opportunity of offering for your consideration the following history:

[1] Read at the thirty-fourth annual meeting of the American Neurological Association, May 20, 21 and 22, 1908.

The patient, Ralph A——, a lad of 11 years, was seen with Dr. William H. Haskin at the Manhattan Eye, Ear and Throat Hospital on October 16, 1907.

Family History.—The father and mother are both living, the former a drunkard. The mother has two sisters and one brother, all insane. She has had seven children and five miscarriages. Three children died during infancy from diarrheal disorders. Of the four living children our patient is the youngest. An examination of the others does not show any form of nervous disorder.

The mother states that he was healthy during infancy and childhood, with the exception of a mild attack of measles when four years old.

In his seventh year he had an acute attack of otitis media, followed by a purulent discharge from the left ear; this received treatment and soon ceased. In April, 1906, he was operated on for adenoids and from this dates the onset of his present illness. Within a few days following the operation he became easily tired, was listless and complained of a general muscular weakness, which would disappear after lying down for a time and then return on exertion or after any excitement. He was bright mentally, standing well in his class at school, but his attendance was interrupted by frequent periods of extreme lassitude and weakness, during which he would remain at home, lying down most of the time. In April, 1907, he complained of his eyelids feeling heavy and these finally drooped; then there rapidly developed a progressive weakness of the facial muscles and a difficulty in raising the arms and going upstairs. He also talked in a peculiar voice and had difficulty in chewing and swallowing his food. Any shock or excitement seemed to increase this disability, and at such times he would become almost powerless. Recently his neck muscles have become weak.

Examination.—The patient is poorly nourished, but not emaciated. The mentality is good and he answers questions intelligently, though in rather a feeble voice with a nasal tone. The expression of the face immediately attracts attention, being flat and expressionless, accompanied by a bilateral ptosis, and he says this drooping of the eyelids is worse at night than in the morning. The lips are not held together and he is not able to pucker them or whistle.

Muscular Status.—Bilateral ptosis and diplegia facialis. The muscles of the arms, shoulder girdle and thighs are flabby, but there is no degenerative atrophy or fibrillary twitchings. The calf muscles seem firm and hard compared to other muscles of the body; the right calf measuring 9½ inches and the left 9¾ inches. All voluntary movements are performed slowly and are soon followed by exhaustion. Lying down on the floor and requested to get up, he does so at the first attempt quickly, but

after one or two repetitions, he is unable to accomplish this without artificial aid. This same condition of induced fatigue is present in the speech and swallowing mechanism. The grasp of the hands is weak, only five of the dynamometer in each hand. The gait is slow, but there is no dragging of the feet or any evidence of spasticity. The pupils are equal and react to light and accommodation. The fundus is normal. Reflexes: All reflexes present and normal. The knee jerk response varied, being sometimes present and then again absent, especially after six to eight taps.

Sensation.—There is a slight loss of tactile sensation over the inner side of right arm and right face. No analgesia or loss of temperature or muscular sense. With the exception of a slight defect of hearing in left ear, all the special senses are normal.

Electrical Reactions.—To galvanism all muscles react normally and to faradism there is a contraction and finally exhaustion of the response in the sterno-mastoids, trapezii, deltoids and extensor communis digitorum of each side.

Diagnosis.—In the absence of hereditary disease of similar kind in the family history, the sudden onset following shock and the presence of marked bulbar symptoms, with the results of the electrical reactions, we can eliminate the pseudo-hypertrophic or Landouzy Dejerine type of myopathy. On the other hand, taking into consideration the variability of the symptoms, increased by emotion and excitement, together with the clinical history, seemed to warrant the conclusion that the symptoms were those of myasthenia gravis pseudo-paralytica.

With this diagnosis he was transferred on November 12, 1907, to St. Luke's Hospital for further observation. Here the rapid variation in the severity of the symptoms from time to time was marked. I may mention, to illustrate this, that I attempted to bring him before the Neurological Society, but he had an attack of respiratory failure on the way to the meeting, his condition became alarming and I was forced to return to the hospital. After this he grew rapidly worse and had several similar attacks, in one of which he died on November 18, 1907, at 5 P. M. During the six weeks he was under observation the temperature was normal, the pulse varied between 60 and 90 and respirations between 20 and 40. Just before death the temperature rose to $100\frac{2}{5}$, pulse 100 and respirations 40.

Post Mortem Report.—The autopsy was performed one hour after death by Dr. Francis C. Wood, pathologist of St. Luke's Hospital and Columbia University: Body emaciated. No rigor mortis. Cartilages and ribs rather prominent. Skeletal muscles not well developed but apparently normal. Lungs and heart present nothing abnormal on section. The thymus gland is unusually well preserved for the age and is considerably enlarged; extending from the upper border of the sternum, as a long, flat

boat-shaped mass and measuring about 11 by 5½ centimeters in width and one centimeter thick. Thyroid gland normal and mediastinal glands slightly enlarged. Abdominal organs: Coils of intestines collapsed. Liver, spleen, stomach, pancreas, kidneys and suprarenals are all apparently normal. Head: Brain apparently normal and weighs 3½ pounds. Pituitary body normal. Spinal cord normal. Specimens from different muscles, nerves, brachial and sacral plexus taken for further study.

Microscopical Examination.—The muscles, both voluntary and involuntary, including that of the heart and tongue; the detoid, brachial, extensors of the thigh, abdominal and pectoral, show no changes from the normal; there being no evidence of lymphocytic infiltration or degeneration. The thyroid and parathyroids show no lesion.

The thymus gland, which was enlarged, is normal except for an occasional area of focal necrosis. The Hassall's bodies are well marked. The lymphoid tissue is normal in distribution and amount. There is a moderate growth of connective tissue between the lobules, but no more than is normal in a child of this age. No lesions could be found in the nerve trunks. The lungs and heart are normal. The liver and kidneys show slight parenchymatous degeneration; the latter being slightly congested. The pituitary body and suprarenals are also normal.

Central Nervous System.—On inspection the pons, medulla and spinal cord were normal. Sections were made and examined from the following situations; through the fourth and first cervical segments, at the lower part of the medulla, through the medulla at the level of the eleventh and twelfth nuclei and four others higher up, of which one was through the vagal nucleus. The cells of the nuclei, the fibers, vessels and all the tissues in every section appear normal, hence any organic lesion of these parts of the nervous system may be safely excluded.

Summary.—Considering the clinical history, the symptoms and course of the disease in the case just cited; the diagnosis of myasthenia gravis pseudo-paralytica was justified. The initial symptoms came on after an operation for the removal of adenoids; they were rapidly progressive and later any emotional excitement caused an increase in their severity. During the six weeks he was under observation he had three attacks of respiratory failure after slight exertion and each was due to this cause. Examination of various muscles, done very thoroughly, did not show any tendency to muscle degeneration or the presence of lymphoid infiltration. The brachial plexus, the sacral and the nerves themselves were also normal. With the exception of a simple slight hypertrophy of the thymus gland, the glandular

structures were normal, including the thyroid, parathyroids and pituitary body. The examination of the central nervous system revealed no lesion or pathological condition to which the paretic symptoms might be attributed.

Conclusions.—In view of the above facts and the various pathological findings recorded by others in similar cases, we must conclude that the symptom complex of the disease is best explained on the basis of its being a nutritional disorder impairing the vital processes in the muscles from some unknown toxine.

In conclusion, I wish to express my thanks to Dr. F. C. Wood and members of the pathological staff of St. Luke's Hospital and also to Dr. M. G. Schlapp for his examination of the central nervous system.

TROPHIC HEMIATROPHY: COMPLETE

A Trophoneurosis[1]

By Thos. J. Orbison, M.D.

of Los Angeles, Cal.

I have found in literature six cases of hemiatrophy that were more or less complete, *i. e.*, that involved the face, arm and leg, or the arm and leg only, of one side. Of these the one described by Paul Broca[2] in 1859 was similar in its details to the one here presented. He said of it—a boy eleven years old—it was "as if the two halves of the body were different sized persons joined together."

In none of the references could I find any satisfactory data relative to the pathology, electrical reactions and reflexes.

Hemiatrophy of the face, including the progressive type, has often been described. Romberg is given the credit of having described the first case. Up to February, 1907,[3] 170 cases of the progressive type had been reported. Oppenheim[4] found one case in which the leg of the opposite side was involved, but at that time had never seen one in which the entire half of the body was involved—as is the condition in the case here presented. He believed the etiological factor to be "an unknown agent acting only in neuropathic individuals." Most authors believe the condition to be a trophoneurosis.

The first case found reported that resembled this one was that of Paul Broca in 1859 (vide supra). A. Stadfeldt[5] and Jacobi[6] considered, in their cases, the causal factor to have existed prior to the sixth month of fetal life. Howden[7] reported an insane woman, aged thirty years, who exhibited atrophy of the left side of the

[1] Case presented before the Clinical and Pathological Society of Los Angeles.
[2] Canslatt's Jahresbericht, 1859, Vol. IV, p. 6.
[3] Deutsche Zeitschrift für Nervenheilkunde, February, 1907.
[4] "Diseases of the Nervous System."
[5] "On the Asymmetry of the Body of the Human Skeleton."
[6] Trans. N. Y. Obst. Soc., 1871.
[7] Jour. Anat. and Physiol., Vol. IX, p. 288.

body; in this case post-mortem findings showed atrophy of the right hemisphere of the cerebrum and left side of cerebellum. In 1878[8] a case of hemiatrophy was reported and shown before the Clinical Society of London. In the same year another was shown before the Societe Anatomique by H. C. Beyer.[9] Another was reported by S. Pixley in 1883.[10] In the next year R. C. Burrell[11] reported and described a case similar to mine except that the face was not involved.

Of these six cases of hemiatrophy four showed it on the left side. Of the remaining two, I find no reference to the side involved.

I have not been able to find any case that is similar to this one reported since 1884 except one referred to in a late number of this Journal.

The points of interest attached to this case are—its rarity; the fact that the hemiatrophy is complete, *i. e.*, it involves the face, arm and leg; there is an hypertrophy of the trapezius muscle on the atrophic side; the psychic symptoms accompanying.

There are two unsatisfactory elements in it: (1) The short time that could be given to watching it because the man had to leave for the East (2) the unreliableness of a man of his type and his statements. However, the case presents much of interest, especially from a clinical standpoint.

CASE.—N. G., male, 26 years of age, unmarried. His occupations have been various: clerk, waiter, hospital orderly.

His family history is not of interest except that he has two brothers that are normal and that his mother died when he was five years old and his father when he was seven years old, when he was placed in an orphans' school.

Previous History.—He says that as a child he was not like others and would not play with them unless forced to do so. He was inclined to be solitary. Other than this he remembers nothing out of the ordinary except that during his first year at the orphans' school he began to have certain peculiar sensations; the first one came on while he was in the class room. He describes it as being like a shock accompanied by vertigo. It frightened him and he gave a cry and "staggered" to the teacher's desk. In a few moments he felt all right again. Since then there have been periods in which these "shocks" have come on daily or

[8] Lancet, 1878, Vol. I, p. 387.
[9] Le Progress Médicale, 1878, p. 242.
[10] Phil. Med. News, 1883, Vol. XLIII, p. 146.
[11] Boston Med. and Surg. Journ., Vol. CXI, p. 462.

oftener and others when they have been absent for months. During the attack he has never been unconscious or fallen. He describes it as an electric shock that he feels all through his left side only, but without any convulsive movement whatever being visible—no muscular twitching, no biting, no involuntary micturition or the like. After the "shock" he is slightly dazed, nervous and mentally clouded: he describes it as a "fluttering." All this will last only a few minutes. The attacks have all been very much alike, a one sided psychic epilepsy, as it were.

So much for the psychic element in the case.

For the last seven years he has noticed that his left side has been smaller than the right; he is not sure whether the left side has grown smaller or the right side larger, leaving the left as it was. He has evidently not been much exercised about this, as he did not volunteer any information concerning it and it was only noted by me when I stripped him for my routine examination. He says that for the last four or five years he has noticed that the left side of his neck has been larger than the right.

He does not remember having any serious illness during his life.

Present State.—He complains that the "shocks" interfere with his ability to attend to any kind of work because of the momentary weakness and mental cloudiness. He says he is in a constant state of apprehension and complains of inability to sleep. He says he is easily fatigued and complains of lack of appetite and constipation. Underneath all his words and acts runs the intense egotism and self pity and complaining criticism of those who have tried to help him and self-distaste of his class.

Physical Examination.—He has a markedly neuropathic facies. The eyes are slightly bulging and their expression heavy. The orbicularis oculi muscles are lax and the palpebral fissures sag widely on both sides alike. The pupils are equal and react promptly to light and in accommodation. The visual fields are not cut. The tongue is protruded slightly to the left. The teeth are good and there is no deformity in the mouth. The shape of the head is good and no ill-placement of the ears or any other marked stigma of degeneracy.

The head is held tilted with the chin pointing to the left and the top of the head to the right. While there is present the distinct difference in the size of the two sides of the face there is no loss of function of the facial muscles on either side. The neck: the trapezius on the left is much larger than that on the right (and is hard, giving a suspicion of tumor formation)—a paradoxical hypertrophy. The arm and leg: on the left side they are uniformly smaller than on the right. The bones seem to be of equal size on both sides. The discrepancy is not limited to any muscle or group of muscles. The grip on the left side is good and nearly as strong as on the right. There are no fibril-

lary twitchings of the muscles on either side of the face, trunk or extremities.

Reflexes.—Knee jerks are prompt and equal; biceps jerk is very prompt on the left and less so on the right; Achilles jerks

FIG. 1.

are equal; triceps jerks are equal; Babinski reflex is absent on both sides; muscle jerks are much more prompt about the shoulder girdle on the left than on the right—this is true of the heavier muscles all over the left side; skin reflexes are more prompt on

the right. There is considerable tilting of the pelvis with consequent spinal deflection that is corrected by raising the right heel five eighths of an inch. Heart and lungs are normal. The abdominal and genital organs are normal.

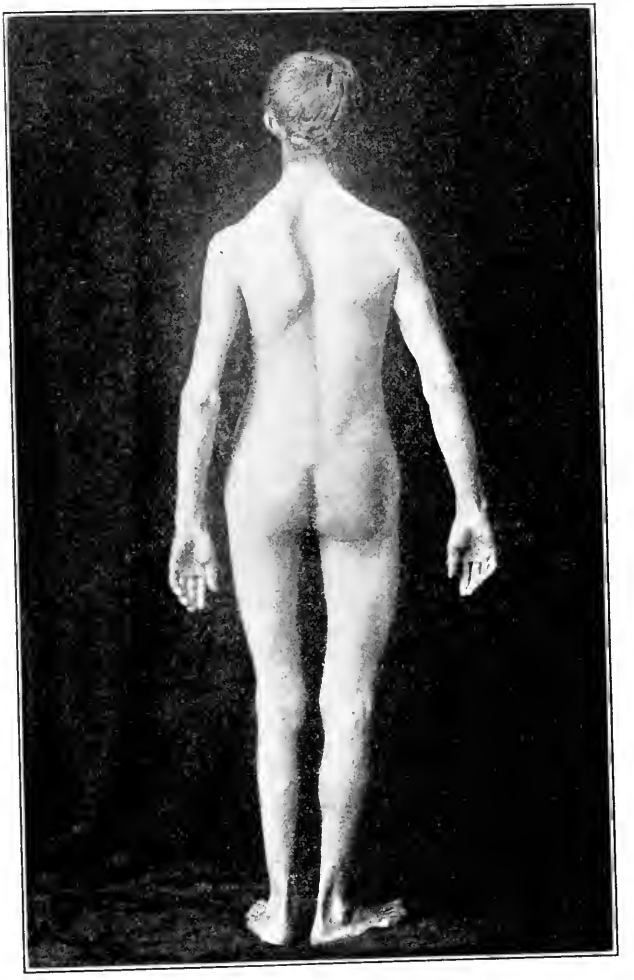

FIG. 2.

Electrical Reactions.—There is no failure to respond to the faradic current on the part of any muscle of the face, arms, legs or thorax. The amplitude is not so great nor the response so prompt on the left side. The reaction time was quicker on the right.

Stigmata of hysteria were not found. The voice is slow, low pitched and listless. There is an evident play for sympathy on the part of the patient that has been taken into account. During the examination he had a "shock": there was no tremor, convulsion or any other outward sign. He was perfectly conscious and talked perfectly connectedly throughout it.

It is noteworthy that he uses the left hand as little as possible: he says he cannot rely on it, and yet his grip is almost as strong on the left as on the right and can be sustained for a long time. The atrophied muscles are not flabby or flaccid.

Sociologically the case is that of a poorly mentally-accoutred, illy-developed, one-sided human inept—to coin a word. Psychologically he would be classed as a malingerer, inasmuch as he consciously and from choice fails to employ what powers he possesses. Neurologically he does not adhere to any one type; there is present the accentuated muscle jerk on the atrophic side that resembles amyotrophic dystrophy, but there is lacking the increased knee jerks, bulbar symptoms and gait. The case is not one of anomaly in which there is present only the failure to develop that happens to be more or less symmetrical: for the differences in the electrical reactions and the skin reflexes together with the marked exaggeration of the muscle jerks on the atrophic side would point to a pathological process, as would also the "shocks" that were felt only on one side. The case is not one merely of asymmetry; the symptom complex would seem to preclude this. The question of asymmetry has been thoroughly worked out. Hunt[12] and Morton,[13] and Roberts,[14] T. Dwight[15] and J. G. Garson[16] have all made reports that correspond closely. They investigated the subject from various points of view. Morton examined over 500 boys and youths and found that about two or three out of every five were asymmetrical, inasmuch as one leg would be shorter than its fellow. They did not report a single case of complete hemiatrophy. In Wight's[17] report of his findings the ratio of asymmetry is placed at one to five.

The case seems to be a trophoneurosis accompanied by a psychosis that may be dependent on it.

As to the probable pathology: "According to de Watteville, trophic changes in muscle may be independent of paralytic phenomena, may affect both muscle and nerve or muscle alone."[18] The trophic, voluntary and reflex influences converge in the multipolar ganglion cell. There may be lesion of one or more of them. If, for example, the trophic and motor influences are

[12] Am. Journ. Med. Sci., January, 1879.
[13] Boston Med. and Surg. Jour., April 15, 1880.
[14] Phila. Med. Times, August 3, 1878.
[15] Mass. Med. Soc. Commu., 1878, p. 125.
[16] Journ. Anat. and Physiol., London, 1878–9, pp. 502–7.
[17] Archi. Clin. Surg., Vol. I, No. 8, February, 1877.
[18] Mills, "The Nervous System and its Diseases," p. 195.

affected there will result a paralysis with atrophy of the muscle. This is the condition in amyotrophic lateral sclerosis. If the lesion is confined to the trophic centers the muscle atrophies but is not paralyzed and presents qualitative alterations in reaction.

It is conceivable that the "unknown agent" of Oppenheim selects the trophic centers for the point of attack. It would seem from the reported cases that the left side, for some reason, is the favorite point.

Mensuration.—The differences in the two sides at various levels are as follows: circumference of head just superior to eyebrows, right half 11.5 in., left half 10.75 in.; of face just inferior to ears, R. 8.5 in., L. 8 in.; of body about the shoulder level: R. 20 in., L. 18.5 in.; about the thorax at nipple level, R. 16 in., L. 14 in.; upper arm, R. 10 in., L. 8.75 in.; thighs, R. 17.5 in., L. 15 in.; calves, R. 12 in., L. 10.5 in.

Conclusions.—The condition is a pathological entity; its beginnings are prenatal in time; the etiological factor is an unknown agent, possibly a toxemia in the fetal circulation that attacks neuropathic individuals and selects the trophic elements, preferably of the left side; the symptoms are atrophy of the muscular structures (or a retardation of their growth); quickening of the muscular reflexes with slowing of the electrical reaction time and without affecting the tendon reflexes materially; asymmetry with accompanying spinal deflection.

Society Proceedings

AMERICAN NEUROLOGICAL ASSOCIATION

The Thirty-fourth Annual Meeting held at the College of Physicians of Philadelphia, May 20, 21 and 22, 1908.

The President, Dr. Charles W. Burr, in the Chair

(*Continued from p. 655*)

Dr. H. H. Hoppe read a paper entitled: Localized Epileptic Convulsions without Gross Lesions of the Cortex of the Brain. (To be published in this journal.)

Discussion

Dr. L. Pierce Clark said he was rather surprised to see the number of idiopathic cases that Dr. Hoppe reports dying of status hemiepilepticus. In studying temporary palsy in epilepsy in a series of cases at the Craig Colony in the 14 cases of this type independent of organic lesion, only one died. It is not an uncommon experience to see these patients with status hemiepilepticus have 10 or 15 status periods before finally dying. He has one patient whom he has had under watch for years, who has had 28 attacks of this sort. It was a common practice in the Colony as well as in his Randall's Island service in New York, not to notify the relatives of possible fatal outcome of status hemiepilepticus. The staff from experience knows, even though the status is very severe, the patient will not die. Dr. Clark said he had looked over the cases Dr. Hoppe mentioned from literature, and to his mind they were not without suspicion of being of an organic basis. There was one particular point in Dr. Clark's one case that came to autopsy, that was of special interest to him, a post mortem rise of 2° of temperature on the side that had been constantly convulsed was noted. He thinks nearly all cases of focal manifestations really come under the head of genuine epilepsies in spite of the fact that they have this focal type. Dr. Clark believes the microscopic evidence will show the condition of cell degeneration, possibly replacement gliosis. A maximum of intense change will show on some one spot. His idea is that the part of the brain entering into the convulsion is the focus of maximum change. However, in his case the entire cortex showed the changes that Dr. Prout and he thought to be characteristic of the anatomic pathology of the brain cortex in epilepsy. Dr. Clark stated that he now has under treatment 3 cases of Dr. Hoppe's type of genuine epilepsy with focal manifestations with no tendency to progression. In fact in 2 of them the disease has steadily improved independent of medication, the treatment being based entirely on hygienic lines. In the light of his pathologic findings Dr. Clark entirely agreed with Dr. Hoppe that operation in these cases is to be discouraged. He has seen several unsuccessfully operated cases in private practice and there are at least 6 or 7 more at Craig Colony. They are much less amenable

to medical treatment after these operations. Two cases he had in mind were cases of this sort; the disease had been moderately progressive and operation was performed. For two or three weeks the patients remained free from attacks, then the attacks returned worse than before; some of the worst types of epilepsies at the Craig Colony were those idiopathic epilepsies with focal attacks where operation had been done. Dr. Clark thinks that the underlying pathology of these conditions will be found to be a chronic cellular degeneration of the cortex and secondary gliosis.

Dr. E. E. Southard said that the post mortem analysis of such cases as that of Dr. Hoppe's servant girl would under ideal conditions include careful bacteriology. This suggestion does not mean that the problem of idiopathic epilepsy can be solved by bacteriology alone. Nevertheless bacteria are found in the blood and tissues of man far more often than was formerly thought. Bacteriemia in non-febrile conditions requires careful investigation. A "cured" patient is sometimes sent from hospital with Bacillus mucosus capsulatus in the blood (See Duval and Lewis "General Septicemia," *Jour. Med. Research,* 13, 1905, p. 535). Internists are beginning to speak of "low grade sepsis" in these conditions.

At the Danvers Insane Hospital the problem of terminal infections has been attacked in a series of 100 cultures from cerebrospinal fluid and blood in autopsied cases. The fluids were positive in 75 instances, the bloods in 64 (read at the 1908 meeting of the American Medico-psychological Association, Cincinnati, and to be published shortly: Gay, Richards, and Southard). Bearing on the superior numbers of bacteria in the cerebrospinal fluid as against the blood, is the observation of Gay (to be published shortly in the *Proceedings of the Society for Experimental Biology*) that the cerebrospinal fluid of normal persons, whether in life or post mortem, fails to contain that substance, the alexin, which is essential in bacteriolysis.

In some cases with cultures from the mesenteric lymph nodes, in addition to blood and cerebrospinal fluid cultures, the nodes and fluid contained bacteria, whereas the blood was sterile. In one instance, the Bacillus coli communis was found in these relations, in another the Bacillus typhosus (read at American Association of Pathologists, 1908, Ann Arbor: "Typhoid Meningitis," Southard and Richards). The work of Ford Robertson with diphtheroid organisms in paretics perhaps belongs in this category. The relation of such findings to convulsions and crises in general paresis has yet to be worked out.

One practical point is that a single negative culture from the brain may not yield the required data. In one case of streptococcus meningitis, the pus at the vertex was sterile, that of the flanks of the brain showed a moderate number of organisms, and that of the base yielded tremendous numbers. Multiple cultures may be necessary (Southard and Stratton, "A Study of Acute Leptomeningitis (Streptococcus pyogenes)," *Jour. American Medical Association,* 1906).

Dr. W. G. Spiller said he had one case of status hemiepilepticus operated on about a year ago in which paralysis had developed on the side of the convulsions, probably as a result of exhaustion. The brain was exposed, nothing organic was discovered. The boy recovered from the operation and status hemiepilepticus, and has done very well since. The convulsions have been less frequent than they were previously.

Much more important is the point Dr. Hoppe has raised regarding operation where there have been no convulsions during many years, and when they developed were strictly confined to one side, sometimes to one limb. He did not refer to those cases where the convulsions begin

in one limb and later implicate the whole body. If we are to be successful in removing brain tumors we must operate at an early stage. If we postpone operation until we can make a diagnosis that would be easy for a second or third year student, we shall not be successful in our operations. Dr. Spiller said he recalled several cases in which unilateral convulsions were the only important sign of brain tumors revealed by operation. It is Dr. Spiller's practice to recommend operation early when the surgeon is experienced in such operations. He said they had been very successful at the University Hospital in such cases, and find it wiser to expose the brain by an osteoplastic flap, in a doubtful case even if nothing abnormal be found. The flap is readily replaced and no damage is done.

Dr. B. Sachs stated that the question as to localized convulsions is very important. Distinction must be made from cases in which the patient is suffering from long continued form of epilepsy in which the individual attack begins in localized fashion. He thinks we see innumerable cases of that sort. No one would ascribe these cases to a recent lesion or one that we would consider operable, but even before the French observers that Dr. Clark mentioned, Dr. Sachs was one of the first to prove, taking a large number of cases of epilepsy, that a large percentage if traced back as to their clinical history will be found to be due to a lesion very early in life. There has been very satisfactory evidence on that point. Dr. Sachs thinks the diagnosis on the non-organic character of a focal epilepsy is a very difficult position to establish. For the present he thinks the safe plan to proceed upon is, that if there is a localized lesion there must be some circumscribed change in the brain. That change may, however, be of a microscopic character as we have also shown in some cases and it would not be evident if the patient were put on the operating table. But that there is some change either microscopic or macroscopic is a point we will have to maintain until there is very complete proof to the contrary. On the other hand, the only cases of focal epilepsy in which we are justified in urging operation, are those in which the focal epilepsy comes on in acute fashion, the person never having had attacks before. In spite of all this everyone of us has encountered cases of focal epilepsy coming on in acute fashion in which there were all the symptoms of a focal lesion, and yet when the operation was undertaken no focal lesions were found. The explanation of these cases is a very difficult one, unless we assume that the change is microscopic and not macroscopic. At one of the New York hospitals a number of years ago focal epilepsy was found in several cases of acute renal disorder, an operation was urged and one of the cases came to autopsy, and it was found that there was absolutely no lesion in the brain but there was acute renal disease. The only point Dr. Sachs insists on whenever called to advise in acute focal epilepsy is that we meet now and then with acute cases, particularly acute renal cases in which convulsive seizures occur of the focal type, not necessarily dependent upon gross organic lesion.

Dr. Putnam said the subject was very interesting to him, that he recognized entirely the importance of Dr. Clark, Dr. Sachs and Dr. Hoppe's views of the subject about the possibility of the focal lesion, but it seemed to him that it was possible to misunderstand, to state the method in rather an imperfect way. We have to deal possibly with a focal lesion, but more often with conditions not so much of maximal change at a given point, but of maximum irritability that has often been urged, but not too often. These cases we are all familiar with, not simply where the epilepsy begins in one part, but where it is confined to special attacks

for a long period. There may be some peculiar disturbance of vision, or of sensation in one finger. One might suppose there were focal lesions, but more probably from what we know these disturbances simply represent areas of maximum irritability, and there may be no focal lesion. Two cases he has in mind compare strongly on that point, one of especial interest in connection with what Dr. Sachs has said, a case of a lady recovering from influenza of rather severe type who was not under his care, was getting well, sitting up and able to knit, when she was attacked suddenly with spasm involving first the hand alone, then in twenty minutes she had an attack involving the face with the hand, and then a final attack of hemiplegia, still without loss of consciousness, without paralysis a day or so, then she became hemiplegic and died. Dr. Putnam was present at the autopsy and made a very careful examination of the brain, microscopic and macroscopic. The whole brain was edematous and was doubtless the seat of lesions in a toxic sense, but they certainly were not focalized.

The other case was that of a young man who had a temporary hemiplegia, which came on during typhoid. The hemiplegia passed away, but was followed by seizures of this sort which were confined to the hand. He called them "rushes." Dr. Warren operated. A large area of the cortex larger than that covered by the trephine opening was the seat of a soft, velvety material, of an organized clot one might say, and a portion of it was excised. In the third case focal signs were present. A bone flap was laid back, the cortex appeared perfectly normal, and yet the hand was left weak simply from the exposure. The weakness passed away. There was no other disturbance from the operation.

Dr. F. X. Dercum, said that while the differentiation between cases of epilepsy beginning in an apparently limited manner and yet rapidly becoming diffuse, from cases clearly focal in character is often very difficult, the patient should even in doubtful cases be given such chance as surgical exploration may afford. Nowadays it is safe to make an osteoplastic resection, and although there are cases in which nothing is found, there are notwithstanding others in which something *is* found. Besides if our cases are carefully selected the percentage of failures will be comparatively small.

Dr. Wm. M. Leszynsky spoke of a young woman under his observation who entered the hospital for operation on the uterus. She recovered from this operation, and in the course of two weeks developed status hemiepilepticus which lasted four days. She made a complete recovery, but a week later began to have epileptic attacks involving the arm and face. These continued at brief intervals for three months and ultimately Dr. Frank Hartley operated on her and explored thoroughly the motor cortex, but no lesion was discovered. There was profuse hemorrhage from the pia, and the patient was completely hemiplegic for three or four days, but gradually recovered normal power. Subsequently the attacks of focal epilepsy returned.

Dr. C. K. Mills said that with regard to Jacksonian epilepsy and idiopathic epilepsy so-called, every neurologist knows that a species of Jacksonian epilepsy often occurs, so to speak, inside the general epileptic attack and it is of importance to be able to recognize this inside occurrence of Jacksonian attacks in deciding the question of operation. The reason for these focal epilepsies resides in the fact that a focus of irritation, some lesion, however minute, is present in the region of representation of the movements concerned in the spasm. This is by no means a new

idea. It is as old as the early statements of the great man for whom this Jacksonian epilepsy has been named. He was the first to point out not only the general features of Jacksonian epilepsy, but to call our attention to the fact that in many cases of generalized epilepsy the initial symptom or some part of the disseminated spasmodic manifestation, assumed a focal type. Jacksonian epilepsy, like other forms, does occur in renal disease, and it may occur in any toxic or infectious disease. It is the duty of the neurologist by his close study of the history of the case and of all its special manifestations to separate cases of such causation from cases due to gross lesions of the motor cortex.

Dr. Patrick said he agreed with Dr. Hoppe that there are cases clinically not to be distinguished from idiopathic epilepsy presenting the purely Jacksonian type. There was a time when Dr. Patrick thought that whenever he had a patient with a Jacksonian fit there was a gross lesion. He doesn't believe it now. He has seen enough cases followed a sufficient number of years to convince him that there are cases of idiopathic epilepsy which show the initial irritability in a way to make them Jacksonian: not only such fits as Dr. Hoppe described, but the little fits, such as tingling and contraction of the fingers of one hand. One of these patients Dr. Patrick followed for at least 10 or 12 years and he has made a practical recovery. These cases should be kept away from the surgeon. They are much better off with what they have than with what they are likely to get on the surgical table.

Regarding Dr. Mills's quotation of Dr. Jackson, Dr. Patrick said he would like to go one step further. Two years ago in conversation Dr. Jackson had said, "Of course I don't quite approve the name Jacksonian epilepsy, but, using it, I would say that every epilepsy is a Jacksonian epilepsy. That is, in every case there is a zone or group of cells which begins the discharge and from that group of cells as a focus the irritation or discharge or whatever it may be spreads with greater or lesser rapidity to the adjoining and surrounding brain tissue or cells."

Dr. Patrick had never looked at it exactly in that light, but the more he has thought of it the more convinced is he that it is a reasonable hypothesis. Every explosion of epilepsy is to be regarded as something starting in certain cells. And he also is inclined to believe another idea of Jackson's, that ordinarily these discharges begin in the smallest cells. Then the reason we have so little of the Jacksonian type in idiopathic cases is because the Jacksonian fit is a manifestation from the largest cortical cells and not from the small cells which are ordinarily the most susceptible.

Dr. Hoppe said that he believed with Dr. Clark that in these cases of so-called idiopathic epilepsy we may, perhaps, in all the cases find microscopic change. He does not believe in cases similar to those described in his paper we will find such a gross lesion as meningo-encephalitis.

As to bacteriology, Dr. Hoppe thinks it plays a great role but that it does not explain the focal lesion or a status hemiepilepticus. It may explain the convulsive character of the case, but not the focal character of the case. As to early operation, he believes in the cases described by Dr. Spiller it is always justifiable, but these are not the cases he described in his paper.

Dr. Dercum called attention to the fundamental difference in the development of these cases between focal organic Jacksonian epilepsy and the idiopathic. In the cases Dr. Hoppe has described as idiopathic epilepsy the clinical picture was complete from the beginning. There is gradual progression in the majority of cases of organic focal epilepsy; whereas

in idiopathic epilepsy the evolution and type of case is practically complete from the first attack. As to the statistics, up to 1899 the only statistics we had on resection of the cortex in idiopathic focal epilepsy were those by Matheolius, who said up to 1899 there was no case on record, in existence more than two years. Dr. Collins's case was on record the longest. The last statistics are by Nast-Kolb and they are very gratifying. He has collected 13 operations for focal idiopathic epilepsy and out of the 13 operations with resection of the cortex 5 have been well from 5 to 12 years. Dr. Hoppe stated that one man under his observation was free for eight years without any treatment whatsoever. There are some cases reported by Rasumowsky in which there has been entire freedom from convulsions and idiopathic epilepsy from 3 to 5 years. In gathering these statistics Dr. Hoppe has ruled out all traumatic cases and cases of resection of cortex where there was any evidence of organic lesion whatsoever.

SANE PROTOTYPES OF INSANE MENTAL PROCESSES.

By Theodore H. Kellogg, M.D.

Insanity is never a spontaneous mental variation but is always evolved on persistent lines of mental action. All insane mental processes have their correlative sane prototypes which have not been adequately studied. It is the object of this paper to traverse this promising field of psychic research, to juxtapose in comparison the insane mental processes with their sane prototypes in the following order, viz.:

I. Delusions of the sane as the causal prototypes of the false beliefs of the insane.

II. Extremes of conduct of the insane as uniformly modelled after sane exemplars.

III. Insane emotions, propensities, and impulses as directly derived from their sane prototypes.

IV. The hallucinations and illusions of the insane as a mere projection of the prototypic sensorial disorders of sanity.

Discussions.

Dr. H. R. Stedman said that tracing the analogy between the sane and insane states is always interesting and rather helpful. The only mental conditions which seemed to Dr. Stedman hard to trace are the perversions of instinct, such as the sexual perversions and the eating and drinking of loathsome things.

Dr. Carl D. Camp read a paper entitled: The Course of Sensory Impulses in the Spinal Cord. (To be published in this journal.)

Discussion.

Dr. Alfred R. Allen asked whether Dr. Camp considers there are any tactile fibers in Gowers's column, as held by some; also, whether he means that the axones and the cells forming Gowers's column contralateral to the starting point in the posterior horn, pass via the posterior commissure or the anterior white commissure.

Dr. Camp replied that he believes that there are no fibers conveying tactile sensations in Gowers's tract. As far as he could measure tactile sensation was tested with a pin.

THE EPILEPTIC VOICE SIGN.

By L. Pierce Clark, M.D.

Dr. Clark presented a paper on changes in the voice occurring in epilepsy, comprising a study of the subject with E. W. Scripture, after the latter's methods. The patient speaks into a mouth-piece connected with a small recording capsule that registers the vibrations on a rapidly revolving smoked drum or kymograph. On examining the records thus made and comparing them with records made by the normal voice, and with records made by voices of patients afflicted with other nervous and mental diseases (in which there is obvious voice defect), it was demonstrated that the voice records of the epileptic are distinctive and peculiar. On analysis it was shown that the melody proceeds by even steps or "plateau speech," as designated by Dr. Clark. There is a tendency to monotony. In the normal speech, Dr. Scripture has well shown, in his researches, that every vowel used has a rising and falling melody, or "unit circumflex," very differently recorded on the kymograph. This voice defect of the epileptic develops with the disease. The more severe the epilepsy and the longer its duration, the more marked the voice sign. The sign is an expression of the underlying brain degeneration. It occurs in patients not treated by bromide and is found in all idiopathic cases in varying degree. It can be detected plainly by the ear, and is of diagnostic and prognostic value.

THE EYE SYNDROME OF DEMENTIA PRÆCOX

By L. Pierce Clark, M.D.

It is well known that dementia præcox forms nearly one-fourth of all the asylum admissions and that next to paresis it has the most hopeless prognosis as regards cure. Kraepelin believes the vast majority of cases undergo an early mental death. Better prognosis ought to be obtained by a much earlier diagnosis. In most cases of dementia præcox it is possible to elicit a pre-asylum history of the disease for months or even years, during which the brain must be undergoing many irreparable alterations. In time it may be possible to make dementia præcox as much an extra-asylum disease as paresis is now. Many cases of the disease are successfully managed in their homes and out-clinics to-day.

The real pathogenesis of dementia præcox would seem to depend in no small degree on physical causes. The evidence of its autotoxic character has steadily increased since Morro advanced the theory in 1900. Thus many of the urine studies show very defective elimination, fully one-half of the subjects die of tuberculosis. Certain toxic dermatoses, such as erythemata and vaso-motor paresis, seen in chronic intestinal putrefaction, occur most frequently in dementia præcox. Since additional facts are at hand, as shown in the co-existence of psychic excesses such as neurasthenia and the like with states of autointoxication. Blood studies usually show a toxic state, especially in the katotonic form of this symptom-complex.

Three years ago Dr. Clark enlisted the interest of Dr. Tyson in the study of the significance of the ocular signs and symptoms of dementia præcox in any efforts to make an earlier diagnosis possible. At that time

no detailed observation had been made upon the eye. They examined 115 consecutive cases from private practice, out-clinics and the metropolitan asylums. Definite changes were invariably found in all the cases. The fundus changes as seen clinically were divided into three stages or types, in the usual order of their occurrence. The first stage was one of congestion of the disks, hyperemia and edema, dilated veins, contracted arteries and blurring of the edges of the disks, all of varying degrees, constituting a low grade of perineuritis of the optic nerve. The second stage was one of congestion of the nasal side with pallor of disks, dilated veins and contracted arteries. The third stage was pallor of disks, dilated veins and contracted arteries, constituting aneurism and partial or complete atrophy of the optic nerve. The other signs completing the syndrome are enlarged pupils, negative sensory, psychic, and Piltz-Westphal reflexes, diminished corneal sensibility and concentrically contracted visual field. No other psychoses presented this eye syndrome. All forms of dementia præcox were under study. The more rapid deteriorating form showed the syndrome more markedly. They believe a form of vascular toxin derived from the liver or gastrointestinal tract is the inciting factor in causing this psychosis. In many cases early and prompt treatment along hygienic lines brings about an arrest of the eye syndrome, but of course does not remove the damage already suffered. This fact is in accord with the arrest seen in the mental symptoms. The eye syndrome which they first studied ought to prove of use in the early differential diagnosis of dementia præcox and to a certain extent in prognosis of this psychic disorder. Finally, the study is a contribution to the autotoxic character of the psychosis.

DISCUSSION.

Dr. E. E. Southard, Boston, asked whether Dr. Clark regards the hypothetical brain change underlying the epilepsy as lying in the cortex or the medulla.

Dr. Clark, in reply, said that he believed the epileptic voice change depends upon cortical disease. The voice sign is only one other expression of the epileptic state, such as the awkwardness of gait and other disorders of motility. It is but a further contribution to rounding out the picture of the cortical disease entity of idiopathic epilepsy.

Dr. William G. Spiller read a paper entitled: The Symptom-Complex of Occlusion of the Posterior Inferior Cerebellar Artery: Two Cases with Necropsy. (See this journal, p. 365.)

Dr. Henry M. Thomas read a paper entitled: Thrombosis of Posterior Inferior Cerebellar Artery with Autopsy Revealing the Lesion in the Medulla.

Reference to the two cases reported to the Association in 1896.

Death in Case No. 2, two and a half years after onset, symptoms having remained stationary; autopsy refused.

Case No. 3, man aged fifty-nine; long neurotic history. About one week before admission to the hospital, intense giddiness, pain in left side of face, difficulty in talking and swallowing, drooping of left eye, and weakness of left face. Examination showed an extremely ill man, with marked vascular, cardiac and renal involvement. Sensorium dull. Constant hiccough. Left pupil smaller than right. Slight left ptosis. Slight divergent squint of left eye. Dissociated sensory disturbance on left face, right arm, and leg. No sweating of left side of face. Death in four days. Autopsy, among much else, revealed thrombosis of left posterior inferior

cerebellar artery. Microscopical examination showed softened areas in lateral aspect of left side of medulla. No secondary degeneration could be determined. Reference to other clinical cases, and some remarks as to cerebral sympathetic control.

These two papers were discussed jointly.

Discussion.

Dr. B. Sachs said he had been interested for some time in collecting in his neurological service unusual forms of apoplexy and among these he has had particularly one case in which the diagnosis of thrombosis of the inferior cerebellar artery was made, which had been presented by Dr. Abrahamson before the New York Neurological Society a few months ago. First of all since we have heard of autopsies in these cases he wished to state that some of the cases suffering from these symptoms do survive the attack. The patient of whom Dr. Sachs spoke is in a much better condition than he was a few months ago. He has practically recovered from the attack of unquestionable thrombosis of the inferior cerebellar artery. The agreement in the symptoms of this case with those of Dr. Thomas' and Dr. Spiller's cases was very remarkable. In a man, 44 years of age, the attack came on without loss of consciousness, speech became slow and difficult, there was marked hoarseness, much mucus in the throat, and difficulty in deglutition. In the course of the illness other symptoms were noted: Anhydrosis of the left side of the face; herpes of trigeminal branches; weakness of the right half of body; ptosis of left lid; no hemianopsia. Dr. Sachs had the eye grounds examined at various times, and each examination showed slight optic neuritis, slight perivasculitis. Changes were probably due only to vascular disturbance; there was no suspicion at any time of any massive lesion, and such slight changes in the optic nerve one often sees after apoplectic seizures; the palpebral fissure on the left side was smaller, and there was distinct enophthalmos. This patient was unable to stand or walk alone, and staggered and fell to the left side on which this lesion was; the knee jerks were diminished, the left more than the right. The trifacial on the left side showed anesthesia and analgesia. There was paralysis of the vocal cord, and slight ataxia of the left hand. There was distinct hypertrophy of the heart with occasional intermittence of a beat or two. This was in association with nephritis. The atypical forms of apoplexy Dr. Sachs has seen have generally been in association with nephritis. There is increased tension. There is remarkable agreement in the symptom-complex, and recognition should be fairly early. The points on which diagnosis may be based are association of what we generally regard as cerebellar and medulla oblongata symptoms, with trigeminal or other cranial nerve symptoms coming on in acute or sub-acute fashion.

Dr. A. R. Allen said in reference to his case that Dr. Spiller referred to in his remarks, it is a case also that did not terminate fatally. The patient was a man 54 years of age, for 30 years he used tobacco and alcohol to great excess, and had half a dozen or a dozen drinks of whiskey or beer a day. Last November following two days of general malaise, he awakened one morning, having gone to bed in a fairly normal condition the night before, unable to phonate, choking when he tried to swallow, and having a regurgitation of liquid through the nose. An interesting point about this case was that four days later he developed seventh nerve symptoms, facial nerve palsy on the right side. There were no eye symptoms, and

the man slowly got better. He is now able to talk in a very hoarse whisper. If he tries hard phonation can be noticed. He told Dr. Allen, with a good deal of pride, that one night not very long ago he won a large jack pot and jumped up and said "O." He thinks, therefore, that his speech is coming back. A rather interesting thing about this case, and something that has attracted Dr. Allen's attention was a wide right palpebral fissure much wider than on the left. He might have been misled by that, except that he noticed the man was atypical in other parts of his body, and he asked for an old photograph, and was given one taken 10 years before, wherein the same condition of affairs prevailed. There was great wasting of the right trapezius muscle which had existed for a long time. Underneath his left clavicle there was quite a marked swelling that was pulsating in character.

Dr. Alfred Gordon said in reference to his case to which Dr. Spiller referred, the patient had a sudden loss of consciousness of several hours duration, which came on acutely. The patient presented symptoms referable to cranial nerves, he presented from the beginning hemiasynergia, had gross syringomyelic disturbances covering half of the body, including face and scalp. The patient presented on several occasions a rise of temperature, had difficulty in swallowing, particularly in bringing the jaws together. The reason Dr. Gordon diagnosed a hemorrhage in the medulla was because all the symptoms but one have almost entirely disappeared. What was striking about the case was the persistence of the syringomyelic disturbances on the opposite side. He saw the patient very recently and made a thorough examination and the only symptom that is present now is the syringomyelic phenomenon. While at the beginning the symptoms presented by the patient were in accord with the syndrome of thrombosis of the posterior inferior cerebellar artery it is difficult to accept entirely the diagnosis owing to the condition presented by the patient now.

Dr. Dana said that Dr. Thomas and others had secured for us a pretty definite syndrome, illustrating the softening of a small part of the medulla. While it represented correctly the results of a blocking up of the inferior cerebellar artery, it should not be forgotten that there are quite a number of other combinations of symptoms which were produced by either more extensive softenings, or by the plugging of other portions of the arterial supply of the medulla. Sometime ago Dr. Dana had gone over the subject of the pons and medullary lesions and it seemed to him that one could rather roughly divide the symptom groups into three kinds: those which characterize the medullary softening, the pons softening and the pons medullary softening. It should be remembered that taken altogether there are more cases of softening of pons and medulla together than pons and medulla separately. The softening which involves both the pons and medulla represents a very large and complicated clinical picture. He did not find that there was any very clear-cut picture which represented a medullary softening, for the symptoms differ so much, according to the degree of softening. The pons symptoms on the other hand, are fairly typical. Either we get more characteristic eye symptoms and always the dissociated symptoms so that forms a rather more definite group. The pons-medullary symptoms are very serious and complicated and usually are fatal.

Dr. Theodore Diller said that two years ago Dr. Walker and he made a study of apoplexies in unusual situations. Among other situations they located one apoplexy in the pons and another in the cuneus. This second

case was that of a priest. One day while standing in front of a door watching the children at play, he was seized with a certain amount of dizziness and mental confusion but did not fall or lose consciousness and continued at his work; and he found that he was not able to see objects to the right and supposed he was blind in the right eye. He suffered no other inconvenience, but examination showed the presence of homonymous hemianopsia, which persisted.

Dr. Diller also related a case of hemorrhage into the pons which he was able to diagnose ante mortem only a short time ago on the teaching that Dr. Dana has laid down. The patient suffered a hemiplegia and had very small pupils and high temperature. The autopsy showed a very large hemorrhage over the region of the pons.

Coming to the subject immediately under discussion, he has seen three or four cases of the sort described to-day. In one patient that he saw only recently, the symptoms corresponded pretty closely to those related by Dr. Spiller and Dr. Thomas. The case was that of a large, burly colored man, about 45 or 50 years of age, who was at work doing plastering one day; and he was suddenly seized with great dizziness and vomiting and some headache. But he did not lose consciousness . He was brought to the hospital. He remained there for a number of weeks and the vertigo and uncertainty in gait persisted as very constant symptoms. The patient complained piteously of them and was incapacitated for labor by them. There was also a fifth nerve involvement.

Dr. Charles E. Beevor said that as far as he knew they do not come across cases in England giving the exact symptoms described by the two readers of the papers. He has never come across an exactly similar case. He had a case of loss of sensation of the fifth and crossed sensation of the rest of the body and limbs. The patient did not die and the findings could not be verified. He has seen a case of paralysis of the soft palate and inability to swallow and that case also recovered. He was much interested in regard to the question of the soft palate because years ago when he was a student he was taught that the soft palate was supplied by the facial nerve. Some years ago with Sir Victor Horsley he stimulated the cranial nerves in the monkey and found no movement of the soft palate from the facial nerve, but got it by stimulation of the accessory to the vagus. Therefore it was interesting to have their findings confirmed by clinical work.

In regard to the injection of the arteries he was glad to hear of the injection of the cerebellar arteries. He left these out. He thought five arteries enough to deal with at one time and therefore to exclude further difficulty he purposely excluded the cerebellar supply.

Dr. D. J. McCarthy, Philadelphia, thought that what Dr. Thomas called attention to should be borne in mind, the wide variation in the branches given off by the posterior portion of the circle of Willis. In fifteen brains recently examined four showed anomalies of the circle of Willis. Another factor is that next to the middle cerebral, the basilar and the branches coming off from the basilar are most frequently affected by thrombotic conditions.

Dr. Dana called attention to the fact that it is not only one group of symptoms but varied groups of symptoms which result from thrombotic obstruction of the pons and medulla. In a case in which the lesion of the medulla was produced by an obstruction of one of the branches high up, from the anterior spinal artery, the only symptoms produced were a com-

plete quadraplegia with complete loss of muscular sense, but without loss of sensation. There was involvement of the median lemniscus on that side. As far as he could determine secondary degeneration did not go above the thalamus.

Dr. Spiller, in closing, said that headache was not prominent in his two cases. In some of the cases reported in the literature pain was present in the areas of disturbed sensation. The vertebral artery is involved in a certain number of these cases. If the thrombus extends into the basilar artery the clinical picture is different and this fact should be borne in mind. In Dr. Spiller's second case the left vertebral artery was thrombotic and much larger than the right. It is difficult when a patient dies soon after the symptoms develop to determine whether the thrombus was formed before death, at the time of death, or a little later. There are not likely to be distinct symptoms from involvement of the cerebellum. The posterior inferior cerebellar artery is often absent on the right side as Dr. Spiller has observed in a number of examinations. Unquestionably recovery occurs. Dr. McCarthy's case Dr. Spiller studied during life; it presented an entirely different symptom complex and was the complement of the symptom-complex he described, inasmuch as another vascular distribution was affected, causing involvement of the anterior pyramids and the lemniscus. If we bear in mind the symptom group of occlusion of the posterior inferior cerebellar artery we can make the diagnosis without difficulty, so striking is it. A man without complaining previously, is suddenly attacked, he may become unconscious but usually does not, has no paralysis or at most temporary paralysis of the limbs of one side, is unable to swallow from that moment, speaks only in a whisper and may have syringomyelic disturbances in the face on the side of the lesion and in the limbs of the opposite side. If the descending root of the fifth nerve escape there will be fewer sensory symptoms. We need not hesitate to make a diagnosis with such symptoms.

Dr. Thomas agreed with Dr. Spiller's remarks; he thought when one could collect both with autopsy and without some 30 cases which show this sharp-cut symptomatology the entity is real, and he would be much obliged if Dr. Dana would give references in which the artery was occluded with diffuse areas of softening.

(*To be continued*)

Periscope

Review of Neurology and Psychiatry

(Vol. V. No. 8)

1. The Pathology of Epidemic Cerebrospinal Meningitis. STUART McDONALD.
2. A Case of Narcolepsy. W. R. GOWERS.
3. Landry's Paralysis. A. L. SHEPPARD and I. W. HALL.

1. *Continued article.*

2. *A Case of Narcolepsy.*—Gowers cites a case of a girl of 22 who had suffered since the age of 16 from peculiar attacks of sleep lasting five minutes or longer, seldom a quarter of an hour. Sleep began after about a minute of yawning and heaviness of the eyes, and was accompanied by vivid dreams, and during it she often talked to herself. She was easily awakened by being spoken to. No headache followed the attacks. She was in perfect health, and slept well at night. Caffein kept her free from attacks.

3. *Landry's Paralysis.*—The case described was a lad of 17 employed as a packer in an oil and color works. The onset of symptoms in both upper and lower extremities was coincident. Tingling occurred, in fact, in the hands before the feet were affected. There was an excessive pressure of the cerebro-spinal fluid. The blood showed an increase of red and white cells and decrease of eosinophiles. No source of infection was found; but a streptococcus, absent from the blood-stream, was present in the cerebro-spinal fluid. A post mortem was performed and a condition of intense congestion of the cord was found with chromatolysis and vacuolation of the cornual cells.

C. E. ATWOOD (New York).

(Vol. V. No. 9)

1. Observations on the Treatment of General Paralysis and Tabes Dorsalis by Vaccines and Anti-Sera. W. FORD ROBERTSON and DOUGLAS M'RAE.
2. The Pathology of Epidemic Cerebro-Spinal Meningitis (*continued*). STUART McDONALD.

1. *Treatment of Parcsis.*—The treatment advocated is based on the theory of the existence of a "threading diphtheroid bacillus" as a cause of general paralysis. Thirty-four cases of general paralysis were given antisera, and two cases of tabes. Reactions were obtained in all. Statistics are given of marked improvement in eighteen cases of general paralysis, and of three who became progressively worse. The pains and ataxia of the tabetic cases treated were reduced.

2. *Epidemic Cerebro-Spinal Meningitis.*—Dr. McDonald in his conclusions states that direct transmission of the disease from patient to patient is rare; that the disease is more or less constantly present, the cause of epidemics being still undetermined; that there exists a marked variation in the virulence of the meningococcus separated from the living tissues, as tested by experimental inoculation in the lower animals. The differences clinically between posterior basal meningitis and its pathological counterpart, acute cerebro-spinal meningitis, is due to this varying virulence on the part of the organism. The organism reaches the central nervous system *via* lymphatic channels from the upper respiratory passages. Pneumonic complications and gastro-intestinal types occur. Besides the obvious meningeal lesions, the general toxemia present brings about grave histological changes in both brain and cord, and in other internal organs. The essential toxine being endo-cellular renders serum therapy impossible.

C. E. Atwood (New York).

Journal de Neurologie

(Vol. XI. 1906. No. 20)

1. *Graphic Stereotypy in a Precocious Dement.* L. Marchand.

Account of a case of dementia præcox, in which stereotypy in construction and expression, was very apparent in the numerous letters written by the patient, all of which showed a monotonous reiteration of the same ideas of persecution.

(Vol. XI. 1906. No. 21)

1. *A Case of Complete Unilateral Ophthalmoplegia with Blindness on the Same Side.* Dr. Bouchaud.

A man of 40 years of age developed a slowly increasing loss of power in the muscles of the right eye, had occasional convulsive seizures, loss of sight and severe pains in the occiput and on the right side of the head, especially over the region supplied by the right supraorbital nerve, there being great tenderness at the point of exit of this nerve from the supraorbital foramen. When he came under the observation of the author, there was complete paralysis of all the muscles, both intrinsic and extrinsic, of the right eye, with complete blindness in this eye while the left eye remained normal. The right pupil failed to react from illumination either of this eye or of the left eye. At this time, ophthalmoscopic examinations showed no change in the papilla. Under antiluetic therapy, the pains and the paralysis disappeared, the right pupil regained the power of contracting for light when the left eye was illuminated, and of reacting when both eyes accommodated, but the blindness persisted, and optic atrophy had developed and become marked.

Considering the seat and character of the lesion responsible for the symptoms, the author concludes that it was a meningitis at the base of the brain, in the anterior part of the middle fossa of the skull near the cavernous sinus, and though no history of syphilis could be obtained the result would seem to indicate its luetic origin. Consulting the literature he can only find two cases of the kind, reported respectively by Hutchinson and by Tacke.

2. *Application of the Mental Tests of Binet to Children in the Schools of Gand.* Dr. Dupureux.

An account of the results obtained by the application of Binet's methods to 32 girls ranging from 6 to 12 years of age. Unsuitable for abstraction.

(Vol. XI. 1906. No. 22)

1. *Acute Mental Confusion and its Peculiarities among Russian Soldiers.* S. Soukhanoff.

The author describes this condition, which he considers identical with Meynert's amentia as he encountered it among Russian soldiers returned from the Japanese war. Among his cases he found stupor, depressive and delirious manifestations but never a maniacal form. The inhibition seemed to affect more the psychical than the physical sphere, and a good many of the patients though much confused could carry out light tasks, not demanding much mental effort. The delusions were mainly unsystematized, vague and of persecutory character. In two of these patients he notes the occurrence at times of certain rhythmical movements and laughing with a sad countenance, which would doubtless be considered as manifestations of stereotypy by the adherents of the Kraepelin classification, and probably some of these cases would be by them considered as examples of dementia præcox.

2. *Lumbar Puncture and Cytodiagnosis, Importance of Counting.* L. Laruelle.

The author urges the necessity of counting the cells in every suspicious case, in which, examination after centrifugation shows only a small number of cells, and gives two illustrative cases. In his opinion there is a pathological reaction, if enumeration several times repeated gives an average of over five leucocytes to the cubic millimeter. He uses the counter of Fuchs and Rosenthal.

C. L. Allen (Los Angeles).

Neurologisches Centralblatt

(Vol. 27. February 1, 1908)

1. Pseudo-Hysteric Hemiplegia. A. Adamkiewicz.
2. Concerning the Eye Movements Caused by Irritation of the Cerebellum. A. Lowrie.
3. Contribution to the Sacral Form of Multiple Sclerosis and Dissociated Disturbance of Sexual Power. H. Curschmann.
4. Sacral Form of Multiple Sclerosis. K. Mendel.

1. *Pseudo-hysteric Hemiplegia.*—A case of hemiplegia is reported having most of the characteristics of a hysterical condition in a woman of 50 years, left arm and leg completely paralyzed, face not involved, complete left hemi-anesthesia, no hemianopsia, onset without unconsciousness following mental excitement. The diagnosis of vascular lesion was made by means of sinapisms applied to anesthetic side. This caused no transference of anesthesia to sound side in corresponding area, which the author claims takes place in hysteria.

2. *Eye Movements in Cerebellum.*—The author has made numerous experiments on dogs in which one side of the cerebellum was entirely

exposed, to determine whether it contains centers controlling the movements of the eyes and face. By electrical irritation very slow movements of the eyes were produced in various directions according to the position of the electrode. He thinks, however, that these movements were due to stimulation of the corresponding nuclei in the brain stem beneath the points stimulated. Nystagmus, strabismus, exophthalmos, or similar eye symptoms were never observed.

3. *Sacral Multiple Sclerosis.*—Curschmann reports a case of multiple sclerosis in which symptoms point to involvement of the conus; diminution in Achilles reflexes, bilateral Babinski's sign, sensory disturbances in the sacral region, perineum and scrotum with partial incontinence of urine and feces. Sexual power: desire nearly normal, erection incomplete, ejaculation slow, orgasm absent. The author has observed two similar cases and refers to two in literature. He considers the libido due to cerebral actions; erection and ejaculation depending upon sympathetic ganglia, and the orgasm upon the intact posterior gray matter of the cord.

4. *Sacral Multiple Sclerosis.*—Two cases of multiple sclerosis in females are reported in which, in addition to symptoms referable to lesions of the conus, the following appeared—active patellar and Achilles reflexes, Babinski's sign, and absence of abdominal reflex, also in case one nystagmus and in case two slight paling of temporal sides of optic nerve discs.

(Vol. 27. February 15, 1908)

1. Concerning the Micro-sympathetic Hypo-spinal Ganglia. MARINESCO and MINEA.
2. Cerebellar Hemorrhage. E. SCHROEDER.
3. A Binocular Pupillometer. KRUSIUS.
4. Anatomical Findings in the Spinal Cord and Nerves of a Morphinist. O. SCHÜTZ.
5. The Changes of the Central Nervous System Appearing after Adrenalin Administration. R. SHIMA.

1. *Hypospinal Ganglion.*—The authors describe minute sympathetic ganglia found in the neighborhood of the subganglionic portion of the spinal nerves close to the corresponding spinal ganglia. They were commonly found immediately outside or beneath the spinal ganglia and never above or in the intra-arachnoid portion of the spinal root. Sometimes one and sometimes several in a group were found and their size appeared to be in inverse proportion to their number; the largest being about the size of a millet seed. The form was spherical or oblong and they were connected by rami communicantes with the subganglionic portion of the nerve. Microscopically they are composed of cells similar to those found in the sympathetic ganglia and are divided into three types. (1) Cells with short dendrites which end in the cell capsule. (2) Cells with long dendrites which may be traced for a considerable distance and subdividing in their course. (3) Cells of a type representing the transition between the two former types and less numerous. The course of the fibers from these ganglia and their function is unknown.

2. *Cerebellar Hemorrhage.*—Reports of a case of cerebellar hemorrhage in a senile dement of 86 years. Patient suddenly became comatose and cyanotic, no vomiting, no convulsions, right side of face drooped, other paralysis not demonstrated on account of coma, no localizing symptoms. Death occurred one hour later. Necropsy showed a large

hemorrhage in the right cerebellar hemisphere and a small one in the left.

3. *Pupillometer.*—The author describes an instrument for studying the size, form and reaction of the pupils.

4. *Morphine Changes.*—A case with necropsy is reported which was considered one of morphin poisoning for lack of other etiological factor. Degeneration shown by Marchi method was present in the posterior columns and direct cerebellar tracts most extensive in the cervical region. The anterior and posterior nerve roots of different levels were degenerated. Root entrance zones unaffected. Gray matter normal. Proliferation of the glia in small spots in the cervical region. No signs of inflammation. Degeneration of peripheral nerves of left hand.

5. *Adrenalin Poisoning.*—Experiments with young animals which were injected with adrenalin daily or every second day and examined after a few days to a month showed the following changes: (1) Shrinkage of nerve cells. (2) Vascular degeneration and thickening of walls. Peri-vascular round cell accumulation and hemorrhage. (3) Increase of ependyma of ventricles. (4) Pia shows productive inflammation in spots under which glia is proliferated.

S. D. INGHAM (Philadelphia).

Revue de Psychiatrie et de Psychologie Experimentale

(May, 1907)

1. Two Asylums for the Criminal Insane. H. COLIN.
2. The Delirium of Persecution Occurring at the Involution Period of Life. L. MARCHAND.
3. Carbonic Acid Baths for the Insane. M. BELLSTRUD.

1. *Asylums for Criminal Insane.*—The article is a plea for special hospitals for the criminal insane. He takes his text from a visit to two German institutions—Düren and Bruchsal. He sees nothing in these institutions to copy—they are too prison like. He does not believe asylums for the criminal insane should be attached to prisons but should be absolutely independent establishments and says that the only countries which have satisfactorily met this problem are England with Broadmoor, and the United States with Matteawan and Dannemora.

2. *Delirium of Persecution in Period of Involution.*—The author thinks that in the realm of mental disease not enough stress has been laid on the influence of age conditions on the type of reaction: Not as much stress for example as in general medicine. He cites three cases in detail of delirium of persecution occurring at the ages of 63, 64 and 67 respectively. In these cases the prodromal period was short, the delirium of rapid development, ideas of megalomanic type soon followed and were mobile and fugitive. They presented psycho-motor hallucinations, doubling of the personality, and neologisms suggesting the psychosis of adolescence. They did not react as paranoiacs do to their persecutions but endured their sufferings and committed no acts of violence, and they differed also from them in presenting enfeeblement of memory at the beginning of the psychosis. The prognosis is grave. These symptoms present an entirely different picture from that of involution melancholia, presenile delusional insanity, or senile dementia, the three psychoses forming the group of involution psychoses of Kraepelin.

3. *Carbonic Acid Baths.*—A series of fifteen observations of the effect

of carbonic acid baths in different types of insanity and a description of the method of giving them. The baths seemed to influence favorably the circulation, the sleep, and especially conditions of hyperesthesia.

(June, 1907)

1. Acquired Idiocy and Dementia in Epilepsy. L. MARCHAND.
2. Latent or Spontaneous Idea of Suicide in a Case of Confusion. H. DAMAYE.

1. *Idiocy and Dementia in Epilepsy.*—The author concludes: Epileptic dementia is a profound intellectual enfeeblement, irremediable, which supervenes in a certain number of epileptics. The moiety of epileptic dements have hereditarily burdened antecedents. Epileptic dementia may supervene at all ages of life, but it appears with a certain predilection at the time of adolescence. As among epileptics in general, one finds, in the personal antecedents of epileptic dementia, infantile convulsions, grave infectious maladies. Supervening before puberty epileptic dementia takes the form of idiocy or imbecility. Supervening at puberty or during adolescence it takes the form of dementia præcox. When the motor disturbances are accentuated it is characterized by two special forms: spasmodic epileptic dementia, and epileptic dementia of the form of pseudo-general paralysis. Epilepsy supervening in the adult and the aged can be accompanied by dementia; in this case one does not give to the patients the designation of epileptic dements; the epileptic attacks and the dementia accompanying other troubles which permit specifying the cerebral lesions that determine the syndrome. Chronic meningo-encephalitis and chronic meningo-corticalitis are the cerebral conditions that one encounters most frequently among epileptic dements, but all extensive and diffuse lesions can determine epilepsy and dementia. In the adult alcoholism, syphilis and tuberculosis frequently determine the meningo-cortical lesions which show themselves clinically by dementia and epilepsy; cerebral tumors and cerebral arterio-sclerosis are equally a frequent cause of epileptic dementia. Epilepsy and dementia can be early symptoms of general paralysis and one can say that there exists a symptomatic epileptic dementia of diffuse, sub-acute meningo-encephalitis.

2. *Suicide Idea in Confusion.*—The report of a case of suicide of a patient who never had expressed any suicidal ideas. The author thinks she may have taken this means to escape her persecutors.

(July, 1907)

1. Frequency and Pathogenesis of Fatal Ictus in General Paresis. A. VIGOROUX.
2. General Pathology of Nerve Conductors. G. DURANTE.

1. *Fatal Ictus in Paresis.*—There is great divergence of opinion both as to the pathogenesis of the apoplectiform and epileptiform attacks of paresis and as to the frequency with which they are a mode of termination of the disease. As to the pathogenesis all sorts of theories have been held, especially those of anemia, congestion and edema which, however, have not been confirmed at autopsy. The author believes the attacks to be of toxic-infectious origin. This is the theory that is coming more and more to the front and has been accepted largely in idiopathic epilepsy, eclampsia, uremic convulsions, senile and late epilepsy, the ictus of dementia præcox, etc. Two conditions are necessary: (1) The existence of hyperexcitability of the motor cortex—the predisposing cause. (2) The

action of toxins at the hyperexcitable zone—the determining cause. The meningo-encephalitis accounts for the increased excitability of the motor cortex and supplies the predisposing cause, the toxins may arise from various sources. In forty-seven cases studied in which death was due to ictus, in nine there was meningeal hemorrhage, in two cerebral softening and in one, cerebral hemorrhage. Eliminating these twelve cases the following thirty-five showed the following conditions: Eleven, infections from the alimentary canal; five, uremia; one, diabetes; two, infections of the urinary tract; eight, acute infections of the pleura and lungs; and eight cases in which neither intoxication nor infection was demonstrable either clinically or at autopsy. In conclusion—ictus is a mode of termination in about one third of the cases of paresis. It cannot be considered as an habitual mode of termination, it is rather a more or less grave complication avoidable so far as it is possible to prevent by rational hygiene chances of infection and auto-intoxication.

2. *Pathology of Nerve Conductors.*—A somewhat extensive article dealing with biology, embryology, anatomy, physiology and pathology of nerves and concluding with certain medical and surgical inferences based thereon. The article is so condensed and treats of so many aspects of the subject that it does not lend itself to ready abstraction. The author repudiates the neurone doctrine and tries to explain certain medical and surgical conditions that appear incongruous on a new supposition that the prolongations of the cell are formed of chains of neuroblasts united at their extremities but preserving a certain autonomy.

W. A. WHITE (Washington).

Revue Neurologique

(Vol. 15. No. 11. June 15, 1907)

1. Some Words Concerning the Work of M. Nageotte; Experimental Research on the Morphology of the Cells and Fibers of the Spinal Ganglia. M. G. MARINESCO.
2. Research in the Psychology of Aphasics, "Memory" in Aphasics. N. VASCHIDE.

1. *Spinal Ganglia.*—Marinesco claims priority for experiments in transplantation of ganglia. The phagocytosis of the degenerating cells is carried on by polynuclear leucocytes.

2. *Memory in Aphasia.*—Memory in aphasics is replaced by a sort of intuition, a "reconnaissance-souvenir" an intellectual phenomenon which implies the existence of a subconscious association of spontaneous ideas, a sort of automatic revival of mental images.

(Vol. 15. No. 12. June 30, 1907)

1. On the Existence of Peripheral Factors in the Genesis of the Pathologic Reflex of the Great Toe. L. BAID.
2. The Sense of Pressure (Baresthesia). MAX EGGER.

1. *Toe Reflexes.*—In some cases of injury to the foot or leg followed by deformity and consequent change in the automatism of walking the reflex movements of the toes may be reversed also in cases where another automatic movement of the feet is developed by long use, such as moving a sewing machine by a treadle. The author cites cases of each sort.

2. *Pressure Sense.*—The author finds that contrary to the views of

Marinesco the sense of pressure is very different from osseus sensation that is best developed over soft parts such as muscles and better over relaxed than over contracted muscles. It is superficial sense, the physiological organ being the skin, and is a total of the sensation of direct contact and of traction on surrounding skin. It is conducted by way of the posterior columns of the spinal cord. Deep tissue such as muscle, tendon, etc., are only sensible to very deep pressure, causing a sensation which approaches pain.

(Vol. 15. No. 13. July 15, 1907)

1. Unilateral Paralysis of Multiple Cranial Nerves. P. LAJONNE and ED. OPPERT.

1. *Cranial Polyneuritis.*—There was complete left facial paralysis. The left fifth nerve was affected in both motor and sensory functions and the left twelfth was affected. Complete recovery by electrical treatment. The diagnosis was a polyneuritis, non-specific.

(Vol. 15. No. 14. July 30, 1907)

1. Hysteria and Trophic Troubles, Simulation. BRISSAUD and SICARD.
2. Musical Amnesia in a Sensory Aphasic, Formerly a Professor on the Violin and Piano. Preservation of Execution of Improvisation and Composition. HENRI LAMY.
3. Spinal Cord Lesions of Zona (Secondary Degeneration—Reaction à Distance, Medullary Congestion and Hemorrhage. ANDRE-THOMAS and LAMINIÈRE.

1. *Trophic Hysterical Disturbances.*—Hysterical-trophic disturbances so-called are the results of simulation.

2. *Musical Amnesia.*—The patient had jargon aphasia and jargon agraphia. He was amnesic to music completely, not remembering or recognizing familiar pieces but he was not music deaf and he preserved a perfect memory for the signs of written music, the technique and the fingering, which the author attributes to their being on a lower plane than the memories of polyphonique sonorities necessary to the remembrance of musical composition.

3. *Zona.*—A report of two cases of Zona, one affecting the cutaneous distribution of the eighth dorsal and the other the ninth dorsal spinal root. The respective roots were found degenerated and the rami communicantes of the sympathetic took part in the process. Within the cord there was found ascending degeneration in the posterior columns corresponding to the position of the fibers of the nerve roots affected. There was found also slight degeneration in the gray matter in the same segment, also a descending degeneration in the posterior column in the tract of Schultz which the author considers proof that the tract of Schultz is composed of descending fibers from the posterior roots. In the eighth dorsal the descending degeneration extended to three segments. In the other case it extended one and a half segments below the ninth dorsal. The gray matter of the affected side was deformed and contained hemorrhages and the cells of the intermedio-lateral tract at that level showed changes indicating the reaction à distance indicating the involvement of the sympathetic system. The author considers that zona is due not only to inflammation of the root ganglia but also the adjacent sympathetic.

(Vol. 15. No. 15. August 15, 1907.)

1. Apropos of Tactile Agnosia. D. DEJERINE.
2. Anatomic Pathological Study of the Localisation of the Motor Cortex, Apropos of Three cases of Amyotrophic Lateral Sclerosis with Degeneration of the Pyramidal Tract Traced by the Marchi Method from the Spinal Cord to the Cortex. ITALO ROSSI and GUSTAVE ROUSSY.

1. *Tactile Agnosia.*—The recognition of an object is due to two factors: First, the integrity of the sensory path which transmits the impressions to the cortex and second, the memory of the object. Recognition being an association of memories brought about by sensory stimuli. Max Egger reported two cases of agnosia (*Revue Neurologique,* May 15, 1907) and regarded the phenomenon in each case as due solely to defect in association. Dejerine disagrees with this opinion holding that the enlargement of the circles of Weber, the only sensory defect found by Egger in his cases, is quite sufficient to account for the failure to recognize objects placed in the hands. Dejerine states that he has found stereognostic disturbances in ataxics who presented sensory disturbances, particularly the enlargement of the circles of Weber.

2. *Amyotrophic Lateral Sclerosis.*—Three cases of amyotrophic lateral sclerosis are reported with full clinical histories and the anatomicopathologic findings characteristic of the disease. The author calls attention to the presence of recent degeneration in the posterior longitudinal bundle, and in the corpus callosum. The degeneration in the pyramidal tract was traced to the cortex by the Marchi method and it was found that the ascending frontal convolution and the anterior part of the paracentral convolution contained many degenerated fibers whereas the ascending parietal and the posterior portion of the paracentral were very slightly affected, thus confirming the previous researches that the motor zone is almost entirely in front of the Rolandic fissure.

C. D. CAMP (Ann Arbor, Mich.).

Journal de Psychologie Normale et Pathologique

(Fifth year. No. 1. January–February, 1908)

1. Responsibility of Criminals (reply to Dr. Grasset). G. BALLET.
2. What is Pathological Psychology? G. DUMAS.
3. Principles of Scientific Physiognomy. P. HARTENBERG.

1. *Responsibility of Criminals.*—This is a reply to the article of Grasset upon the same subject in the last number of the JOURNAL. It is, therefore, largely argumentative, critical and indicative of differences in the use of terms. Ballet maintains that as yet no argument has been advanced by any one to cause him to change the position he took before the Geneva Congress. He says he agrees heartily with Grasset that society has the right to protect itself and to legislate in regard to all forms of dementia. He takes exception, however, to the loose conceptions which are involved in the use of such terms as "madness" and "half madness." These he insists are words belonging to lay medicine and popular psychiatry. They are utterly devoid of scientific accuracy and value. He calls upon Grasset to give a definite description of what he means by "medical responsibility." Is it a function of a psychophysiological sort, demonstrable by scientific means in a laboratory?

If there is *actual disease* present, a veritable histo-pathological state, Ballet declares, the physician must alone, of course, determine the degree of the patient's responsibility; but the determination of this responsibility must be made upon the bases of the disease and not upon anything else. This would be, however, a pathological responsibility and not a physiological medical responsibility, such as Grasset has invented. Like many others, Ballet in his reply to Grasset seems to assume that there is a sharp line of separation between health and disease, between what is physiological and pathological. He argues that not only would it be dangerous to society to submit to the magistracy the determination of a partial non-pathological responsibility in a given individual, but that the very term "physiological medical responsibility" will ere long join the other loose phrases that have from time to time been dropped for want of intelligible meaning.

2. *What is Pathological Psychology?*—Dumas holds that clinical psychiatry is something quite different from pathological psychology. In the former, the physician aims merely at establishing the origin, course, and treatment of a particular malady. This the author illustrates in the clinical analysis of tabes dorsalis and general paresis, such as it is given in the average text-book. He shows that a synthesis is really made from the clinical presentations and that upon these a specific clinical type or disease is established. Beyond the establishment of this clinical type or disease psychiatry does not go. Pathological psychology, on the other hand, according to the author, adopts a process of examination that is more distinctively and truly analytical. This he illustrated in a psychological analysis of some of the more prominent mental presentations of general paresis, melancholia and other forms of alienation. Each mental manifestation is subjected, by itself and in conjunction with the others, to a minute study so as to discover, if possible, the origin of it, the reason for its existence, and the relationship it may bear to the patient's past and present modes of mentalization and environmental influences.

3. *The Principles of Scientific Physiognomy.*—This is the opening chapter of a forthcoming work upon *Physiognomy and Character*, by Hartenberg. It discusses, in rather an elementary way, the interrelationship of mind and body.

METTLER (Chicago).

American Journal of Insanity

(Vol. LXIV. No. 3. 1908)

1. Report of Twenty-seven Cases of Chronic Progressive Chorea. ARTHUR S. HAMILTON.
2. Technical Aspects of Experimental Psychopathology. L. WELLS.
3. Cyst of Dura Mater occupying the Left Middle Cranial Fossa, associated with Anomalous Development of the Left Superior Temporal Gyrus. J. B. AYER, JR.
4. Some Origins in Psychiatry. C. B. FARRAR.
5. Arteriosclerosis in Relation to Mental Disease. C. MACFIE CAMPBELL.

1. *Chronic Progressive Chorea.*—These cases consisted of 13 males and 14 females, and varied in age from 19 to 84 years. In 24 of the 27 cases there was a history of chorea in the immediate relatives; in the remaining 3 cases no history could be obtained. The author has

not been able to differentiate between senile and hereditary chorea and regards these conditions as essentially the same. Several of his cases had originally been diagnosed as senile chorea but investigation later unearthed hereditary predisposition. Ordinary nervous and mental diseases he found rather conspicuous by their absence in these cases. Hereditary occurrence is interestingly exposed in two charts showing the presence of the disease through five generations in one case and through three in another. It appears capable of transmission through either male or female line. Trauma, fright and infectious disease seemed to be possible etiological factors in some of the cases. The author could find among his patients no evidence of descent from the Long Island families in which the original cases of Huntington occurred, nor was it confined to those well on in life. Some of the cases presented stigmata of degeneration. There was complaint of muscular fatigue in some of the cases, but in others, notably in some in whom the movements were violent and lasted all day and a good part of the night, this was absent. The movements were not confined to any one portion of the body, nor could the author make out that they most usually began in any particular muscles. Respiration was frequently affected; in two instances there was irregularity of the heart and in one possibly of the movements of the stomach. Speech defect appeared in most of the cases though sometimes only when the disease was far advanced. In all these patients the movements were absent during deep sleep though not always during light slumber. Alluding to the statement of Vaschide and Vurpas that the movements cease entirely before death, of eight cases which the author observed, in four there was practically no change up to the end, once they were most violent on the day before death, once they disappeared from an infected arm some hours before death, once they were slight for two days previously and in one case which died in coma they had been absent for two days. In fifteen cases in which the tendon reflexes were studied in twelve they were increased. The patellar reflex was once absent, once decreased, and once normal. Pupillary reflexes were almost invariably normal or decreased. Ankle clonus was twice present, the Babinski reflex was never found. Well marked increase in muscular tonicity was found in nearly every well developed case in which it was sought for. Mental impairment was present in all the cases which the author has seen, and while there is some variability in its manifestation the characteristic picture is gradually increasing dementia, irritability and not infrequently delusions of persecution. Hallucinations and illusions the author found uncommon, while delusions were common. The dementia does not usually become extreme. The prognosis is entirely unfavorable.

2. *Experimental Psychopathology.*—Reviewing the more immediate problems of experimental psychology with especial reference to their application to pathological conditions, the author considers that the aim which may be set for the coöperation between psychiatry and experimental psychology is threefold. First, a better organization of the clinical tests with special reference to their comparability between individuals. Second, exhaustive study of the recognized clinical entities by methods more exact than ordinarily available for clinical tests, with a view to (*a*) developing methods for a more precise measurement of the effects of therapeutic agents, and (*b*) to ultimately determining quantitatively the groups into which the various psychoses separate. Third, the application of psychological experiment to a more intimate analysis of pathological mental processes with a view to gaining a clearer insight into the interpretations

which clinical observation has suggested. Considering the various methods which have been used for testing sensation, movement and the higher mental processes, as well as those for organic sensations and fatigue, which he criticizes from the point of view of a psychologist familiar with laboratory methods, he finds that all are more or less defective. He concludes that while conditions for work in this line are at present hardly in satisfactory condition, there is no reason to despair but closer coöperation between the trained clinician and the trained psychologist should cause some at least, of these difficulties to disappear. Especially must pathological psychology comprehend equally with normal psychology, the advantage of dealing with data in the mass.

3. *Cyst of Dura Mater.*—A man of 85 years having one insane brother, showed failing vision and hearing, loss of memory and inability to work. He was confused, had occasionally thickness of speech and several falls. There was marked sclerosis of arteries, and trouble at the lung bases, rather sudden death. The gross anatomical changes found were numerous, the chief being arteriosclerosis, chronic myocarditis, hypostatic pneumonia and cysts in the kidneys and in the intestinal wall. The brain showed chronic pachy- and leptomeningitis, edema of the cortex with pigmentation, with microscopically increase and pigmentation of the glia cells, some decrease in the number of Betz cells and vascular change. In the spinal cord there was some gliosis in the posterior columns and reduplication of the elastic membrane of the arteries. The interesting feature was however a cyst of the dura mater, probably congenital which occupied a considerable part of the left middle cranial fossa. Associated with this there was distortion of the left temporal lobe and considerable loss of substance of the anterior part of the superior temporal convolution presumably from pressure through the intervening tissue. The author could find in the patient's previous history no ground for suspecting a lesion of the left temporal lobe, and does not think that the contortion and hypoplasia of the temporal cortex which was found, can be directly connected with the deafness of four years standing, with which the patient was affected.

4. *Some Origins in Psychiatry.*—Continued article.

5. *Arteriosclerosis in Relation to Mental Disease.*—The author condenses into the following summary his discussion of the above subject: (1) While arteriosclerosis is a commonplace finding in mental disorder, especially in advanced life, there are cases in which the cardio-vascular trouble is the central element in the picture. (2) Certain cases symtomatologically described as melancholia, hypochondria, neurasthenia, etc., could better be etiologically grouped as arteriosclerotic brain disorder, not neglecting however the symptomatic factors. (3) In certain cases of organic dementia the arteriosclerotic changes are obviously the most important element in the process, and the term arteriosclerotic dementia is justifiable; but the relation of certain mental symptoms to similar ones in the presenile and senile psychoses is to be kept in mind. It is premature to correlate the whole symptomatology with the arteriosclerotic part of the findings. (4) The neurological picture in advanced cerebral arteriosclerosis is still ill defined; and for the differential diagnosis of the various organic dementias further clinical material is required. (5) In certain cases of epilepsy with onset late in life, the convulsions and general symptomatology are closely related to arteriosclerosis.

C. L. Allen (Los Angeles).

Allgemeine Zeitschrift für Psychiatrie

(Band 64. Heft 4. 1907.)

1. Acute Exfoliations of the Skin in General Paresis. THEODORE ZAHN.
2. Alcohol and Suicide. KURBITZ.
3. Skull Measurements and Calling. LOMER.
4. Association Tests as an Aid to Diagnosis. BOLTE.
5. Dementia Præcox and Manic-depressive Insanity. THOMSON.

1. *Acute Exfoliations of the Skin in General Paresis.*—Trophic disturbances of various kinds have been observed in general paresis and reported by many authors. As a contribution to the subject the author gives the histories of nine cases which came under observation at the Wurzburg Psychiatric Clinic, during a period of 11 years. Of these, seven were instances of acute exfoliative conditions which came on near the end of the disease, were accompanied by severe general disturbances and ended with the death of the patient. In two other cases similar troubles came on during the course of the disease, but were accompanied by little constitutional disturbance and eventually healed, though there was recurrence later. In all the cases the trouble began as a bullous eruption, usually suddenly, the blebs dried or ruptured and there was gradual healing or the production of a more or less deep ulcer, in either case with much exfoliation of skin. The condition could in no case be attributed to uncleanliness or to trauma and there was no direct evidence of infection. The lesions were in no case limited to a special nerve or segmental area.

In the spinal cords from some of the cases there was degeneration more or less marked in the posterior and lateral columns. The cells of the gray matter were not degenerated. In one severe case in which the lesion consisted in the development of blebs on both feet, the author found in the first right sacral ganglion decided changes consisting in hemorrhagic exudation, proliferation of the capsular epithelium, and paleness and indistinctness of the granulations of the nerve cells. The nerve roots both afferent and efferent showed perineuritis. The corresponding ganglion on the left side was lost but its nerve roots showed perineuritis. The fourth lumbar ganglion on the left showed slight hemorrhage. The author while he thinks these changes not without importance hesitates to attribute the lesions of the skin observed to them. He seems to feel more inclined to regard some not well made out infectious process as at the bottom of both troubles. He adds to his description of these cases reports of a case of herpes zoster and of one of sudden edema of the left leg, both in general paretics. It should be added that in the cases which he examined post-mortem, the peripheral nerves in no case showed change.

2. *Alcohol and Suicide.*—The author studies the mental condition of alcoholics who have attempted suicide, as illustrated in twenty cases from the Konigsberg Psychiatric Clinic. In all of these there was a history of heavy drinking, in some combined with mental or physical shock, in others acting upon a constitution herditarily defective. In nearly all the attempt was directly traceable to a condition of dread, resulting from hallucinations and illusions. In some cases there were epileptiform attacks. In conclusion the author considers the medico-legal aspects of these cases and urges that the authorities should take more immediate

cognizance of those instances in which an alcoholic is threatening his family or neighbors, to the end that the many tragedies which occur may be avoided. In Germany at any rate he thinks there are entirely too few institutions for the care and cure—when possible—of chronic alcoholics.

3. *Skull Measurements and Calling.*—The author made skull measurements on a material of 210 paupers, 382 insane patients, almost all chronic cases, and 58 insane criminals, all males. These people came almost exclusively from the lower walks of life and chiefly from the country. He divides them somewhat arbitrarily into the following classes, "workers, handworkers, peasants, merchants, officials, educated classes and no calling." He tabulates his results giving the average by classes and as to mental condition, of the circumference of the skull, average sum of the greatest transverse and antero-posterior arcs, average sum of the greatest transverse and greatest antero-posterior diameters. Consideration of the results shows that in head circumference the workers stand at the bottom of the scale. Next come handworkers, merchants, officials and educated people (of course under the last there are included only the lower class of officials, and of the educated classes only those who have been unable to keep up and have fallen to a lower stratum). Remarkable is the figure for head circumference among the peasants, the highest in the scale.

Another surprise is that the circumference of the head in the insane workman is on an average 0.6 cm. and in the criminal insane 1.0 cm. greater than in the normal persons. A similar difference of 1.4 cm is found among the handworkers.

He finds in general: (1) That among the insane the cranial measurements were greater than among the sane; that (2) in the class of persons studied, the lowest in the scale, the workingman, showed both sane and insane the lowest average measurements; (3) the skull measurements of the mentally sound peasants were markedly high; (4) the measurements in handworkers, merchants and small officials average about the same. The cranial indices ran from 78.95 for the insane peasants to 83.87 for the insane educated people. Only 162 of the 650 skulls measured showed the mesocephalic type, the rest were brachycephalic. Not one was dolichocephalic.

4. *Association Tests as an Aid to Diagnosis.*—That experimental psychological methods have so far not gained great popularity, as applied to the diagnosis of mental diseases, is due chiefly to the expensive apparatus and the time required in making the minute reaction time measurements which psychologists have insisted upon. With some practice and no more expensive an armentarium than a stop watch the author thinks very valuable information can be gained by testing association as to time and method of reaction, and urges that it should be applied as a routine procedure to each case coming under examination. He has not found Sommers' scheme satisfactory as a whole, and prefers one in which the test words are not arranged in special categories basing the one he uses mainly on that recommended by Jung. To gain a true basis of comparison he thinks that the same list of words should always be used and that the test should be made by the same individual in each case. He sums up the advantages of the method as follows: (1) It is a simple method of fixing in an objective manner many symptoms, especially the general bearing of the patient. (2) In clear and orderly thought the conception of a definite aim preponderates over all the more cloudy psycho-

physical tendencies. In aimless associative thinking on the contrary, the subconscious and indefinite elements of the mental life make themselves more strongly felt. The partial elimination of volition, and loosing of involuntary tendencies tend to bring out symptoms which would otherwise only be found later in the disease or not at all. (3) New symptoms of the disease may be found. (4) Testing of the association may throw some light upon the not yet satisfactorily solved question of intelligence testing. (5) Association testing affords some insight into the course of the disease while of course disturbances in association are not always proportional to the other symptoms. That the affective condition of the subject at the time may exert a powerful influence upon the association reaction is well known and of late it has been proposed to utilize this in the examination of criminals.

According to Jung, as soon as a psychical complex accentuated by an affect is touched upon by the stimulating word: (1) The reaction time is lengthened eventually with the irradiation of the disturbance to the following reaction. (2) It causes the subject to forget what he has already answered. (3) Some conceptions from the complex already touched upon in the course of the experiment force themselves in where they have no apparent connection, often against the will of the subject. (4) In critical positions "cover-reactions" such as citations, sentences, sound-associations ("klangassoziationen") occur. The study of the associations is recommended especially in the investigation of the neuroses. As illustrating this the author gives the results obtained by its application in several cases.

5. *Dementia Præcox and Manic-depressive Insanity.*—Charged with a review of the relations of these two forms of mental disease for the meeting of the Psychiatric Society of the Rhine Provinces, the author brings out the chief points in differential diagnosis. The distinction is important particularly from the point of view of prognosis. The author finds on the one hand that an absolute diagnosis is not always possible, at any rate at the start, and on the other he makes the point that the prognosis in dementia præcox is not necessarily entirely unfavorable, since some cases, at least, practically recover. Summing up he concludes. That the term dementia præcox since it leads often to wrong assumptions and conclusions in relation to prognosis might well disappear from our psychiatric nomenclature. Nevertheless it can not be disputed that we have in hebephrenia, katatonia and dementia paranoides clinical pictures of conditions whose external and internal relationship are shown especially in diagnostic and prognostic indications, provided we only place in this group the cases which begin in the first half of life. If we do this we have a clinical group which comprehends these three conditions. This group is characterized by a number of common symptoms which serve to separate it from the other psychoses of this period of life, especially from manic-depressive insanity. The more marked the "katatonic symptom-complex" is, the more certainly any particular case would seem to belong to this group. The "Katatonia group" seems to the author a better name for these cases than dementia præcox. While compared to the manic-depressive group the prognosis in these cases is in general much less favorable, the author warns that for the prognosis in any one case we have no absolute criteria, hence advises caution in utterances on this subject.

ALLEN (Los Angeles).

METAMERIC DISTURBANCES OF SENSIBILITY. Benedict (Wiener klin. Wochenschrift, 1907, No. 3).

A man of 47 developed headache, dizziness, tinnitus, disturbances of speech, ptosis and analgesia of the left half of the body. An organic disorder of the right hemisphere, from lues, probably a thrombosis, seemed to be present. The chief interest in the case lay in the peculiar distribution of the analgesia, viz., the regions of the inferior branch of the trigeminus, the 1st to 3d dorsal, the 7th to 9th dorsal and the 4th lumbar zones. Therefore, there seems to be no doubt that hemianalgesia in lesions of the sensory centers may present an exquisite segmentation.

C. E. ATWOOD (New York).

EPILEPTIC STATE TREATED BY LUMBAR PUNCTURE. A. Pinchot et P. Casting (L'Encephale, 1907).

A case where the attacks were reduced from 166 in three days and a half to their normal of between 8 and 24 a month with some attacks of "petit mal." The tension of the liquid was very high at each of the five punctures made (with the exception of the last) within twenty-one days. The authors consider the procedure should be practiced, as in the cases of Babinski's voltaic vertigo, hydrocephalus, syphilitic headache and cerebral tumor.

TOM. A. WILLIAMS (Washington, D. C.).

Book Reviews

Popular-Psychiatrie des Sokrates. Von Dr. H. Schafer. Oberartz a. d. der Irrenanstalt Freidrichsberg in Hamburg. A. Stuber's Verlag, Würzburg.

This is a supposed dialogue between Socrates and others upon the elevation of knowledge concerning the mental condition of people for judges and teachers and others whose duty it is to discipline and guide the mind. Jurists should understand the psychology of mind and the character of the people intrusted to their discrimination, one judge will look at the deed, the other at the accused; the purpose is not for the vindication of crime but for the care of the feeble-minded. The cure of crime can only be by the education of the people, by bringing them to a higher plane of thought and teaching them to develop mental and moral equilibrium. It is emphasized that all persons should have some information concerning mental conditions such as are seen in growing children. The author points out that the education of the physician should also be along lines fitting him to give opinions upon the rearing of deviates as well as normal children. A plea is also made for the elevation of the regular physician in the asylum, to free him from the overlordship of the non-medical director of the institution.

S. D. Ludlum.

Die Kontrakturen bei den Erkrankungen der Pyramidenbahn. Von Dr. Otfrid Förster. Verlag von S. Karger, Berlin.

The author studies only those contractures that are the result of muscular conditions, and these he divides into two classes: (1) Those caused by lessening of the elasticity of the muscles and by the formation and contraction of the connective tissue within the muscles; and (2) those caused by increased tonicity of the muscles. The exaggerated tonicity may be from irritation of peripheral motor nerves or of their nuclei, or of the motor cortex or pyramidal tract. He believes that irritation of the cerebellum may produce clonic or tonico-clonic convulsions, but usually the movements are choreiform. Irritation of the pyramidal tracts may cause clonic spasms as well as tonic. Occasionally irritation of the posterior roots or of sensory nerves may cause reflexly increased muscular tonicity. He devotes much attention to contractures following diseases of the pyramidal tract. He believes that contractures are the result of the position in which the limb is placed, and often may be altered by change of position of the affected limb. When complete paralysis exists, contracture is caused by the long duration of one position. Return of function has much influence on the contracture, but then also the position caused by the restoration of function in certain groups of muscles is the determining factor in the contracture, so that it is immaterial whether the limb is brought into a certain position by active or passive movement. Involuntary reflex movements, as flexion of the lower limbs, lead to contracture of these limbs in the position assumed during the movements.

Spiller.

DIE BEDETUNG DER SPINO-CEREBELLAREN SYSTEME. KRITISCHER UND EXPERIMENTELLER BEITRAG ZUR ANALYSE DES CEREBELLAREN SYMPTOMENCOMPLEXES. Von Dr. Robert Bing, Privat-dozent in Basel. J. F. Bergmann. Wiesbaden.

In this short monograph of 100 pages there is compressed an excellent critical analysis of most of the recent work that has been done on the physiology and anatomy of the spino-cerebellar system. Coming as it does from the Frankfurt institute of Edinger and from the laboratories of H. Munk, W. His and Voit it starts with the approval of well-known masters.

Bing shows in an historical summary the reasons for much of the confusion in the interpretation of the physiological experimentation. It lies mostly in the field of technical methods which differing in different laboratories have given rise to conflicting interpretations.

The anatomical work of Foville, Flechsig, Bechterew, Gowers and Van Monakow is next discussed and the general views of Gowers regarding the separation of the ascending tracts into two series is accepted. The author then takes up the dorsal spino-cerebellar tracts, discussing seriatim: (*a*) levels of origin of these tracts, (*b*) the relations of conduction impulses to the cells of origin of these roots, (*c*) the correct topography of the tracts in the cord, (*d*) is there any separation of the fiber tracts originating in Clarke's columns and going through the restiform body to the cerebellum? and (*e*) what are the relations of the end stations with reference to topography? He then takes up the ventral spino-cerebellar tract in much the same manner. The conclusions reached must be read in the original but it should be borne in mind that the older belief in the specificity of the separation as here taught has of late years been denied and the work of Lewandowsky in particular inclines one to doubt the author's position.

A complete summary then follows relative to his results on animal experimentation; the anatomical results of which seem to be very poorly analyzed. A schematic table illustrative of the fiber tracts is suggestive but certainly not in accord with the recent work of VanGehuchten, Lewandowsy and others.

JELLIFFE.

STUDIEN ÜBER DIE NEUROFIBRILLEN IM ZENTRALNERVENSYSTEM. ENTWICKELUNG UND NORMALES VERHALTEN. VERÄNDERUNGEN UNTER PATHOLOGISCHEN BEDINGUNGEN. Von Dr. Nikolaus Gierlich, Specialarzt für Nervenleiden in Wiesbaden und Dr. Gotthold Herxheimer, Prosektor am Städt. Krankenhaus zu Wiesbaden. Verlag von J. F. Bergmann, Wiesbaden.

This rather formidable monograph of 200 pages handsomely and copiously illustrated by cuts and plates is the outcome of the newer studies on neurofibrils first brought into prominence by the work of Apáthy. As the technical methods of Weigert opened up a rich mine of research for both the normal and diseased nervous structures, so it is confidently expected that with the newer methods for studying neurofibrils, more particularly of Cajal and of Beilschowsky, another advance step may be taken into the difficult mazes of anatomical structure and alteration.

The authors have for some years devoted themselves to a study of the nervous system by the Bielschowsky methods and this present splendid

volume constitutes, in part, the collected results of their studies. The study of the alterations in neurofibrils is still in its infancy, and with this truth in mind the authors have confined themselves more or less definitely to the recording of changes rather than to the formulation of hypotheses.

They record their findings in normal embryological material in the first portion of the work and also contribute a discussion on the tenability of the neurone hypothesis in the light of these results. Inasmuch as the authors find a certain grade of continuity of fibrils, they hold that the neurone theory is in need of some modification. To just what camp of anti-neuronists they would ally themselves, however, is not certain.

The second portion of their work takes up the pathological modifications of the neurofibrils, first under thermic, chemic, infectious and toxic influences. The changes in the cord in tabes, in acute myelitis, traumatic myelitis, etc., are then described. A later section deals with the changes following acute brain lesion, abscess, hemorrhages, softening, etc., while two further sections deal with the fibril alterations in paresis, senile dementia, uremia, and delirium tremens, and also with organic tumor, etc. A rich literature is appended.

The monograph is a scholarly production, and will afford to the pathologist particularly a useful basis of comparison in a new and comparatively little-known field.

JELLIFFE.

DIE LARYNGEALEN ERSCHEINUNGEN BEI MULTIPLER SKLEROSE DES GEHIRNS UND RÜCKENMARKS. Von Privat-dozent Dr. L. Rethi, in Wien. Verlag von Josef Safář, Wien. Price 4.80 Marks.

The many-sided picture of disseminated sclerosis enables one to assemble a rich symptomatology and also affords a large opportunity for the pathological study of isolated or partly isolated mechanism.

The author has availed himself of this and in presenting this short monograph on the laryngeal symptoms of multiple sclerosis has brought together a complete record of the hitherto recorded examples, added to it observations of his own and given a résumé of the pathological findings both interesting and unique. He further gives a masterly discussion of speech defects, of laryngeal paralyses and a study of tremors in a variety of affections.

It is a short monograph of 150 pages full of clinical suggestions, and critical observations.

JELLIFFE.

UEBER DEN EINFLUSS DER GESCHLECHTSFUNKTIONEN AUF DIE WEIBLICHE KRIMINALITÄT. Von Dr. Jur. Siegfried Weinberg, Berlin. Juristisch-psychiatrische Grenzfragen, VI, heft. 1. Carl Marhold, Halle. Price 1 Mark.

The author here discusses the relation of the sexual function in woman to criminality. There is nothing original in the book, quotations being freely made from the works of Krafft-Ebing, Ellis, and others, and the author, a jurist, appeals to statistics largely for his deductions. As a beginning to a large and intricate subject it is a praiseworthy contribution.

JELLIFFE.

Aerztliches über Sprechen und Denken. Von Prof. G. Anton, Halle. Carl Marhold, Halle.

A rich reward is offered for the teaching to man the real actions of mind. The normal is frequently enlightened by the diseased. The vocal organs are innervated by the excitations of the mind, the vocal vibrations call up similar mental phenomena in another man. Speech can continue with one side of the vocal cords and tongue paralyzed, but a small portion of certain brain areas destroyed will cause a cessation of all speech. Words, according to the author, before being spoken are heard in the mind, hence with lesions in the first and second temporal gyri, the patient loses the power of choosing words; speech becomes confused, he is unable to think word sound pictures. Words are wrongly used; therefore, correct thinking is difficult. This can be balanced by sign speech. With a lesion of the lower frontal convolution, the patient is unable to pronounce words. With this location affected there is also diminished capacity to write thoughts or to write at dictation, but the patient can often copy. The lower third frontal is a component in writing as well as in speaking, for the symbols used in writing and speaking have a similar mental meaning. In other cases the patient may be word blind and this affects in a proportionate manner the mental content.

S. D. Ludlum (Philadelphia).

Nervous and Mental Diseases. For Students and Practitioners. By Charles L. Potts, M.D., Professor of Neurology in the Medico-Chi College of Philadelphia. Second Edition, thoroughly revised. In one 12mo volume of 570 pages with 133 engravings and 9 full page plates. Price, cloth, $2.50 net. Lea and Febiger, Publishers, Philadelphia and New York.

In the second edition of this well-known manual Dr. Potts has added many new and important features, bringing the subject matter well up to date. We are glad to see, for instance, that he has incorporated the term Serous Meningitis in his article on Meningitis. Few text-books, if any, make reference to this condition. Many other subjects we find presented, as lumbar puncture, concerning which the author discusses in detail the indications and technique. The illustrations here should prove valuable. Aphasia is presented well, the writer giving a special chapter on the development of speech in the child. Among other commendable chapters are those on cerebral localization, spinal localization, the vascular distribution of the brain and dementia præcox.

The book is well balanced, save that we think the author gives too few references to the literature, and that illustrations of the pathology of tabes would have been more appropriate than of Friedreich's ataxia, a disease somewhat rare.

For the student and general practitioner this volume, enhanced by the latest views, should prove most useful and valuable.

S. Leopold.

Speech Disturbances and Speech Therapeutics. Contributions to the Knowledge of the Physiology, Pathology and Therapeutics of Speech. Reprint from the Monatsschrift für Sprach Heilkunde, Bd. XVII. Verlag von S. Karger, Karlstrasse 15, Berlin.

This reprint from the *Monatsschrift für Sprach Heilkunde*, Bd. XVII,

in honor of the 70th birthday of Albert Gutzman, director of the State Institute for the Deaf and Dumb in Berlin, does not lend itself readily to abstracting. It comprises a series of articles by friends and former students of Dr. Gutzman. The articles embrace divers subjects relating to speech disturbance and their treatment.

Among the more noteworthy are to be mentioned, "The True Relation of Divers Forms of Language," by George Rouma. "The Phonomimic Method," by Dr. A. v. Sarbo; and "Speech Disturbances in a Case of Chronic Bulbar Paralysis and the Method of Treatment," by Dr. H. Knopf and Panconelli Calgia.

S. Leopold.

Diseases of the Nervous System. By H. Campbell Thomson, M.D. (Lond.), F.R.C.P. Keener & Co., Chicago, 1908.

In this work the author presents to the third and fourth year student the essentials of nervous diseases in a concise and readable manner. It is clearly written and well arranged, taking up the etiology, pathology, symptomatology, differential diagnosis, and treatment of each disease. The chapter on Tabes is particularly good. Here the author refers to Ford Robertson's researches on the infective origin of general paralysis of the insane and locomotor ataxia, as still an open question. In discussing the sensory neurones he adopts Head's classification of epicritic and protopathic sensibility. This volume should be invaluable to medical students.

Zur Differentialdiagnose der Poliomyelitis anterior acuta. (Myatonia congenita [Oppenheim] und Polyneuritis.) Inaugural Dissertation, von J. Victor Haberman, A.B., M.D. Verlag von S. Karger, Berlin.

As the title would suggest, the monograph is concerned primarily with the clinical picture presented by the three diseases, and their differential characteristics. Unfortunately, little knowledge of real value has been contributed to their etiological or pathological aspects. The subsequent investigations upon myatonia have merely confirmed, but added little new to the facts already described by Dr. Spiller. The author is convinced that a "polyneuritic" form of poliomyelitis does exist and that the peripheral nerves in such cases show no evidence of inflammatory changes. From a clinical point of view the author has presented a very careful and thoughtful study of his own and reported cases. His analytical work is good and the differential points are well chosen. Wickman's studies on poliomyelitis have been freely consulted. The article reads like a good "text-book" chapter, and will no doubt be helpful to the general practitioner in moments of hesitation.

Six cases are reported from Professor Oppenheim's clinic; one in which no diagnosis was made, two of myatonia congenita, one of idiopathic polyneuritis, and two of poliomyelitis. The first resembled poliomyelitis, with its acute onset, fever, and slight disturbance of function, but the entirely negative objective findings made the diagnosis uncertain. Patient recovered after eight days without electrical changes. He emphasizes the not infrequent occurrence of such cases, and the necessity of waiting to determine whether the muscle weakness be permanent or transient.

The two cases of myatonia congenita with marked paralysis of the

lower limbs in one case, and involvement of all four extremities in the other; absence of tendon reflexes, and vasomotor changes in one conducted themselves quite like poliomyelitis. The following differential points served to make the diagnosis clear; both conditions existed from birth, no reaction of degeneration in the muscles, the paralysis did not tend to become localized, all the affected muscles showed some mobility, muscles were atonic rather than atrophic. The condition is also differentiated from myxedema, mongolismus, and rickets. The facts that myatonia is always congenital, reflexes diminished or absent, and that the muscles show quantitative changes are strongly emphasized. Some importance is attached to the early manifestation of symptoms—usually the first day of birth—their nonprogressive nature, and the escape of cranial and sensory nerves. Ten cases, studied in the literature, showed some improvement after thirty-one months.

The remaining half of the monograph is devoted to the study of poliomyelitis and polyneuritis. The possibility that the two diseases may coexist, is admitted, but the author seems inclined to the view that polyneuritic symptoms can and do manifest themselves in the course of a poliomyelitis, independently of any inflammatory condition in the peripheral nerves. In fact, such cases, studied microscopically, failed to show any evidence of neuritis, but rather a widespread inflammatory condition involving not only the anterior horn cells, but the posterior horn, meninges, medulla, pons, and even cerebrum. Upon these findings he explains the occasional sensory disturbances observed in poliomyelitis, such as, hyperesthesia, hyperalgesia, and even anesthesia.

In conclusion he mentions a number of differential points; the more important of which are tabulated below:

Poliomyelitis.—(1) Paralysis reaches its acme at the beginning. (2) Absence of swollen nerve trunks. (3) Frequent in children. (4) Cranial nerves seldom involved. (5) Paralysis usually not widespread, may be bilateral, but not symmetrical. (6) Involvement of bladder and rectum not infrequent. (7) Complete recovery rare.

Polyneuritis.—(1) Often several days or weeks before paralysis is fully developed. (2) Can usually be palpated. (3) Rare in children. (4) Not at all uncommon. (5) Usually bilaterally symmetrical. (6) Rare. (7) Quite common to a great degree.

C. M. Byrnes (Univ. of Va.).

The Borderland of Epilepsy. Faints, vagal attacks, vertigo, migraine, sleep symptoms and their treatment. By Sir Wm. R. Gowers, M.D., F.R.C.P., F.R.S. P. Blakiston's Son & Co., Philadelphia.

This unpretentious volume of 118 pages is a welcomed contribution to the more stable literature of preepileptic states and neuroses. Whoever has made patient inquiry into the corelationship between many constitutional nervous disorders, cannot help being impressed with the fact that before he studies epilepsy proper, he must studiously investigate a large category of nervous phenomena less distinct than true epilepsy, generally less harmful in results, but quite as important in many ways as the essential disease.

The six chapters in "The Borderland of Epilepsy" deal with faints and fainting, and their relationship to true epilepsy; Chapter 2 takes account of vagal, vaso-motor, and tetanoid spasms; while Chapters 3

and 4 consider vertigo with its characteristic symptoms and points of differential diagnosis from true epilepsy. It is quite plain the author believes there is a positive relationship between migraine and epilepsy since he devotes more than 25 pages to migraine alone. His consideration of sleep symptoms—such as "night terrors," "somnambulism," "half waking" and "narcolepsy" are of much interest and not to be ignored by the neurologist who would acquire a true conception of certain phenomena that precede, accompany, or follow manifestations of essential epilepsy.

Dr. Gowers' latest book has real value for physicians who have been some years out of medical college and who are tending to specialism. The student and general practitioner will hardly find its advanced teachings adaptable to their requirements.

WILLIAM P. SPRATLING.

HYPNOTIC THERAPEUTICS IN THEORY AND PRACTICE. By John Duncan Quackenbos, M.D. New York and London. Harper & Brothers, 1908.

There is no reason especially why this book should be either reviewed or read by the physician. It is apparently written with the view of placing the author's thoughts before a public in whom discrimination, critical judgment, and a sense of the ridiculous are entirely absent. The place where this book touches the physician is in the class of patients to whom the ideas advanced in it are received as though they were novel and true. Against such an influence medical criticism can with justice be directed. It is a pertinent question as to the reason why books of this kind are written nearly always in a certain extravagant, intense and rather inflammable style. Does the practice of hypnotism, as the author of this book uses it, so dissociate the mind of the operator that the normal and average regard for accuracy, proportion and fact is lost sight of? The possibility of this book and others like it being the products of a subliminal consciousness is a fascinating thing to contemplate. To arouse investigation into this new field may be regarded as the chief virtue of this work on hypnotic therapeutics.

SIDNEY I. SCHWAB.

TECHNISCHER FORTSCHRIFT UND SEELISCHE GESUNDHEIT. Von Willy Hellpach, Dr. Med. et Phil., Privatdozent der Psychol. Carl Marhold, Halle.

The author argues that psychology should have an equal share in the curriculum of the technical school with other branches of study; psychology builds no bridges or machines but what concerns everyone, be he technically engaged builder or machinist, is the care of mental health. What influence has the rapid change in the technical world had upon the mental health of man? Insanity is not perhaps markedly increased but the neuroses have become decidedly more common. It is not so much the result of strenuous life on the individual as on the progeny, who do not stand the strain, but the technical avalanche of the last hundred years has been the cause of many a sound mind being crushed. Workers at monotonous trades and pursuits all contribute a quota to neurasthenia, and it is for this reason that the care of mental health should be taught in all technical schools.

S. D. LUDLUM.

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Original Articles

IDIOPATHIC STATUS HEMI-EPILEPTICUS AND GENUINE FOCAL EPILEPSY[1]

By Herman H. Hoppe, M.D.

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In bringing the subject of localized convulsions or rather epileptoid convulsions before you, I wish to confine the scope of the paper to that group of cases which are of genuine epileptic character in contradistinction to symptomatic epilepsy of the Jacksonian or reflex type.

The material for this paper is partly anatomical, but chiefly clinical, and I feel that there may be two opinions as to the underlying pathological changes in the cortex of the brain in these cases. I have, however, been very careful to choose, after a most minute examination, only such cases in which the physical examination has been entirely negative.

If there was a history of trauma with unconsciousness, even through there was no fracture of the skull, I have ruled out the case.

If the previous pathological history was such as to point to the possibility of an encephalitis during the course of an acute infectious disease, the case was not included in the group. We have been careful to enquire into the presence of congenital or acquired syphilis. All cases which presented any deviation from the normal, such as slight degree of ptosis, inequality of the pupils, difference in the muscular innervation of the two sides of

[1] Read at the thirty-fourth annual meeting of the American Neurological Association, May 20, 21 and 22, 1908.

the face, tongue and body, changes in the reflexes, no matter how slight, even though no relation could be shown between the attacks and the supposed lesion producing the above slight deviation, were not included, because of the suspicion of the presence of an organic lesion in brain or nerve trunks.

I have known these cases from a few years up to fifteen years, wherever possible, have watched them up to the present time, and I feel that I can safely say that as far as our methods of physical examination go, I have ruled out all cases with apparent cortical lesions.

I would like to categorize these cases as cases of genuine epilepsy in the sense that the attacks are of unknown origin in contradistinction to those cases which are symptomatic. These cases are of interest in a two-fold manner; first, as a study of one of the manifold phases of epilepsy, and secondly, from the standpoint of the question of surgical intervention.

The cases comprised under this group can be arranged into two classes:

1. Hemi-epilepsy and more especially the status hemi-epilepticus.

2. Localized epileptoid seizures in the form of partial convulsions or the initial manifestations on the part of the special senses or of the power of speech.

Binswanger defines genuine epilepsy as a "well-defined chronic disease of the central nervous system, which can be produced by manifold causes. It manifests itself either by frequent attacks of convulsions with unconsciousness or in partial attacks of this kind or in psychopathic states occurring either in or after the attacks."

There is some question whether some of the cases of status hemi-epilepticus can be conformed to the above definition of epilepsy.

Müller was the first to call attention to a status hemi-epilepticus, which ends fatally without any discoverable lesion of the brain or its cortex, and named the condition status hemi-epilepticus ideopathicus. The condition is not very frequent and there are but few cases on record. Of these there are two groups, either the patient has never had any convulsive attacks before or the hemi-epileptic status occurs intercurrent in a case of genuine epilepsy, or the case, if it recovers, subsequently becomes one of general epilepsy.

Most cases on record have terminated fatally, usually after

surgical interference, and the autopsy revealed no cause for the hemi-epileptic character of the convulsions.

The following case may serve as a type for this form of localized convulsions:

CASE No. I.—Status hemi-epilepticus. M. F., æt. 22. Female, servant—parents are nervous. Father alcoholic. Mother neurasthenic. Brother and sister are well. No epileptics or insane in any branch of family.

Was injured at age of seven, struck on head, no loss of consciousness, right leg seemed weak for some time afterwards. Has never been sick afterward and never had a convulsion.

Grew up to be a very strong, muscular woman.

Has been well absolutely and a hard worker, never sick, no headaches. Eight days before death arose in the morning with a violent headache; this soon passed off, and patient felt well all day until she retired at 9 P. M., when she began to vomit and had a chill; vomiting continued at intervals for two hours. Patient then fell asleep and was found unconscious next morning.

A physician was called and gave her a hypodermic of strychnine; after regaining consciousness in half an hour she was unable to speak, felt drowsy, fell asleep easily. Pulse was weak, temperature was normal. At noon she felt well. All next day and the day following she was absolutely well, attended to her household duties, cleaned windows, cooked dinner, etc.

On fourth day arose feeling well up to noon, then had vertigo, sinking spells, fear of losing mind and seemed somewhat incoherent in her speech.

On same night she had a convulsion with loss of consciousness. On fifth and sixth days no convulsion but patient seemed half dazed, staggered about the house, complaining of weakness and vertigo. Forty-eight hours preceding death patient was in convulsions continually and did not regain consciousness.

All convulsions began on the right side of face, then extended to right arm and right leg, after which they became general. The convulsions lasted about two minutes and were followed by a complete paralysis of the right side of body, face, arm and leg. In the interval between convulsions patient was restless, tossed about with left arm and leg, gradually movement began, after the lapse of a few minutes, in the right arm and leg and these movements became more and more marked until the muscular activity of the right side seemed entirely restored before the onset of the next convulsion. The patient was observed by me twenty-four hours before her death and in one half hour I observed three convulsions.

The physical examination showed a well developed, strong young woman, of good height and in good flesh, no edema anywhere.

Mental Condition.—Entirely unconscious and could not be aroused. Pupils equal in size, reaction to light sluggish between attacks. Papilla somewhat injected. Passing paresis of both arm and leg of right side, reflexes abolished on both sides.

Slight amount of albumin in urine with some granular casts. Temperature normal, pulse 90. Patient was removed to a hospital, but after a surgical consultation, no definite reason could be assigned for surgical intervention except the Jacksonian character of the convulsions. The acute character of the case and the status epilepticus seem to be contraindications to an operation.

Patient died on the following day, the convulsions diminishing in frequency toward the end.

Autopsy.—Brain. There was an intense venous congestion over the entire surface of brain, more marked on left side than on right. Especial attention was paid to the various sinuses in the dura-mater but all were found to be normal. The middle cerebral artery on both sides and all their branches, especial care being paid to the left artery, as far as the ramifications could be followed were found patulous and normal.

The cortex of the brain, after a most minute examination, especially over the left psychomotor areas, was found to show no macroscopic changes; punctate hemorrhages and small areas of softening were looked for, but none were found. The pia mater showed no adhesions anywhere. The dura mater was normal. The sections of the cortex were misplaced and no microscopic examination was made.

The kidneys were macroscopically normal, microscopically slight fatty changes were found in the cells.

In a word, the autopsy was negative. The kidney changes were probably secondary to the general condition, not the primary cause.

Here we have a case where the entire previous history was known.

The first attack, one week previous to her death, came on during sleep, the patient being found in a comatose condition from which she recovered in a few hours, then went about her work for two days. The attacks then coming on again, first few in number, with conscious intervals of hours during which the paresis of the affected limbs disappeared, then a gradual increase in the number of attacks until there was no return of consciousness between the attacks; finally the attacks became so numerous that she practically passed from one attack into another.

There can be no doubt but what some general intoxication produced all the symptoms, including the albuminuria; that this

condition was entirely uremic is doubtful. We see similar general intoxication, say, for instance, of la grippe, followed by a general infection, including the albuminuria with granular casts, and we have no uremic manifestations at all, to say nothing of the convulsions.

Numerous similar cases are on record without the albuminuria. Then, again, the localized convulsions in uremia are not of the same character and the cases do not yield to the treatment for uremia. In this particular case the kidneys did not show the changes of acute parenchymatous nephritis. On the other hand, the important points under consideration are the half-sided convulsions and the negative autopsy, leaving the cause for the half-sided convulsions unexplained. The injury at the age of seven, with a doubtful paralysis of the right leg might, before death, point to some old scar in the left leg area, but the convulsions always began in the right side of face. Again, the cortex in the left leg area was found normal macroscopically. The entire illness lasted for nine days, and if any gross lesion had caused the half-sided convulsion, even though they had been punctate hemorrhages or minute areas of softening in the sense of Spiller and McCarthy, they should have been visible to the naked eye.

Cases of this kind belong in a category by themselves. It is true that in their mode of origin, the spread of the convulsive seizure and the secondary paralysis following the return of consciousness or in the restless period between the convulsive seizure when patient was comatose, and the rapid disappearance of the paralysis were in a very close resemblance to the ordinary Jacksonian epilepsy. But here the resemblance ceases. In ordinary Jacksonian epilepsy, except in the acute traumatic cases, I have never seen a condition of status epilepticus; moreover, the previous histories of the cases point to a gradual onset over a period of months and even years, whereas all the recorded cases of status hemi-epilepticus usually have a history of acute sudden origin and a rapid fatal termination or an equally rapid recovery in the few instances where recovery has occurred. There are quite a number of these cases on record; I mention those of Bernard, Bonnhoff, J. S. McKendrick, and especially Leononere and Henneberg.

McKendrick's case is interesting on account of the fact that his patient had numerous attacks without loss of consciousness,

limited usually to right side, followed by aphasia, paresis and hemianesthesia, in one attack the left side alone was involved. This patient died in status hemi-epilepticus and the autopsy was absolutely negative, both macroscopically and microscopically.

Leo Müller reports a series of eight cases of this character with autopsy negative both for macroscopic and microscopic examination.

In each of these eight cases no evidence could be found that the individuals were formerly epileptic; all died in a state of status hemi-epilepticus with negative autopsy. Müller, after a review of the literature, comes to the conclusion that in both of these so-called cases of Jacksonian epilepsy as well as in ordinary genuine epilepsy we can have a status epilepticus which terminates fatally. This does not, however, explain the case; it merely states a fact.

That we might have minute foci of inflammation and degeration throughout the cortex, as is shown by Spiller, McCarthy and others for uremic cases, would hardly explain the cause of death, nor would the finding of such foci explain the hemi-epileptic character of the seizures; they themselves are merely the effect and not the primary cause; we might suppose an embolic invasion of the various branches of the arteria fossa sylvii so numerous and minute as to effect a widespread area on one side of the brain. But in our case, at least, where a week elapsed between the first attack and the fatal outcome, we would suppose that macroscopic evidences of minute hemorrhagic or degenerative areas ought to be found.

It is possible, too, that these cases represent suddenly developed acute epilepsy which rapidly passes to a state of status epilepticus and death.

The half-sided attacks could be caused by the fact that one hemisphere is more vulnerable, functionally weaker than the other and that as a result of a toxemic condition the irritations begin in the weaker side of the brain in the form of localized convulsions which rapidly spread and end in a general convulsion. We have an analogy for this explanation in those cases of genuine epilepsy in which the general convulsion is followed by monoplegia, hemiplegia, aphasia, deafness, etc., which quickly disappear after the attack. These secondary manifestations are explained by an excess of exhaustion in the corresponding cortical

areas. There must, however, be some local weakness of these centers, which explains this excess of exhaustion. In a similar way we might explain that the half-sided origin and nature of these convulsions may be due to a functional weakness of one hemisphere compared to the other.

The most important lesson, however, that is taught us by the study of above and similar cases of status hemi-epilepticus is the futility of surgical interference. In fact, the very character of these seizures, that they have all the ear-marks of Jacksonian epilepsy, that they occur in individuals whose previous history has been negative, and the rapid recurrence and number of seizures must be looked upon as rather indications against surgical interference than for such procedure.

There is an air of mystery about these cases which only future investigation can clear up.

I have seen three other cases of half-sided convulsions, which have had at times so many convulsions in twenty-four hours that we can speak of a passing status epilepticus. In one of these cases there was never any loss of consciousness, but in the other two loss of consciousness occurs during the night attacks, especially if the attacks during the day have been very numerous. The question of hysteria can be raised in all three cases, but in two of the cases the loss of consciousness with secondary sleep and amnesia and especially the fact that the patients are aroused from deep sleep by the localized convulsions and become unconscious after the involvement of the whole side, would speak in a positive manner for epilepsy rather than hysteria. One of the last-named patients is the sister of a physician, who vouches for the fact that she is really unconscious, and the other has been observed repeatedly by nurses in a hospital under my care.

The first case whose history I give in a concise manner is the son of a physician and may seem a little doubtful. The history is given by the father.

Alfred B., æt. 21. Had never had any of the ordinary diseases of childhood. Parents are well. At four years of age, began to have spasms in the left arm, which continued daily, and after several months increased in extension until the entire left side, including the face, became involved, at times he has had as many as twenty attacks per day. Patient never has less than eight to ten attacks per day; has frequent attacks during the

night. At the end of one year the left side of body was paralyzed. Patient, six years of age, was subjected to anal dilatation (orificial surgery!!!), was isolated in a hospital and after two months the attacks gradually subsided and the paralysis disappeared. Never any loss of consciousness.

During the next seven years patient was free from attacks.

During the past eight years patient has had frequent localized convulsions in the left arm, without any paralysis and without loss of consciousness.

Examination.—Patient is a tall, handsome, well-built athletic young man. Mental condition good, is bright and cheerful, never has any headache or vertigo. Stands well in his class at Yale and is fond of all out-door sports. General muscular development good. No change in optic disks. Pupils equal in size and react to light. There is an absence of both patellar reflexes, otherwise the examination for muscular power, sensation and reflexes is negative. After the examination patient was seized with a spasm of the left arm, tonic in character, somewhat painful, the fingers were flexed, the hand almost closed and flexed on the forearm, the forearm in supination and bent upon the arm, the biceps also involved in the spasm. Duration about one minute. There was no weakness after the spasm subsided.

Patient was given twenty grains of strontium bromide at bedtime and took the medicine for one month and has had no return of the spasm for the past twenty months.

Résumé.—Onset at five years of age, spasm of left arm, gradually involving the whole left side, with paralysis. Patient has as many as twenty attacks per day; after fourteen months of treatment was completely cured by dilatation of anus and a rest cure without use of any drugs. Recurrence of local spasm in the left arm after seven years, countless attacks during the next eight years and then sudden cessation after the use of twenty grains of strontium bromide at night for one month.

I have seen the other two cases of a similar character, without, however, the happy result, grow up from childhood to womanhood with constant attacks of a hemi-epileptic character without permanent paralysis, with loss of consciousness occurring occasionally and yet without the usual stigmata of hysteria.

Case III.—Miss M., æt. 7, of Hebrew parentage. The child has always been well, has not had any serious illness with the exception of whooping cough. About two years after the whooping cough she began to have fainting spells for which there was no adequate cause.

Present trouble began at the age of five with spasms in the right hand which extend to the leg and are painful in character.

During the attack patient becomes very nervous, is unable to speak, but does not lose consciousness except on rare occasions. The attacks occur often during the day; frequently during the night the patient is awakened from her sleep by the attacks; the attacks last but a few minutes and are not followed by weakness in the arm or leg. On rare occasions patient will not have attacks for a day or two at a time, but the rule is to have numerous attacks daily.

Examination.—Mental condition only fair. General development good. No loss of muscular strength, no anesthesia on right side, reflexes normal, special senses normal.

During the past thirteen years she has grown to be a large, well-built woman, suffers no headaches or vertigo. Is never ill, but the local spasms of the right side of body continue. The right side of body has grown and developed as well as the left, and there has never been any paralysis. Patient at times loses consciousness, especially so during night attacks, when the number of attacks during the day preceding have been especially numerous. In one attack the patient fell and sustained a Colles fracture. The attacks in the arm continued during the time the fracture was in plaster of paris.

Her mental state is somewhat backward, due in a measure to the fact that she has never been able to attend school.

A similar case in every respect is the following:

Bessie C., æt. 26, small town, Indiana. Family history negative. Fourteen years ago had a fall. Since then has not been well. Did not strike head and did not lose consciousness. She says that her right arm and leg were weak periodically, before the onset of the convulsions, for years after the fall, before the convulsions began. Patient has attacks of convulsions which begin in the right arm, then extend to the right leg and right side of face, duration only a few minutes. Usually attacks pass off in a few minutes without loss of consciousness, but has lost consciousness on several occasions, but very rarely. The attacks are followed always by a weakness of the right arm, but not always of the leg and never of the face, which quickly passes away. These attacks occur with great frequency during the day, attacks with loss of consciousness occur only at night. Patient is awakened from her sleep with a spasm in the right forearm or the right foot. The spasm is at first tonic, then clonic, very severe and painful; in a few seconds the entire right side is affected, patient is unable to talk or cry out and then loses consciousness, from which she awakens in an hour or two, has a bad headache, feels badly all next day.

In the attacks observed at the hospital there has been no convulsion at all on the left side during the period of unconsciousness. After the attack patient at times arises, walks about, does automatic acts of which she afterward has no recollection.

Most of the spasms are limited to arm, leg and face; they are tonic in character for a few seconds, then clonic, the spasmodic contractions continuing for several minutes, often so slight that they can hardly be seen, but can be distinctly felt if the arm is grasped. The initial tonic spasm is always severe.

There is no marked headache, no vomiting and no vertigo.

For days at a time patient passes from one paroxysm into another. These are of the character described below. Each attack, both clonic and tonic, does not exceed two minutes in duration. At times the attacks are as numerous as ten per hour, usually beginning in the arm, but at times simultaneously either in arm and foot or face and arm. Then for days at a time the right side is entirely free from attacks.

Bromides reduce the number of seizures, but the above condition has continued now for ten years. Patient can attend to the ordinary household duties on a farm.

Examination.—A well-developed and well-nourished woman of medium height. Has a bromine eruption all over face and back.

Mental condition good, memory quick and active.

There is an exaggeration of the reflexes but no evidence whatever of any organic disease of the brain.

Résumé.—This case has gone on in about the same way for the past fourteen years, spasms numerous, in character always beginning in the right arm and extending to leg, with at times a passing paresis and at times a loss of consciousness, but neither the history of the case nor careful observation reveals the presence of any organic lesion of the cortex. The fact that the severe attacks with loss of consciousness occur at night and that patient is aroused from her sleep by the spasms in the arm or foot proves that this case is epileptic rather than hysterical in character. It is rather a good point in differential diagnosis between genuine epilepsy on the one hand and Jacksonian on the other, that the local spasms in the arm awakens patient to consciousness out of a deep sleep before the convulsion has had time to become general on the right side and before consciousness is lost. But all proof of an organic lesion for the Jacksonian seizure is wanting.

I have no hesitation in designating these three cases as epileptic and also believe that the numerous attacks, in one case continuous for days at a time, constitute a condition of status hemi-epilepticus, even though there is no continuous loss of consciousness. The question as to the functional character or organic origin of these attacks in these cases will be taken up later.

The question as to whether a localized convulsion, limited to one group of muscles, followed by paresis and aphasia, is really due to a cortical lesion is often very difficult to decide at the time

of the first examination. We must often wait for weeks and even months, even with such gross lesions as tumor or syphilitic meningitis, before a second attack occurs.

Our knowledge on the subject of Jacksonian epilepsy is undergoing some rearrangement. The line of division between convulsive seizures of a functional character and those due to an organic lesion of the cortex is not as sharp as it might be.

The opinion is gaining more and more ground that there is a goodly number of borderland cases in which it is difficult to say even after a long study of the case, whether the localized convulsion is caused by an organic lesion, whether it is to be looked upon as a motor aura, whether it itself is to be classified as genuine epilepsy, or whether it is purely a reflex contraction, or whether it is hysterical in character. We must also remember that every Jacksonian attack need not be necessarily caused by a gross lesion of the cortex of the psychomotor area. Diseased conditions of the vessel walls, local circumscribed changes of the bony or membranous covers, subcortical scars, scars or areas of softening in the neighboring parietal or the under surface of the frontal lobe can cause Jacksonian seizures, if other causes such as alcoholism or the various intoxications have been at work to produce an epileptogenic condition of the cortex. Even atrophic conditions of the cerebellum have been found as the only cause of Jacksonian convulsive seizures. We can see therefore how difficult it is in a given case of localized epileptic seizures, to rule out organic causes as the underlying condition of the attack. When we consider that there are brain lesions which may occur during the embryonic period, slight attacks of cortical encephalitis producing temporary delirium during the acute infectious diseases of early childhood and which may disappear without leaving a scar beyond the slight changes in the ganglionic cells of the cortex, but which may nevertheless be the point of origin of cortical irritation leading to Jacksonian attacks, we can readily understand the position which has lately been taken by numerous authors, Freud and Rie, Rosenberg, Nurerricht, Koenig, Murutoff, Lukacz, Starr, Sachs, Pick, Heilbronner, etc., who hold that epilepsy is an organic disease of the cortex. The advocates of the organic theory of the origin of epilepsy have not been without their opponents, prominent among whom have been Oppen-

heim and Binswanger. While they admit that there may be an organic basis for epilepsy, they maintain that the latter has not as yet been demonstrated and that the distinction between genuine and symptomatic epilepsy must still be maintained. We can also see how difficult it is to assert even after prolonged observation in clinical cases that these localized convulsive seizures are not dependent on some organic change in the cortex.

The study of this subject and the review of the literature and the experience of most of us will lead us to the conclusion, however, that there are undoubtedly cases of localized convulsions which are not produced by organic lesions, and this conclusion will have an important bearing on the subject of surgical interference in epilepsy. The question then arises, where can we draw the line, between organic cases which might be benefited by an operation and those cases where an operation would not only be useless, but might damage the patient.

In considering localized convulsions we must in the first place draw a sharp line between convulsions of a local character which precede and those which follow the loss of consciousness; we must also distinguish in the first group between such cases in which there is no loss of consciousness at all, at any time during the history of the case, and those cases in which either in the beginning or later during the course of the disease the local convulsions become general and are attended with the loss of consciousness.

I will take the second group first. Whenever the loss of consciousness occurs first and convulsions follow, limited to a small group of muscles, the cases are undoubtedly epileptic in character and are not Jacksonian in type. These cases are not numerous and should be grouped under the heading of petit mal moteur. It is at times difficult to distinguish such attacks from hysteria. Lately I saw such a case develop on the basis of hysteria.

Miss B., æt. fifty-six, always nervous, about one year ago, after an attack of grippe, began to have stomach symptoms, loss of appetite, intestinal fermentations and gradual emaciation, with occasional vomiting. Six months ago a brother died with hemorrhage from both stomach and bowel. On the day of the funeral patient began to vomit and has continued to do so ever since. Three months ago patient fell and struck the left fore-

head and temple against the marble floor of the bath. One month later began to have attacks, during vomiting, viz.: loss of consciousness, pallor of face and turning of head over to left shoulder; the loss of consciousness occurs first. At times has had as many as six such attacks per day. It seems to me that we have here a case of traumatic epilepsy with localized convulsions, but not in the sense of Jacksonian epilepsy, but a case of genuine epilepsy, traumatic in origin, engrafted on a neurotic basis, but with localized convulsive seizures.

In other cases of genuine epilepsy we have similarly limited convulsions.

Lilly L., æt. twenty-one, has had epilepsy for six years. The attacks begin without an aura, always with sudden loss of consciousness. The convulsion is usually limited to the left arm. If the attack spreads it involves the left side of face and then the right leg.

This patient has been under observation for two years and the most careful examination has always been negative.

In some cases of genuine epilepsy we have only occasionally a local spasm, say of an arm or the face, the convulsions usually being of a general character.

In other cases of petit mal, which usually have no convulsions, we have attacks occasionally in which there is a local spasm of the arm. Other cases of genuine epilepsy will occasionally have an attack in which, during a few seconds preceding consciousness, an attempt is made to perform a definite act requiring coördination of movement, such as an attempt to brush the hair, but these attacks occur rarely, the usual attack being of the ordinary genuine epileptic type. All of the above variations and irregularities are simply manifestations of genuine epilepsy. It seems to me that the condition of the cortex in the foregoing cases can be compared with those cases of genuine epilepsy in which the epileptic convulsion is followed occasionally by a monoplegia, hemiplegia, aphasia, deafness and other losses of function of the cortex of a focal character. They are so-called cases of abortive epilepsy. The fact that the loss of consciousness precedes the localized convulsion proves them to be genuine epilepsy. Why the convulsion when once begun does not spread is difficult to explain. We may, however, remove these cases entirely from the realm of Jacksonian epilepsy and therefore out of the province of surgery.

In these cases, a most careful examination at various times and a prolonged observation, as well as a careful consideration of the previous history of the case, failed to reveal any evidence of any local change in the cortex which might account for the post-epileptic loss of function. They seemed to present as far as any organic lesions were concerned an absolutely normal condition of the brain.

I mention these cases only to call attention to the fact that even in genuine epilepsy we may have motor manifestations limited to certain areas of the cortex, either always or at times without any apparent reason and with an entire absence of gross organic changes in the cortex, as far as our present methods of physical diagnosis will enable us to determine, during life.

I will now pass on to a group of cases which, either on account of localized convulsive seizures or on account of the onset of the attack with disturbance of function of one of the special senses, I look upon as idiopathic focal epilepsy.

I will begin with the history of two adults who have had but a single attack without any recurrence:

Case IV. Mr. McK., æt. 24, was examined in March, 1895. He has always been nervous, but has never been seriously ill. On the day before the examination while standing at his door he was seized with a sense of numbness in the left leg which quickly spread to the left arm, at the same time there was a cramp in the arm and leg, he fell, remembers that he was assisted to a couch and then lost consciousness. There was no general convulsion. After regaining consciousness, he found that the left side of the body was paralyzed. The loss of function disappeared in a few hours. Patient came to Cincinnati, a distance of forty miles, by rail, on the following day.

Examination, negative with the exception of neurasthenia. This patient remained under close observation for months, was seen at intervals for years and was again seen a few weeks ago, without a second attack and with no treatment whatever except for neurasthenia.

Résumé.—A local convulsion beginning with numbness, in left leg and arm followed by a spasm, then unconscious with a subsequent passing paresis of the left arm and leg. After thirteen years no recurrence.

Case V. Mary K., æt. 31, has been under my care for neurasthenia for a few months. Always has been nervous, from overwork, as a clerk.

On November 19, 1906, while at work and in a pleasant frame of mind, suddenly found a difficulty in speaking, could

not answer a customer, almost immediately there followed a spasm in the right arm and leg and then she fell, in a state of unconsciousness, was removed to her home.

Examination two hours later: Patient is crying, hysterical, afraid that she is going to die.

Examination reveals still some disturbance of speech of motor character and some weakness of the right arm. The right leg is normal. Sensation and reflexes of right side of body normal. Pulse 96, temperature normal. A few days later patient was back at work.

A second examination three weeks later failed to show any evidence of any organic disturbance of brain.

Patient is well to-day and has never had a recurrence.

Résumé.—A sudden attack beginning with motor aphasia, followed by a localized spasm of the right arm and leg, unconsciousness, then a subsequent partial aphasia and weakness of the right arm, passing off in a few hours. No recurrence in eighteen months. While both these individuals were neurasthenics, I am convinced that the attacks were not hysterical in character, but were due to a temporary and sudden irritation of the cortex of the brain, beginning on a small area and spreading over the entire cortex of one hemisphere. These individuals are not epileptics, but have had a localized spasm of cortical origin, functional in character without a single recurrence.

From the history of the following cases, the manner of the onset of the convulsion and the spread of the seizure with secondary loss of consciousness, we would think at the first examination to have typical cases of Jacksonian epilepsy before us.

CASE VI. W. C., æt. 14. Parents healthy, has always been well, never seriously ill. Attacks began in 1895. Patient was seized with a convulsion of the left arm which rapidly spread over entire left side. Then lost consciousness. The convulsions then became general. Has had three attacks of this character.

Examination.—Well developed boy, bright, quick, with good memory.

Examination negative.

In the past thirteen years patient has grown into manhood, over six feet tall, very strong and muscular. He never has any headaches, vomiting or vertigo. Still has occasional attacks, which are possibly brought on by taking a few glasses of beer. The attacks, however, invariably begin in the left arm, extend over left side of body and then loss of consciousness, after which the attacks become general.

CASE VII. J. H., æt. 13. Parents neurotic. Never has been sick until six years of age, sudden onset of epileptic seizures. The attacks always begin with tonic and clonic spasms of the right facial, then falls, becomes unconscious and has a general convulsion. Attacks come on during sleep with a scream, but

never knows when he has an attack. Often has as many as six in one night and has had twenty per day.

Examination Six Years after Onset of Trouble.—Strong well-developed boy of average intelligence. Physical development good. Examination in every respect negative.

Five years later patient's condition remains unchanged. He has grown into manhood and is in every way normal except for the convulsions.

Résumé.—Healthy young man, epileptic attacks for thirteen years. Onset, always in the region of right facial, no change in the character of attacks since first one. No paralysis.

CASE VIII. C. K., æt. 10, girl, parents normal. In infancy always had "spasms" when sick with a fever. For the past six years has had convulsive seizures, averages one attack per week, has seizures both night and day. At times as many as eight to nine in one night.

The attacks always begin in right hand before they become general. Also has attacks of petit mal.

Examination.—Well-developed girl, stands well in her class.

Examination negative, no weakness or exaggerated reflexes in the right arm, no change in patient at time of writing.

Résumé.—Case of six years standing, local convulsions of right arm usher in each attack of general epilepsy.

CASE IX. The following case has not been under observation long enough (twelve months), but on account of the absence of all symptoms between attacks and the negative character of the examination is of great interest.

John A., æt. 43. Farmer, has three healthy children.

Trouble began ten months ago. Patient complains of a subjective sensation of numbness in the right hand. "The arm has not the right feeling." Attacks begin with scintillations, "sparks," before the eyes then a sensation of tingling in the right side of face, right arm and right leg, loses his ability to talk, "seems to have no mind at all." There is no feeling in the arm and leg, drops whatever he may have in his hand and he can't walk. Never any spasms. He has never during the twelve months lost consciousness. Between attacks there is no headache, no vomiting, no vertigo, there is no loss of strength in the arm or leg.

During the attack has palpitation of heart. Duration of attack two minutes. Frequency. At times several attacks per day, often only one or two attacks per month.

Examination.—Medium height, average development. Intelligence normal. No albuminuria. There was found no change in the optic nerve, no hemianopsia, no loss of strength on right side, no objective disturbance of sensation in any of its qualities and no aphasia, no exaggeration of reflexes.

Résumé.—Attacks, periodic in character affecting entire right

side of body, with loss of speech, loss of muscle power and subjective disturbance of sensation, beginning in face and extending to whole right side of body. Typically Jacksonian in character.

CASE X. The next case is somewhat similar in one respect, the numerous attacks limited to attempts at speaking, followed by loss of consciousness and at times general convulsions. Under observation three years.

Miss T., æt. 30. Music teacher, unmarried. As a child had diphtheria and scarlet fever. In the past five months has had five general convulsions with loss of consciousness. One attack began in the right arm and was followed instantly by loss of consciousness. All attacks begin with speech disturbance, numbness and spasms in tongue and lips, during which patient mutters unintelligently and almost immediately loses consciousness. Patient is conscious for a second or two after the onset of the attack. She is conscious of an effort to speak and thinks that she is calling loudly for help, at times the attack passes over in a few seconds, at other times there follows a general convulsion. Patient has frequent attacks of "speech trouble," as described above, which she says she can stop by taking a drink of water, always carrying a small bottle of water with her wherever she goes.

Suffered much with headache. But never has vomiting or vertigo.

Examination.—Negative in every respect, except that right pupil was little larger than left. Patient has been under observation for three years with no change.

CASE XI. The following case presents a feature of local disturbance which is unique.

Otto W., æt. 24, single, draughtsman. Family history negative. Has never been seriously ill.

Onset of trouble five and a half years ago. All attacks begin with a subjective feeling of movement in the right eyeball; at times a loss of vision in the right field (right-sided hemianopsia) precedes the loss of consciousness. This is followed at once by loss of consciousness. Some attacks are limited to the above sensations of a movement in the right eyeball with a "flash of thought through his head" which he cannot recall after the attack.

In the majority of instances the loss of consciousness is followed by a general convulsion, biting of tongue, etc.

Examination.—Mental condition slightly morose and depressed. The examination is in other respects negative.

The following case presents a combination of disturbance of sense of taste, which precedes loss of consciousness, with local convulsions of the right arm and face.

CASE XII. P. H., æt. 17. Has never had any acute illness which could have caused present trouble.

Had first spasm when a year old, one each year until six

years old, then none for four years. When a child the right arm was paralyzed after an attack. Up to a few years ago all attacks were as follows: First there is a sensation of having a nasty taste in the mouth. "The taste of worms" nauseating in effect; the boy then runs to washstand or waste-jar and begins to expectorate saliva from the mouth, almost immediately there is a spasm in the right arm and face. Then follows loss of consciousness, is not sure that the turning of head to right and convulsion of face and arm precedes the loss of consciousness. But the convulsion always begins with the spasm in the right side of face, turning of head to right, then right arm and leg are affected and then the convulsion becomes general.

For the past five years attacks begin with the exclamation, "Oh, stuff is coming up," which is quickly followed by a loss of consciousness and a general convulsion. Patient states that the above cry is always the result of a foul taste in the mouth which seems to rise from the throat. Sometimes patient has attacks only of disturbance of taste with loss of consciousness.

The majority of his attacks now are those in which he has the conscious feeling of a vile taste in the mouth, then expectorates. There is a flow of saliva and then loss of consciousness and a general convulsion. At times, however, the convulsion begins in the right side of face, head turns to right, then a convulsion in the right arm and leg, after which the convulsion becomes general.

Between attacks he is well, has no headache, vomiting or vertigo. Sometimes has no attacks for weeks. Has had sixteen spells in the last thirty-six hours.

Examination.—Mental condition is sluggish, otherwise negative, including sense of taste and smell.

In the foregoing group of cases I was very careful to include only such which were entirely negative as far as any objective examination can reveal the presence of slight organic changes in the cortex. I do not overlook the fact that these changes may be present in the neighboring latent or silent areas of the frontal, parietal or temporal lobes. Horsley operated on five cases of the above character and in two found organic cortical lesions. Years ago I had a case of so-called idiopathic epilepsy operated upon and found that a small diploic vein had worn through the inner table and had caused a perforation of the dura and an irritation of the cortex. However I believe that, unless we wish to become Nihilists in our clinical work, we must hold as a working proposition, hard and fast to the rule, that if the objective examination is negative, especially in cases which have ex-

isted for years, there is no gross lesion of the cortex. Although therefore, as to the subject of Jacksonian epilepsy, we are often standing on uncertain ground, we have some good rules for guidance to distinguish idiopathic from Jacksonian seizures.

There are two points of distinction between the so-called idiopathic local epileptic convulsions and the symptomatic Jacksonian.

The idiopathic partial epilepsy shows no progression. In all the cases which I have cited above, there has practically been no change in all the years during which they have been under observation. They always begin in the same way and the secondary paresis has never become permanent or progressed to a real paralysis. In the symptomatic Jacksonian attacks, however, we have a steady progression. The symptomatic spasms have a gradual beginning; at first almost unnoticeable, muscular twitchings occurring at long intervals. Then a gradual increase in severity, number and groups of muscles involved until after weeks, months or years they attain their height and maximum. Then again the secondary paresis in symptomatic Jacksonian epilepsy sooner or later becomes permanent, progressive, persisting during the entire interval between the attack. In the second place, in the idiopathic Jacksonian seizures, the objective physical examination is negative, whereas in the symptomatic form, sooner or later we will find objective signs, which point to an organic change in the cortex.

The symptomatic form is less apt to be nocturnal than the idiopathic, but since symptomatic Jacksonian attacks do arouse patients from sleep, we cannot rely very much on this point, although the presumption would be in favor of the idiopathic form.

Are we justified in looking upon cases of the above character as idiopathic epilepsy? Does the fact that we are unable to find on physical examination any deviation from the normal prove that there is no gross change in the cortex of the brain?

The history of epilepsy abounds with cases of supposed idiopathic epilepsy in reality caused by gross pathological lesions, even brain tumor, which were not suspected during life. Horsley reports that in two out of five cases of idiopathic local epilepsy, he found on operation gross lesions of the cortex. Until we have careful clinical investigation on the line followed by Red-

lich, we cannot but hold that for practical working purposes, when the physical examination is negative, there is no gross lesion of the cortex. Investigating the widely promulgated theory, that a vast number of cases of youthful epilepsy are caused by cerebral palsy in early childhood, Redlich carefully examined 150 cases of genuine epilepsy in young subjects. Of these, three showed manifest evidence of hemiplegia and he excluded them as not being cases of genuine epilepsy, but cases of cerebral palsy with epilepsy. He insists that we must not look for marked physical signs caused by gross lesions, but slight deviations produced by finer anatomical changes. He found slight evidences of paresis on the part of the cranial nerves and of the extremities, brought out by the use of the dynamometer, rapidity of exhaustion of one or both limbs on one side as compared with the other, and differences in the reflexes, especially the skin reflexes, of the two sides. He also found not only passing, but more or less permanent states of weakness or paresis of extremities after an attack.

Basing his opinion on these clinical findings, he came to the conclusion that 40 per cent. of cases of genuine idiopathic epilepsy show the presence of organic change in the cortex, in another 15 per cent. the indications were doubtful, and in 45 per cent. the cases were negative.

Research of this character is of value, if only in the sense that when painstaking investigation shows an absence of all clinical signs, we can presume that gross cortical changes are not present.

Two opinions can readily be held as to the cellular changes found in the cortex. Clark and Prout's investigations as to the changes in structure of the ganglionic cells of the cortex have been widely quoted as having an important bearing on the pathology of epilepsy. Collins in his case of excision of the cortex found microscopic cellular changes in an apparently normal cortex, and the recent publication of Jelliffe's experiments indicate that cellular changes of a widespread character may underlie these apparently idiopathic cases. Osler, on the other hand, believes that these minute changes can result from and be caused by the epileptic seizures themselves, resulting from the circulatory changes which accompany the attack. This is further supplemented by the angioneurotic theory of Quinke, who supposes

that a local spasm of a small vessel may cause the focal symptoms in local idiopathic epilepsy. Perhaps the explanation may be found along the lines of work of Spiller, McCarthy, and others, who found minute areas of softening, perhaps the result of capillary embolism in uremic and arteriosclerotic conditions. Redlich, Chaslin, Bleuer, Buchholz, Altzheimer, Weber, Orloff, believe that we may have localized increase of the neuroglia tissue. On the other hand, Weber has shown that this localized increase of the neuroglia tissue does not bear any constant relation in its cortical location to the point of origin of the muscular spasms.

That the lesion, whatever it may be, is not necessarily progressive is shown by the course of the majority of the above-cited cases.

In considering the surgical aspect of these cases, I wish to confine myself entirely to the discussion of cases of the above type, namely: localized idiopathic epilepsy.

It resolves itself therefore into two questions, first, the question of an exploratory operation, which necessarily means the opening of the dura mater and the electrical examination of the cortex, and secondly the excision from the cortex of the area corresponding to the seat of the initial convulsion.

The surgical opinion on this subject is pretty well expressed by Roswell Park and J. Chalmers Da Costa.

Roswell Park, in speaking of the surgery of epilepsy, divides the appropriate cases into three groups: (1) Those presenting reflexes from peripheral irritation, (2) those presenting gross anatomic lesions within the cranium, (3) cases of so-called genuine epilepsy of non-traumatic origin presenting localizing phenomena.

In our discussion, the third group alone is of interest. It covers a wide group of cases and makes no distinction as to clinical findings.

J. Chalmers Da Costa says: "That a correct decision cannot be reached intuitively, a conclusion must not be jumped at. An opinion to be of value must emanate from some one who possesses a scientific knowledge of epilepsy and has examined the patient with painstaking care." Concerning idiopathic focal epilepsy he says: "If from the very start the convulsive attacks have a local beginning and if the surgeon has seen the case within two years of the first attack, it is proper to trephine and excise

the irritated portion of the cortex. This is true whether the convulsions remain partial or spread to the rest of the body, whether consciousness is lost or not. He advocates operations in all cases whether the convulsions are local in the beginning as well as in cases of general convulsions which afterward assume a local character, if the attacks are very numerous, are uninfluenced by ordinary treatment and in any way threaten life. On the other hand, Horsley has lately expressed the opinion that idiopathic focal epilepsy is not amenable to surgical treatment.

In considering the advisability of surgical interference, therefore, we must be absolutely satisfied that we have ruled out reflex epilepsy, the motor aura and hysteria. According to Starr, 23 per cent. of all cases show a local or focal origin.

The first point to be considered is the exploratory operation. This involves necessarily the opening of the dura, the examination of the cortex, both mechanically and electrically. In the first place the operation itself has quite a large mortality in idiopathic cases where there is no intracranial pressure. In cases of this kind Horsley lost one case out of five, viz.: 20 per cent. Matthiolius' statistics show a mortality of 24 per cent. When we consider that the disease for which the operation is performed is mild and rarely terminates fatally, the above results should make us halt and consider. As to the actual mortality in operations in a large number of idiopathic cases of focal epilepsy, I have not been able to find statistics. If the cortex is not excised it cannot be examined without causing some local damage, and this damage is not necessarily confined to the immediate area involved, but the surrounding cortex must necessarily be explored; then we always leave a local imperfectly repaired dura mater, danger of adhesions between the cortex and membranes, and finally a large bony defect. In a word, the operation itself substitutes for at most microscopic defects of the brain gross surgical defects and local contusions and hemorrhages in the cortex. Rasumowsky, however, states that not one of his seven cases was damaged mentally or physically by the ablation of the cortex.

Let me introduce a case here which serves to illustrate the dangers of surgical interference.

Case XIII.—Mrs. W., æt. 34, married, six children.

Has been of a nervous disposition; some years ago had an

injury, a piece of iron fell from a tree and struck her on the left side of head. Was unconscious for a few minutes. Later complained much of headache and developed some weakness in the right side of body, with loss of sensation.

On the supposition that there was a depression or splintering of the inner table, patient was trephined over the left parietal region. The operation was successful, but no defect of bone, except an unusual thickness, was found.

The symptoms remained unchanged and a second operation was performed, another section of bone was removed. The headache and weakness and loss of sensation on the right side remained unchanged. Patient became pregnant and during delivery had a severe general convulsion. Gradually she lost vision in left eye.

Later on she developed convulsions in the right arm, which extended to right side of face, and then became general after losing consciousness.

A third operation was performed, a large area of bone removed but nothing was found except a normal cortex.

After third operation patient had more convulsions, the vision of left eye was entirely lost and the headaches continued; there was a weakness of right arm and leg with loss of consciousness. Patient was brought to Cincinatti in this condition and placed in the Good Samaritan Annex for observation.

There is no vomiting nor vertigo, constant headache for past seven weeks.

Examination.—Mental condition very changeable, exaltation varying with depression.

No optic neuritis, left papilla unchanged. But patient cannot see with left eye. Left pupil is larger than right.

Loss of sense of smell on right side, weakness and increased reflexes of right side, but no Babinski, Oppenheim, or ankle clonus. Sensation in all of its qualities is lost on right side of body, including the face. There is hemianesthesia, hemianalgesia, thermanesthesia, loss of muscle sense and astereognosis.

After ten weeks all the symptoms disappeared with the exception of fainting spells; since her return home eighteen months ago, patient has born another child and has had three general convulsions.

This case is instructive in several respects. The paralysis of the right side of the body was hysterical in character and was presumably the same before the operation; at least, one of our best neurologists who saw her before and after the third operation was convinced after the operation that the paralysis and loss of sensation were hysterical. It yielded to suggestive treatment. In the second place, the epileptic seizures occurred only after the second operation; there is no doubt as to the genuine character of the epileptic seizures, and, to say the least, if the original

injury was the cause of the epilepsy, the extensive surgical interference failed to find a cause and failed to prevent the development of both the Jacksonian and genuine epileptic attacks. My judgment of the case is, that the surgical interference was futile and ineffectual, to say the least.

What is the experience as to excision of the cortex? We must remove from consideration at once all these cases whose point of origin seems to be in the special senses, especially those with initial visual and speech symptoms. This leaves for consideration only those with initial sensory or motor symptoms. The sensory cases can also be dismissed on account of our imperfect knowledge of sensory localization of the cortex and the impossibility of verifying the center involved unless we follow Cushing's suggestion and use no anesthetic. However, there could not be sufficient accuracy to justify cortical excision in these cases. This narrows down the field of operation to cases with motor manifestations. We know that some of the earliest experiments in epilepsy were those of Hitzig, who produced epilepsy in dogs by excision of portions of the cortex of the motor area. Jacksonian epilepsy followed, but soon the convulsions became general, losing the Jacksonian type, beginning with initial loss of consciousness. Moreover, the experiments of Munk and Sherrington on apes has been apparently verified in man. While most operators have reported a quick disappearance of the paresis and paralysis following excision of the motor area, Da Costa says: "That the finer movements of the fingers are never regained."

Another question arises in these cases; even though we should decide to extirpate the cortex, we can never be sure that the source of irritation is the region indicated by the origin of the convulsions.

Drivet (These de Bordeaux) collected thirty-five cases, from the literature, of Jacksonian epilepsy beginning in the face or arm; out of thirty-five cases twenty-six times the lesion was in the motor area, but not always in the area indicated (viz., the face area) nine times outside of the motor area and twice in the cerebellum.

Cramer and Weber publish a typical case of Jacksonian epilepsy caused by atrophy of the cerebellum.

The above are the objections to the excision of the cortex based upon experimental and clinical observations. It seems to me that any one of the above cases, with a normal condition of

the hands and face, for instance, is better off than if these regions were left permanently paretic or deprived of the finer functions of coördinate movement.

Now what are the surgical results in the above class of cases as gleaned from tables of statistics?

There are very few cases on record of excision of the cortex in idiopathic localized epilepsy. Bergman, Dejerine, Braun, Horsley and Gowers, Raymond, Donath, Putnam, Pick and others are opposed to the operation, Collins and Sachs are in favor of it. Da Costa says that only one per cent. of cases are cured. Braun collected thirty cases of ablation of the cortex in traumatic cases, thirteen were reported cured, but only three had been observed more than three years. Braun says that there is not a single case of operation for focal idiopathic epilepsy with ablation of the cortex, which has been under observation more than three years (up to 1899).

Mattheolius has been able (up to 1899) to collect fourteen cases of the above character, viz., idiopathic focal epilepsy, in which the cortex was excised with but one cure, and this case had not been observed more than two years.

Rasumowsky publishes two cases with brilliant results. These, with Collins' case, certainly hold out some hope. Nast-Kolb's tables bring up the statistics to 1904; of eighty-six cases, fourteen were apparently cases of idiopathic focal epilepsy in which the cortex was removed, in eleven of the cases after electrical investigation. In five of these cases the freedom from convulsions lasted for from three to twelve years, a result which may once more raise our hopes.

Raymond, basing his views upon a review of thirty-one collected cases of Jacksonian epilepsy with only three cures, is opposed to the operation. He bases his opposition on the presence of the epileptogenic condition being present beyond the seat of origin in the cortex, a widespread change, which is present in a quantitative manner to a greater degree in the affected zone, but nevertheless present over the entire cortex. Horsley says that no general laws can be laid down, that it is impossible to argue as to the surgery of epilepsy except as to individual cases.

My own judgment, based upon the experience of cases operated upon and those not operated upon, both being observed for a long time, is, that the presence of localized convulsions alone should never be an indication for an operation. These individuals, in

idiopathic focal epilepsy, already possess the epileptogenic changes (Jolly) of the cortex in areas beyond the first focus of discharge. The removal of that focus will not change the remaining areas of the cortex. When the attacks have been general and frequent, with complete loss of consciousness, no cure can be expected from any surgical operation. If, after prolonged observation, there is no marked progress of the disease and the case is more or less amenable to medical treatment and no localizing physical signs develop, I believe that no surgical measures should be resorted to.

If, however, the case rapidly becomes worse in the sense of numerous attacks of a violent character and the individual's life work is handicapped by the disease and if the medicinal treatment fails to give relief, an exploratory operation should be recommended in the hope of finding some local disease of the brain or its coverings. Whether the cortex should be removed is a debatable question, although the statistics of Nast-Kolb would seem to be favorable to the operation. In gross lesions, I believe we can say that the number and severity of the attacks are diminished and made more amenable to treatment after the operation.

Under the above restrictions Da Costa advocates the ablation of the cortex in idiopathic focal epilepsy, stating that after the operation the attacks are less numerous and more amenable to treatment, even though no absolute cure is effected.

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DELAYED APOPLEXY (SPAETAPOPLEXIE) WITH THE REPORT OF A CASE[1]

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In 1878 Duret (1) published his great work, "Études expérimentales et clinique sur les traumatismes cérébraux," founded upon seventy-one experiments on the lower animals, many of which are irrelevant to the subject at hand. The first chapter deals with twenty-five experiments, mostly on dogs, in which the cranium was subjected to one or more blows, or in which wax or some other substance was injected into the cranial cavity through a small trephine opening, in this way producing a cerebral compression. The future effect of this work was so far-reaching that it seems to me advisable to quote a pertinent part of his conclusions: "Au moment d'une chute sur la tête, ou par un coup sur le crâne, un *flot* de liquide est formé autour des hémisphères et dans les ventricules, qui répercute la violence subie en un point, dans toutes les régions des centres nerveux, et plus particulièrement au niveau du bulbe rachidien.

"L'action vulnérante du flot aqueux exerce ordinairement ses effets les plus graves et les plus étendus, dans les lacs arachnoïdiens de la base du cerveau, autour du collet du bulbe, et principalement au niveau du plancher bulbaire, et sur les corps restiformes." [Translation: "At the moment of a fall on the head or a blow on the cranium a flow of liquid is formed about the hemispheres and in the ventricles which reflects the violence

[1] Read at the thirty-fourth annual meeting of the American Neurological Association, May 20, 21 and 22, 1908.

sustained at one point, in all the regions of the nervous centers, and more particularly at the level of the medulla oblongata.

"The damaging action of the aqueous flow ordinarily exercises its most grave and extended effects in the arachnoidal spaces at the base of the brain, about the collet of the medulla and particularly at the level of the floor of the ventricle and on the restiform bodies."]

Although many times there was shown at autopsy a damaged or hemorrhagic condition of the floor of the fourth ventricle, there were nevertheless other considerable lesions in the brain well removed from the locality. This work of Duret's was followed in 1880 by an article by Gussenbauer (2) in the main confirmatory in tone of Duret's conclusions. He gives a résumé of theories held prior to Duret's experiments which is interesting historically. For three years he conducted experiments on the heads of cadavers in order to arrive at some conclusion as to the mechanics of concussion of the brain. To this end he made a number of trephine holes in the skull, all in one direction, and then introduced deeply into the brain substance pieces of straw, sticks of wood and long Karlsbad insect needles. He found that when he struck the head in the direction of the trephine openings there was a movement in the brain substance of the introduced bodies, but when the direction of force was not in the long axis or axis of insertion of those bodies, they remained stationary. Now experiments of this kind fail to reproduce the physical hypothesis of the living animal, on account of the changes in tissue after death. Moreover, these three years were spent in proving what is so obvious that it is practically an axiom, *i. e.*, it is easier for a body to move through a medium in the direction of its long rather than its short axis, or to use a simile, a boat makes easier headway if it go either forward or backward than if it move sideways.

In speaking of general convulsions immediately following a trauma to the head, as in Duret's experiments, he says: "Sie sind die Folge der mechanischen Reizung, welche der durch die äussere Massenbewegung hervorgerufene Strom in der cerebrospinal Flüssigkeit am Boden des 4 Ventrikels, insbesondere aber in den sensiblen Corporibus restiformis bewirkt." [Translation: "They are the result of the mechanical irritation, which the stream of cerebrospinal fluid, caused by the external massive

impulse, works on the floor of the fourth ventricle, but particularly on the sensory restiform body."] How he reaches this conclusion he does not say and I fail to see. Another statement which is hardly borne out in cases of lesions of the floor of the fourth ventricle in man is: "Langere Zeit andauernde Muskelcontractionen in dieser ersten Periode der Erscheinungen, deuten nach den Untersuchungen Duret's durchwegs auf erheblichere Verletzungen am Boden des 4 Ventrikels." [Translation: "Persistent muscular contractures in this first period of the symptoms point, according to Duret, to important injury to the floor of the fourth ventricle."]

In discussing unconsciousness in trauma to the head Gussenbauer holds that at the time of and immediately following the injury there is a decrease in pressure in the veins and an increase of pressure in the arteries, possibly due to general arterial contraction. This condition produces a temporary anemia, which in turn leads to unconsciousness. As soon as the circulation is restored to normal, consciousness returns. In severe cases there follows a reflex arterial paralysis which leads to venous stasis. This can lead to a second loss of consciousness. The reason for dealing with these views of Gussenbauer will be more apparent later on.

In 1891 Bollinger (3) advanced his great hypothesis on late apoplexy. This was received by all practically without dissent, and so closely is his name identified with this reasoning from cause to effect that one frequently meets the term *Spätapoplexie Bollingers*. In his article he cites four cases, all the subjects of injury to the head. After a latent period, during which symptoms were either absent or inconspicuous, the late apoplexy came on. Bollinger's hypothesis is summed up as follows: First, a trauma to the head; second, a degeneration, particularly necrotic softening, in the cerebrum, pons or medulla; third, an alteration of blood vessels secondary to the necrotic softening; finally, the apoplexy due to three factors: (*a*) alteration in the vessels, (*b*) the weakening of the tissue immediately surrounding the vessels, thereby decreasing their resistance to internal pressure, and (*c*) the raising of arterial pressure.

For twelve years Bollinger's views won nothing but acceptance and approbation.

In 1903 Langerhans (4) published his monograph. This

revolutionary work, a most brilliant critique, might be said to have for its text the following, which I quote: "Da Bollinger der geistige Vater der traumatischen Spätapoplexie ist, so lege ich damit gleichsam die Axt an die Wurzel der ganzen Lehre und setze mich in Gegensatz zu der allgemein herrschenden Anschauung." [Translation: "Because Bollinger is the spiritual father of the Traumatic Spätapoplexie, therefore I lay at once the axe to the root of the entire doctrine and set myself in opposition to the generally prevailing opinion."]

He first takes issue with Bollinger on account of the title of his paper: "Ein Beitrag zur Lehre von der Hirnerschütterung." He calls attention to the fact that Bollinger bases his arguments on Duret's experiments, in which, after blows on the head, there were found small punctiform hemorrhages in the aqueduct and immediate vicinity situated just beneath the ependyma, frequently the ependyma being torn. Bollinger thought that he had bridged the gap between these experimental findings of Duret's and concussion of the brain. Langerhans says, in the first place, Bollinger's cases failed to show the symptoms of concussion. Moreover as concussion does not necessarily entail the changes in the aqueduct above mentioned, and as these changes are by no means constant in their connection with the symptom complex, therefore one must come to the conclusion that these changes are collateral in nature only, and in no sense form the anatomical basis of concussion. For these reasons Langerhans very properly concludes that Bollinger's title is a misstatement.

In dealing with Bollinger's cases Langerhans' criticisms are as follows: In Case I the fact that there was a considerable fracture of the skull with meningeal hemorrhage makes it extremely doubtful what part if any the lesions of the aqueduct played. This is particularly emphasized by the fact that the microscopical examination of the brain stem was meager in the extreme. In Case II there was found in the brain no alteration of the blood vessels, no area of softening and no traumatic degeneration to account for the hemorrhage. In Case III the injury must have been one and a half hours before death or else is merely supposititious; there was no alteration of blood vessels and no area of softening. In Case IV there was no *Spätapoplexie* and no microscopical examination of the isthmus. There was, however, an area of softening in the isthmus.

The utter demolition of Bollinger's views is so complete that Langerhans' simile, "die Axt an die Wurzel," is well taken. How it has been possible for the medical fraternity, usually so exacting in their demands when anything new is advanced, to accept without question these utterly fallacious lines of reasoning, the glaringly inadequate data; how it has been possible for twelve years to pass before the man comes forward to fairly riddle the preposterous structure of pseudo-reason, are things which must ever remain wonderful and inexplicable in the history of medicine.

Langerhans' discussion of Seydel's (5) case is masterly. He comes to the conclusion that the patient did not suffer a traumatic late apoplexy (Spätapoplexie Bollingers), but that on account of the hypertrophied heart and the arterial condition, miliary aneurismata most likely being present, the injury to the head was enough to cause an increase of blood pressure sufficient in force to burst one of the lenticulo-striate arteries.

From a study of the case of Maurermeister Lorenz Gerbl (6), which Bollinger thought one of Spätapoplexie, Langerhans draws the following conclusions: "(1) L. G. an spontaner Hirnblutung gestorben ist; (2) ein Zusammenhang der Hirnblutung des L. G. mit dem Hinfallen, dem angeblichen Unfall nicht bewiesen und auch nicht wahrscheinlich ist." [Translation: "(1) L. G. died from spontaneous hemorrhage into the brain; (2) a connection between the hemorrhage into the brain and the fall, the alleged accident, is not proven and also not likely."] These statements seem altogether too strong to me. When one suffers from an apoplexy, the immediate cause of the broken artery being an increased blood pressure, and the increased blood pressure having been preceded by such an accident as L. G. suffered, it is questionable whether the term "spontaner" can be used with accuracy. Also the microscopical examination of the brain was such that miliary aneurism cannot be excluded and therefore I think Langerhans' second conclusion too strongly stated.

In 1903 also appeared the work of Stadelmann (7). This paper is particularly valuable on account of three conditions which he advances, coincidence with which he holds as necessary to the correct diagnosis of late apoplexy. They are important enough to quote verbatim. "(1) Der betreffende Kranke muss nachweislich vorher gesund gewesen sein, keine Zeichen von Gefäss-

veranderungen dargeboten haben, Lues, Nephritis, Potus, Herzerkrankungen müssen ausgeschlossen sein, auch darf es sich nicht um ältere Leute handeln, die sowie so schon an arteriosklerose leiden können, resp. bei denen sie sich spontan entwickeln kann. (2) Das Trauma muss erheblicher gewesen sein, wenn es auch nicht nöthig zu sein scheint, dass es direkt zur Bewusstlosigkeit geführt hat. (3) Die Erscheinungen der Gefasserkrankungen, resp. der weiteren Gehirnerkrankung müssen sich in kürzerem Zeitraume und unter unseren Augen entwickelt haben. Liegen erst Jahre dazwischen, in denen die ärztliche Beobachtung fehlt, so werde ich mich nie entschliessen können, ein irgendwie bestimmtes Urtheil über den Zussammenhang des Trauma mit den jetzt zu beobachtenden Erscheinungen abzugeben." [Translation: "(1) The patient concerned must without question have been in good health, no signs of alteration in blood vessels having existed, syphilis, nephritis, alcohol and heart disease must have been excluded; also old people who so frequently suffer from arteriosclerosis cannot be considered; in other words, those who can develop it (apoplexy) spontaneously.

"(2) The injury must have been considerable, although it does not seem necessary that it should have caused unconsciousness.

"(3) The symptoms of the vascular or brain lesion must have developed within a short space of time and under our own eyes. If years have intervened in which there has been no medical surveillance I cannot determine with any kind of precise judgment a relationship between the trauma and the symptoms now appearing."]

According to Stadelmann, the development of a late apoplexy is as follows: (*a*) Disturbance of circulation; (*b*) softening, and (*c*) late hemorrhage in the softened area of the brain.

Inferences drawn from cases such as presented by Bohne (8), Wimmer (9), and Rupp (10), in which the patients did not come to necropsy, are all of doubtful value.

Kurt Mendel (11) divides cases of traumatic late apoplexy into two classes: Class A, those cases in which the vessel in question is the seat of an arteriosclerotic process at the time of injury; Class B, those cases in which the trauma causes a disease of the wall of a blood vessel, previously normal.

(*ad* A.) The trauma either causes an immediate rise in blood pressure, in which case we have a traumatic apoplexy, or it causes

an increase in blood pressure which takes place some time afterward, this leading to the late apoplexy. He says that any trauma, even though it does not directly affect the skull, can cause an hyperemia of the brain. Also an emotional shock, such as sudden fear, can cause a vasomotor disturbance with increased blood pressure.

(*ad* B.) He advances the following theories:

1. On account of softening in surrounding tissue the vessel dilates and its walls become thin.

2. The vessel wall takes part in the concussion and suffers from fatty degeneration.

3. Miliary aneurisms are formed on account of changes in the walls of blood vessels.

4. A disturbance in the nutrition of the walls of the blood vessels secondary to circulatory disturbance with consequent formation of aneurisms.

5. A certain relation exists between accident (trauma) and arteriosclerosis, especially of the vessels of the brain and spinal cord.

Kurt Mendel has watched the development of arteriosclerosis after trauma. He has seen patients who have suffered a traumatism of one side in which headache has been confined to the injured side and in which the temporal artery of that side has become tortuous and hard. His final conclusions are that whereas the clinical history shows a clear relation between trauma and late apoplexy, pathology has, up to the present, failed to do so. He thinks that the cases in which late apoplexy are caused solely by trauma without any preëxisting arterial degeneration are exceedingly rare.

Late apoplexy is found in literature according to Kurt Mendel in patients from seven to seventy years of age. The latent period, the time elapsing between the injury and the apoplectiform seizure, can vary from four days to nine months. Usually this latent period is from one to six weeks and can be absolutely free from symptoms or show mild mental hebetude and some headache.

I am indebted to Dr. William G. Spiller for the history and pathological material of the following case:

Annie M., white, female, aged 36 years, occupation, cook; presents the following history: Father is living and well; mother is dead, cause unknown. Two sisters and one brother living and

well. No brothers or sisters dead. No history of tuberculosis or malignant disease. Her family were of nervous and excitable temperament. The history of diseases of childhood could not be elicited. There is a history of influenza sixteen years ago, also anemia when a child. Fourteen years ago she was operated upon for some uterine trouble the exact nature of which is not known. She has had four children, the youngest of whom is ten months old. One child died of diphtheria. No miscarriage and labors normal. Two years ago she became suddenly violently insane and had to be confined in an institution for three months, at the expiration of which time she had recovered sufficiently to be discharged. Her husband states that she frequently complained of nervous headaches, although her general health had been good. She never used alcohol immoderately. All venereal history was denied.

On February 27, 1907, the patient had a quarrel with a neighbor and was struck repeatedly, ten or fifteen times, over the head and face with the fist. From this time she began complaining of severe headache which was not localized. Ten days after the quarrel, while she was lying down with her baby, her husband heard a sound as if something had fallen and found that she had dropped her baby to the floor and was lying with the right arm and leg completely paralyzed and was motor aphasic. She was not unconscious and had lost control of her bladder and rectum.

She was admitted to the hospital March 12, 1907, in a semi-stuporous condition. The one word she was able to say was "No." There is a history of cough, expectoration and pain in her chest between the time of her injury and her apoplectiform seizure and her family physician said in this time she had pleurisy and probably pneumonia.

As she was completely motor aphasic except for the single word "No," and as she used this word in answer to every question, it was hard to determine whether she was word deaf, but the chances are that she was not; if asked whether her name was Annie she would say "No." On two occasions she gave her left hand when asked, but it was impossible to get her to do anything else. She had a right facial paralysis of central type. She opened and closed both eyes firmly and equally. It was impossible for her to protrude her tongue but it deviated to the left while in the mouth. The biceps and triceps reflex were not very distinct on the left side, but were much more distinct on the right. Pin prick caused pain over the entire right side. Once or twice the Babinski reflex seemed to be obtained on the right, but it was not constant. Patellar tendon and Achilles tendon reflexes were present and equal on each side.

On March 15 it is recorded that "her lungs were full of fluid and her pulse extremely rapid."

The patient died on March 18, at 8 A. M.

Sections of the spinal cord at the level of the fourth lumbar and fourth thoracic segments were stained by the Weigert hematoxylin, hemalum-acid fuchsin and von Lenhossék-Nissl methods, and showed no pathological alteration. The amount of central glia substance in the fourth lumbar segment was much greater than is usually seen. An examination of sections in the mid-olivary region showed that the ependymal lining of the fourth ventricle was, at this level, in perfect condition. Many of the blood vessels in the sections from the mid-olivary level presented a lumen packed with erythrocytes. The perivascular lymph spaces were greatly distended and filled with an unstaining detritus which in several instances could be seen to contain erythrocytes, but by far the largest part of this detritus was albuminous coagulum. In several instances the point of escape from the vessel of the erythrocytes could be detected. This condition of affairs was not limited to the immediate subependymal part of the floor of the fourth ventricle, but is also seen far anterior to this, one such vessel being located in the hilus of one of the inferior olives. In this latter case blood pigment was found in the detritus filling the lymph space. A section at the level of the decussation of the fourth cranial nerves showed a few of these distended vessels in the neighborhood of the aqueduct. But the chief point of interest here is an area anterior to the decussation of the superior cerebellar peduncles. This area is located in the mid-line and shows several large blood vessels with the surrounding unstained detritus and in addition a pronounced hemorrhagic condition. This lesion is easily seen with the unaided eye and measures approximately two millimeters transversely by one millimeter antero-posteriorly. The ependymal lining of the aqueduct at this level is unaltered. A section through the cerebral peduncles at the level of the mammillary bodies and the middle of the red nuclei shows a slight tearing at one point of the ependymal lining of the aqueduct and also a microscopical hemorrhage into the ependymal lining at another point. Here, as in sections lower down, one finds the distended perivascular lymph spaces but not in so great numbers. A section through the optic chiasm and anterior part of the optic tract shows many erythrocytes free in the ependymal lining of the third ventricle. The chiasm, optic nerve and optic tracts are normal.

In the left lenticular region sections were made from blocks of tissue, the highest level of which corresponded to a plane passing just above the highest part of the lenticular nucleus. Over four hundred and fifty sections were made, the lowest plane being reached when the middle cerebral artery was cut longitudinally. Sections were stained at different levels of the series by means of the Weigert hematoxylin, Van Gieson's, Weigert's elastica and hemalum-acid fuchsin methods.

The lowest sections, those having the middle cerebral artery

cut longitudinally, show the small vessels filled with blood. In some instances there is a tearing of the intima and media with a passage of erythrocytes into the perivascular lymph spaces. In some of the small vessels there is thrombus formation and the presence of fatty granular cells about the vessels. As successive levels higher up are examined, the occluded vessels are of more frequent occurrence and are located chiefly in the globus pallidus and putamen of the lenticular nucleus. There are found also a few occluded vessels in the optic thalamus. At about the mid-level of the lenticula there is found in the globus pallidus a vessel, the muscular portion of whose wall shows calcification. This is the only vessel which I have examined in this case which showed this process. Within the lumen there is seen an hyperplasia of the endothelial lining. At about the mid-level of the lenticula there is found a process of softening which is chiefly confined, at this level, to the globus pallidus and in which are frequently seen fatty granular cells. At the highest level of the lenticula the area of softening has greatly increased and extends well through the white matter external to the caudate nucleus and optic thalamus for a distance antero-posteriorly of about five centimeters. There are also small areas of softening in the caudate nucleus.

Sections of the right and left paracentral lobules were stained by the von Lenhossék-Nissl method. The large motor cells of the right side were normal but those of the left side showed marked degenerative reaction.

Among other things the general post-mortem notes state that the cortical epithelium of the kidney presents a moderate degree of cloudy swelling. There was some general kidney congestion and a slight increase of connective tissue between the tubules was noted. There seems to be no evidence from the histological examination of undue arterial change.

Cases of this kind are particularly interesting from a medico-legal standpoint. There are so many instances on record of trauma to the head followed after a greater or less time by late apoplexy that we may be morally certain that a definite relation between trauma to the head and the apoplectiform seizure does exist. But are we in possession of every link in the chain of pathological circumstances so that we can swear on the witness stand that a given case of apoplexy is a late apoplexy due to the trauma? This question must be answered unqualifiedly in the negative.

My conclusions, drawn from the above case, together with the many on record, must of necessity be theoretical and are as follows:

(*a*) Traumatic delayed apoplexy (Spätapoplexie), in the sense of the original Greek, ἀποπλήσσειν, is in all probability, an entity.

(*b*) Delayed apoplexy is not of necessity a condition in which hemorrhage takes place, but the stroke can have as its immediate etiological factor the occluding or thrombosis of one or more arteries.

(*c*) The cerebro-spinal fluid does not play a necessary part in the production of delayed apoplexy and injury to the region of the aqueduct and fourth ventricle is a collateral circumstance of no etiological moment.

(*d*) In cases of delayed apoplexy in which hemorrhage takes place, the hemorrhage is not necessarily preceded by a process of necrotic softening about the artery in question, this removing the outside support (Widerstandsfähigkeit) but the artery itself is injured as Langerhans holds, and the secondary rise in arterial pressure, or the normal pressure causes the hemorrhage.

(*e*) The mechanics of many cases of delayed apoplexy is as follows: The trauma to the head causes a mechanical agitation to the brain substance, which falls with greatest severity on the arteries, small and large, they being filled with an incompressible fluid. The particular location of the chief action on the vessels cannot be determined by the external impact of the blow or the direction of the force and is impossible of determination until revealed by symptomatology. At first there is in all probability a general vasomotor constriction of the cerebral arterial system followed very shortly by a paresis of the vessel walls. The vessels particularly injured undergo endothelial proliferation, and thrombotic processes are set up. Then occlusion, if in a functionally important area of the brain, can cause an apoplectic attack. To this class belongs my case.

In considering a case of what may be traumatic delayed apoplexy, a possible incompetence on the part of the kidney must be borne in mind and the action of a consequent uremia must be given full weight. The case I report had a slight amount of chronic interstitial nephritis and had she not come to necropsy one could not have positively stated whether there was a hemorrhagic or thrombotic condition on the one hand, or a uremic attack.

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THE SYMPTOM-COMPLEX OF A LESION OF THE UPPERMOST PORTION OF THE ANTERIOR SPINAL AND ADJOINING PORTION OF THE VERTEBRAL ARTERIES[1]

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In the June, 1908, number of this journal I described the symptom-complex of occlusion of the posterior inferior cerebellar artery, as it appeared to me from a microscopical study of the material from two cases and the reports of cases in the literature. The symptom-complex is sufficiently characteristic to make a diagnosis easy. In the paper referred to the following occurs:

"Wallenberg sums up Duret's investigations on the supply of the vertebral arteries. Each of these arteries, the left the larger, gives origin about two cm. below their union to the posterior inferior cerebellar artery, and higher to the anterior spinal artery. Branches from the latter artery enter the raphe and nourish the interolivary bundles, posterior longitudinal bundles, hypoglossus nuclei and other nuclei below the floor of the fourth ventricle. The pyramids are nourished by the anterior spinal arteries and frequently by the vertebral arteries. [It is understandable therefore that when the anterior spinal arteries are not occluded the central and anterior parts of the medulla oblongata are not affected.] Branches from the posterior inferior cerebellar or vertebral artery supply the lateral portion of the medulla oblongata."

It has seemed to me desirable to emphasize a clinical picture—the complement of that caused by occlusion of the posterior inferior cerebellar artery—alluded to in my former paper and in my concluding remarks on the discussion of this paper (see this journal, p. 713); especially as I believe I have observed cases

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that belong to this group. I hope that this brief article may arouse sufficient interest to make the symptom-complex a source of observation and study by others.

The anterior spinal arteries arise from the vertebrals about a centimeter before the latter unite to form the basilar. The left anterior spinal artery is frequently the larger, and according to Duret the right may be absent. I have found that these arteries sometimes descend several centimeters before uniting, in other instances they unite shortly after their origin to form one vessel. Many of the fibers of the hypoglossus nerves leave the medulla oblongata above the origin of these arteries. Branches of these arteries supply the median portion of the medulla oblongata, especially the lemniscus and the anterior pyramids, although the latter may be nourished also from the vertebral arteries. Duret believed that branches of the anterior spinal arteries supply the hypoglossus fibers, but it seems probable to me that this nerve as well as the vago-accessorius is nourished more from the vertebral arteries. I have seen complete absence of the right vertebral and anterior spinal arteries.

The important fact is that the area of supply of the upper part of the anterior spinal arteries is chiefly the anterior and middle portions of the medulla oblongata. Occlusion of these arteries or hemorrhage from them, including the adjoining part of the vertebral arteries, should therefore give a very definite symptom-group. This may be unilateral or bilateral, depending on the union of the two arteries near their origin or several centimeters below. As these arteries are side by side shortly below their origin, even when they do not at once unite, the symptom-complex is likely to be bilateral; whereas in occlusion of the posterior inferior cerebellar artery it is always indicative of an unilateral lesion.

We should expect to find paralysis of all the limbs, trunk and neck, whereas the reflexes necessary to life would be preserved. The face would not be affected. The tongue might escape, as the hypoglossus nerve has at least a portion of its origin above the origin of the anterior spinal arteries, but it might be involved by implication of the vertebrals. As the lemniscus probably conveys fibers of deep sensation, this form of sensation would probably be disturbed. Henschen believes the lemniscus contains tactile fibers. If this opinion be correct tactile sensation would be

affected. If the symptom-complex were unilateral the disturbance of sensation referred to and that of motion would probably be on the same side of the body, as all the fibers concerned with these functions have decussated at the level in question. If the lesion extended lower sensation might be affected on the side opposite to that of the disturbance of motion, or even on both sides of the body. The tendon reflexes are not infrequently lost when the cerebellum or fibers connecting with it are involved, and as in the lesion under consideration the cerebello-olivary fibers would be implicated, it is possible that the tendon reflexes would be lost instead of exaggerated, although this loss might be due to other causes. The vagus and glossopharyngeus should escape, at least in great part. Ataxia probably would not be observed because of the motor paralysis or the escape of important cerebellar fibers.

Occlusion of the vessels in the posterior part of the brain is more likely to occur in syphilitic arterial disease, and transitory quadriplegia might be caused by temporary interference with this vascular supply.

I am well aware that a certain degree of uncertainty is to be expected in the description of a symptom-complex such as this without sufficient clinical and pathological material, and I have delayed the publication of this article many months. The anatomical foundation seems to be reliable, and I am not without some clinical observation. It remains to be seen whether the symptom-complex could be unilateral, as both the motor fibers and the sensory fibers of the lemniscus decussate at about the level under consideration.

I have been able to study a case in the service of Dr. Mills that probably belongs to this type: A woman, forty years of age, previously in good health, attempted to rise from her bed during the night, in April, 1908. She fell to the floor and had the sensation of having no legs, as she expressed it, these limbs feeling numb and dead-like. She was unconscious about two days, and then understood what was said to her, but could not speak for about three weeks. She had incontinence of urine and feces only during the first few days of the attack. She has had a dull aching, tired sensation in all the limbs since the attack. Her speech has been peculiar in that certain words are indistinct and she has difficulty in finding the word she wants at times, and

yet she does not appear to be aphasic. She is distinctly weak in all her limbs and equally so in the limbs of the two sides, although the upper are weaker than the lower. The grip is much impaired on both sides, and the fingers are held in partial flexion, but can be straightened by the patient with difficulty, although the wrists remain slightly flexed. The toes are scraped a little on the ground in walking, especially the right, and the lower limbs are slightly spastic; the upper are not so. The tendon reflexes of upper and lower limbs are exaggerated. There are no localized atrophies, and no incoördinate movements. Sensations of touch, pain and temperature are normal everywhere, but the sense of position is distinctly impaired in the toes and fingers, especially on the right side. Babinski's reflex seems to be present at least on the right side.

The patient is intelligent and is positive that she was in good health before the attack, which occurred suddenly and affected both sides of her body simultaneously and equally. The escape of the tongue and face is noteworthy. The important features of the case are spastic paresis of all the limbs with some disturbance of the sense of position.

Society Proceedings

THE AMERICAN NEUROLOGICAL ASSOCIATION

The Thirty-fourth annual meeting held at the College of Physicians of Philadelphia, May 20, 21 and 22, 1908

The President, Dr. Charles W. Burr, in the Chair.

(*Continued from p. 713*)

Dr. J. Arthur Booth read a paper entitled: Report of a Case of Myasthenia Gravis with Negative Pathological Findings. (See this journal p. 690.)

Discussion.

Dr. N. E. Brill said that the case mentioned by Dr. Booth, which was reported by Dr. Sachs and which occurred in the latter's service at Mount Sinai Hospital, could throw but little light on the etiology and pathogenesis of this disease. However, it was worth reporting as another case of myasthenia gravis pseudo-paralytica with a positive thymus tumor and with a completed autopsy and pathological record. Dr. Brill had seen the patient with Dr. Sachs during life and had examined the organs and sections of the tissues from the autopsy. He could not speak too highly of the work done over these tissues by Dr. Mandelbaum, the pathologist, and Dr. Celler, the associate in pathology, of Mount Sinai Hospital, who reported the results in the last number of the *Journal of Experimental Medicine*.

In this case there was present the small lymphocytic infiltration in the muscles which has been the most constant finding in the reported cases. One should not think that these infiltrations are scattered over every area of the muscles. It requires many sections of muscles, much patience and industry to find these infiltrations. On this account, unless much time was given to the examination, unless many sections of muscles were cut, it would not be at all improbable to miss these infiltrations completely, and the case go on record as one with negative findings.

As to these infiltrations, they were composed of small round cells, with relatively large nuclei, which stain deeply with basic dyes, and look exactly like lymphocytes. They were irregularly scattered throughout the muscles and usually about a blood vessel among the fibers. Similar infiltrations were found in the liver, the adrenals, about a blood vessel in the gray matter near the nucleus of the tenth nerve and one lateral to the olivary body in the white substance of the medulla. The findings of these infiltrations in the central nervous system are the first to be recorded in literature. The infiltrations are usually perivascular and seem to be in the perivascular lymph spaces.

The thymus tumor in this case was very peculiar. It was composed of cells which seemed to arise from the endothelium of the visceral and not the vascular periphery of the perivascular lymph spaces, so that the

tumor would be best designated as a perilymphatic angio-endothelioma, or as one belonging to the group of tumors formerly known as peritheliomas.

As to the thymus in Dr Booth's patient, if it measured, as Dr. Booth reported, 11 cm. by 5 cm., it ought certainly, even at the age of eleven years, the age of his patient, to be regarded at least as a persistent thymus. Dr. Brill said he would consider a thymus of those dimensions at the age of eleven as a *hypertrophied* thymus, because the thymus undergoes the evolutional regression shortly before the age of five years, and under normal conditions should not be much more than one-half the size at that age, as reported by Dr. Booth.

In conclusion he wished to say a word as to positive findings in these cases. We are prone to consider them in the light of causative factors of disease, forgetting that they themselves may be secondary to some undiscoverable factor. The most constant of the gross findings in these cases has been a tumor of the thymus gland; thus in 45 cases reported in literature, a thymus tumor or abnormality occurred in 11. On the other hand myasthenia gravis cases have been reported without thymus involvement, but with tumor formation in other so-called ductless glands; thus, one last year, by Tilney with a tumor of the hypophysis cerebri. In literature, cases with thyroid tumor, and with adrenal tumor, are noted. Taking into consideration these facts we are only justified in suspecting that some toxin arising from perverted function of these glands may be the agent giving rise to the generally distributed muscular lesion of this disease, and that future work in the bio-chemistry of this disease may throw some light on its pathogenesis and nature. Perhaps this conclusion would seem to be the more justified by the analogy existing between this disease and the disease producing sudden death, called variously lymphatism, thymus tod, and thymus death. In the latter the course is very acute, in myasthenia less so. In thymus death, there is a persistent thymus gland in addition to general hypertrophy of the lymphatic structures of the body. The persistent thymus and the lymphatic enlargement on the one hand, the tumor of the thymus and the general lymphocytic infiltrations in myasthenia on the other should be suggestive.

Dr. Spiller said he had had a pronounced case of myasthenia gravis in the University Hospital. On laboratory examination by Dr. Edsall and Dr. Pemberton an excess of calcium chloride in the excreta was found. The relation of this finding to the disease is uncertain.

Dr Booth, in concluding, said in regard to Dr. Brill's assertion that the simple hypertrophy of the thymus in this case was pathological, that he did not quite agree with that statement, as we know that the thymus is present sometimes up to the age of 14 to 16 years. He has seen autopsies confirming this without any evidence of symptoms such as are present in myasthenia gravis. In regard to Dr. Spiller's remark concerning the urine, Dr. Booth had the urine carefully examined and there was absolutely nothing abnormal. Also the blood was examined with negative results. It suggested to him that perhaps there was some connection between the operation for adenoids and the onset of the myasthenia gravis. Whether the removal of adenoids has anything to do with the subject, he is not prepared to state, but in the case reported these symptoms all came on after the operation for the removal of the adenoids. Whether the exposure of that raw surface permitted some toxines to enter or whether the adenoid tissue has some protective influence he is not prepared to state.

Dr. S. W. Mitchell, Philadelphia, read a paper entitled: Rest Treatment in Relation to Psychotherapy.

Discussion.

Dr. F. X. Dercum, Philadelphia, said it seemed a work of supererogation to attempt to discuss this subject after so full, so brilliant, so satisfying a presentment. To us in Philadelphia the story of the rest treatment is not a new one. It is one which is a source of local pride to us and of individual and personal satisfaction. We laugh at any suggestion that the method was not original with Dr. Mitchell. Its originality consisted not only in the combination of the various procedures employed, but especially in the substitution of physiological methods for medicines in the treatment of disease. This fact, which constituted a true innovation in therapeutics, cannot be too strongly emphasized.

In regard to the modern so-called psychotherapy a number of us have only recently at a meeting of the Philadelphia County Medical Society expressed our opinions. It is not a new method but an old one—thousands of years old, as we have just heard. The one great danger in its application lies in relegating other and tried methods of treatment to the dust heap. It leads to neglect of physical and real methods. The persons employing it lose sight of the fact that disease has a physical, an organic basis—organic or physical in the sense that there is always in the affections termed functional some nutritional or other disturbance, some change in the biochemistry of the tissues, some change which cannot be corrected by merely talking at the patient. No one, least of all, the reader of the paper, decries the use of psychotherapeutic methods. We talk to a patient in the way that has been hinted at by Dr. Mitchell, but we do not keep alive his symptoms by constantly reminding him of them. We turn the mind into other channels than those which deal with his malady and make use of a host of other expedients, mental rest, isolation, occupation and what not to aid us in bringing about a result. We have not found it necessary to resort to hypnotism excepting under very unusual circumstances and still less have we found it necessary to adopt that newest of all fads, psychoanalysis. The fact that a patient feels some relief after having talked over his case with a doctor hardly justifies one in going into so elaborate a method as has been devised by Breuer and pursued by Freud of placing the patient upon a couch upon her back and inducing her to talk for from one to three hours at a time about her case. Besides the very conditions of the seance are such as to bring about a form of autohypnosis so that in the end we cannot depend on the statements which the patient makes. Symptoms, we are told, disappear after they have been dug up from the recesses of the past, but it requires many hours of often daily sessions, and we are told that at the end of six months, perhaps at the end of three years, a patient will get well. These methods, Dr. Dercum was frank to say, do not appeal to the profession at large. He was glad that they did not. The matter however assumes a more serious aspect in the peculiar views of pathology adopted by Breuer and Freud. Freud believes that the ideas of the hysterical patient, the obsessions of the neurasthenic or to use the more fashionable term psychasthenic, have their origin in some sexual aggression or sexual assault in which the patients have been active participants in childhood. We are told that it is the pain of the recollection of such an event which, attaching itself to some action of life other than the past sexual act, gives rise to an obsession. This strange pathology we are

asked to accept to explain the findings in hysteria and psychasthenia. No one need dwell upon the unwholesomeness of endeavoring to elicit such histories from patients, no one need dwell upon the harm that may be done, nor upon the unpleasant and unethical attitude which the physician must assume to elicit such histories. Again we are assured by at least one experience of Loewenfeld of the utter valuelessness of the histories obtained. A woman who had been a patient of Freud's, subsequently came under Loewenfeld's care and she stated to the latter that the sexual events that she had related to Freud when under the latter's treatment had really never occurred, that they had been wholly imaginary.

Finally a word as to the use of new terms. Dr. Decum stated that to his mind nothing is gained by using the word psychasthenia instead of neurasthenia. It is neither more scientific nor more expressive to say soul exhaustion than nervous exhaustion. The word psychasthenia is more high sounding but also more mystifying. Again the word psychotherapy offers no advantage over the old term of treatment by suggestion, and lastly psychoanalysis conveys an utterly fallacious meaning. Some recondite and excessively refined procedure is suggested, an expectation which is scarcely realized when the method is inquired into. Instead we find supposed cures brought about by the unearthing of memories alike abhorrent and fictitious.

Dr. Putnam said he wished to speak for another city, Boston, and to express his pleasure in the great treat we had had. The contribution which Dr. Mitchell had made to medicine was such a great one and so complete, as complete as Minerva springing from the head of Jupiter, that it is difficult to realize that Dr. Mitchell ever was seriously criticized in the beginning, or that his teaching met with great opposition. He thought that in estimating the value of the different parts of it those of us who have had for some time the pleasure of Dr. Mitchell's personal acquaintance cannot help feeling that his glorious courage and inspiring personality, which we all admire, have such an affection for, contributed not little to the result that he gained. There are one or two points to which Dr. Mitchell referred that Dr. Putnam dwelt on. First, in relation to neurasthenia as a bodily disease, Dr. Putnam strongly upholds that view and discussed it with Dr. Janet when he was here a couple of years ago, and Dr. Putnam was pleased to find he held the same position, believing that the bodily conditions which were understood as a species of stigmata of neurasthenia, the tendency to slender build and the enteroptosis and various other conditions of that sort, would tend to disappear as the patients improve.

As regards the psychotherapeutics, Dr. Putnam entirely sympathizes with what Dr. Mitchell said. Of course what we should do is not to study methods of treatment as such, we don't want to make ourselves electricians, but we wish to study the diseases for which electricity or whatever it may be, is one of the things that is useful. Nevertheless it is only natural that when methods of treatment are being studied that too much emphasis should sometimes be laid on them. It seems to be impossible to avoid that result, and for his part he is glad that these methods have come up for discussion. He disbelieves in the Freud method, nevertheless he thinks it has been a distinct contribution, and he thinks that Dr. Meyer, whom he understood has had some successes founded more or less on that treatment, would agree with him that we have been led to think about something which it is well we should think about, so these over-stated claims have the value of psychoanalysis.

Dr. Putnam also agreed with Dr. Mitchell in regard to what he said of Dubois, although his book contains much that is stimulating. He has done what so many others have done, in claiming exclusive value for his

methods. This of course we do not sympathize with. In that respect Dr. Prince has not erred. He has readily appreciated what others have done, and has tried to disparage no one, and has attempted to educate neurasthenics in a sensible way.

Dr. Dana said he also wished to express the pleasure and gratification he had experienced in hearing Dr. Mitchell recite the story of his development of this plan of treatment. Dr. Dana thought so far as his colleagues were concerned, they had always received it sympathetically, and not critically. They had never questioned the value of it as an original contribution to therapeutics. In almost every detail regarding the subject of psychotherapeutics Dr. Dana would follow along Dr. Mitchell's views. After all, the question is not whether Freud's view is correct or Dubois's plan is good for anything, or whether we should use psychotherapeutics, hypnotism or suggestion; we as neurologists are confronted with the fact that an enormous number of mentally sick people are running around and get their psychotherapeutics from the wrong well. They go the seven different kinds of cures which Dr. Mitchell has spoken of, and a great many of them are injured by them. We do not believe that this is the best way for these people to be treated. We feel that there ought to be some definite forms of psychotherapeutics approved by the profession so that people would not go after "soul-massage" or other faked forms of psychotherapeutics. What are we going to do with the large number who won't come to us and will go to anyone else who will raise his psychic standard? We must find out the good behind these false methods and organize it into some wise scientific measure which we can prescribe. Until we do this there will be a continual succession of new cults, Christian Science, osteopathy, etc., to the discredit of medicine and more especially to the discredit of neurology and psychiatry. Therefore we ought to accept the word psychotherapeutics and try to treat these cases in a way that is wise and efficient. For that reason he believed in opening clinics for the regular, systematic treatment of these people.

Dr. E. C. Spitzka, New York, said he was afraid the love for the occult and mysterious would exist as long as the human species, but they can't be done away with by any ideal force such as that suggested. He believes the step taken by Dr. Mitchell would prove efficacious in correcting the error he reprobated. Years ago he would have taken it for granted that it would be impossible for any number of physicians to advocate such psychotherapy as in now popular. That they have done so all know, but a paper like Dr. Mitchell's will cause them to reflect and retrace their steps. It really requires someone of his authority to do so. The demand so often made that the patient should combat his ideas is an absurd one. Dr. Spitzka in his practice, says to the patient, "I don't ask you to fight your ideas yourself, that is my business." By taking any other attitude with these patients as the medical adviser you place the patient himself in the attitude of medical adviser and therapeutist. With another class of patients any maneuvers are justifiable though we often experience ridiculous failure in using such. For instance, a patient came to Dr. Spitzka's office with the statement that the sutures of his skull were coming apart. He was becoming bald and the usual superficial appearance of the sagittal suture was manifest. When he came the second time Dr. Spitzka told him (showing him a large New Zealand war club, made of iron wood), "The next time you ask that question I will hit you with this" (the club). The man seemed somewhat dazed but said, "Thank you, Doctor, I never shall," and he went. Two hours afterward the telephone was rung up and the same question was asked.

Dr. B. Sachs said he wished Dr. Mitchell's conclusions might stand forth as the sane sentiment of this Association, in opposition to the psychotherapy idea. He was very thankful to Dr. Mitchell for having put everything in his inimitable way. We feel that this whole matter of psychotherapy and psychoanalysis has been horribly overdone. The psychotherapy that is of especial value has been practised by all of us more or less intentionally for years past. He does not believe that we are going to gain very much by giving these methods an especial label. In a busy professional life, Dr. Sachs has not had to worry over the influence of mind over body; he has been very much busier in hospital and private practice in trying to find out what are the physical conditions which have given rise to mental disturbance more or less severe.

As to Dr. Dana's statement regarding the seven cults, we are above the other cults. Let those who want to go to Christian Science go, we are not seeking patients. Let them go. A certain number of them will go. There will be plenty left. We cannot keep people from consulting quacks of every description; he thought it was a rather undignified position for the neurologists to take them into account at all. He said he didn't care about the osteopathists, Mrs. Eddy, or other faddists, more or less honest, more or less unscrupulous. He is in favor of psychotherapy but such as he can practice without labeling it or calling attention of his patients to it, and saying to them, "Here I am a practitioner in psychotherapeutics." Dr. Sachs continued, "I am a neurologist first. I am not going to call myself a psychotherapeutist any more than I would call myself an electrician, though I do use electricity occasionally."

Dr. Beevor said he had had the most delightful treat in his life. It was the greatest pleasure and if he had to cross the Atlantic simply to hear this one address and go back to-morrow he would have thought he had spent his time exceedingly profitably.

In regard to psychotherapy, it had not yet invaded England's shores. He said he must confess that he has not studied the French books, and he doesn't know that any of the English have very much, but they still use the Weir Mitchell treatment. They use it with the greatest success in the world and as far as psychotherapy is concerned he does not know of any neurologist who uses it in England.

Dr. Adolf Meyer said he must make almost an apology for putting in a word in this discussion largely on account of the contrast that Dr. Sachs has raised by speaking of the difference of his opinion from that of Dr. Dana. It may not be right to divert from the discussion of what Dr. Mitchell has brought before us so splendidly and in terms which, of course, may be misinterpreted, but to all of us who have taken them in as they were spoken, apply to the psychotherapy which we are trying to practice, consciously, not merely unconsciously, as Dr. Sachs says. We can look forward to a decided transition in this whole question as soon as we take a different attitude as to what we understand by mind and mental activity and then formulate our efforts accordingly. The mental therapeutics which worked in Dr. Mitchell's methods and are successful in the methods of practically every one of us, are the training of activities, not merely as words or abstract states of mind, but as things which show in the action and attitude of the patient; and any method which will produce correction of the erroneous attitude and ways of living is a proper form of psychotherapeutics. It is a very unfortunate thing that we think of psychotherapeutics only when we hear of so-called "psychoanalysis" and other special formulas. Proper mental activity, and proper conduct and proper attitudes

are the aim and essence of psychotherapy, and also training of the individual to meet certain difficulties. Dr. Meyer does not see why we would not be able to put these things in better form for teaching and application, if we make determined efforts and do not simply leave things to chance and to the unconscious. It has been a great inspiration to hear what Dr. Mitchell has said and also what the other speakers have said. Dr. Meyer thought the matter one certain of growth and he believes what Dr. Dana has proposed will lead to an evolution and further development of this issue, and is a much better stand to take than simply that of "there is no chance for improvement," and "it does not concern us."

Dr. P. C. Knapp endorsed absolutely the position Dr. Mitchell had taken in his most admirable address. He must confess that all these questions of words derived from psyche, which seem to have such a mystic and wonderful significance, to him were often a little fatiguing. He had had assistants who had become most enthusiastic over psychoanalysis and psychotherapy and yet who found it impossible to test the knee jerks properly or to test sensation correctly. In the craze for matters psychical they have neglected the fundamental principles of the art of medicine. The whole tendency of this present move in favor of psychoanalysis and psycotherapy has been to neglect, as Dubois cheerfully neglects, the physical basis which is so often at the bottom of the nervous disorder. The first thing for us to do is to put the patient on the best physical basis. Then we have solved part of the problem in regard to mental conditions. Very often by explaining to patients their errors, by assuring and encouraging them we can do very much, and he was a firm believer in an intelligent education of the patient by every method. There is, however, an important point which Dr. Dana has hinted at. There are a large number of patients who are the victims of some incurable disease, who will catch at any straw. They go to the seven or seventy-seven different mental sects in the hope of relief. Then there is another large class of sufferers for whose devotion to these false gods the medical profession itself is to blame. That is due to the fact that the patients are treated by the general practitioner whose knowledge of nervous diseases and proper treatment of nervous diseases is lamentably defective. In a recent number of one of our medical journals an eminent surgeon was expressing himself with some contempt regarding "merely neurasthenic symptoms." Such neurasthenic patients are really looked upon with some contempt and fail to receive treatment from the family physician, so that, instead of consulting a competent neurologist, they will seek for help elsewhere which they fail to get from the medical profession to whom they apply.

The President, Dr. Burr, asked Dr. Philip Coombs Knapp to give the Chairman's report on the Committee appointed to collect data in regard to the existence of general paresis and general diseases of the brain in railway employees.

Dr. Knapp reported that the Committee had collected data on general paresis and other diseases of the brain in railway employees which show such diseases are a source of very considerable danger to the traveling public and the Committee believes that persons in responsible positions, such as railroad engineers and train dispatchers, should be examined at regular intervals by competent neurologists. By such examinations the dangers from such diseases can be greatly diminished. The Committee desired to report progress and asked to be continued so that they might have time to make an elaborate report.

(*To be continued*)

NEW YORK NEUROLOGICAL SOCIETY

May 5, 1908

The President, Dr. B. Sachs, in the Chair

DISCUSSION ON THE PRESENT-DAY LIMITATION OF THE CONCEPTION OF PARANOIA

(*Continued from p. 660*)

Dr. Hirsh said Dr. Gregory had clearly pointed out the great confusion that existed on the subject of paranoia. There was no reason why there should be such confusion regarding it, because there was scarcely any mental disease which could be as well defined as paranoia. One reason for the confusion was the fact that many men called anything paranoia that presented some paranoiacal symptoms during the course of a disease. Such a course was so unscientific that it really called for no comment. Another reason that existed for so much difference of opinion about paranoia and which was largely responsible for the manifold classifications of the various types of the disease lay in the fact that paranoia was a mental condition which was entirely distinct from what we generally called disease. As a rule, by disease we understand physiological functions to be altered in such a way as to become unfavorable to the preservation of life. Paranoia, however, consisted in something entirely different. It consisted, essentially, in a different mechanism of thinking. This did not imply that the disease must manifest itself from childhood; on the contrary, it usually manifested itself only in adult life, although not always. In most cases, however, we are able to trace back the disease to early childhood. We almost invariably got the history that the individual was always "peculiar"; that he or she differed in many respects from other human beings; that the individual was known as a "crank," etc.

The reason why so many different types of paranoia were recognized, Dr. Hirsch said, was because the character of the individuals differed so widely, and paranoia would show itself in different ways, according to the characteristics of the individual, and to the environments in which he developed.

As to the frequency of paranoia, the speaker thought the statistics on that point were very unreliable, and were necessarily based on what the individual observer regarded as paranoia. If the observer happened to be a hospital man whose experience was limited to hospital material, his percentage of cases of paranoia would naturally be low, because paranoia was not a hospital disease. The number of paranoiac patients in the hospitals was comparatively small; probably not one out of ten of all paranoiacs would ever find his way into a hospital. In order to study paranoiacs we had to look for them in general life. These individuals were to a large extent able to earn their living, and were often productive. They were to be found in the ranks of the professions, and among prominent people. History was full of paranoiacs who had exerted a remarkable influence on civilization. They were to be found among the prophets of old; many of the poets were paranoiacs; Rosseau was a paranoiac. Coming down to modern times, Dowie was a good example of a paranoiac, as were also the founders of Mormonism and Christian Science. Paranoia was a very common disease, and its chief characteristic was an abnormal process of thinking, which manifested itself in what we term clinically "delusions."

Dr. Louis C. Pettit said he thought Dr. Hirsch had drawn the line too broadly in giving paranoia such a prominent place among individuals in general life. Many persons, doubtless, were eccentric, but we could scarcely call them paranoiacs.

Dr. Pettit said that the number of paranoiacs received at the State Hospital with which he was connected was not large. He had seen the disease develop during the secondary stage of syphilis. It might be due to an organic disease, and he recalled one case that developed late in life in a man with advanced arteriosclerosis. In its development it was not unlike some cases of dementia præcox. He did not agree with the statement that it was always an intellectual process at the onset but said that in the majority of cases there was primarily some sense deception in the various special fields, and cited instances of noted paranoiacs exhibited at the clinics, showing hallucinations in the fields of general sensation, taste and hearing; that ideation was affected through these more frequently than through the emotions and internal feelings. He also recalled cases where manic-depressive symptoms and other affective disorders developed during the course of paranoiac conditions. Very few of the cases he had seen deteriorated mentally.

Dr. B. Onuf said he agreed in the main with the views of Dr. Hirsch. He believed that true paranoia was a disease with which the patient was born, and consisted primarily in a wrong method of thinking, and required a certain degree of mental development before it could properly manifest itself. It might also, perhaps, remain latent, in the same manner that certain disturbances of ocular refraction remained latent until some particular condition brought them to light The faulty process of thinking was the essential feature of paranoia. The variations of the disease were dependent on the special attributes of the individual, and had no direct bearing on the disease itself. Their development depended on the degree of intelligence and other individual factors. Kraepelin, who had studied paranoia among the Japanese, noted the rudimentary character of the delusions among those people. In paranoia, hallucinations might be either present or absent, and they were usually the result of external stimuli or of very strong emotional influences. Auditory hallucinations, when present,—and they are the most frequent ones in paranoia—simply represent the thoughts of the patient which have become loud. It might at times be very difficult to distinguish paranoia from paranoid conditions, for example, dementia præcox. It might also be difficult to distinguish the alcoholic paranoiac from true paranoia, especially in those cases where the paranoia simply manifested itself through the abuse of alcohol. In distinguishing between these conditions, we should bear in mind that in alcoholism the hallucinations were apt to be primary, and the delusions built upon them, whereas in paranoia we had just the opposite; namely, the delusions were primary and the hallucinations secondary.

REPORT OF A CASE OF BRAIN TUMOR, WITH AUTOPSY

By Dr. Morris J. Karpas

The patient was a married woman, 44 years old, who was admitted to the Manhattan State Hospital on August 20, 1907. Her family history was negative and her previous history unimportant. As far as could be determined, the first evidences of her malady became manifest in 1904,

when she began to have convulsive seizures which varied in frequency and intensity. At first they appeared once a week, and later she would have them several times a day. For the past three years she suffered with headaches, and was often troubled with emesis. It was also noticed that she became seclusive and somewhat cranky, and would frequently make peculiar remarks to her husband. In November, 1906, the symptoms of her disease became aggravated. She commenced to talk in a bizarre manner, her speech became affected and the convulsions were more frequent. She was unable to find proper words for the expression of her thoughts. She could not name objects, but identified them correctly when their names were suggested to her. She lost her ability to read or write French and English. The vomiting became more frequent; the headaches more severe. Evidences of right-sided paralysis were observed in the early part of November 1906, and she gradually grew ataxic in gait and station.

When the patient was admitted to the hospital, an examination revealed the following: Slight facial asymmetry; left side of the face somewhat elongated, and its muscular contraction was apparently more active than the opposite side. Tongue protruded in the median line; grips equal; the left lower extremity was decidedly shorter than the right, and its general musculature was in a state of atrophy; this was probably due to an injury she had received in early childhood. Elbow, supinator and wrist reflexes were active. The knee jerks were unequal, but brisk. Ankle jerks were present; no ankle clonus; Babinski's reflex was present on the right side. Corneal reflexes were much diminished. Stereognosis was doubtful. Reaction to pin-pricks more marked on the left than on the right side. Distinct tenderness to percussion on the left side of the cranium. Internal strabismus of the left eye; pupils more equal; irregular in outline; reaction to light was sluggish, but accommodation reflex was good. A slight optic neuritis was subsequently demonstrated. According to Dr. Adolf Meyer, there was a suspicion of a right hemianopsia. Hearing was more acute on the left side. An examination of the cerebrospinal fluid revealed the presence of serum albumin and a moderate leucocytosis. Heart and lungs negative. No arterio-sclerosis. Blood pressure, 125. Mentally, the patient was quiet; she did not seem to recognize her new environment, and showed no acute depression. She showed peculiar awkwardness in handling objects, especially in the right hand. During the months of September and October, she remained drowsy, fatigued and somnolent, at times moaning as though in pain. On November 2 1907, she had a general convulsion, with coma; several hours later she had a second convulsion, and five hours after this death occurred from exhaustion.

Dr. G. Y. Rusk, who made the autopsy in the case reported by Dr. Karpas, gave a detailed account of the pathological findings. The left hemisphere of the brain was distinctly larger than the right, and on palpation one could roughly outline a solid mass, underlying the lower third of the left posterior central convolution, and extending upwards and backwards beneath the supra-marginal and angular gyri, then downwards to the point of separation of the parietal and occipital lobes, then inwards and forwards towards the uncus, where an irregular colored soft tumorous mass projected from the mesial end of the Sylvian fissure, compressing and causing considerable distortion of the left optic tract, and also compressing the third nerve. There was considerable distortion of the temporal lobe; the pons appeared flattened, and the cerebellum was also compressed. The limitations of the growth could not be made out macroscopically;

its center and apparent origin, however, lay in the region of the left amygdala, and from this point the growth infiltrated the remainder of the corpus striatum, the overlying island of Reil, the adjacent surface of the temporal lobe, the internal capsule, the thalamus, and apparently the centrum ovale. Macroscopically, the growth was a glioma, chiefly of the small-celled type.

Dr. Abrahamson, in discussing the case reported by Dr. Karpas, said that the tumor, growing, as it did, from within the brain instead of from the cortex, should have given some indication of its location from the characteristics of the convulsions.

Dr. Karpas, in reply to Dr. Abrahamson, said the physicians who had charge of the case did not have any opportunity to study the character of the convulsions. The only convulsion observed in the hospital occurred shortly before the patient's death, and that was general in character.

A CASE OF GLIOMA OF THE BRAIN

By Dr. I. Strauss

The case was one of glioma probably originating in the wall of the ventricle and infiltrating the meninges. This condition was first described by Klebs, and up to a few years ago was not thought to exist.[1] The symptoms in the case were largely those pointing to an internal hydrocephalus. There was marked exophthalmus, blindness and slight weakness of the left facial. As a rule, in these cases, the cranial nerves were not involved.

Dr. Sachs said the German writers, even those of recent date, referred to certain sarcomatous tumors, particularly those springing from the spinal meninges, as non-malignant. This was not in accordance with the view taken of them here.

Dr. Strauss, in reply to Dr. Sachs, said the tumor in this case was regarded as a glioma starting from the embryonic cells, and which, by its rapid growth, had taken on the evidences of malignancy.

STREPTOCOCCEMIA, WITH SEPTIC ENDOCARDITIS AND INFARCTS

By Dr. Strauss

The case was one of streptococcemia, with septic endocarditis and infarcts. The patient developed a flaccid paralysis of all extremities, and died in a state of coma two hours after the onset of the hemorrhage. The autopsy showed a very extensive hemorrhage of the brain, involving the surface of the island of Reil on one side, extending out through the Sylvian fissure to the under surface of the brain and then breaking through into the third and lateral ventricles. The hemorrhage was entirely sub-pial.

[1] Schlesinger in his monograph on tumors does not mention such a case. Since then there have been about nine cases reported.

Periscope

Deutsche Zeitschrift für Nervenheilkunde

(Vol. 31, Nos. 3–4.)

11. Carcinoma of the Frontal and Parietal Bones and of the Cerebellum in a Seventeen-year-old Girl Metastatic from Colloid Adenoma of the Thyroid Gland. FLATAU and KOELICHEN.
12. A Contribution to the Clinical Course and Histopathology of the Extramedullary Tumors of the Spinal Cord; A case of Tumor of the Spinal Cord whose Course was without Considerable Pain. FLATAU and STERLING.
13. The Pathological Anatomical Findings in a Further Case of Familiar Spastic Paraplegia. NEWMARK.
14. Family Type of Paralytic Amaurotic Idiocy and of the Cerebellar Ataxia of Childhood. HIGIER.
15. Myasthenic Paralysis with the Report of Two Cases. LADISLAUS V. KETLY.
16. The Diffuse Extension of Malignant Tumors, Especially Gliosarcoma, in the Leptomeninges. GRUND.
17. Acute Multiple Sclerosis with Transverse Lesion. KARL WEGELIN.
18. Note upon the Article in Vol. 30 of this Journal, page 167. "Hysterical Fever."

11. *Carcinoma of Brain.*—The patient had severe headache, vertigo, and dimness of vision for about a month. There was tenderness over the left thyroid, choked disks, and the knee jerks were diminished. There was a soft tumor in the posterior region in which a murmur could be heard. The gait was swaying. There was some variation in the degree of headache, paralysis of the muscles of the neck, some vomiting and finally the patient died.

Tumor was found in the temporal and occipital bones growing into the right half of the cerebellum. The thyroid gland was enlarged and contained nodules which under the microscope proved to be an adenoma similar to the metastatic tumor in the brain. A discussion of the metastasis of these tumors is given. Patel has reported 18 cases, 14 in women. Flatau and Koelichen discuss the symptoms in the reported cases and in their own, and regard the murmur as systolic in time and as arterial in origin. The subjective symptoms seem to vary. Treatment is without effect. Potassium iodide did no good. Spinal puncture was tried twice. At first it was followed by improvement, and the second time by marked aggravation.

12. *Tumor of the Spinal Cord.*—A man of thirty had pain in the right hypochondrium worse at night, then he developed paresis of the lower extremities more severe on the right side, and there was incontinence of urine. The paresis increased to spastic paralysis. There was also a sensory disturbance more pronounced on left side. Diagnosis of a tumor pressing on spinal cord was made and a lesion localized at the fourth dorsal vertebra on account of tenderness at this point. Later the

patient consented to an operation. No tumor was found at the operation. The patellar reflexes were lost immediately afterwards. Two weeks later the patient died. At the autopsy a tumor was found at the point of emergence of the sixth dorsal root which partially compressed the spinal cord. The characteristic ascending and descending degenerations were observed. The changes in the cord suggested to Flautau and Sterling that they were produced largely by mechanical pressure and edema and not by inflammation.

13. *Spastic Paraplegia.*—The author reports an autopsy upon a second case of spastic paraplegia in the same family. Although symptoms had lasted for years they were slight. The alterations found in the spinal cord consisted of a moderate degeneration in the pyramidal tracts and a considerable degeneration in the median and posterior tracts. In the other case degeneration in the two tracts were about equal. He therefore, concludes that in this family the posterior and median tracts are exceptionally vulnerable.

14. *Paralytic Amaurotic Idiocy.*—In a Jewish family without neuropathic heredity, but in which the husband and wife were close relatives, two daughters developed atrophy of the optic nerve with partial blindness, but with no other symptoms. Two other children showed the following changes: A girl of 9 at the end of the fourth year began to develop slowness of speech, disturbance of gait, uncertainty in the movements of the hands and arms, diminution in vision, and arrest of the mental development. Ultimately the gait was that of a drunken person, there was intense tremor with occasional explosive speech. The reflexes were exaggerated and there was Babinski's sign, and dorsal flexion of the toes even under normal conditions. There were no disturbances of sensation, vision was impaired. The second child was normal at 7 months, at 13 months it began to be apathetic, moved less vigorously, and was not able to sit up without support. There was some paresis of the legs and arms. The reflexes were increased and there was Babinski's sign. There was atrophy of both optic nerves, and convergence of the eyes without fixation. There was apparently marked diminution of vision. Patient would eat normally, but made no effort to speak and did not seem to understand sounds. In the diagnosis of these two cases Higier discusses their relation to the ordinary form of disease of the central nervous system, and concludes that the first belongs to the Marie type of family spastic disease and the second to the Tay-Sachs type. He prefers Edinger's functional theory to others for the explanation of these conditions.

15. *Myasthenic Paralysis.*—Ketly gives a very careful description of two cases of myasthenic paralysis, in the first of which an autopsy was obtained but proved negative as far as the central nervous system and some muscles were concerned. He then gives an analysis of 134 cases collected from the literature. The paper includes two valuable tables upon the differential diagnosis between myasthenic and progressive bulbar paralysis and acute apoplectic bulbar paralysis. A list of the literature not given in Oppenheim's article is appended.

16. *Diffuse Extension of Malignant Tumors in Leptomeninges.*—A boy of 11 years of age noticed gradually increasing weakness of various muscles and corresponding atrophy, and loss of sensation. Later pains were felt in various regions. There were attacks of headache, and vomiting. Choked disk developed in both eyes and there was paresis of the cranial nerves. He finally died two and one half months after

the onset. Miliary grayish nodules were found in brain and cord. These at first were supposed to be tubercular, particularly as miliary tubercles were found in liver and kidney. The other tumors resemble the glioma sarcomatodes of which five cases of diffuse distribution upon the leptomeninges in the central nervous system have been recorded up to the present time.

17. *Acute Multiple Sclerosis with Transverse Lesion.*—Wegelin reports the case of a man 34 years of age who had a condition resembling spastic paraplegia which ran a rapid and progressive course and terminated fatally in about six months.

Autopsy: Multiple sclerosis of the central nervous system was found. The cerebro-spinal fluid showed an excess of lymphocytes.

18. Controversial.

SAILER (Philadelphia).

(Vol. 31, Nos. 5–6.)

20. Contributions to the Knowledge of Tumors of the Central Nervous System. ROSENBLATH.
21. Two Cases of Traumatic Disease of the Cervical Cord. MÜLLER.
22. The Treatment of Tetany with Parathyroid Preparations. LOEWENTHAL and WIEBRECHT.
23. Disturbances in the Region of the Median Nerve. WANDEL.
24. Peculiar and Apparent Typical Symptom Group in Bulbar Paralysis (with remarks upon the perverse sensation of temperature and bulbar paresis of the sympathetic). MÜLLER.
25. Accumulated Small Convulsive Attacks. HEILBRONNER.

20. *Tumors of the Central Nervous System.*—This article consists of a series of articles each dealing with a particular case. Briefly these are:

Case 1.—A man of 32 for three months had had headache and shortly before his death developed exophthalmus more pronounced on the left side. At the autopsy a tumor was found involving the head of the caudate nucleus and the white substance forward and above, probably a glioma. Rosenblath is inclined to believe that the exophthalmus is due to obstruction to the return of the blood from the bulb by pressure upon the sinuses.

Case 2.—A man 53 years of age, five weeks before death, fell and struck the back of his head. He was obliged to stay in bed. He had epileptic attacks and later developed hemiparesis. There were no choked disks. At the autopsy a large glioma of the left frontal lobe was found which had undergone calcification and cystic degeneration. The epileptic attacks were associated with turning the head to the right. Conjugate deviation of the eyes is becoming more generally accepted as a symptom of lesion of the frontal lobe.

Case 3.—First developed symptoms of headache and vertigo, three weeks before death. There were no paralyses. At the autopsy a glioma of the right frontal lobe was found, of which a detailed histological description is given.

Case 4.—A man of 56 first developed symptoms two months before his death. There were bilateral choked disks, impairment of the movements of the right hand and paraphasia. A tumor of the left island of Reil was found.

Case 5.—A man of 22 complained of headache and vomiting three months before his death. There was weakness of the right leg and hand,

choked disks. A large cyst was found in the white substance of the left hemisphere, sending processes to the right, which appeared to be composed of epithelium of the ependyma and also of structures belonging to the blood vessels; so that it was finally called perithelioma.

Case 6.—A man 36 years of age noticed diminution of vision in 1902. A year later he had headache, vertigo, vomiting and complete blindness. He died about five months after the onset of the severe symptoms. Tumor was found involving the temporal lobe on the right side growing from the arachnoid.

Case 7.—A man of 43 years had various symptoms of which the most important were immobility of the pupil. A large tumor was found involving the right parietal lobe. Diagnosis of endothelioma was made. An interesting feature was the reflex immobility of the pupils, evidently the result of distant action of the tumor.

Case 8.—A man of 49 in addition to other tumor symptoms had complete paralysis of the left side of the body. In the course of some weeks it involved the muscles of respiration. A tumor attached to the membranes of the medulla was found pressing upon the cervical cord. The greatest damage was done at the decussation of the pyramidal tracts. The tumor was a melanotic sarcoma.

Case 9.—A man of 22 had headache, vertigo, loss of vision, weakness of the legs, incontinence and a bed sore over the sacrum. Symptoms had been progressing for about two years. There was a diffuse tumor of the soft membranes of the spinal cord throughout its entire extent. The substance of the spinal cord was very soft. In addition a tumor was found in the region of the fourth ventricle. This appeared to be a primary tumor and was probably round-cell sarcoma.

21. *Traumatic Disease of the Cervical Cord.*—A man after a severe fall developed pain in the arms, and dyspnea. There was rapidly, but not immediately, developed paraplegia. From the fourth day after the injury there was severe pain in both legs. There was prominence of the sixth cervical spine and the symptoms of a slightly irregular transverse lesion of the spinal cord. At the autopsy dislocation of the body of the sixth cervical vertebra was found. There is a description of secondary degenerations.

The second patient was struck in the back by a heavy object. There were immediately complete paraplegia, and symptoms of a transverse lesion in the lower cervical segment. There was a loss of all reflexes below the lesion, slow pulse, and narrow pupils. Patient died of edema of the lungs. The lateral process of the fifth cervical vertebra was found to be broken and the spinal cord was crushed in this region.

22. *Treatment of Tetany with Parathyroid Preparations.*—Loewenthal and Wiebrecht treated several cases of tetany with thyroid preparations that were supposed to contain parathyroid substance. They conclude that in many cases a favorable influence is observed which they assume is due to the parathyroid. In these cases there are no loss of body weight and no symptoms of thryoidism. In general, cases of tetany depend upon functional weakness of the glands.

23. *Disturbances in the Region of the Median Nerve.*—The author reports a series of cases of injuries to the median nerve, due to fracture, another group which is apparently professional and includes masons, smiths, cow milkers and various other occupations. He also reports some exceedingly interesting cases in which vasomotor and trophic disturbances

occurred, involving, in one instance the total absorption of the terminal phalanges of the first and second fingers.

24. *Symptom Group in Bulbar Paralysis.* Case 1.—A man of 49 with unimportant history awoke about midnight after having gone to bed apparently perfectly well at 10 P. M. and complained of a disagreeable vertigo and a peculiar twisting of the body. The left side felt weak and asleep. In a very short time severe headache and vomiting occurred. The next morning the whole of the left side of the body felt warm objects as cold. There was difficulty in micturition. The movements of the left side were normal but there was a subjective sensation of weakness. In four weeks he was able to return to work. Three months later there was paralysis of the right sympathetic. On the left side of the body there was total anesthesia and profound disturbance of the temperature sense. This involved the face, but the middle and lower branches of the trigeminus nerve were less affected than the upper. There was a subjective sense of warmth over the left side. Cold objects were felt as warm, but he occasionally discriminated between warm and hot objects. The blood pressure was high.

Case 2.—Tailor, 54 years of age. He woke in the morning with a curious sensation in the parietal region, and vertigo upon attempting to move. There was an ataxic gait. Towards evening he had difficulty in swallowing, and became weaker. Cold objects were felt as warm on the right side. The following morning he vomited and was unable to swallow. When examined there was found paralysis of the left sympathetic nerve, some disturbance in the region supplied by the upper branch of the right trigeminus, total anesthesia of the right side of the body with a temperature disturbance similar to that in the previous case. Power in the right side was not disturbed.

He regards these cases as due to disturbances of circulation in the region of the posterior cerebellar artery. Careful study of the disturbance of the temperature sense showed that the patients were capable of perceiving temperatures of 28° to 29° C. correctly. All temperatures above this were regarded as cold, and below as warm. In general the symptoms resemble those of a Brown-Séquard unilateral disturbance with its focus in the region of the sensory nucleus of the trigeminus nerve. There is some discussion to determine the exact point at which the lesion occurred.

25. *Accumulated Small Convulsive Attacks.*—Heilbronner reports a number of cases in which there were attacks of temporary unconsciousness, sometimes occurring with great frequency. Often the patients failed to have these attacks when under observation or when removed from their homes to the hospital. He also discusses the difficulty of the differential diagnosis between epilepsy and hysteria, and calls attention to some important features in the treatment, the avoidance of any forcing process in children who are intelligent, and in adapting the course of education to children who are less intelligent and particularly in the avoidance of examinations. SAILER (Philadelphia).

Archiv für Psychiatrie und Nervenkrankheiten

(42. Band, 2. Heft.)

15. Protoplasmic and Fibrillar Supporting Substance of the Central Nervous System. W. SPIELMEYER.

16. Investigation of the Cerebrospinal Fluid of Persons suffering from Mental and Nervous Disease. HENKEL.

17. The Mentally Caused Narrowing of the Field of Vision. N. KLIEN.
18. Exploratory Puncture of the Brain after Trephining in the Diagnosis of Brain Tumor. B. PFEIFER.
19. The Pathological Anatomy of the Senile Spinal Cord. KNICHI NAKA.
20. The Condition of the Reflexes in Paralyzed Parts after Total Division of the Spinal Cord. (Conclusion.) LAPINSKY.
21. The Question of Air Douches and the so-called Water Air Douches. PRENGOWSKI.
22. Atypical Forms of Thomsen's Disease. PELZ.
23. Further Contributions to Poriomania. DONATH.

15. *Protoplasmic and Fibrillar Supporting Substance of Central Nervous System.*—Spielmeyer discusses the histological questions underlying the relation between glia fibers and glia cells, with particular reference to Held's work on gliasyncytium and gliareticulum. The details of this discussion, which is largely a critique of other men's work, are beyond the scope of a brief review. For students of the neuroglia problem the paper will be of interest.

16. *Cerebrospinal Fluid.*—In the much investigated subject of the cerebrospinal fluid Henkel offers the results of his investigation of a series of fluids taken from persons suffering from various structural diseases of the nervous system. His results are in part as follows: that in progressive paralysis, tabes, cerebrospinal syphilis, various forms of meningitis, and in tumor of the brain and myelitis in less degree there is a constant, marked increase of cellular elements with serum albumin and increased serum globulin. In myelitis there is a relatively marked increase of albumin over cells. The results are not constant in arteriosclerotic conditions, multiple sclerosis and syringomyelia. The findings were negative in infantile cerebral paralysis and in all functional conditions. Although the cause of the cell increase in the cerebrospinal fluid cannot at present be definitely determined, it appears that various inflammatory processes tend to its production. The value of lumbar puncture for diagnosis is regarded as considerable, but the warning is given that in estimating its significance all other symptoms must be considered. A table and a bibliography of fifty-one references conclude the paper.

17. *Mentally Caused Narrowing of the Field of Vision.*—Recognizing the wide differences of interpretation concerning the causation of so-called functional disturbances of sensibility, Klien believes that much may be learned from a detailed study of the disturbance of function associated with concentric narrowing of the visual field, inasmuch as the eye represents a highly differentiated organ and one also amenable to various methods of investigation. On the symptomatic side simulation is one of great importance regarding which there is much diversity of opinion. An extremely detailed study of the entire subject follows through nearly fifty pages in which both theoretical and practical considerations are taken up. The discussion is far too detailed to permit of an adequate abstract within reasonable space.

18. *Exploratory Puncture of the Brain.*—As a result of a study, both clinical and microscopic, of twenty cases of brain lesion Pfeifer arrives at the following general conclusions: That puncture of the brain for diagnostic purposes is a very important and relatively safe measure by which it is possible, first, to render clear the general clinical diagnosis of a brain tumor as contrasted with other diseases of the brain, and especially through the demonstration of an internal and external hydro-

cephalus to arrive at conclusions regarding the difficult question of localized disease through brain atrophy; secondly, clinical local diagnosis of a brain tumor may be modified, verified and refined by this means; finally to assist in the operative treatment of brain tumors and to bring direct therapeutic results through the evacuation of cysts and ventricular fluid.

19. *Pathological Anatomy of the Senile Spinal Cord.*—Naka has undertaken an investigation of the pathological anatomy of the senile spinal cord following various methods. The investigation covers the microscopic study of seventeen cords, in which certain changes in cells, fiber tracts and blood vessels were found. Regarding the possible relations between the changes in senility and in paralysis agitans Naka thinks that in the senile cord the increase of small vessels and the overgrowth of glia about the vessels is of a higher degree than exists in paralysis agitans, nor does he think that the symptoms of paralysis agitans can have any connection with such spinal cord alterations.

20. *Condition of the Reflexes after Total Division of the Spinal Cord.*—Lapinsky concludes an exhaustive study of the condition of the reflexes in paralyzed parts with the following essential statements: Bastian's law concerning flaccid paralyses in cases of complete destruction of the spinal cord in man does not contradict a physiological explanation. The assumption that in all such cases of flaccid paralysis the reflex arc of the paralyzed parts remains normal is not as yet proved. The loss of the reflex cannot at present be attributed to loss of the reflex exciting influence of the cerebellum, since such cerebellar influence has also not yet been proved. In many cases of flaccid paralysis the real cause must lie in a structural affection of the reflex arc itself. This affection of the reflex arc may be explained through extravasation of blood in the gray cord substance and to edema, as well as through a variety of other deleterious influences. In some cases the loss of reflexes in total transverse lesion must be explained through dynamic causes, demanding an investigation of the irritability of the cut-off portion of the cord. The possible summation of several irritating causes in relation to the activity of the reflex arc must be considered.

21. *Air Douches.*—Prengowski offers an experimental and clinical study regarding the question of air douches and so-called water-air douches. The physiological action of the various forms of douches administered is described in detail.

22. *Atypical Forms of Thomsen's Disease.*—Pelz offers an interesting discussion on atypical forms of Thomsen's disease. He finds a striking diversity of symptomatology with many aberrations from the classical picture. The most constant sign is the disturbance of mechanical and electrical irritability, which is practically always present. On the other hand he finds that the myotonic disturbance is subject to very marked alterations. It may, in fact, wholly fail, so that like paralysis agitans *sine agitatione*, one may have myotonia *sine tonu*. In many cases an hereditary history is lacking. Nevertheless the opinion is expressed that the condition is still a congenital one and is better so designated than as acquired myotonia. The course of the disease also shows wide variations. The conclusion is finally reached that there is absolutely no single absolutely pathognomonic symptom.

22. *Further Contributions to Poriomania.*—Donath, on the basis of three further cases discusses again the question of poriomania or a sudden, uncontrollable desire to wander away, at times occurring in epileptics.

E. W. Taylor (Boston).

Book Reviews

THE PSYCHOLOGY AND PEDAGOGY OF READING. With a Review of the History of Reading and Writing and of Methods, Text and Hygiene in Reading. By Edmund Burke Huey, A.M., Ph.D., Professor of Psychology and Education in the Western University of Pennsylvania. The Macmillan Company, New York. $1.40.

This is a charming little book tracing the origin and development of the signs which have come to stand as symbols of communication and the physiological work of the eye and its mechanisms in the process of reading. It would be complete if the author had done a little more in the analysis of the sensory defects of word blindness, etc., but the pathological features are entirely neglected, nor are the anatomical considerations, save those of the eye muscles themselves, at all adequate.

But it is assumed that the author has not had in mind any of these things. In four sections he discusses the Psychology of Reading; The History of Reading and of Reading Methods; the Pedagogy of Reading and finally the Hygiene of Reading.

The section on the psychology of reading is full of physiological details and some consideration of the psychic side but it is a one-sided psychology—the discussion of the relation of inner speech to reading being almost the only association psychology touched upon, and as is well known there is a rich vein of association psychology connected with the subject of visual perception of word symbols.

The chapter on the History is well told; the genesis of alphabets interestingly presented, the evolution of printing and the printed page told with good effect.

In the chapter on the Pedagogy of Reading emphasis is laid on the desirability of a later acquisition of reading than is usually considered the best thing. The authors quoted all advocate eight, nine, or ten as the proper years for the acquisition of reading. It is doubtful if eye-mindedness is a good substitute for ear-mindedness in pedagogy.

The section on Hygiene is rational. There is no eye-strain clap trap in the author's hygiene.

JELLIFFE.

THE ANIMAL MIND. A Text-Book of Comparative Psychology. By Margaret Floy Washburn, Ph.D., Associate Professor of Philosophy in Vassar College. The MacMillan Company, New York. $1.60 net.

This is the second of a series of works on Animal Behavior which the authoress says might more appropriately be termed The Animal Mind as Deduced from Experimental Evidence. In her opening chapter a full discussion of the difficulties and methods of comparative psychology is given. The fallacies of the anecdotal method of Romanes and the world at large is gone into fairly and comprehensively and the shortcomings of the experimental methods of Lloyd Morgan, Thorndike and others not overlooked.

It is a fair statement of the problems encountered. The inferences

of mind in the lower animals from their behavior and from anatomical structure is then taken up. This is a very fragmentary discussion, mostly confined to Jenning's work and might with profit have branched out to the higher fields so well covered by Sherrington and his school.

The work goes on to take up the Mind of the Simplest Animals; Sensory Discrimination of Taste, Hearing and Vision; Spatial Relations and Space Perception; Conscious Experience; Memory and Some Aspects of Attention. In these chapters the lower forms occupy the chief position in the discussion and indeed the whole tendency of the work is to deal with the invertebrate types.

It is well written and with its very complete bibliography will make a welcome addition to the library of the biologically inclined physician.

JELLIFFE.

NEUROPATHOLOGICAL PAPERS, 1906–1907. Harvard University Medical School. 1908.

Ten reprints from the pens of Doctors Putnam, Taylor, Knapp and Smith, done into a book at Boston, represent the work of the Neurological Department of Harvard University for the past season.

A detailed criticism of each and every article in this collection is uncalled for, but the general estimation of the sum total is altogether in order and pertinent, and to this end it is necessary to very briefly touch on the titles and values of the component papers.

E. W. Taylor has written six of the articles, one of these ("A Simple Method of Reconstructing Nerve Plexuses") being in collaboration with R. M. Smith.

Taylor's first article—"Tumor of the Spinal Cord Leading to Destruction of the Lumbar Region, Hydrocephalus, Double Optic Neuritis and Painless Labor"—will well repay one for careful perusal. His second article—"Painless Labor: Report of a Case Due to a Destructive Lesion of the Spinal Cord"—is founded upon the case presented in his first paper, the subject being treated from the obstetrical point of view.

Taylor's third paper—"Senile Trepidant Abasia: Report of Cases"—is a short discussion of three cases of a frequently observed condition.

"Medical Expert Testimony" is the title of Taylor's fourth paper. It discusses in a very general way some of the difficulties which beset the medical expert.

In "A Simple Method of Reconstructing Nerve Plexuses," R. M. Smith and Taylor give a lucid and much to be recommended plan for the diagrammatic teaching of the constitution of the several plexuses.

"The Attitude of the Medical Profession Toward the Psychotherapeutic Movement" is the title of Taylor's sixth and last paper.

Knapp has two contributions: "Heredity in Diseases of the Nervous System with Especial Reference to Heredity in Epilepsy" and "The Mechanism of the Plantar Reflex with Especial Reference to the Phenomenon of Crossed Reflex." These papers, particularly the first, show careful thought and well repay close attention.

Putnam contributes two papers: "Pulse Pressure Estimation" and "Treatment of Psychasthenia from the Standpoint of Social Consciousness."

ALFRED REGINALD ALLEN.

FOLK WAYS. A Study of the Sociological Importance of Usages, Manners, Customs, Mores and Morals. By William Graham Sumner, Professor of Political and Social Science in Yale University. Ginn and Company, Boston.

The psychiatrist is destined to be a leader of the people. In his workshop lie the dismembered parts of mental mechanisms, the study of which constitute his ability to grasp the whys and wherefores of mental reactions. Some of the most elusive factors in this estimation, however, are those ideas held by mankind which in spite of the wisdom of the professors still continue to sway human conduct just as they did in the days of David, of Homer, of Epicurus, of Plato, of Thomas Aquinus, and down to the present day. It is to these folk ways that Dr. Sumner has wisely turned his attention and has given a work which no true student of mental processes would care to overlook.

Adopting the biological point of view the author himself confesses that he must needs go out and gather his material before generalizing. Would that other sociologists might do the same, and some psychologists. He presents a work of 700 pages with 20 chapters, dealing with the fundamental notions of human society. The mores, struggle for existence, labor, wealth, societal selection, slavery, abortion, cannibalism, sex customs, marriage, incest, kinship, blood revenge, the evil eye, harlotry, sports, drama, asceticism, these and others are among the subjects discussed—all in a thoroughly simple, first hand manner that is delightful.

The customs that have originated from disease are unfortunately overlooked. No philosopher has yet had the necessary facts and ability to interpret but the time is not far distant when the important relations of disease to social customs and ideas of morality will be written. Even Sophocles would attempt to excuse Ajax for his mad outbreak on the ground of an unknown hereditary factor. In the meantime this work of Sumner's will prove stimulating.

JELLIFFE.

NEUROLOGICAL AND MENTAL DIAGNOSIS, A MANUAL OF METHODS. By L. Pierce Clark, M.D., Senior Attending Physician, Hospital for Nervous Diseases, New York; Visiting Neurologist to the Randall's Island Hospitals and Schools, New York, etc.; and A. Ross Diefendorf, M.D., Lecturer in Psychiatry in Yale University; Assistant Physician and Pathologist, Connecticut Hospital for the Insane, etc., New York. The Macmillan Company, 1908. All rights reserved. Price $1.25 net.

The authors of this practical little volume find justification for its appearance in their statement in the preface that the necessity and desirability of forming correct habits in case examination is in line with the advanced teachings of modern medicine. Definite methods of procedure are laid down in the book both for cases of nervous disease and for those of mental disease, so that a proper analysis may be made, as a matter of routine, either in hospital or private practice. These examinations are both thorough and systematic. There are thirty-one illustrations which are helpful and for the most part original. The chapter on neurological diagnosis is by Dr. Clark and follows largely the scheme of the National Hospital for the Paralyzed and Epileptic of London. The newer "signs and phenomena," as yet unproven, mention of which might confuse the student, have been wisely omitted. Those which are really helpful are

fully described. The chapter by Dr. Diefendorf on mental examination is on Kraepelinian lines, and is followed by a much-needed glossary of modern psychiatric terms. It is doubtless much more difficult to outline a definite scheme for mental examinations than it is for neurological. Dr. Diefendorf has accomplished much in this direction. May we hope that some day he will give us a tabulated scheme in connection with his excellent descripiions.

Notes and News

An examination will be held on December 15, 1908, for the position of internes on the staff of the Hospital for Nervous Diseases of the City of New York, which is situated on Blackwell's Island. Two internes to be appointed. All applications should be addressed in writing to Dr. Edward Livingstone Hunt, Secretary, 54 West 50th St., New York.

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Figures with asterisk * indicate original articles, and are accompanied with title. Figures unaccentuated, accompanied with title, indicate abstracts.

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