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COMPLETE TRANSECTIONS OF THE SPINAL CORD AT DIFFERENT LEVELS

THEIR EFFECT ON SWEATING *

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Knowledge of how and to what extent the sweat glands are controlled by central and peripheral nervous factors is still somewhat limited. Langley [1] established the fact that sweating is controlled peripherally through the sympathetic nerves, which leave the cord at definitely localized segments. Winkler [2] in 1908 produced sweating by applying stimuli to the floor of the fourth ventricle, and Karplus and Kreidl [3] were able to activate the sweat glands by stimulation of the tuber cinereum. Bechterew,[4] Gribojedow [5] and Winkler produced it by stimulation of definite areas in the frontal lobes. It is clear, then, that sweat mechanisms are located in the spinal cord, medulla, tuber cinereum and hemispheres. The relative importance of each of these mechanisms and the rôle played by each is more difficult to determine. This problem may be approached most simply through an investigation of how the spinal mechanisms are affected by the removal of all impulses from higher centers, and to what extent they are able to function independently. An opportunity for such an investigation is found in preparations with complete transection of the spinal cord.

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* Submitted for publication, April 18, 1930.

* From the Phipps Psychiatric Clinic, Johns Hopkins Medical School.

1. Langley, J. N.: The Arrangement of the Sympathetic Nervous System, Based Chiefly on Observations upon Pilo-Motor Nerves, J. Physiol. **15**:176, 1894.

2. Winkler, F.: Die zerebrale Beeinflussung der Schweissekretion, Pflügers Arch. f. d. ges. Physiol. **125**:584, 1908.

3. Karplus, J. P., and Kreidl, A.: Gehirn und Sympathicus. 1. Mitteilung. Zwischenhirnbasis und Halssympathicus, Pflügers Arch. f. d. ges. Physiol. **129**: 138, 1909.

4. Bechterew, W.: Der Einfluss der Hirnrinde auf die Thränen-, Schweiss und Harnabsonderung, Arch. f. Anat. u. Physiol., Physiol. Abth., 1905, p. 297.

5. Gribojedow: Ueber Rindencentra der Schweissecretion, Behandl. der Psychiatr. Gesellsch., St. Petersburg, Dec. 13, 1903.

Head and Riddoch,[6] in 1917, took advantage of the opportunity to study this question during the World War when many soldiers were returned from the front with complete transections of the cord. They found that the immediate effect of the transection was the absence of all reflexes, paralysis of the rectum and bladder, and total loss of the activity of the sweat glands, as shown by dryness of the skin below the level of the lesion. This condition persisted usually for several months or longer, when, however, the reflexes returned and became exaggerated, the rectum and bladder began to function regularly, and the skin became soft and moist. Since even slight stimulation was sufficient at this time to produce visible sweat all over the body, the sweat glands must have been hyperactive.

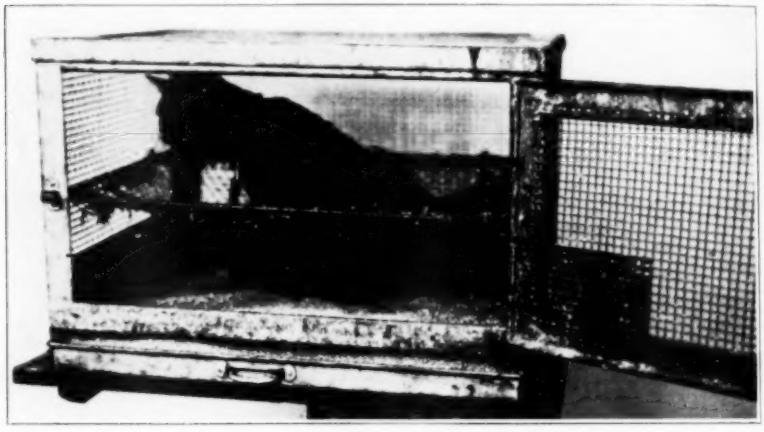

Fig. 1.—Cage and hammock in which the spinal cats were kept.

The work of Head and Riddoch has shown that the sweat mechanisms of the spinal cord cease their activity only temporarily after removal of impulses from higher centers, and that ultimately they not only resume their functions but actually become hyperactive. However, because they had no method of measuring the activity of sweat glands, these workers could not demonstrate the magnitude of the change produced by the transection beyond the observation that the skin was dry during the first stage and excessively moist during the second. By means of the electrical skin resistance and galvanic skin reflex methods, we have been able to make a quantitative study of the effects produced on the activity of sweat glands by transection of the spinal cord at various levels. Such data may constitute a basis for further study of

6. Head, H., and Riddoch, G.: The Automatic Bladder, Excessive Sweating and Some Other Reflex Conditions in Gross Injuries of the Spinal Cord, Brain **40**:188, 1917.

the rôle played by the different nervous mechanisms in the control of the sweat glands.

METHODS

Our experiments were performed on twenty-four cats with spinal lesions at different levels from the sixth cervical to the fifth lumbar segments. The animals were kept in hammocks of the type shown in figure 1, suspended in individual cages. Such hammocks greatly facilitated the sanitation problem in the cages and helped to keep the cats in a healthy postoperative condition. In consequence,

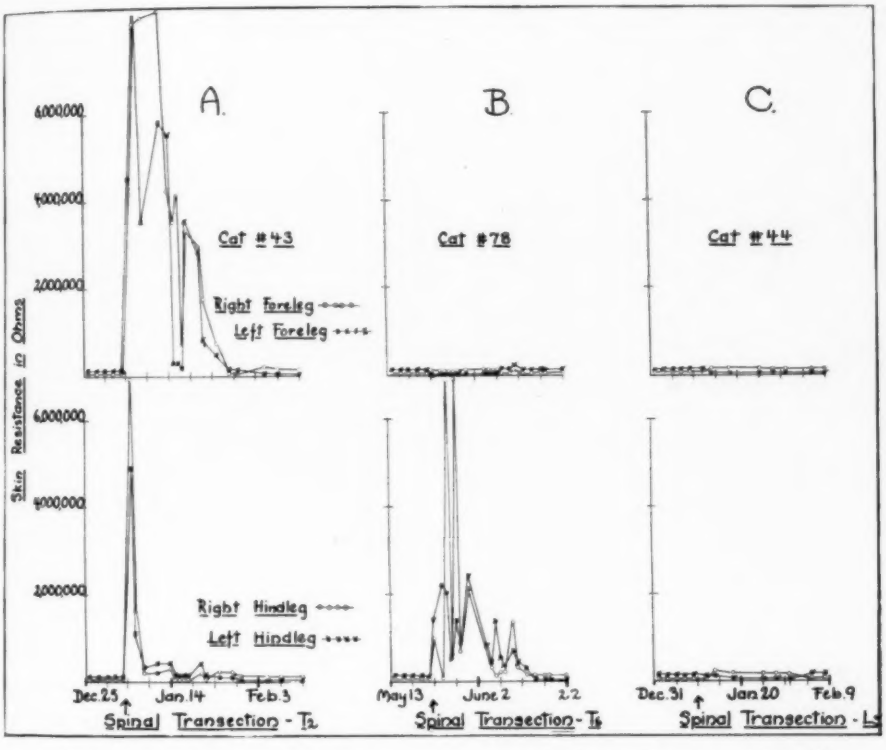

Fig. 2.—Graphs showing the increase in the electrical resistance of the skin on the fore and hind feet produced by transection at three different levels of the cord, second thoracic (*A*), sixth thoracic (*B*) and third lumbar (*C*).

most of them were kept alive for a significant interval, some of them for as long as eight months.

The technic of measuring the activity of the sweat has been described in detail in previous papers.[7] It consists, in brief, of a measurement of the resistance offered by the skin to the passage of a small constant galvanic current. In these

7. Richter, C. P.: Physiological Factors Involved in the Electrical Resistance of the Skin, Am. J. Physiol. **88**:596, 1929; Nervous Control of the Electrical Resistance of the Skin, Bull. Johns Hopkins Hosp. **45**:56, 1929.

experiments with cats, the electrodes (kaolin paste and zinc sheeting) were attached to the central pad on each of the four feet.

In addition to the daily readings on the resistance of the skin, daily observations were made of the body temperature and the condition of the skin, and a record was kept of the presence or absence of reflexes.

RESULTS

Spinal transection in the cat produced great changes in the electrical resistance of the skin. These changes could be separated into two periods. In the first, during which the skin was dry, just as Head and Riddoch found in men with transections of the spinal cord, the resistance was extraordinarily high. In some of our animals it increased from a preoperative level of 40,000 to more than 25,000,000 ohms. During the second stage, during which the skin became soft and moist, the resistance dropped to a level even lower than the normal, which is low in the cat, and this subnormal level was maintained permanently.

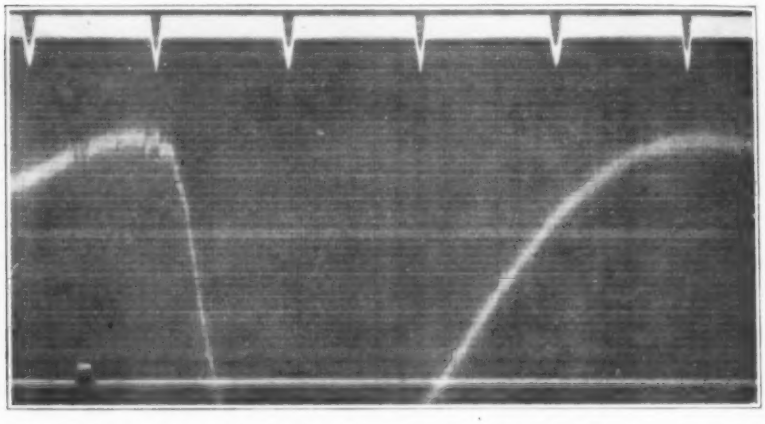

Fig. 3.—Record of galvanic skin response from the hind foot produced by an electric shock applied to the tail. Time in seconds is given at the top of the record, and below this, the galvanic response. The notch in the line at the bottom indicates when the shock was applied.

A typical curve is shown in figure 2 *A*. The resistance in ohms is given on the ordinates for both the fore feet and the hind feet, and the duration of the experiment in days is given on the abscissae. The transection was made at T 2. Before the operation, the resistance was approximately 45,000 ohms on all the feet. After the transection, it increased on the fore feet to 5,943,000 and 14,993,000 ohms, and on the hind feet to 4,893,000 and 6,963,000 ohms. At the end of twenty-five days, however, it had decreased to a final level slightly below the preoperative average. This low resistance was maintained for 180 days, at which time the animal was killed.

The inactivity of the sweat glands during the first stage and their hyperactivity during the second is brought out by observations of the galvanic skin reflex. During most of the first stage, at least until the resistance had reached a fairly low level again, the galvanic skin reflex was absent even to painful stimuli, and all spontaneous galvanic currents were lacking. During the second stage, however, the galvanic reflex could be obtained from stimuli as mild as touching the hair on the tail, and spontaneous currents were given off almost constantly. A record of the galvanic skin reflex produced by pinching the tail is presented in figure 3. The galvanic skin reflex response obtained from cats with a decerebrated spinal cord has been described in detail in a previous paper.[8]

GENERAL CHARACTERISTICS OF THE EFFECTS PRODUCED ON RESISTANCE OF SKIN BY TRANSECTION OF THE SPINAL CORD

Onset.—With one exception, the increase in resistance began within a few hours after transection of the cord. In this instance the galvanic change occurred two days after the transection. In all but the aforementioned animal, the resistance of the skin reached its highest point within the first three days after the operation.

Magnitude.—The magnitude of the increase produced by the transection is shown in the accompanying table. The identification number of the animal is given in the first column; the levels of the transection, in the second; the highest point reached by each of the fore feet and hind feet before the operation, in the third; and finally, the highest point reached by each of the four feet after the transection. Thus, the first animal, with a transection of the spinal cord at the sixth cervical shows an extraordinary increase in resistance from an average of 40,000 ohms on each foot, to 6,963,000 ohms on the right fore foot; 19,593,000 ohms on the left fore foot; 25,193,000 ohms on the right hind foot, and 22,393,000 ohms on the left hind foot. The other animals with transections in the upper thoracic region show a correspondingly large increase. With the lesions in the lower part of the cord, the change is not so great. The significance of this fact will be discussed later.

Duration.—The interval during which the resistance was increased could be measured in only seven of the twenty-four animals. The others either died or were killed before the beginning of the second stage. In these seven animals, the resistance remained high for periods varying from twenty-five to seventy-five days. The duration was roughly proportional to the magnitude of the effect; that is, when the initial increase was great, the effect was prolonged.

8. Richter, C. P.: Galvanic Skin Reflex from Animals with Complete Transection of the Spinal Cord, Am. J. Physiol. **93**:468 (May) 1930.

Final Level.—In the observations of Head and Riddoch on men with transections of the spinal cord, the excessive sweating during the second stage received the most emphasis. In the cats with transections this phase of the second stage is not reflected satisfactorily in the resistance readings owing to the fact that the resistance of the skin of the intact animal is usually low under normal conditions. However, the increased activity of the sweat glands is clearly manifested in the hyperactivity of the galvanic skin reflex and in the spontaneous currents that are given off.

Magnitude of the Increase Produced by Transection

No.	Level of Transection	Highest Level of Resistance of Skin Reached Before Transection				Highest Level of Resistance of Skin Reached After Transection			
		Fore Feet		Hind Feet		Fore Feet		Hind Feet	
		Right	Left	Right	Left	Right	Left	Right	Left
86	C 6	40,000	40,000	40,000	40,000	6,963,000	19,593,000	25,193,000	22,393,000
74	T 1	40,000	40,000	40,000	40,000	19,593,000	21,993,000	21,993,000	21,993,000
122	T 1	30,000	30,000	30,000	30,000	25,193,000	25,193,000	6,963,000	25,193,000
43	T 2	45,000	45,000	45,000	45,000	5,943,000	14,993,000	4,893,000	6,963,000
100	T 2	40,000	40,000	40,000	40,000	6,293,000	2,150,000	2,200,000	2,100,000
42	T 2	40,000	40,000	40,000	40,000	5,593,000	8,393,000	4,193,000	4,193,000
61	T 2	40,000	40,000	40,000	40,000	2,950,000	9,793,000	2,750,000	2,950,000
75	T 2	40,000	40,000	40,000	40,000	14,993,000	6,893,000	2,800,000	2,800,000
107	T 2	25,000	50,000	75,000	110,000	4,200,000	27,993,000	27,993,000	27,993,000
110	T 2	40,000	40,000	40,000	40,000	12,593,000	12,593,000	14,273,000	8,393,000
126	T 2	25,000	25,000	25,000	25,000	14,993,000	27,993,000	27,993,000	25,993,000
47	T 3	40,000	40,000	40,000	40,000	2,160,000	5,593,000	5,593,000	5,593,000
101	T 4	40,000	40,000	40,000	40,000	12,593,000	6,963,000	6,963,000	8,393,000
78	T 6	40,000	40,000	40,000	40,000	35,000	20,000	2,400,000	6,163,000
123	T 8	10,000	50,000	15,000	10,000	45,000	50,000	6,963,000	6,963,000
59	T 9	45,000	45,000	45,000	45,000	25,000	25,000	700,000	1,100,000
53	T 10	40,000	40,000	40,000	40,000	100,000	110,000	5,593,000	5,150,000
79	T 12	40,000	40,000	40,000	40,000	25,000	25,000	2,600,000	2,400,000
45	L 2	40,000	40,000	40,000	40,000	40,000	40,000	4,193,000	6,963,000
51	L 3	40,000	40,000	40,000	40,000	40,000	10,000	50,000	25,000
57	L 3	40,000	40,000	40,000	40,000	20,000	20,000	40,000	10,000
44	L 3	40,000	40,000	40,000	40,000	30,000	20,000	30,000	50,000
91	L 4	40,000	40,000	40,000	40,000	20,000	40,000	50,000	25,000
134	L 4	100,000	100,000	60,000	90,000	110,000	70,000	100,000	110,000

RELATION BETWEEN THE MAGNITUDE OF THE INCREASE IN RESISTANCE OF SKIN AND THE LEVEL OF THE TRANSECTION OF THE SPINAL CORD

In the table it can be seen that the increase in resistance has a definite relationship to the level of the transection. Thus, in the fore feet the large increase occurs with all transections down to the sixth thoracic segment. At that point it disappears completely, and all transections at lower levels are without effect. The transection at the sixth thoracic, however, still produces a large increase on the hind feet, as do all other transections at levels down to the third lumbar. At the third lumbar and below, the increase fails to occur on the hind feet also. These changes are shown in the graphs in figure 2 in three animals with transections at the second and sixth thoracic and the third lumbar, respectively.

The significance of these results becomes clear at once if one considers them in connection with the spinal levels from which the skin on different parts of the body receives its sympathetic innervation. Langley has observed that the fore paw of the cat receives most of its sympathetic impulses through segments of the cord above the sixth thoracic, and the hind paw through segments above the third lumbar. Similarly, Head and Riddoch found that in men with transections of the spinal cord, the distribution of areas of excessive sweating was determined by the level of the spinal lesion with relation to the thoracicolumbar outflow of the sympathetic nerves. In accordance with this observation, we have found that the galvanic effect is, in general, greater with transections in the upper parts of the cord. Thus, one sees in the table that transections at the level of the sixth cervical and first and second thoracic nerves produce a much greater increase in resistance of the skin on the hind feet than did the transections from the ninth thoracic to the second lumbar nerves.

These results indicate further that the resistance of the skin is independent of influences from the somatic nerves, since a spinal transection at the second thoracic nerve, which has no effect on the motor and sensory fibers of the fore legs, produces profound changes in the electrical resistance of the skin, and a transection at the third lumbar, which completely paralyzes the hind legs, leaves the resistance entirely unaffected.

SPINAL TRANSECTION, SYMPATHECTOMY, AND TOTAL SECTION OF THE NERVE COMPARED ACCORDING TO THE EFFECTS ON RESISTANCE OF THE SKIN PRODUCED BY EACH

Our experiments thus far may be summarized as follows: Total section of the spinal cord produces a large increase in the resistance of the skin (from 20,000 to 25,000,000 ohms), an increase which is only temporary since after from twenty-five to seventy-five days, the resistance curve resumes its initial low level. This large increase is due to the severance of sympathetic pathways in the cord coming from suprasegmental mechanisms to the sweat glands. The glands ultimately resume their function, however, through segmental sweat mechanisms, as is indicated by the return of the conductivity to a low level and the reappearance of the galvanic skin reflex.

These results lead at once to a significant question. How do the effects produced by section of the suprasegmental sympathetic pathways in the cord compare with the effects that follow section of the more peripheral sympathetic pathways in the thoracic and abdominal cavities and in the nerve trunks in the extremities? Although in previous experiments on monkeys it was shown that the two latter opera-

tions have a marked effect on the resistance of the skin, we have repeated this work on twelve cats to insure strictly comparable results.

For purposes of comparison, a typical graph showing the effect of complete spinal transection is given in figure 4 *A*. In this instance, the resistance rose shortly after the operation from 40,000 to more than 14,000,000 ohms, remained high for forty-three days and then returned to the flat low preoperative level. The typical galvanic change produced on the right hind foot by removal of the abdominal sympathetic chain

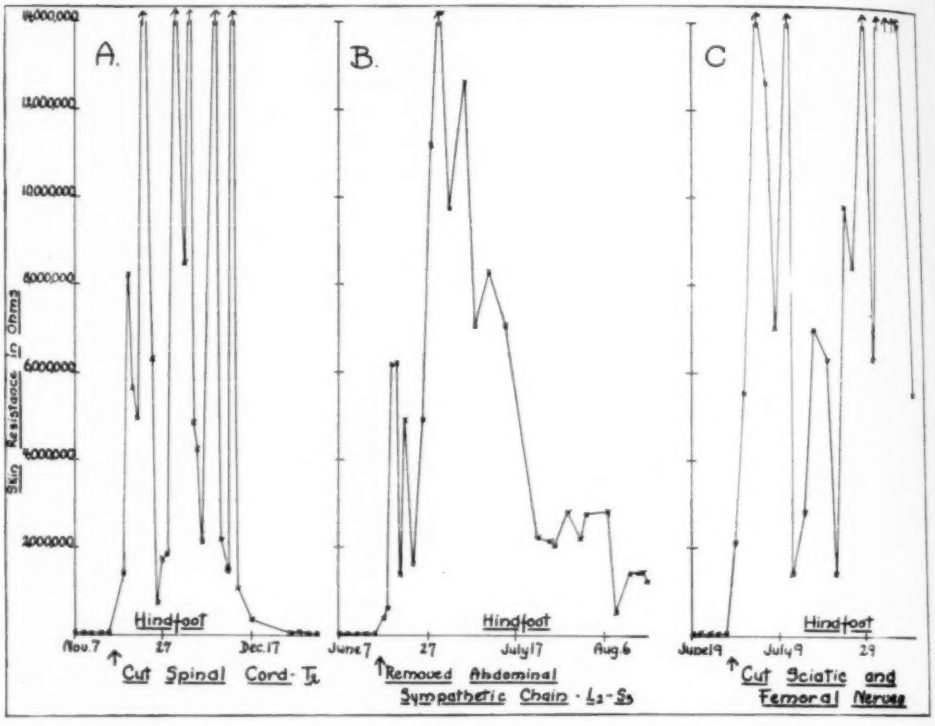

Fig. 4.—Graphs showing the different effects produced on the electrical resistance of the skin by spinal transection (*A*), sympathectomy (*B*) and total nerve section (*C*).

is shown in figure 4 *B*. Here one sees that during the period immediately following this operation, the effect was as great as that which follows spinal transection, but there was a marked difference in its duration. After sixty-two days, the resistance was still well over 1,000,000 ohms, and from previous experiments we know that this level would probably have been maintained for many months before the preoperative average was reached again.

Spinal transection and sympathectomy differ in one other respect not apparent in the curves: The former causes the galvanic skin reflex

to disappear temporarily, while the latter causes it to disappear permanently.

The fact that after sympathectomy the resistance of the skin finally returns to a lower level suggests that some peripheral mechanism in the sweat glands may assume control of the glands; much as the segmental mechanisms assume control after their connection with higher centers is interrupted, or as the enteric plexus is able to take control after all nerve connections to the gut have been destroyed. Favoring this view is the fact that when the resistance has reached a low level after sympathectomy, the skin again becomes soft and feels more normal to the touch.

It remains, then, to consider the effects produced by total nerve section (i. e., section of the brachial plexus and of the sciatic and femoral nerves). A record showing the typical resistance curve obtained by section of the sciatic and femoral nerves is presented in figure 4 *C*. The change again is of the same magnitude as that produced by spinal transection or by sympathectomy, an increase of from 50,000 to more than 14,000,000 ohms being recorded, but it differs from the effect produced by both of these operations in that the increase is permanent. The same result was obtained in five other cats and corroborated our observations in the previous experiments on monkeys. In spite of the fact that the resistance remains at a high average level, it does show marked fluctuations from day to day, and occasionally drops very low. Consistent with this high average level throughout the postoperative period to death is the permanent dryness of the skin. It would seem, therefore, that after total nerve section the sweat glands do not resume even a part of their function as they seem to do after sympathectomy. This observation argues that the somatic nerves must have some influence on the glands, probably of a trophic nature.

In summary, then, we may say that after section of the suprasegmental sympathetic pathways in the cord, the sweat glands regain their functions through the fact that the segmental sweating mechanisms in the cord take over the control. After sympathectomy, the peripheral mechanisms within the glands themselves, under the influence of the somatic innervation, seem to take over the control; and after total nerve section, no control is ever established again.

COMMENT

Thus far, we have had no opportunity to study the effects produced in man by lesions causing complete or partial transections of the cord. From experiments on cats we know that even the slightest injury to the cord causes an increase in the resistance of the skin. It may be possible, therefore, ultimately to apply this method to man to determine not only the extent of spinal lesions but also their exact location.

SUMMARY

A complete transection of the spinal cord in cats produces profound changes in the skin below the level of the lesion. These changes, which are similar to those found by Head and Riddoch in men with transections of the spinal cord, can be divided into two distinct stages. During the first stage, which lasts from twenty-five to seventy-five days, the skin is dry, while during the second stage, which is more or less permanent, it becomes soft and moist and often is actually covered with visible sweat.

By means of the electrical skin resistance method, it is possible to obtain a continuous picture of the onset, magnitude and duration of these changes throughout the two stages.

During the first stage, when the skin is dry, the electrical resistance is high—much higher than would ever be suspected on the basis of palpation or inspection. Thus in all of our animals, it increased from a preoperative level of 40,000 to at least 2,000,000 ohms, while in some it rose to more than 25,000,000. During this stage, the galvanic skin reflex is absent.

During the second stage, when the skin is soft and moist and the sweat glands are obviously functioning again, the electrical resistance is as low or lower than before transection. At this time the galvanic skin reflex and spontaneous currents are present.

The magnitude of the increase in the resistance of the skin was shown to depend on the level of the lesion with relation to the thoracicolumbar outflow of the sympathetic nerves. A similar observation was made by Head and Riddoch regarding the distribution of areas of excessive sweating in men with transections of the spinal cord.

By measuring the conductivity of the skin, it was possible to compare the effects produced on the sweat glands by transection of the spinal cord, sympathectomy and total nerve section, and in this way to throw more light on the question of what central and peripheral factors are involved in the control of the sweat glands.

It was suggested that this method may be used in the future to study the localization and extent of injuries to the spinal cord in man.

CEREBRAL PSEUDOTUMORS *

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Of the 1,209 cases of brain tumor, on file in our clinic, both verified and unverified, 30 have been classified as pseudotumors or arachnitis. In 8 of these the data are insufficient or the patient cannot be traced, and therefore of this number I have reviewed the records of only 22 cases; all but 4 were first observed before 1929, so that at least two years have elapsed, a period which has been proposed as a criterion by which to exclude tumors proper. Of these 22 cases, 11 presented posterior fossa symptoms and 11 pretentorial symptoms. Of the pretentorial group, 4 suggested a pituitary lesion and 1 a suprasellar lesion. In the 22 cases we have information of 1 patient covering a period of twenty-five years, 1 nineteen years, 2 thirteen years, 1 twelve years, 1 eight years, 3 six years and the remainder four years or less. Of the total number but 3 have died. From the entire series will be selected for presentation in detail only a few individual cases, each of which is illustrative of a distinctive clinical picture.

NOMENCLATURE

The term "arachnitis," frequently used to imply pseudotumor, is rather loosely used, and one finds it frequently in the reports of roentgenologists. By them it is used to indicate an unusual collection of fluid, often circumscribed, over the cerebral or cerebellar hemispheres, as revealed by collections of air in the encephalogram. With them there has been no opportunity for verification by either inspection or pathologic studies. Terms other than arachnitis or arachnoiditis have been used as synonyms for pseudotumor. As a matter of fact, in the first observations on circumscribed collections of fluid, such as that by Schlesinger [1] in 1898, and that of Spiller [2] in 1903, the lesion was diagnosed not as arachnitis, but as circumscribed serous meningitis. In both of these cases the lesion was in the spinal canal. The term "pseudotumor" was applied first by Nonne to lesions of the brain. In the accumulated literature, already extensive, the condition under consider-

* Submitted for publication, May 2, 1930.
* From the Neurosurgical Clinic of the University Hospital.
* Read at a Meeting of the Philadelphia Neurological Society, April 25, 1930.

1. Schlesinger: Beiträge zur Kenntnis der Rückenmarks und Wirbeltumoren, Jena, 1898.

2. Spiller: Univ. Pennsylvania Med. Bull., March-April, 1903.

ation is variously referred to as "pseudotumor," "meningitis serosa," "meningitis serosa circumscripta," "meningo-encephalitis serosa," "serous effusions of inflammatory origin," "arachnitis" and "generalized cisternal arachnoiditis." Donner,[3] under the term pseudotumor, described in detail the pathologic process as represented by a chronic cerebral edema, especially over the medulla oblongata. His patient died suddenly, apparently in a cerebellar crisis as a result of a foraminal hernia.

There has been some discussion as to what justifies the diagnosis or designation of a lesion as pseudotumor. As the underlying pathologic lesion may be varied, and since in a number of cases recorded as pseudotumor no histologic studies were made and in others the macroscopic or naked eye appearance, apart from the collection of cerebrospinal fluid, was not distinctive, it seems to me that the distinguishing features justifying the term pseudotumor should be (1) the course of the disease and (2) the symptomatic picture. Finkelnburg [4] laid down as the first prerequisite for the diagnosis "pseudotumor" a sufficiently long period of observation, at least two years, although this is not infallible, as gliomas, he said, may be present for ten years, often with long remissions of the cerebral symptoms. But on the time basis alone he excluded six of Nonne's cases and several of other sources.

Puusepp [5] referred to several cases of brain tumor in which there were remissions of years and to cases of brain tumor described by Redlich in which a spontaneous retrogression occurred. Hence Puusepp said that recovery alone cannot be taken as a criterion until several years have elapsed.

Finkelnburg,[4] in a critique of twenty-four articles that had appeared prior to 1912, rejected certain cases diagnosed as pseudotumor on the pathologic observations. Many, he thought, should have been designated as meningitis serosa, or meningo-encephalitis serosa. On the other hand, Muskens [6] emphatically disagreed. In agreement with Raymond, he maintained that pseudotumor and meningitis serosa are identical, and referred to a series of cases in which he could demonstrate at operation localized foci of meningitis serosa and encephalitis of the cerebral cortex. Thus it seems apparent that if one attempts to limit the term pseudotumor to a certain pathologic picture, confusion and disagreement will arise. It is much better, it seems to me, to accept the convention that the clinical course and the symptomatic picture shall be the determining factors.

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3. Donner: Deutsche Ztschr. f. Nervenh. **72**:48, 1921.
4. Finkelnburg: Zentralbl. f. d. Grenzgeb. d. Med. u. Chir. **15**:515, 1912.
5. Puusepp: Tumoren des Gehirns, 1930.
6. Muskens, quoted by Finkelnburg (footnote 4).

ETIOLOGY

What of the etiology of pseudotumors? No doubt in the majority of cases there must be an infective agent. In case 1, to be described, the patient had had influenza a year before the onset of the symptoms. In a number of histories it is recorded that the patient had had in childhood one of the exanthems and later an infectious disease. Thus in fourteen of the series, measles, diphtheria, influenza, pneumonia, whooping cough, scarlet fever, typhoid and tonsillitis are mentioned in the record of previous illnesses.

CASE 1.—*A young woman developed signs of intracranial pressure without focal symptoms. A subtemporal decompression revealed excessive pressure and a large quantity of free fluid. Eleven years after the operation the patient was symptom-free.*

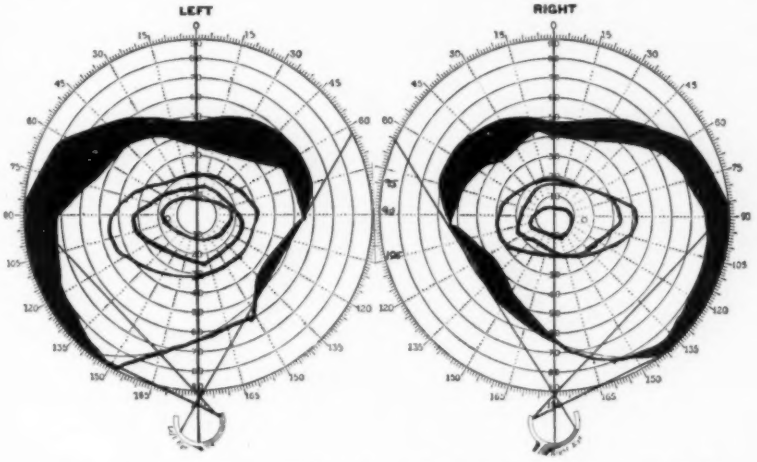

Fig. 1 (case 1).—Preoperative visual fields taken on Nov. 12, 1919.

History.—M. L. F., a woman, aged 22, was referred to the neurosurgical service of the University Hospital by Dr. George E. de Schweinitz on Nov. 27, 1919. She had had pneumonia when a child, diphtheria in 1911, and influenza in 1918. She was a junior at college. In May, 1917, she began to have headaches, mostly at night. In September, the headaches became much more severe; they were mostly on the right side, at times frontal and again occipital. In October, she began to vomit and was taken to a suburban hospital. Sleep was disturbed and the physician said she was "flighty" at times. She had had dizzy spells.

Examination.—There were no signs of a focal lesion. The features were coarse, arousing a suspicion of pituitary dysfunction. Roentgen examination revealed some enlargement of the pituitary fossa with slight atrophy of the posterior clinoid processes. The patellar reflexes were exaggerated. There was no disturbance of gait and no incoordination.

In both eyes the swelling was plus 4 diopters. Vision in the left eye was 2/5. There was concentric contraction of the fields (fig. 1).

Operation.—A subtemporal decompression was performed on the right side on Nov. 28, 1919. The dura was under considerable tension and when it was incised a large quantity of cerebrospinal fluid escaped and continued to flow until the wound was closed.

Course.—Immediately after the operation a large hernia cerebri developed at the site of the decompression, and for a while there was weakness of the left side of the face, and of the left arm and leg. This subsequently disappeared.

On Feb. 19, 1920, three months after the operation, the vision of each eye was 6/7. The choking of the disks had practically disappeared.

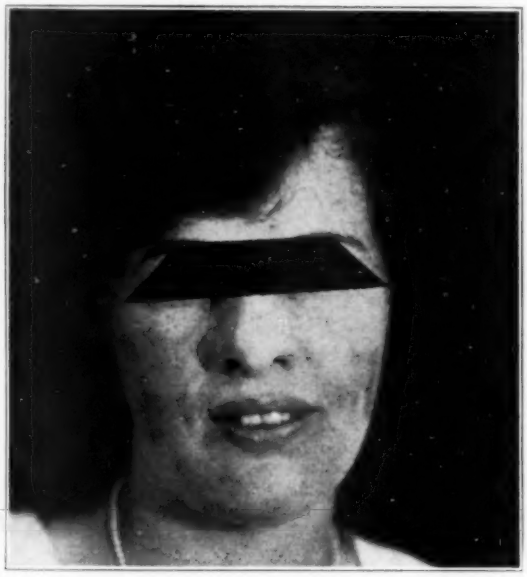

Fig. 2 (case 1).—Patient ten years after operation.

Ten months later, the papilledema and the hernia cerebri had disappeared. The patient resumed her studies at college and graduated in the following spring.

In 1925, the patient was married, and in March, 1930, eleven years after the operation, she was entirely well (fig. 2).

Comment.—From the standpoint of time and the positive signs of increased intracranial pressure, there can be no question that this case belongs to the pseudotumor group. From the evidence at hand one cannot hypothecate the pathologic process. Whether this was an arachnitis or a serous meningitis is immaterial. A preoperative diagnosis of brain tumor was justified, and the subsequent course of events unquestionably eliminated this as a possibilty.

In one instance, that of a child (case 2), convulsions began one month after a fall down stairs. Whether this was a coincidence or a responsible factor is conjectural.

CASE 2.—*In a child convulsions, headache, vomiting and papilledema developed. Eighteen years after the evacuation of a circumscribed collection of fluid and subtemporal decompression the patient was symptom-free.*

History.—L. G., a boy, aged 3 years, was referred to the neurosurgical service of the University Hospital by Dr. J. P. Crozer Griffith on Oct. 7, 1911. Delivery had been normal, and the child was perfectly well until the recent illness. One month before admission, the patient fell down a flight of stairs but suffered no apparent ill effects. One week later, convulsions developed and since then he had had eight seizures. For the past two weeks, he had complained of headache and vomiting.

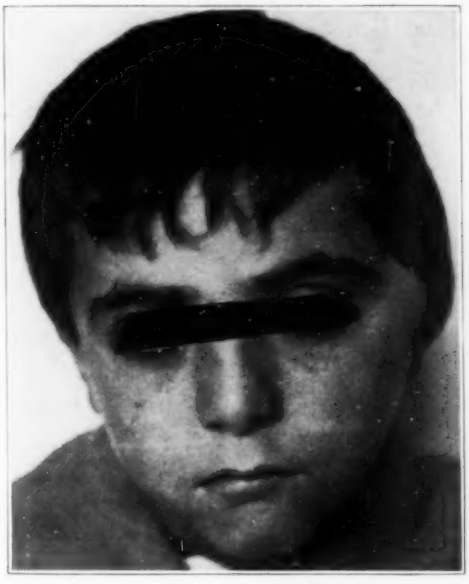

Fig. 3 (case 2).—Patient following operation in October, 1911.

Examination.—There were diminished patellar reflexes, no pathologic reflexes and a doubtful Kernig sign. There were signs of postpapillitic atrophy; the swelling on both sides was plus 6 diopters.

Operation.—A subtemporal decompression was done on the left on October 23. On incision of the dura a circumscribed collection of fluid was found. A large quantity of clear fluid, which was under great tension, escaped. The surface of the brain was displaced more than an inch from the dura, and when the fluid escaped the brain slowly resumed its normal relation to the dura.

Course.—Convalescence was uneventful, but there developed at the site of the decompression a marked hernia cerebri (fig. 3). On October 31, eight days later, the disks measured plus 4 diopters, the general condition had improved, and the patient was wide awake, alert and less irritable. On November 23, the papilledema had disappeared.

The patient has been under observation at various times from the day of discharge, Jan. 21, 1912, until April, 1930. On Feb. 11, 1920, a neurologic examination gave entirely negative results, with the exception of failing vision. The

hernia cerebri had entirely disappeared, and the patient was self-supporting, with enough vision to enable him to get about without assistance (fig. 4).

Comment.—I included this case in the series of pseudotumors as one with a large circumscribed collection of fluid over the left temporal lobe, with all the signs of increased intracranial pressure but without any localizing symptoms. The patient has survived the operation and has remained symptom-free for eighteen years. Because of the post-papillitic atrophy there is marked deterioration of vision.

The question has arisen whether the presence of a positive Wassermann reaction and the presumption of syphilis should or should not

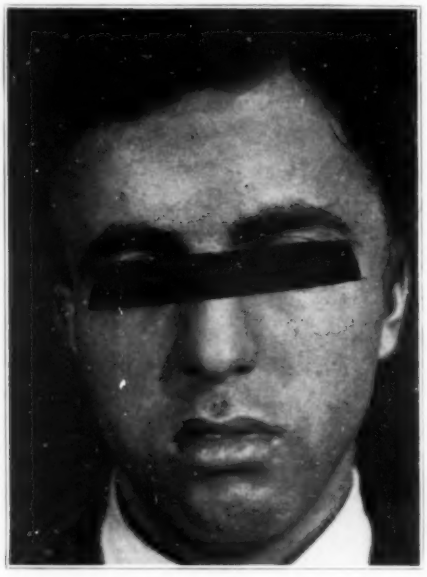

Fig. 4 (case 2).—Patient nineteen years after operation.

exclude a case from the category of pseudotumor. In the literature one finds cases reported as pseudotumor in which there was positive evidence of syphilis. In one of my cases, which I shall present in abstract, there was a plus 4 Wassermann reaction.

CASE 3.—*A young woman had a series of convulsions four years prior to admission; there were signs of increased intracranial pressure and dysfunction of one cerebellar hemisphere. Following a subtemporal decompression the symptoms entirely disappeared, and she was symptom-free eight years later.*

History.—E. K., a girl, aged 16, was referred to the neurosurgical service of the University Hospital on Feb. 24, 1922, by Dr. Charles Potts.

Examination.—There were partial paralysis in the distribution of the right facial nerve, a positive Romberg sign, a tendency to fall to the right, finger-to-nose test clumsy on the right, station poor, walking with the feet wide apart.

staggering to the right, hypesthesia of the right side of the face, and right patellar, biceps and triceps reflexes absent.

Roentgen examination showed increased convolutional markings.

A binasal superior quadrantopsia for form and colors was present and there was an enlargement of the blind spots.

Vision on the left was 20/30; on the right, 4/100. Papilledema was present on both sides, with swelling of plus 7 diopters (fig. 5).

The cerebrospinal fluid pressure was 260 mm. of water.

While before admission there had been reported a positive Wassermann reaction, after admission both blood and spinal fluid gave a negative Wassermann reaction.

Operation.—A right subtemporal decompression was done on March 8. An excessive quantity of cerebrospinal fluid escaped.

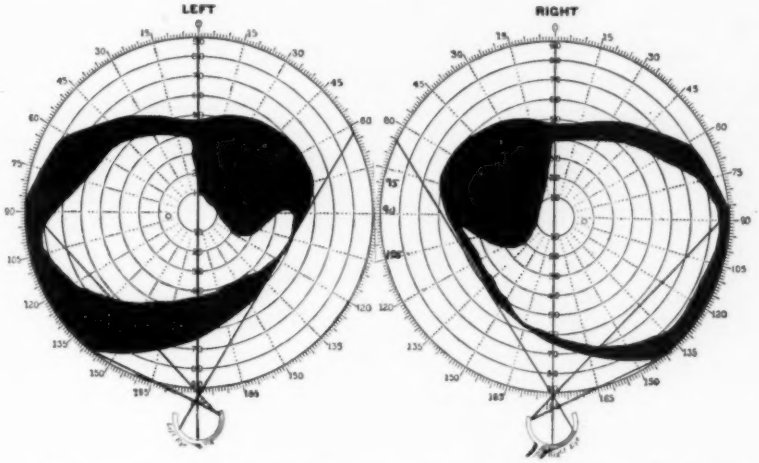

Fig. 5 (case 3).—Preoperative visual fields taken on Feb. 24, 1922.

Course.—A marked hernia cerebri developed. She received seven doses of neoarsphenamine. On October 22, the headache and vomiting had entirely disappeared and a neurologic examination gave negative results.

On April 15, 1930, the patient was found to be entirely free from any focal signs or evidence of increased pressure; it was eight years since the operation. The hernia had subsided (fig. 6).

As to the etiology of pseudotumor from the pathologic point of view, I shall refer later to the many pathologic processes involved. Mention may be made here of ependymitis, as described in 1910 by Merle,[7] and of changes in the choroid plexus. These structural changes no doubt interfere with the circulation of the cerebrospinal fluid and determine a dilatation of the ventricles and stasis of lymph in the cerebral tissues. Thus the foundation for increased intracranial

7. Merle, quoted by Puusepp (footnote 5).

pressure is established, and there follow the symptoms of increased pressure which simulate tumor.

In some instances, especially those with focal symptoms, the infiltrating process extends beyond the membranes to the brain tissue itself. A number of instances of meningo-encephalitis, as the background of pseudotumor, have been recorded. Other physical factors no doubt may operate to produce the picture. If the perivascular spaces are penetrated under pressure with cerebrospinal fluid, the capillaries may be compressed and the lymph circulation obstructed. Obstruction to the capillary circulation may of itself interfere with the function of

Fig. 6 (case 3).—Patient eight years after operation.

the area involved, and thus another factor in the creation of focal symptoms is evolved. If this hypothesis is accepted, one can understand, as Puusepp pointed out, why a decompressive operation or lumbar puncture alone may be effective.

PATHOLOGY

The pathologico-anatomic cerebral changes leading to the picture of pseudotumor cereberi are still unknown, as in many cases coming to autopsy no marked anatomic changes were noted. Macroscopic examination of the brain usually gives negative results in these cases. The process has been attributed to a serous meningitis, inflammatory

gliomatosis or edema of the brain. In Bailey's case there was a hemorrhagic leptomeningo-encephalitis.

Microscopic examination has revealed mild inflammation of the meninges, changes in the cerebral nerves (neuritis, perineuritis and interstitial changes), lymphocytic infiltration of the pia and proliferation of the lymphocytes in the perivascular spaces, enlarged glia cells and thickening of the vascular walls. Puusepp noted a peculiar chronic process in the form of a vaguely defined serous meningitis with a fibrin deposit. He explained the changes in the vascular wall as being due to extension of the process inward from the surface of the brain. He found changes also in the plexus and in the ependyma of the ventricle which he believed to be of primary significance in the pathogenesis of pseudotumor. Marked changes were noted in the cells of the choroid plexus, some being completely destroyed, some enlarged or swollen and others without nuclei, while in some several nuclei could be distinguished. In other words, there existed both a destructive and a hyperplastic process as may be observed in glandular tissue during pathologically increased activity.

There was a perivascular infiltration of the large celled elements found also in the subepithelial connective tissue. Here and there small cystic formations appeared. These occurred also in the ependyma. There was a slight subepithelial infiltration of the ventricular walls. Beneath the ependyma the infiltration was limited chiefly to the perivascular spaces. Lymphocytes were found in most cases.

In Bailey's case, in which there was a definite cerebellar hemisphere syndrome, the combination of interstitial and parenchymatous changes proved that he was dealing with a case of hemorrhagic leptomeningo-encephalitis. In view of the widespread involvement of the brain in cases of meningo-encephalitis, he found it not surprising that it is difficult clinically to locate the lesion and that striking pathologic changes should be found in areas not indicated by clinical observation. Bailey thought that the term "pseudotumor cerebri," if used at all, should be restricted to purposes of clinical classification, without attributing to it any etiologic or pathologic significance.

CLINICAL TYPES

It has been suggested[8] that cases of pseudotumor be classified according to localizing symptoms; thus: a group with symptoms pointing (1) to the motor cortex, (2) to the posterior fossa, (3) to a cerebral hemisphere and (4) to the base of the brain. In my series of twenty-two cases there were four in which I was inclined to believe

8. Finkelnburg: Deutsche Ztschr. f. Nervenh. **38**:35, 1910.

that the patient had a pituitary lesion. At least there were field distortions and signs of pituitary stigmas. In each of these, as in case 4, a solid lesion was not found. In an additional case in which an operation was performed recently, with similar observations, the patient is now under observation, but sufficient time has not elapsed to justify a final statement as to the diagnosis or the result. The others have improved since the operation, and their cases have been classified as pseudotumors of the pituitary group.

CASE 4.—*A patient with failing vision, hemianopia and certain pituitary stigmas was believed to have a pharyngeal duct cyst or suprasellar lesion. Exploration failed to disclose a solid lesion; six years afterward, the patient's vision was perfect in one eye and improved in the other.*

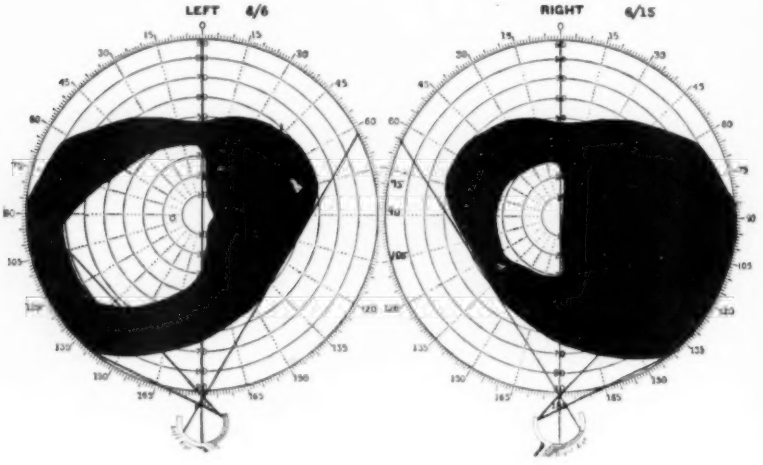

Fig. 7 (case 4).—Preoperative visual fields taken on Jan. 15, 1924.

History.—O. G., a man, aged 21, was referred to the neurosurgical clinic of the University Hospital on Jan. 14, 1924, by Dr. T. D. Casey. He had had measles as a child, and at 10 was struck under the eye with the bung of a beer barrel. At 7 he could not see well with the right eye, but during the past three years vision had grown worse and he had had visual hallucinations (zigzag lines). From 7 to 14 years of age he had had convulsions. Diplopia appeared at 14. Appetite was excessive, and he was fond of sweets.

Examination.—The patient was obese, with tapering fingers. He was mentally clear and cooperative. The reflexes were not exaggerated, and there were no motor or sensory disturbances.

There were partial optic atrophy and right homonymous hemianopia. Vision in the right eye was 6/15; in the left eye, 6/6 (fig. 7).

A ventriculogram showed no defect of the lateral ventricles and a patulous third and fourth ventricle.

The cerebrospinal fluid pressure was 130 mm. of water; it gave a negative Wassermann reaction.

Possible ethmoiditis was suggested. The basal metabolic rate was minus 22.

Preoperative Diagnosis.—The diagnosis was extrapituitary lesion, possibly a cyst of the pharyngeal duct.

Operation.—A transfrontal craniotomy was performed on January 23. The exploration failed to reveal any evidence of a solid growth, and the symptoms were attributed to an arachnitis.

Course.—Before the patient was discharged from the hospital the fields had improved 50 per cent. In a communication dated April 4, 1930, six years after discharge, the patient wrote that for a year after the operation he had felt an eye strain, but since then vision had improved and is now "100 per cent in the left eye, and 50 per cent in the right." Generally speaking, he felt better than he had for many years.

In my series, as with most cases recorded by others, the majority of patients had symptoms of cerebellar dysfunction. In only one, case 5, were the symptoms suggestive of a lesion of the motor cortex.

CASE 5.—*A man with jacksonian seizures was found at operation to have a circumscribed collection of fluid, with thickened arachnoid, near the mesial surface of the motor cortex.*

History.—W. F., a man, aged 46, was referred to the neurosurgical clinic of the University Hospital on Jan. 18, 1928, by Dr. Charles Kelly and Dr. C. D. Vernooy. He had had measles, mumps, whooping cough and scarlet fever in childhood. Later he had had typhoid fever. Four years before examination, tremors and weakness appeared in the left leg, followed by a period of unconsciousness. Four months later, the patient had a second attack, with convulsive movements of the left leg and unconsciousness as before. The attacks recurred at intervals of three months for the next two years. Since the onset of these attacks, the patient had had frequent headaches, mostly occipital, and for a day or two after the attacks his memory was defective. At times while lying in bed the left leg and arm twitched. Occasionally he complained of paresthesias in the left hand and foot.

Examination.—Neurologic, roentgen and other studies gave entirely negative results.

Operation.—A craniotomy was performed on the right side on January 26. On reflecting a flap to expose the motor cortex, there was revealed a circumscribed collection of fluid near the mesial surface, with a thickened arachnoid. The fluid was evacuated and a strip of thin celloidin tissue introduced into the cavity for drainage.

Course.—Convalescence was uneventful, and up to the time of discharge, on February 10, there was no recurrence of the attacks. Letters have been received from time to time, the last on April 10, 1930. The attacks have reappeared; the patient is irritable and seems to be developing a psychosis.

DIAGNOSIS

Whether one can by any means differentiate between true tumors and pseudotumors seems questionable. The encephalogram might be of value were the collection of fluid not definitely encapsulated. Donner [3] said that pseudotumors may be distinguished from true tumors

by the slow course and the benign nature of the lesion. There is no doubt that the duration of the symptoms may be a useful differential sign, as in some of my cases the onset of symptoms preceded observation by a number of years. If one includes syphilitic lesions with pseudotumors, the serologic reactions may be the determining factor.

In most cases of pseudotumor the symptoms of general intracranial pressure usually predominate, and focal symptoms may be wanting. In the life history of brain tumors I have observed two modes of onset: In one the initial symptoms are those of increased intracranial pressure. In the other the initial symptoms are definitely focal. With pseudotumor, however, the signs of intracranial pressure almost invariably precede the focal signs, and as a rule there is a long interval between the onset of one and the onset of the other. It has been said that pleocytosis in the cerebrospinal fluid is not uncommon in pseudotumor, but is rare in real tumors.

The mimicry of the true tumor picture is in many instances striking, and this holds true particularly of the cerebellar group. Of these I have a number of illustrative cases, some of them of extraordinary interest. One only will be cited.

CASE 6.—*A history of headache and vomiting for eighteen months, loss of hearing on the left for six months. Encapsulated collection of fluid at the internal auditory meatus. Excellent health one year later.*

History.—G. W. C., a man, aged 34, was admitted to the neurological service of the University Hospital on Feb. 12, 1928, referred by Dr. W. C. Payne, complaining of nausea, vomiting, tinnitus, failing vision and dizziness. For eighteen months there had been headache, nausea and vomiting, and for six months impairment of hearing in the left ear, and later complete deafness. The patient was irritable; his station and gait were unsteady.

Examination.—The following positive observations were noted: staggering to the left; positive Romberg sign; ataxia and dysmetria of the left arm and leg; held head to the left and lay on the left side; in the horizontal plane past-pointed to the left with the left hand; total deafness in the left ear, and loss of the left corneal reflex. The vestibular reactions indicated a lesion of the cerebellopontile angle, and roentgen examination revealed some erosion of the left petrous bone.

Operation.—A suboccipital craniectomy was done on March 6. An unusual quantity of fluid escaped when the dura was opened, and an encapsulated collection was found at the internal auditory meatus.

Course.—When the patient's physican last reported one year after the operation, he wrote: "The patient is apparently in excellent health, and states he never felt better in his life."

TREATMENT

The treatment for pseudotumors in the majority of cases has been operative, chiefly because the diagnosis has not been established before the lesion was exposed on the operating table. Some improvement has been noted in patients treated by Nonne and Hoppe by the administra-

tion of mercury and iodides. Obviously, in the presence of a positive Wassermann reaction antisyphilitic treatment is indicated. Redlich said that irradiation has proved of value in some cases, and good results have been recorded after the use of hypertonic salt and dextrose solutions. Finkelnburg [8] saw no improvement after lumbar punctures, and referred to several deaths following this procedure. On the other hand, Puusepp reported two recoveries after lumbar puncture. In one of my cases there has been a decided improvement following tonsillectomy, the tonsils having been suspected as the source of infection.

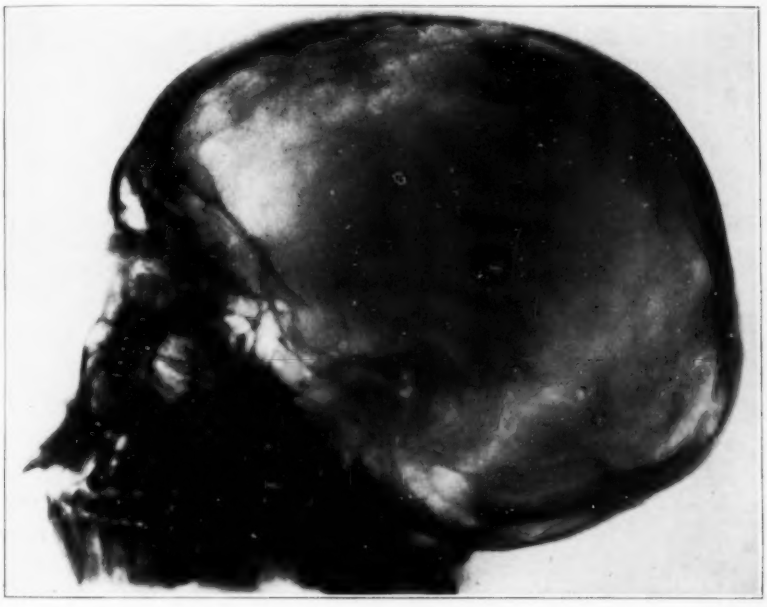

Fig. 8 (case 7).—Roentgenogram, showing general convolutional atrophy, atrophy of the dorsum sella and some enlargement of the pituitary fossa.

Case 7.—*History.*—M. F., a girl, aged 14, was referred to the neurosurgical service of the University Hospital on May 25, 1929, by Dr. S. A. Rulon. She had had scarlet fever at 6 years, followed by otitis media and several attacks of tonsillitis. Since the age of 6, after the scarlet fever, she had had attacks of headache and vomiting, and vision had begun to deteriorate. There was a bilateral papilledema of plus 2 diopters. The pressure of the cerebrospinal fluid was 312 mm. of water. The roentgen rays showed some enlargement of the pituitary fossa and convolutional atrophy. There was an homonymous hemianopia and a positive Romberg sign (fig. 8).

Course.—On April 21, 1930, the papilledema had subsided, the tonsils had been removed and the patient was symptom-free; an exploratory operation had not been performed.

In two cases of my series the diagnosis was suggested by an encephalogram. Sufficient time has not elapsed to warrant a final report to confirm the diagnosis and record the end-result. But the question arises as to whether, in all cases in which there is a reasonable doubt as to the diagnosis, encephalograms should not be the first step in the management of the case. When the collection of fluid is definitely encysted an encephalogram would not differentiate between a solid and a cystic lesion, but the fluid is not always encysted.

In my twenty-two cases the following surgical procedures were carried out:

Suboccipital decompression	1
Subtemporal decompression	6
Suboccipital craniectomy	6
Transfrontal craniotomy	3
Callosal puncture	2
Craniotomy	2
Encephalogram	2
Ventriculogram	2
Ventricular estimation	1
Ventricular puncture	2
No operation	2

RESULTS

I shall not attempt a comprehensive statement of the results in other clinics. In my series of twenty-two cases, nineteen patients are alive after periods varying from one to twenty-five years. Three have died; one immediately following a subtemporal decompression, one at an unknown date following a suboccipital craniectomy and one more than seventeen years after a suboccipital craniectomy. The last patient had been in perfect health for seventeen years when he developed some gastro-intestinal disturbance for which an operation was necessary. He died a few months later following a second operation for this condition, which is reported to have been carcinoma. I have in no instance been called on to operate a second time, although I have advised a second operation in one case with definite symptoms of recurrence. Apparently relief from pressure suffices, although one wonders, especially in the encysted cases, why there should not be more recurrences. The longest period of relief in the series, as recorded in the following case, was twenty-five years.

CASE 8.—*A patient with signs of increased intracranial pressure and cerebellar dysfunction, following a suboccipital decompression is symptom-free, with vision preserved in one eye, twenty-five years after operation.*

History.—S. L., a man, aged 20, was referred to the neurosurgical service of the University Hospital on Oct. 25, 1905, by Dr. T. H. Weisenburg. His family and social history were without significance. He had measles in infancy;

otherwise he had always been in excellent health. One year before examination, he began to have attacks of nausea and vomiting, with sharp pains in the neck and back of the head. These attacks were more frequent at first than at the time of examination. Two months previously, vision in the left eye began to fail and at the time of examination he had only light perception. At the same time he noticed that he was deaf in the left ear and staggered while walking.

Neurologic Examination.—He was unable to converge with the left eye. Associated ocular movements to both right and left seemed affected; he could look to either right or left to the normal extent, but in a short time the eyeballs had a tendency to jerk back toward the median line. There was no diplopia. Hearing was diminished on the left side, and the patient staggered in walking. Both disks were swollen, and hemorrhages were profuse.

Operation.—A suboccipital craniectomy was done on October 26. On removal of the bone, the dura appeared under great tension, yellowish and opaque. It was dotted with yellowish-white specks. The possibility of a tuberculous lesion was suspected. The dura was not opened, and the wound was closed.

Course.—Examination of the eyes gave the following results: October 31: right eye, swelling, plus 11 diopters; left eye, swelling, plus 9 diopters; vision: right eye, 6/22; left eye, shadow; November 16: right eye, swelling, plus 8 diopters; left eye, swelling, plus 8 diopters; December 22: right eye, swelling, plus 7 diopters; left eye, swelling, plus 7 diopters; vision: right eye, 6/12; left eye, atrophy; Feb. 1, 1906: right eye, swelling, plus 1 diopter; left eye, swelling, plus 1 diopter; vision: right eye, could read and write; left eye, count fingers.

The patient was discharged from the hospital on Dec. 23, 1905.

Six weeks after discharge, he was entirely relieved of the symptoms of which he had complained and the papilledema had practically disappeared.

Readmission.—On May 16, 1906, the patient was readmitted after a recurrence of the symptoms, including nausea, vomiting, dizziness, headache and ataxia.

Second Operation.—On June 15, the wound was reopened, and free radiating incisions were made in the dura. The cerebellar hemispheres bulged a little through the opening. No further exploration was made, and the wound was closed.

The patient was entirely relieved by this operation, and was discharged on June 30.

Subsequent Course.—The patient was examined on June 12, 1916, and on Nov. 17, 1920, at which times, with the exception of occasional attacks of vomiting, he was entirely well. On the latter date vision in the right eye was 6/12 and in the left eye it was diminished to hand movements. The fields of the right eye were normal; of the left eye barrel vision.

On April 14, 1930, twenty-five years after the operation, the patient wrote that his condition, if anything, was better than when he was examined on Nov. 17, 1920.

Comment.—Although at the operation, a suboccipital decompression, no search was made for the lesion, and although its precise nature and pathology are undetermined, it seems that the condition is with propriety recorded as pseudotumor. Measured by the standard adopted, the symptom complex and the duration of the period of relief, it qualifies as a pseudotumor. Not only have the pressure and cerebellar symptoms disappeared, but vision in one eye has been conserved. From

Oct. 3, 1905, to Feb. 1, 1906, the swelling of the disks subsided from plus 11 to plus 1 diopter. Vision in the good eye, twenty-five years after the operation, is 6/12.

Whether or not it is proper, as in this case, to include in the category of pseudotumor cases in which the precise nature of the lesion has not been revealed may be questioned. Yet one notes that Finkelnburg,[8] in classifying the cases hitherto reported, divided them into four groups, the first of which includes those with apparently negative observations, both macroscopically and microscopically. This case might well be included in that group. The second group includes those with negative macroscopic but with satisfactory microscopic studies; the third those with negative macroscopic but with positive microscopic studies, and the fourth those with macroscopic cerebral changes.

TUMOR OF THE FILUM TERMINALE, WITH CYSTOMETRIC STUDIES

REPORT OF TWO CASES *

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AND

ABRAHAM KAPLAN, M.D.

ST. LOUIS

Tumors of the filum terminale have been observed so rarely that we have been prompted to report two cases that were studied and in which operation was performed in the last four years.[1] These cases have been of particular interest because of their rarity and because of their symptomatology. While we do not claim that the symptoms of such a lesion are so characteristic as to constitute a separate clinical entity, it nevertheless is noteworthy that the onset of the illness in the two cases was identical. These two cases, furthermore, emphasize the great value of cystometric studies of the bladder in tumors of the spinal cord. These tumors help to throw light on the mechanism of the function of the bladder and also on the sexual center of the conus.

LITERATURE

An extensive search of the literature has brought to light only three cases, one by Lachman [2] in 1882, which he reported as a glioma but of which he published no microscopic picture, though he gave a detailed account of the microscopic appearance. The gross appearance of his tumor was identical with that of one of ours. The plate that he published is reproduced in figure 1. The initial symptoms in this case began two years before the patient's admission to the hospital; there was dribbling of urine, followed by dysuria and accompanied by

* Submitted for publication, June 11, 1930.

* Dr. Howard A. McCordock furnished the photomicrographs.

* Read at the Fifty-Sixth Annual Meeting of the American Neurological Association, Atlantic City, N. J., June 10, 1930.

1. Steinke in his report of 330 cases of spinal tumor (Spinal Tumors: Statistics on a Series of 330 Collected Cases, J. Nerv. & Ment. Dis. **47**:418, 1918) observed no such case. Frazier and Elsberg in their books on spinal surgery also did not record such cases.

2. Lachman, B.: Gliom im obersten Teil des Filum terminale mit isolierter Compression der Blasennerven, Arch. f. Psychiat. **13**:50, 1882.

increasing constipation. Six months before entering the hospital, he had epigastric distress with vomiting and loss of weight. In the history on admission, it was learned that he had had paresthesia in both legs for a year. On physical examination, the outstanding features were a greatly distended bladder, a relaxed bladder sphincter, obstipation for six days before admission, exaggerated patellar reflexes with fibrillary twitchings of the calf muscles, arteriosclerosis and secondary anemia.

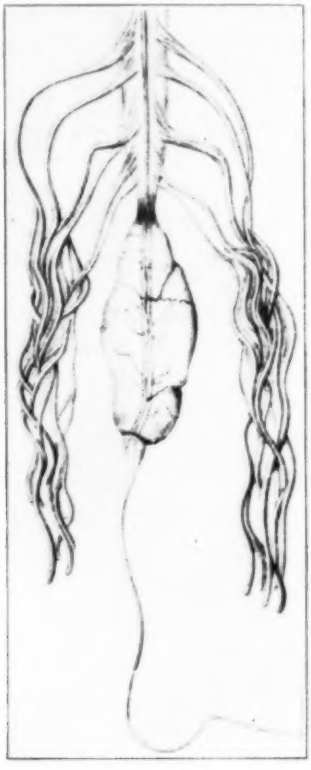

Fig. 1.—The tumor in Lachman's case. This is identical in location with the tumor in case 1.

The diagnosis was carcinoma of the bladder. The neurologic observations were negative, though there can be little doubt that a careful sensory examination would have revealed at least a partial saddle anesthesia, as in our cases. Autopsy revealed a tumor of the proximal portion of the filum terminale and not a tumor of the bladder.

In 1875, Gowers reported a small lipoma in this region, accidentally observed in a case of tabes dorsalis.

In 1899, Spiller,[3] while studying a large series of cases of tabes dorsalis in Dejerine's laboratory, found a small lipoma, 0.5 cm. in size, at the junction of the conus and the filum terminale. With Weigert-Pal hematoxylin he was able to show small groups of nerve fibers within the fatty tissue of the tumor, which, he stated, represented the delicate nerve fibers that may be separated from the periphery of the filum in many normal cases. With his usual thoughtful insight, he predicted that a tumor of the filum terminale would cause paralysis of the nerves that innervate the bladder, since they occupy the most central position and are in closest proximity to the filum. He went on to say, "An enlarged tumor of the filum terminale may first manifest itself by vesical symptoms, then possibly loss of sexual powers, and not show any evidence of motor or sensory disturbance of the lower limbs."

ANATOMY

The description of the anatomy of the filum terminale is meager. The best embryologic studies are those of Streeter,[4] from whom the following quotation is taken:

> The part played by the dedifferentiation in the caudal region of the spinal cord is more apparent in the younger stages of development. The so-called absorption of the tail is completed before the embryo reaches a length of 30 mm. In the case of the spinal cord dedifferentiation is well demonstrated in the period represented by embryos between 11 and 30 mm. long. The spinal ganglia show a regression varying from arrest in development to complete disappearance. All but two of the coccygeal ganglia have disappeared in the 30 mm. specimen, and the remaining two are of about the same size as the same two ganglia in the 11.5 mm. specimen.
>
> While the precoccygeal cord has continued to increase in thickness of its walls, and in the elaboration of the mantle and marginal zones, the coccygeal region is less advanced in its development than it was in the younger stage. While in the 11 mm. embryo there existed a distinct elaboration into ependymal, mantle, and marginal zones, the mantle zone is completely missing in the 30 mm. embryo, and we find thin walls consisting only of ependymal cells covered by a thin marginal zone. The coccygeal spinal cord in the 30 mm. embryo is in an earlier embryonic state than that of the 11.5 mm. embryo; that is, it has undergone dedifferentiation. In later stages the process still goes farther, and the ependymal tube becomes converted or redifferentiated into a fibrous strand.
>
> About opposite the first coccygeal nerve in the 30 mm. embryo the mantle zone abruptly disappears, and there is a corresponding enlargement of the lumen of the cord, thereby producing the thin-walled ventriculus terminalis. The transition is quite abrupt involving only one segment. Opposite the second coccygeal nerve the mantle zone is entirely gone. The ventriculus terminalis at this stage tapers caudally and may be said to extend to the third coccygeal segment. Caudal to

3. Spiller, W. G.: Lipoma of the Filum Terminale, J. Nerv. & Ment. Dis. **26**:287 (May) 1899.

4. Streeter, George L.: Formation of the Filum Terminale, Am. J. Anat. **25**:1 (Jan.) 1919.

this the dedifferentiation of the cord is more complete and results in the gradual obliteration of the lumen and the replacement of the ependymal substance by a fibrous strand, embedded in which can be found isolated groups of persistent ependymal cells. At its extreme tip there is often found a more or less detached group of cells which undergoes cystic enlargement and constitutes the coccygeal medullary vestige. The interval of cord lying between this and the ventriculus terminalis constitutes what is later known as the filum terminale. Its subsequent growth and elongation is accomplished by an interstitial increase of its constituent fibers.

There is no further dedifferentiation of the sacral region of the cord after the embryo has attained a length of 30 mm.

In the first twenty-five weeks of foetal life there is an ascent of the ventriculus terminalis from the level of the second coccygeal to the third lumbar vertebra, or a distance of nine segments before the adult position is reached.

The dura mater and its relations can be plainly recognized in the 67 mm. foetus, where it can be seen to reach and adhere to the filum terminale at the lower border of the fourth sacral vertebra, thereby sealing off the lower end of the subdural space. It undergoes very little change from its position here and that which it occupies in the adult.

According to Keibel and Mall,[5] as the central canal extends through the conus medullaris to the beginning of the filum it undergoes a conical expansion at its lower end. From this enlargement, which is termed the ventriculus terminalis, irregular side pouches open, and an occasional elongated blind sac may be found giving the lower end of the canal the appearance of being bent on itself. This can be seen in the adult specimen of the filum (fig. 2) which was cut serially to bring out this variation.

Because of the relatively fixed position of the cephalic end of the spinal cord, and because of the unequal increase in the rate of growth of the vertebral column and the spinal cord during the latter part of fetal life, at birth the caudal end is seen drawn upward, and that part of the cord from which the coccygeal nerves arise is opposite the third lumbar vertebra. In the adult it is opposite the first lumbar vertebra.

Gross and Microscopic Anatomy.—The filum terminale consists of two parts: (1) the upper, called the filum terminale internum, which is a direct downward prolongation of the conus medullaris and ends at the bottom of the dural sac at the level of the second sacral vertebra; this portion is on the average about 15 cm. long; (2) the lower, termed the filum terminale externum, which starts at the pointed closed end of the dural sac as a continuation of the filum internum, receives a covering of dura and extends downward to the first segment of the coccyx, attaching itself to the periosteum on the posterior surface of the vertebral column.

5. Keibel, F., and Mall, F. P.: Manual of Human Embryology, Philadelphia, J. B. Lippincott Company, 1912, p. 57.

The filum terminale internum can readily be distinguished from the surrounding nerves of the cauda equina by its bluish-white glistening appearance. Adhering to the outer surface of the filum there are often several fine nerve fibers, which, according to Rauber, represent one or two rudimentary coccygeal nerves (the second and third).

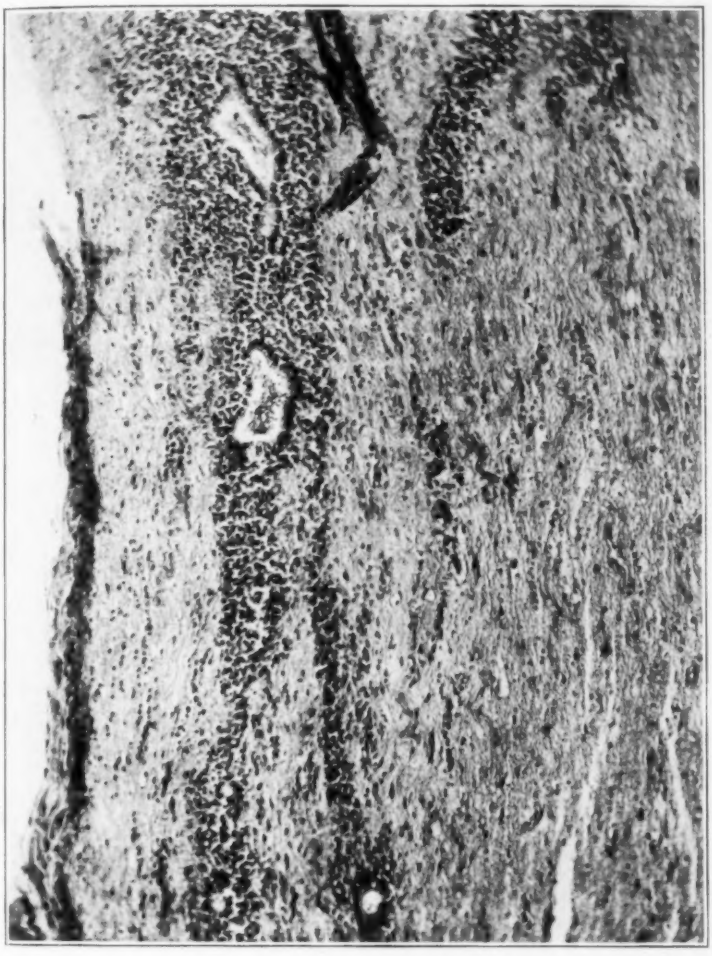

Fig. 2.—Longitudinal section of filum showing the medullary tube cut partly in cross-section and partly longitudinally: the ventriculus terminalis.

The central canal of the medulla spinalis extends for a variable distance, from 5 to 6 cm., down the upper portion of the filum internum. This portion of the central canal is surrounded by a layer of nerve substance in which nerve cells are usually present. A longitudinal section and a cross-section of the filum internum (figs. 2 and 3) clearly

show portions of the central canal, in one place bending on itself, also the columnar cells lining the canal, the nerve cells, fibrous tissue, neuroglia cells and the outer fibrous coverings which are derived from a continuation of the fused pia and arachnoid. The terminal branches of the anterior and posterior central arteries and accompanying veins can readily be identified.

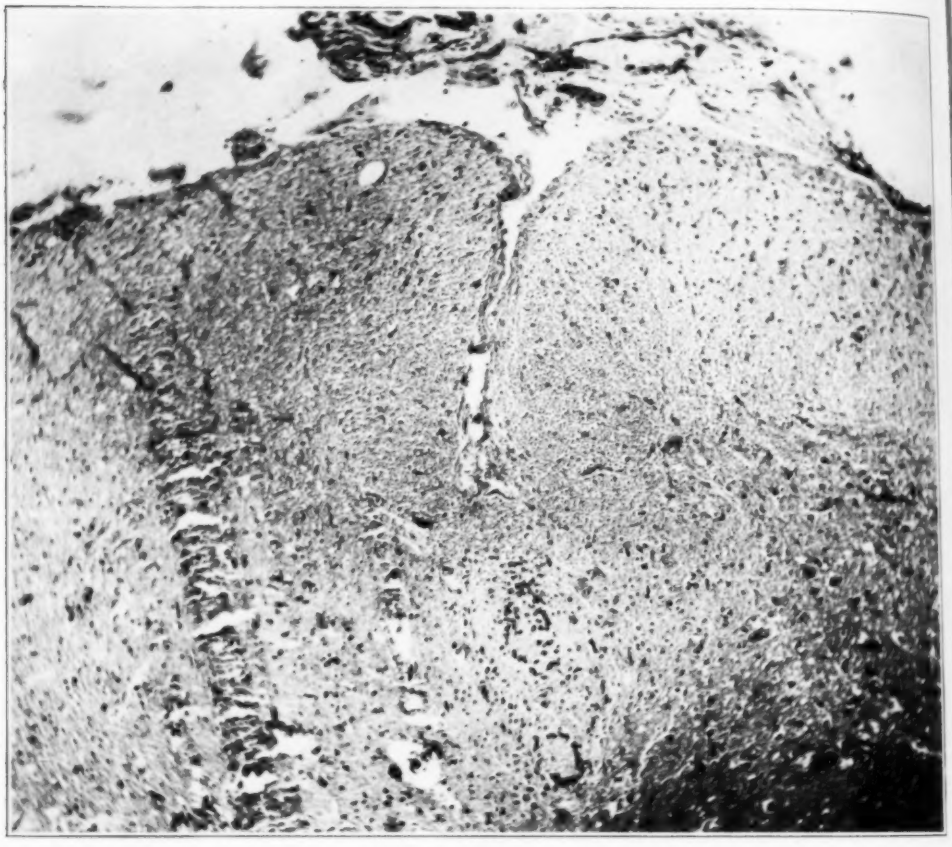

Fig. 3.—Cross-section above the tumor in case 1 showing the lowest portion of the filum terminale internum. This contains glial tissue and an occasional ganglion cell.

REPORT OF CASES

CASE 1.—*History.*—E. C. T., a court stenographer, was admitted to the hospital on Sept. 15, 1926, on the recommendation of one of us (D. K. R.), with the complaint of "bladder trouble." There was nothing significant in the family or marital history. At the age of 12, he had had a mastoidectomy, and on several occasions he had been treated for mild attacks of fever which were thought to be malaria. He said that he had not had venereal disease.

His illness started while he was taking a short vacation in the southern part of Missouri, thirteen days before his entry, with a sudden attack of severe diarrhea. This was followed shortly afterward by generalized abdominal pains, nausea and vomiting, which subsided after three days. The following day, he noted hematuria for the first time and experienced some difficulty in voiding. Two days later, he developed acute retention. He returned to the city and, after a cystoscopic and cystometric examination (by D. K. R), was referred to the neurosurgical service with the diagnosis of a "neurogenic bladder."

Examination.—The physical examination on entry to the hospital showed a well developed man resting comfortably, with no systemic abnormalities except

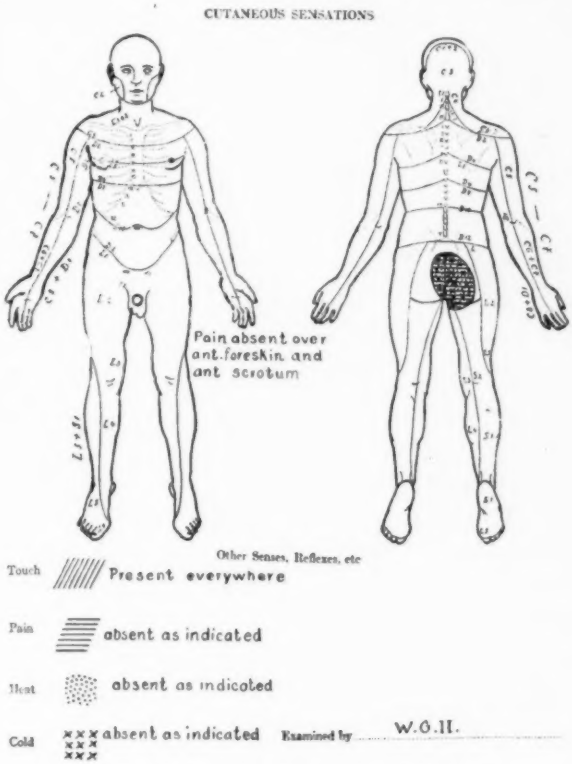

Fig. 4.—Sensory disturbances in case 1.

those referable to the genito-urinary tract and nervous system. The knee jerks and ankle jerks were hyperactive on both sides; there was slight spasticity of both lower extremities, with a bilateral Oppenheim sign, ankle clonus on the right and a positive Babinski sign on the left. Sensory examination revealed a saddle-shaped anesthesia corresponding to the third, fourth and fifth sacral skin segments (fig. 4).

Laboratory Studies.—The patient had to be catheterized twice daily because of a residual cloudy urine varying from 360 to 400 cc. It showed a trace of albumin, some red and white blood cells and epithelial cells but no casts or sugar. Culture of the urine showed staphylococci. The nonprotein nitrogen content of

the blood was 35.7 mg. per hundred cubic centimeters, and on a subsequent examination, 41.6 mg. The blood pressure was 100 systolic and 70 diastolic.

Cystoscopic and Cystometric Examination (by D. K. R.).—Cystoscopic examination showed a cystitis, large bladder capacity and relaxed internal sphincter and prostatic urethra. Cystometric examination showed a very large capacity (1,000 cc.) with low involuntary pressure (6 mm. of mercury), a normal voluntary pressure (54 mm. of mercury), and the pain of overdistention developing at 800 cc. The cystometric chart is shown in figure 5. The cystometric diagnosis was low spinal cord lesion of a destructive type.

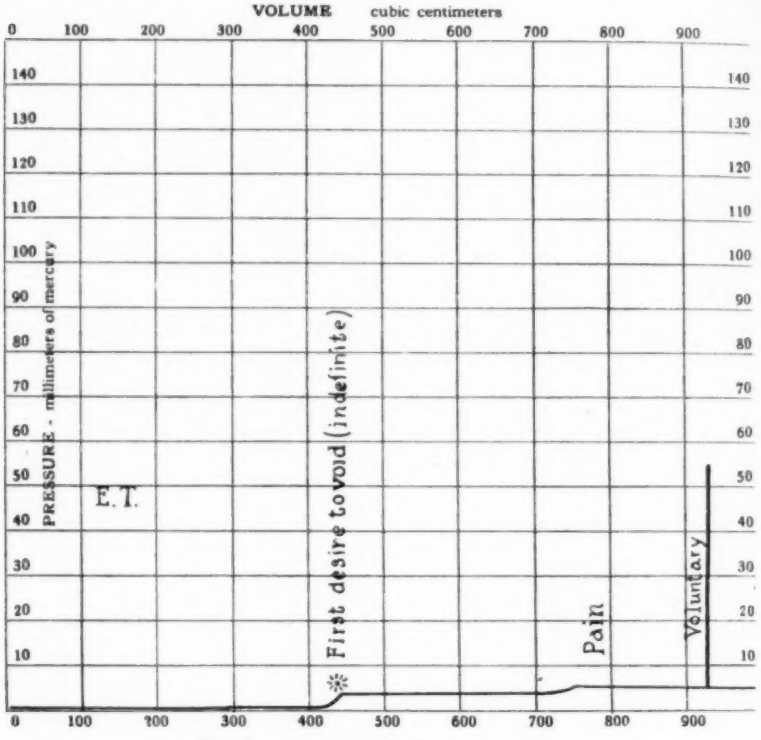

Fig. 5.—Cystometric chart in case 1.

Queckenstedt Test (by Dr. R. M. Klemme).—The fluid rose to 90 mm., and after bilateral jugular compression for ten seconds rose to 170 mm. After release, it dropped in "steppage" fashion to 110 mm. at the end of ten seconds. This was repeated with practically the same results. The spinal fluid was clear and colorless; the Pandy test was two plus; there were only 3 cells per cubic millimeter; the Wassermann test was negative, and the colloidal gold curve was 1122210000.

Roentgen Examination.—1. The urinary tract gave only a partial outline of both kidneys, but there was no clinical evidence of any calculi. 2. A stereoscopic (anteroposterior) picture of the dorsal and lumbar spine suggested the possibility of a destructive process in the lower portion of the body of the eighth dorsal vertebra.

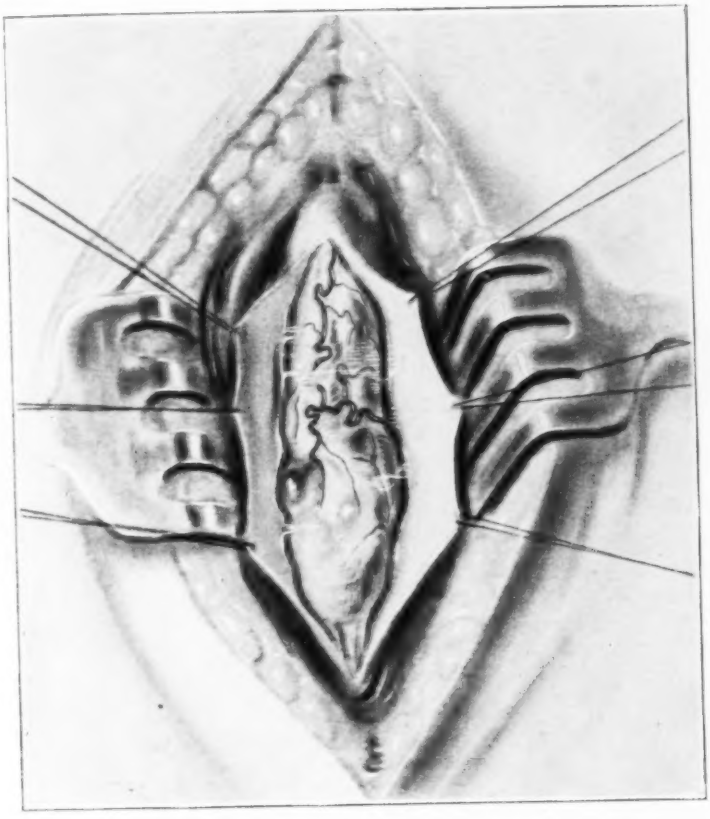

Fig. 6.—Appearance of the tumor in case 1.

Course.—The patient was told that he had a tumor of the spinal cord, but he was permitted to go home for a few days. He returned to the hospital on Sept. 27, 1926, for an operation. The observations on second admission were practically the same as previously, with the exception that the residual urine had increased to 800 cc. and he had a suggestive Babinski sign on the right side instead of on the left side, as had previously been found.

Localization Diagnosis.—All examinations left one of us (E. S.) uncertain regarding the exact location of the lesion. It might have been in the lower portion of the cauda equina at the exits of the spinal roots, or it might have been at the conus; consequently, iodized poppy seed oil 40 per cent was injected, but this threw no light on the case, as all the iodized oil dropped to the bottom of the canal.

Operation.—On Sept. 30, 1926, a laminectomy was performed by one of us (E. S.) under ether anesthesia. The spinous processes and laminae of the twelfth dorsal and first lumbar vertebrae were removed. There was a moderate amount of extradural fat, which when peeled away revealed a pulsating cord. When the dura was opened, one could see floating in the cerebrospinal fluid a tumor attached to the end of the cord, surrounded by the roots of the cauda equina. In order to reach the lower end of the tumor, it was necessary to extend the laminectomy as far down as the third lumbar vertebra. The subarachnoid space was opened and there presented a nodular tumor, cystic in places, extending from the conus down between the roots of the cauda equina. Projecting from the lower end of the tumor was the filum terminale. The portion nearest the lower end of the tumor was divided between two clips. Using the upper end as a handle, the tumor was raised from its bed, and it was found necessary to clip and cut some of the roots on the left side of the mass. The cord was then cut across at the upper end of the tumor (fig. 3). The dura was closed with interrupted silk; silver wire was used for the muscles; then there followed three layers of silk for the fascia, subcutaneous tissue and skin.

Postoperative Course.—The patient had to be catheterized several times a day; the bladder was irrigated, and a 25 per cent solution of a mild silver protein was instilled. On the sixth day following the operation, all the skin sutures were removed. The wound healed by primary union, and five days later the patient was able to sit in a chair. At the end of two weeks he walked about; he complained of slight pains in the calf of the right leg and a tired feeling over the lower portion of the back. He was discharged sixteen days after the laminectomy. The paralysis of the bladder gradually cleared, and after two months he had regained perfect control of the bladder. He has had no return of symptoms.

Pathology.—The tumor (fig. 6), 4 by 1.5 cm. in size, was irregularly fusiform, with a portion of spinal cord at one end and at the other a portion of a thin fibrous cord. It was definitely encapsulated, showing on its surface several cystic areas varying greatly in size. On section the cystic areas were found to be filled with dark brownish material resembling colloid material (fig. 7).

Microscopic Description.—In the cephalic portion of the tumor one could readily distinguish the nerve tissue of the lower end of the spinal cord, which was cut across. This consisted mainly of neuroglia cells. Without any line of demarcation, the nerve tissues fused with the main tumor, which showed huge cystic spaces filled with colloid material. These spaces varied greatly in size and were lined with flattened endothelial cells. Between these large cystic areas were numerous pink staining cells with oval nuclei, all tending to form small

spaces, which in some places could be seen to fuse into larger ones. There was evidence of recent hemorrhage, although the markings of old hemorrhage were seen in places. Curiously enough, in one portion of the tumor one saw psammoma bodies. Phosphotungstic acid and Perdrau's stain brought out the vascular nature of this tumor. The diagnosis was hemangio-endothelioma.

Comment.—In view of the patient's recovery of bladder function, and in view of the histology of the filum as described by Streeter, it seems probable that what was cut across and taken to be spinal cord really was that portion of the filum terminale which contains nerve tissue.

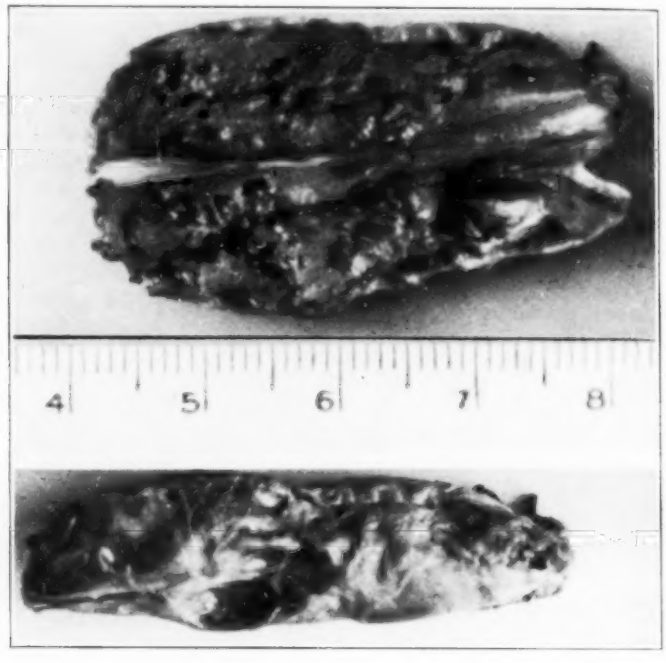

Fig. 7.—The cystic areas in this specimen are well seen.

Case 2.—*History.*—J. H. R., a fireman, aged 38, came to Barnes Hospital on the recommendation of Dr. S. I. Schwab because of progressive atrophy and weakness of the lower portion of both legs. The family and marital histories were irrelevant. At the age of 20 he had a gonorrheal infection and six months before entry an attack of malaria. He dated the onset of the illness from 1918, while he was serving overseas; at that time he suffered an injury to the lower part of the back and shortly afterward noted a cold spot over the sacrum. One year later, he noted soreness and a drawing sensation of the calf of the right leg, and discovered that he was unable to raise himself on his toes. Progressive atrophy and weakness of the lower part of the right leg followed. For a period of eight or nine years he had recurrent attacks of pain in the left lumbar region, lasting from one to three days and

accompanied by moderate chills and fever. Pain, soreness and atrophy of the calf of the left leg started about three years before entry, and progressed more rapidly than in the right. He soon found that he tired easily and had difficulty in balancing himself. On various occasions both feet became cold and cramped.

For four months before admission, he had attacks of dysuria and frequency of urination. During this period he had erections but no ejaculations.

Examination.—The patient's general condition was robust, but he had an evident atrophy of the buttocks and thigh muscles, which showed almost constant fibrillary twitchings and was more marked on the right side. There was weak-

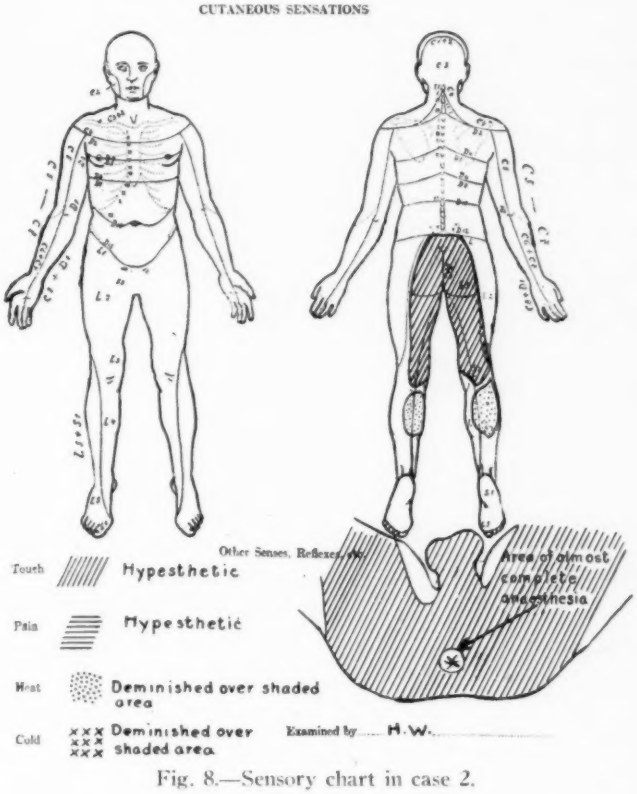

Fig. 8.—Sensory chart in case 2.

ness of both legs. The ankle jerks were absent, and a sensory defect corresponding to the fourth and fifth sacral skin segments could be demonstrated, as well as hypesthesia to pin prick from the posterior surface of the knee downward to the lower portion of the gastrocnemius muscle. The posterior surface of the scrotum was anesthetic (fig. 8). Subsequent tests also showed involvement of the third lumbar, and possibly of the first sacral, skin segments; there was an Oppenheim sign on the left and a normal ankle jerk on the right side.

One of us (D. K. R.) reported as follows: The patient was unable to void following an injection of mercurochrome six months ago. Since that time he has had considerable urgency, with incontinence resulting at times, frequency,

day and night, and dysuria. The stream starts well; the force is fair, but the patient is unable to contract the muscles to eject forcibly the last few drops. He has been able to correct this weakness by pressing on the perineum with his hand. He stated: "It seems to me that there is some loss in the power of the rectal muscles. I am unable to lift up the rectum for the terminal squirts of urine." The residual urine is 250 cc., and there is evidence of a marked cystitis, infected with *Bacillus coli*. The rectal sphincter is relaxed; the rectal mucous membrane is markedly redundant; the prostate is normal in size. Excretion of phenolsulphonphthalein in two hours was 45 per cent. The Wassermann reaction was negative.

Cystoscopic Examination.—The cystoscope was held at an angle of 90 degrees to the body by continuous spasm of the muscles holding the prostate in this plane. There was a marked cystitis, but the bladder otherwise was normal.

Cystometric Examination.—The temperature sense was normal. Capacity in the first and second curves could be considered normal; however, with an associated low pressure, the sensations were slightly exaggerated. In the first cystometrogram, the temperature sense was normal; the "first desire to void" as indicated by the asterisk was normal (150 cc.), but was followed rather suddenly by a sensation of fulness (*F*) at 210 cc. with low pressure, of pain (*P*) at 310 cc. with a mounting pressure and of severe pain (*S.P.*) at 410 cc. This severe pain was carried to the point of distinct discomfort of overdistention. The effect of the first cystometrogram on the second was evidenced by the increased myogenic tone, the resultant higher pressure and slightly more irritable bladder, which pointed to normal autonomic and a fair degree of muscle compensation. Voluntary pressure, taken at the end of the second curve, was 72 mm. of mercury (fig. 9).

The diagnosis was peripheral (nerve) sacral lesion external to the cord.

Spinal Fluid.—On Feb. 11, 1930, Dr. Sachs performed a Queckenstedt test, which showed no evidence of block. Spinal fluid removed at that time was clear and showed: Pandy test, three plus; lymphocytes, 2; red cells, 8, 2 of which were crenated; colloidal gold curve, 0000221000.

Roentgen Examination.—The lateral and anteroposterior views of the lumbar spine and sacro-iliac region showed no evidence of any changes in the bone.

Operation.—On February 17, a laminectomy, under ether anesthesia, was performed by one of us (E. S.), with removal of the spinous processes and laminae of the first, second and part of the third lumbar vertebrae. The extradural fat about the region of the first lumbar vertebra was practically gone. As soon as the dura was opened, the tumor came into view; it lay anterior to the roots and below the conus. The lower pole of the tumor could be raised up; running caudally was a narrow band, evidently the filum terminale (fig. 10). This was coagulated with the Bovie knife and cut. It was then possible to lift the tumor out from among the roots, where it was lying free. By a little traction the upper pole of the tumor was exposed, and a similar thin band could be traced to the conus. This was clipped and cut. Closure was made in the usual manner by layer suture.

Postoperative Course.—On the third day following the operation, the patient voided spontaneously, but because of some residual urine it was thought best to keep up bladder irrigations and instillations of mild silver protein through an in-lying catheter. Skin sutures were removed on the following day; the wound healed per primum. The catheter was removed two weeks after the operation. Two days before the patient's discharge from the hospital, sensation over the

involved area had returned to normal and power was gradually returning to the calf muscles. No pathologic signs could be elicited in the toes. Eighteen days after the operation, a second cystometrogram was made by one of us (D. K. R.), the results of which, compared with the first cystometrogram, were a better bladder tone, a higher sense of irritability and a fair degree of muscle compensation, all indicative of normal autonomic control. The patient was discharged on March 7.

In the patient's most recent note (April 23), he stated: "Though my legs are far from being restored to their normal condition, they really seem to be a little stronger. The soreness has left my back almost entirely, and when I cough

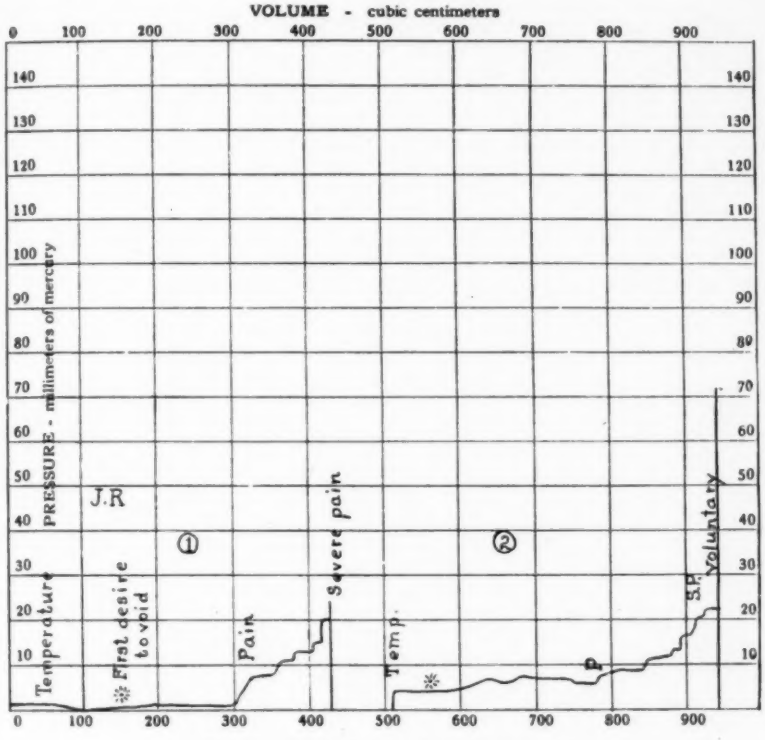

Fig. 9.—Cystometric chart in case 2.

or sneeze, my legs do not feel as if they had received an 'electric shock,' as was the case before the operation. I can walk two or three times as far. I feel as if I could handle my job all right and hope to be called before long."

Pathology.—Grossly, the tumor was about the size of a hazelnut, measuring 3.5 by 2 by 1 cm., and weighing 2 Gm. (fig. 11). Its capsule was white and shiny; to it was attached a portion of the filum terminale. The tumor was soft and spongy; on cut section it appeared gelatinous and gray and several hemorrhagic areas could be seen.

Microscopic Description.—Sections showed an encapsulated tumor containing many blood spaces which were lined with a single layer of cells resting on a delicate fibrous wall. Most of the spaces contained a pinkish colloid material,

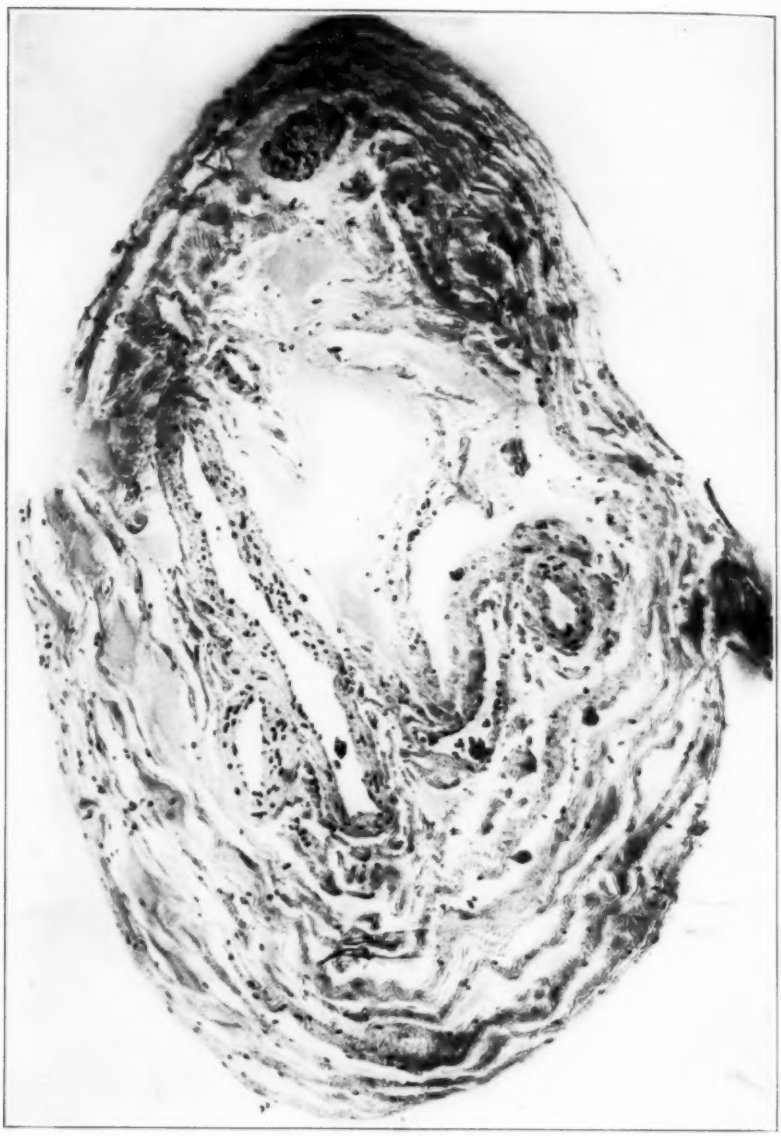

Fig. 10.—Cross-section of the filum terminale distal to the tumor. This is the filum terminale externum.

but there were also areas filled with red cells, besides those indicating recent hemorrhage. The cells lining the spaces were irregular in shape, with frayed edges and tails, and contained a round or oval dark-staining nucleus. Some of the cells appeared to be vacuolated. The colloid material was fairly homogeneous and was seen to lie free in many of the spaces. No mitotic figures were seen. With Mallory's connective tissue stain the vascular walls and the thin endothelial spaces were evident (fig. 12). Perdrau's method brought out these vessel spaces more strikingly.

The diagnosis was hemangio-endothelioma.

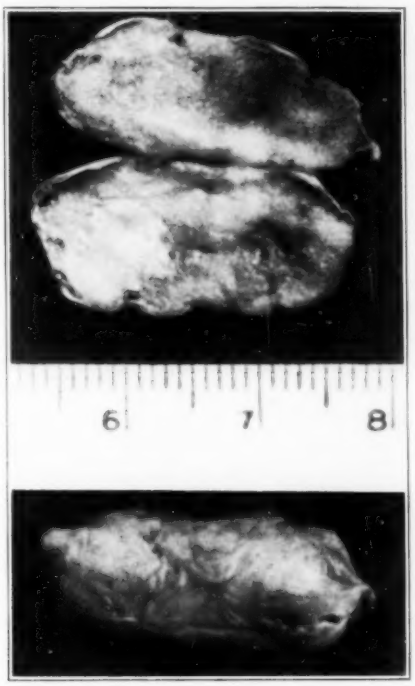

Fig. 11.—The tumor in case 2. Note the small bit of filum terminale attached to the lower end of the tumor.

Comment.—In this case there were some slight root symptoms prior to the bladder symptoms, but they were not prominent. In this case also, just as in case 1 and in Lachman's case, the outstanding symptom was inability to void urine.

COMMENT ON THE CYSTOMETRIC OBSERVATIONS (BY D. K. R.)

In the interpretation of the neurologic factors of a cystometrogram, I consider the capacity of the bladder a balance between the nerves of active dilatation of the bladder wall (sympathetic) and those of active contraction (parasympathetic) and, in addition, the nervus pudendus

which controls the voluntary external sphincter. The mechanics consist chiefly in the ability of the patient to depress the posterior urethra and with it the internal sphincter, this act being accomplished by the anterior portion of the levator ani muscle. The sensation of overdistention is transmitted through the sympathetic, and the "first desire to void"

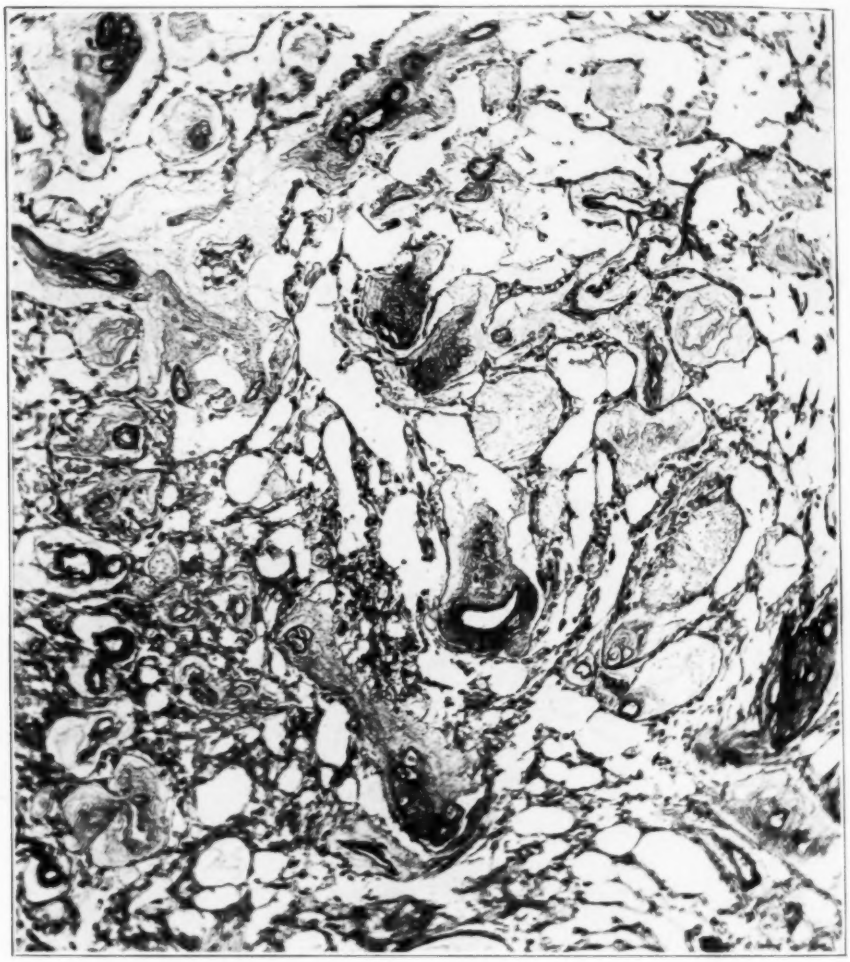

Fig. 12.—A typical hemangio-endothelioma seen in case 2.

through the nervus pudendus. The latter point is of great value and establishes the filling capacity with the associated intracystic pressure at which the patient first appreciates that there is a sufficient amount of fluid in the bladder to enable him to void. It is definitely not the point at which there is a marked desire to empty the bladder. The nervi

erigentes (parasympathetic) are sensory in that the sensation of early pressure in the bladder is perceived through them, and this starts the first reflex of urination; this first reflex is an involuntary slight contraction which stimulates the nervus pudendus in the upper posterior urethra and thus notifies the patient of a "first desire to void." On experiencing this sensation the patient empties the bladder. The first act of this process is a voluntary one, and consists in a depression of the posterior urethra. That is followed by an opening of the internal sphincter and a contraction of the bladder wall. With the first flow of urine the pudic nerve is further stimulated throughout the entire urethra, the motor portion of the reflex continues through the erigentes and further contracts the wall, which thickens, and consequently strengthens, as its contents lessen, with the result that the greatest force comes at the height of the stream. The bladder now empties down to the internal orifice, and the urethra is emptied in terminal squirts by contraction of the prostatic muscle and the bulbocavernosum, by changes in the position of the posterior urethra in an up-and-down direction, and also by contractions of the anterior portion of the levator ani (nervus pudendus). Elevation of the urethra is associated with elevation of the rectum, which helps to empty the posterior urethra. Initiation of voiding or depression of the urethra is associated with a slight depression of the rectum. Voluntary retention is associated with a voluntary elevation of the perineum and rectum, thereby elevating the prostatic urethra and increasing the resistant kink at the internal sphincter and urogenital trigone. In addition, one must consider the factor of the myogenic bladder wall separately, because it thickens when offered increased work by an obstruction which, if continued and increased, will eventually cause a breaking down of the strength of the muscle wall to an ultimate state of complete decompensation. I consider a muscle wall which carries a residual an anatomically compensated wall, in that it is thicker than normal yet unable to complete its duty of emptying the bladder, and therefore is functionally or physiologically decompensated. The effects of infection in increasing irritability must also be considered.

Voluntary pressure, produced by the abdominal wall and diaphragm, has a high innervation and is also a factor in the physiology of urination.

Using these points for the diagnosis of a lesion in the lower part of the spinal cord in case 1, we found that the pressure was low and the sensation of "first desire to void" was set over to 450 cc. A lesion in the cord would produce such damage more directly than a peripheral lesion, although the low pressure is partly due to myogenic decompensation secondary to the large residual. The chief injury was to the nervi erigentes (motor) and to the pudic nerve (sensory for the first

desire to void), both of which are of sacral origin. The prostatic urethra was flaccid, which further indicated damage to the pudic nerve. Severe pain occurred at 800 cc., at a very low pressure, which suggested normal sympathetics. The lesion was found to involve the conus medullaris and filum terminale.

Comparing case 2 with case 1, one finds the capacity smaller in case 2, sensations slightly heightened though well distributed over the first pressure curve, and pressure slightly increased in the second curve, which suggests myogenic decompensation with normal autonomic control. Since voluntary pressure should be sufficient to empty the bladder and as cystoscopic examination shows no obstruction and yet there is a residual of 250 cc., I conclude that there must be a neurogenic obstruction. One notes further that the cystoscope is held almost at right angles to the body, showing irritation of the nerve which supplies the muscles that control this motion (levator ani). In a destructive lesion, sensory and motor fibers are usually equally damaged, but as the sensory fibers are intact I conclude that the obstruction is due to an irritative peripheral lesion which maintains the external sphincter in spasm and causes an obstruction. The myogenic failure is secondary. Other lesions (metastatic carcinomas and tuberculomas on the nerve roots) producing such a cystometrogram have always been found to be peripheral.

SUMMARY AND CONCLUSIONS

Two cases of tumor of the filum terminale are reported. The outstanding symptom was loss of the function of the bladder. This would seem to be the outstanding symptom of such lesions. If patients are seen late in the course of the disease, other symptoms of the cord and root may be present.

The study of these cases would seem to confirm the theory of the mechanism of the bladder as described by one of us (D. K. R.). According to this theory, active dilatation of the bladder wall is carried on through the sympathetic fibers, active contraction is carried on by the parasympathetic fibers, and the voluntary control of the external sphincter is taken care of by the nervus pudendus.

These cases emphasize the following points:

1. Tumors of the filum terminale, though rare, do occur and have a characteristic symptomatology which distinguishes them from other tumors of the cauda equina.

2. Cystometric examination should be a regular method of examination in all suspected spinal tumors.

3. All tumors of the filum terminale thus far reported are benign and readily removable.

4. The physiology of micturition is not fully understood, and studies such as these are much more likely to clear up this problem than are experiments on animals.

ABSTRACT OF DISCUSSION

DR. STEPHEN W. RANSON, Chicago: The paper by Dr. Sachs and his co-workers is of obvious diagnostic and surgical importance. Less obviously, but just as truly, it possesses a good deal of physiologic interest.

The cystometer which has been developed and used so effectively at Washington University has made it possible to study with detail and precision the physiology of micturition in man. It is well known that three pairs of nerves are concerned in the innervation of the urinary bladder and urethra: the hypogastric nerves belonging to the sympathetic, the visceral branches of the second and third sacral belonging to the parasympathetic and the pudendal belonging to the somatic division of the peripheral nervous system.

The hypogastric nerves favor the retention of urine by stimulating the internal sphincter and inhibiting the rest of the bladder musculature. These nerves have their connection with the spinal cord through the upper lumbar and thoracic nerves, too high to be involved in lesions of the cauda equina.

The visceral branches of the second and third sacral nerves bring about the expulsion of urine by stimulating the musculature of the bladder and inhibiting the internal sphincter. The fibers of this system are involved whenever lesions of the cauda include the second and third sacral nerves, and paralysis of these nerves will lead to retention of urine and dilatation of the bladder, but only if the fibers are involved on both sides. Dr. Sachs' first case was of this type, and the lesion obviously included these nerves on both sides.

The visceral branches of the second and third sacral nerves contain sensory as well as motor fibers. It is of interest that the same symptoms are produced if only the sensory fibers are destroyed. I have found that in cats after bilateral section of the dorsal roots of the sacral nerves the bladder becomes greatly distended. This never occurs after unilateral root section. Similar observations have been made by Barrington.

The pudendal nerves supply the deep transverse perineal and the sphincter urethrae membranae. These muscles are not supposed to be of much value as sphincters, but Barrington has shown that a section of both pudendal nerves in the cat always causes more or less incontinence, and it would not be surprising, therefore, if irritation of the corresponding fibers in the nerve roots should bring about retention of urine back of the spastic external sphincter in spite of normal sympathetic and parasympathetic innervation of the bladder. That, I understand, is the interpretation which was placed on the cystometric observations in the second case. That is to me a new conception. Is this type of neurogenic urethral obstruction common?

DR. WILLIAM G. SPILLER, Philadelphia: The cases reported by Gowers and Lachman were discussed in my paper in 1899 and, with my case, made three cases of tumor of the filum terminale on record. It is extraordinary that during the thirty-one years that have elapsed until the present time no other cases have been reported. It must be that such tumors occurred and were overlooked. Possibly they were considered as tumors of the cauda equina. With the two cases reported by Drs. Sachs, Rose and Kaplan, five cases are now on record. The first case seems to have been described by Gowers in 1876; the tumor was a lipoma, as in my own case, and as in my own case also, the specimen

was from a tabetic patient. Lachman's tumor reported in 1882 was a glioma, and the two tumors described by Sachs, Rose and Kaplan were hemangioendotheliomas. The two cases of lipoma were without symptoms attributable to the tumors, but in the other three cases disturbance of the function of the bladder was among the first signs in two cases and was a prominent sign in the third, in which disassociation of the sexual functions was also observed. This case was more advanced in symptomatology when it came under observation. In two cases, exaggeration of the patellar reflexes occurred which came from a lesion below the reflex arcs. I have known this to occur in other instances. It seems as though a lesion below the reflex arcs may stimulate these arcs and cause hyperreflexia.

The prognosis of tumor of the filum terminale as shown by these cases should be good if operation is performed early. This is different from the prognosis of tumor of the cauda equina, as tumor here may be infiltrating about the nerve roots and inoperable.

On account of the early and, possibly, isolated, paralysis of the bladder which may occur in these cases, there is danger that the lesion may be attributed primarily to the bladder; this was true of Lachman's case, in which the diagnosis of carcinoma of the bladder was made.

I have seen one other chance discovery of a growth of the filum terminale, namely, a small calcareous mass, the size of a small pea.

Dr. Rose's method seems to me so exceedingly important that I discussed it with Dr. Alexander Randall, professor of urology at the University of Pennsylvania. He has given me authority to repeat some remarks that he sent me.

"I think all urologists have been watching Dr. Rose's work with a great deal of interest. He has developed a manometric method of studying bladder tension which can be applied both to varying degrees of bladder distension and again under varying stimuli both voluntary and involuntary.

"It is his belief that he can differentiate lesions of the three nerve trunks which appear to take active part in the urinary act. If this proves to be true and is of easy application, it will certainly be a most valuable contribution to diagnosis. Today it seems its greatest drawback lies in the clinical interpretation of sensory reactions, which naturally hold a fallacy in clinical diagnosis. Nevertheless, back of the whole method is an earnest effort and probably a very definite contribution toward the appreciation of very difficult, poorly handled and rarely diagnosed conditions."

Dr. Charles A. Elsberg, New York: The importance of the two cases reported by Dr. Sachs and his collaborators cannot be overestimated, and the significance that he and those who have spoken have laid on the fact of early disturbances of the bladder in tumors of the filum cannot be emphasized too strongly. Of course, early disturbances of the bladder signify that a tumor in the region of the cauda equina is located in such a manner that the sacral nerves are first involved; in other words, it is a mesially placed tumor.

I believe there were probably many cases of tumor of the filum terminale that were not recognized. I believe that I have missed some myself, in cases in which I have not more carefully looked for the relations between the tumor and the filum terminale.

I have had one of my associates look up the subject of early disturbances of the bladder in the last thirty cases of tumor in the region of the cauda equina in our clinic. In one case the tumor was large and mesially placed; in another the tumor lay entirely on one side, having crowded the roots of the cauda equina over to the other side. In the latter instance the tumor was attached to the dura

on one side, which had probably been its site of origin; it could not have been primarily a mesially placed tumor. In both of these patients, symptoms in the bladder were the first to appear. In patients with tumor in this region, which begin with symptoms in the bladder, one should always think of tumor of the filum terminale—as Dr. Sachs has mentioned in reporting these cases, and Dr. Spiller has emphasized—still one should not be too certain that it is a tumor derived from the filum. Such tumors are probably rare.

The paper just presented is instructive and stimulating from another point of view. We are accustomed to accept the statement that in the adult the spinal cord ends at the level of about the first lumbar vertebra. The actual fact is that while grossly the spinal cord does end there, in reality the spinal cord extends downward to the lower end of the arachnoid sac, to what Dr. Sachs described, following Dr. Streeter, as the filum terminale internum. All along this portion of the filum, as Dr. Sachs mentioned in his paper, one can find not only a canal extending down a considerable distance toward the lower end of the arachnoid sac, but nerve tissue, glial tissues and membrane. We ought to become accustomed to the point of view that while grossly the spinal cord ends in the adult at about the level of the first lumbar vertebra, actually it extends down almost to the lower end of the arachnoid sac.

Dr. Alfred W. Adson, Rochester, Minn.: There is no doubt that many tumors in the filum terminale have been operated on but not reported. In going over the records, it has been found that fifteen tumors in the filum terminale have been explored at the Mayo Clinic. Kernohan has stated these to be five ependymomas; two astroblastomas; two astrocytomas, (*a*) one protoplasmic astrocytoma, (*b*) one fibrous astrocytoma; two spongioblastomata multiformia; one oligodendroblastoma; three hemangio-endotheliomas.

In addition, three chordomas of the sacrum have also been explored. The beginning symptoms of this group were pains, usually radiating along one or both sciatic nerves. The next common symptom was disturbance of the function of the bladder, and then motor and sensory disturbance. Early spinal punctures and Queckenstedt studies demonstrated incomplete blocks, but when the diagnosis was in doubt, studies made with iodized poppy seed oil 40 per cent proved of great value. The chordomas all produced erosion of the sacrum, which was recognized by roentgenologic examination. Urinary retention and saddle anesthesia were the early accompanying observations.

Dr. Percival Bailey, Chicago: Are there any peculiarities in the blood supply of the filum terminale? It is easily understood how a glioma might arise from the filum terminale, but not a hemangioma.

I should like to know also whether it is probable that the so-called giant tumors of the cauda might arise from this structure.

Dr. Ernest Sachs: I was not aware of Dr. Adson's case, and so I do not know whether it was reported as a tumor in the filum terminale or not.

The bladder in our cases was trabeculated, but this occurs in a number of conditions and is merely an indication of a prolonged pressure in the wall of the bladder.

Regarding Dr. Bailey's question: I do not know anything unusual about the circulation of the filum except that there are amazingly large blood vessels in it.

A NEW DEMONSTRATION OF HORTEGA CELLS*

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AND
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Since del Rio Hortega published his silver carbonate method for the demonstration of the neuroglia cell which now carries his name, there have been complaints from research workers and technicians that the method "simply does not always work." I have been interested in silver methods for histologic purposes since the same attitude has arisen as regards their application to *Spirochaeta pallida*. Experimentation with spirochetes resulted in the presentation of my "colloidal" method for demonstrating them in single microscopic sections.[1] Following this work, I applied certain modifications of the procedure to the Hortega cells and, as is common experience with silver methods, there was obtained, through certain mysterious incidences, the best demonstration of rod cells in dementia paralytica that I had seen. The inconstant results, however, were as discouraging as with Hortega's original method, and I accordingly dropped the investigation in spite of the occasional exceptionally excellent pictures resulting with the colloidal medium used in my spirochete method.

When Kubie and Davidson[2] and later Kubie[3] published their excellent work and took silver staining solutions out of the kitchen technic of existing methods and put them into the scientific chemical laboratory, their suggestions were substituted by me for all the commonly used silver methods of neuropathology, with the most gratifying results.

First of all, I recommend the use of Kubie's solutions for the Bielschowsky, Hortega and Achúcarro methods, including the Klarfeld modification of the latter and the Perdrau modification of the Biel-

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* From St. Elizabeth's Hospital, Blackburn Laboratory, Washington, D. C., and in part during tenure of John Simon Guggenheim Memorial Foundation Fellowship, 1928-1929.

1. Dieterle, R. R.: Method for Demonstration of Spirochaeta Pallida in Single Microscopic Sections, Arch. Neurol. & Psychiat. **18**:73 (July) 1927.

2. Kubie, L. S., and Davidson, D.: The Ammoniacal Silver Solutions Used in Neuropathology, Arch. Neurol. & Psychiat. **19**:888 (May) 1928.

3. Kubie, L. S.: Staining of Tissues of the Central Nervous System with Silver, Arch. Neurol. & Psychiat. **22**:135 (July) 1929.

schowsky principle for connective tissue staining. Technicians will do well to discard the old guess work ways of the cook and chef in making ammoniacal silver solutions. Kubie's formulas are so easily made and can be kept in stock so nicely that half the work and doubt of the technician is thereby removed. Furthermore, the application of this work in a theoretical way points to a future of developing silver technic on a scientific basis. All of the usual methods are made easier, simpler and more constant than by the original formulas of their authors. There is no silver method existing that cannot be modified toward improvement, and every technician should familiarize himself with the chemistry of the solutions described by Kubie. Through them the researcher will become more productive.

I shall present a method for Hortega cells which, though not perfect, gives such excellent results that details about the morphology of the rod cell are brought to the foreground in such a way that one is given new ideas and insight into their structure. Its technic shows how a practical application of theory works satisfactorily. The method is as follows:

1. Fix small pieces of brain tissue, from 2 to 3 mm. thick, in ammonium nitrate, 10 Gm.; formaldehyde (Merck's Blue Label), 70 cc.; distilled water, 430 cc.

There is no definitely limited optimum time for the best results. The consistency of the tissues, after one day's fixation for example, varies according to the individual case. The senile brain or the brain affected by dementia paralytica varies from the normal and from the schizophrenic brain regarding this factor alone. Whether other intrinsic factors, as of disease, alter the extrinsic one of fixatives cannot be doubted in this particular field of colloidal physical chemistry. The colloidal medium varies, furthermore, with reference to the thickness with which sections are cut on the freezing microtome. This apparatus should be one which cuts accurately in a serial way, and the suggested thickness for sections fixed in the given solution is 20 microns. The physical qualities of the sections naturally vary with the hardness of the fixation, and this becomes, after fixation, the second variable factor in the conditions to be overcome. Practically, then, trials may be begun after twenty-four hours in the nitrate-formaldehyde solution. On the average, good results are obtained in from about three to eight days. Cut the sections into fresh fixative as for Hortega's method.

2. Put a dozen sections into 50 cc. of distilled water plus 5 drops of ammonium hydroxide and leave for ten minutes.

3. Handling and rinsing the sections singly in 100 cc. of distilled water, transfer them to Kubie's ammoniacal silver carbonate solution.[4]

Kubie's solutions are prepared as follows:

Ammonia: Add 8.09 cc. of 28 per cent ammonium hydroxide with a specific gravity 0.90 to a 100 cc. volumetric flask and dilute to 100 cc.

Sodium Carbonate: Dissolve 3.2 Gm. of the anhydrous salt in distilled water; make up to a final volume of 100 cc. in a volumetric flask and keep in a paraffined flask.

4. Kubie and Davidson (footnote 2, p. 899).

Silver Nitrate: Dissolve 10.2 Gm. in distilled water; make up to a final volume of 100 cc. and transfer to a brown paraffined bottle.

Put 10 cc. of the silver nitrate solution with a graduated pipet into a 100 cc. volumetric flask; add the diluted ammonia solution from a buret, rapidly at first, shaking vigorously between additions toward the end-point. Add 10 cc. of the

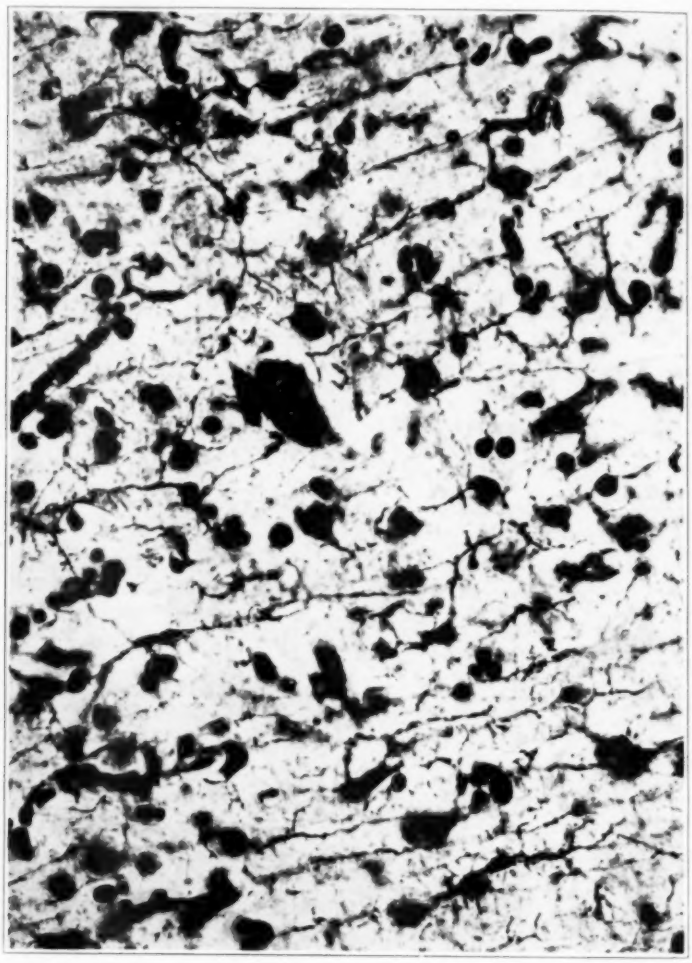

Fig. 1.—Hortega rod cells, radial arrangement of cell axis, extremely fine collateral dendrites producing syncytium from motor cortex in a case of dementia paralytica.

sodium carbonate solution and make up to a final volume of 100 cc. This is theoretically equal to a 1 per cent solution. Next place the dozen sections into 50 cc. of this solution contained now in a closed ground-glass Stender dish at room temperature.

In spite of the standardization of the silver solution by Kubie, one now comes into conflict with the prevailing room temperature. I have tried to control this

by placing the sections at 37 C. and at 55 C. The intensity of the brown staining naturally advances with time and exposure to heat. The silver solution tends to reduce metallically at 55 C., even after from ten to fifteen minutes, perhaps owing to escape of ammonia. Though I have obtained good results on different occasions

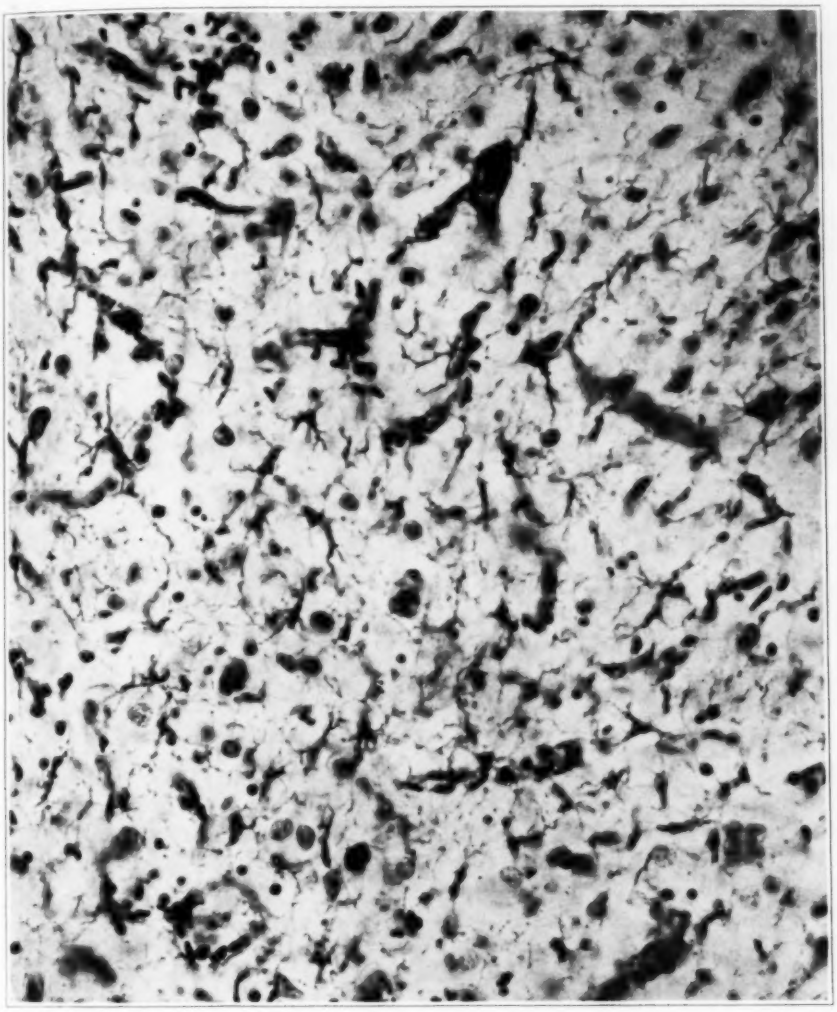

Fig. 2.—Hortega cell focal gliosis, profuse network of dendrites, excellent differentiation of fibroglia nuclei from Ammon's horn in a case of dementia paralytica with extreme convulsive seizures.

with oven temperature, I usually leave the sections at room temperature in a closed box for darkness, and begin trials after fifteen minutes. Results usually start at this time, and if control use of the microscope is relied on, the individual case may be found to appear up to one hour. After an hour this experiment

may be continued, but I consider that negative results after one hour and a half indicate that some other uncontrollable variant is present.

4. Reduction may be performed in the following method, and my advice for this and for all ammoniacal silver methods is to approach the problem of reduction

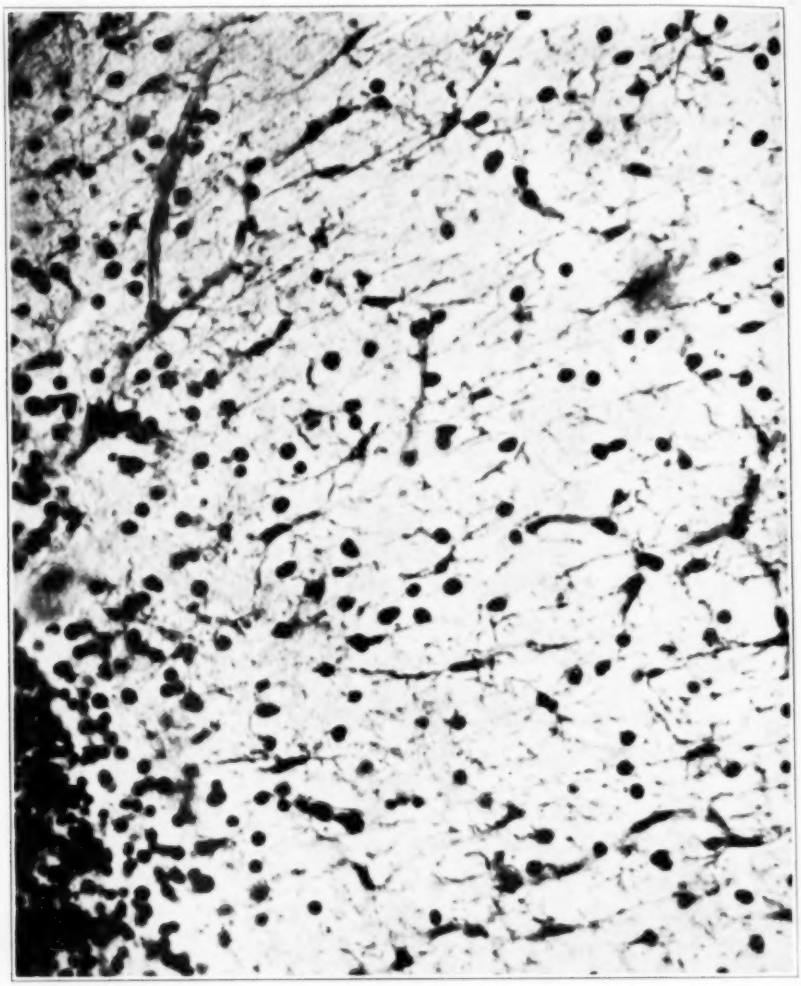

Fig. 3.—Hortega rod cells in molecular layer of cerebellum. Note the differentiation of the nuclear shade from the dendritic processes. Same case as in figure 1.

with an attitude of "research with the method at the time." Following Kubie's suggestions in his second paper,[5] the technician will learn to be on top of the trouble. Thus, if one takes a section exposed to the silver solution for fifteen minutes and places it in 50 cc. of distilled water to which is added 1 drop of 37 per cent neutral formaldehyde (Merck), the silver slowly reduces with a light

5. Kubie (footnote 3, p. 137).

yellow color. This I call the "colloidal color" and attribute it to an actual colloidal union of the silver with the tissue. It is really a stain in the dye sense. With the use of 1 drop of formaldehyde I go farther than Kubie, whose extreme

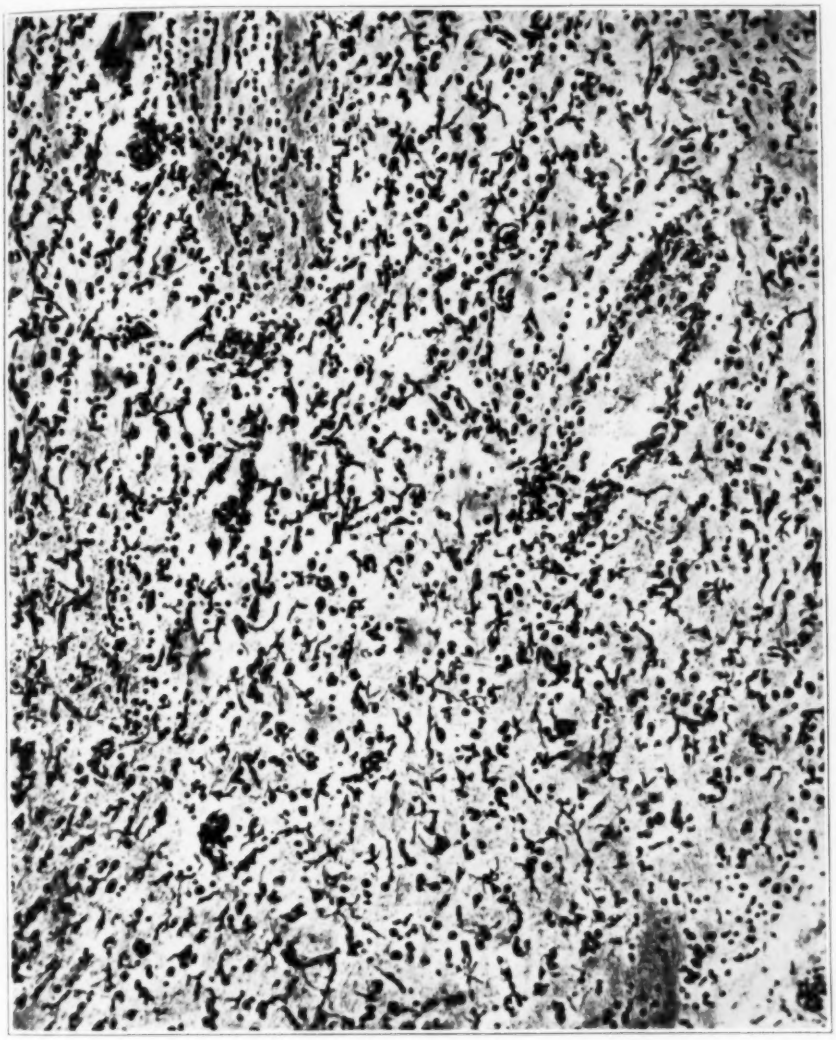

Fig. 4.—Diffuse Hortega microgliosis from putamen in a case of juvenile dementia paralytica, with cerebral atrophy and internal hydrocephalus. Very low magnification.

dilution was 0.25 cc. of formaldehyde. This test serves as an indication of the intensity of the staining, and with stepped dilutions to 1 cc. of formaldehyde in 100 cc. of distilled water should show at the end of fifteen minutes whether Hortega cells are present or not. It is really the method of Hortega. If these

cells are present, one can continue. This continuation is driven at to improve the pictures already obtained, and its purpose is to stain the Hortega cells colloidally and to get away from the superficial precipitates which the original Hortega method gives.

In the course of this work and experiments with spirochete-containing tissues, it has been found that sections exposed to 1 per cent silver nitrate will not stain with a yellow tinge after an attempt at reduction with formaldehyde. Sections

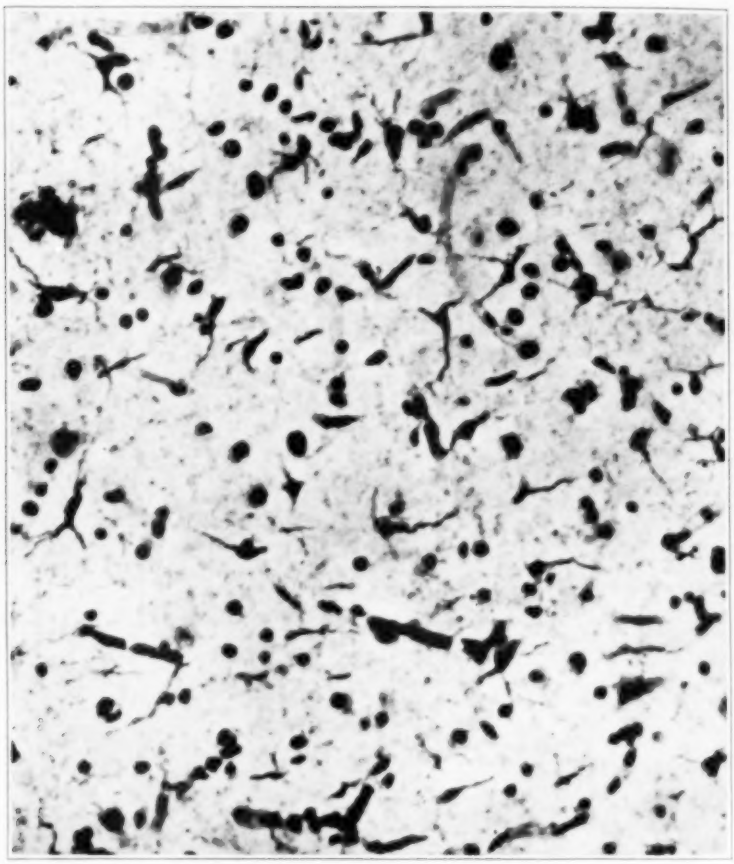

Fig. 5.—Hortega cells from a case of acute hyperactive amentia with death six days after admission. A Nissl stain showed acute nerve cell changes with some typical axonal reaction forms.

treated with any ammoniacal silver solution, however, will stain this way, and the colloidal effect can be produced with dilute formaldehyde. From 1 to 10 per cent of formaldehyde, however, produces a surface impregnation, and the resulting sections are grayish, as with Hortega's and Bielschowsky's methods. On the other hand, when pyrogallol or hydroquinone are used either alone or with formaldehyde to reduce silver nitrate preparations, a yellow color is produced such as occurs with my spirochete method, the Achúcarro and the Perdrau

methods for connective tissue. Furthermore, in a medium such as the dilute mastic solution, used in the developer for my spirochete method, formaldehyde alone in such solution will not bring about the desired result, and hydroquinone in a strength of 1 to 1.5 per cent causes a diffuse and not selective staining and a granular precipitation of silver. The latter solution reduces it too suddenly and intensely. After much experimentation with dilutions, the following modification of my spirochete developer has produced results:

	Cc.
Formaldehyde (Merck)	10
Acetone	10
Pyridine	10
Mastic (10 per cent absolute alcoholic)	10
Distilled water sufficient to make	100

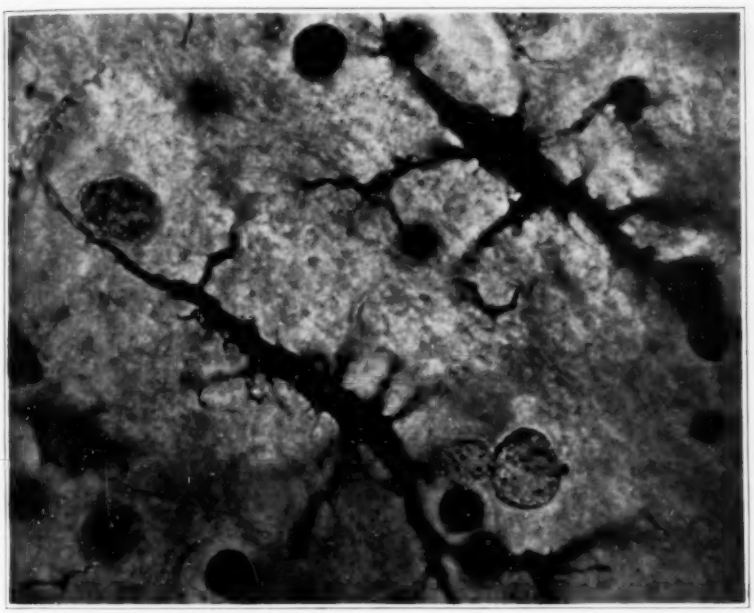

Fig. 6.—Extremely high magnification of two Hortega rod cells, to show differential staining from three fibroglia nuclei lying adjacent to the larger one; black round nuclei, oligodendroglia (?). Oil-immersion objective.

To develop the sections in Kubie's silver carbonate, take 50 cc. of this developer in a Stender dish and add from a pipet 1 cc. of 1 per cent hydroquinone solution. The hydroquinone should be weighed accurately, dissolved and made up to 100 cc. in a volumetric flask. The sections should be put directly into this solution after remaining for from fifteen minutes to one hour and a half at room temperature in Kubie's silver solution. Development occurs almost immediately. The mastic is then dissolved in 96 per cent alcohol; the section is blotted on the microscopic slide, cleared in 10 per cent phenol-xylene and mounted in balsam. The usual after-toning in gold chloride is superfluous.

RESULTS

The best result is a differentiated staining of the Hortega cells. The nucleus ranges from a dark brown to black; the processes show varying shades of brown to deep yellow against a background of golden-rod yellow. The nuclei of all other elements stain, but the method remains selective for the entire structure of the Hortega cell. The procedure is especially excellent for rod cells, not only in cases of dementia paralytica, but also in other organic processes. As has been noted by others, pathologic alterations in the Hortega cells, especially hypertrophy, increases the chances of its demonstration in microscopic preparations. In brains of normal and of schizophrenic persons I have been unable to show these alterations by the means of such a colloidal medium. However, it is possible to demonstrate them with the simple dilute formaldehyde reduction. This method, then, becomes a kind of indicator of the pathologic change in the Hortega element. Other forms than the rod cell variation can be shown. In the tissue in dementia paralytica the iron granules within the rod cell processes are beautifully stained, and the relation of the Hortega cell to capillaries and nerve cells has led me to deduce that the diffuse iron of dementia paralytica comes from the destruction of the nerve cells themselves and from them is transported to the capillaries. This is so interesting to observe in the "dead" specimens thus stained that the process of phagocytosis almost becomes "living" on serial study under an oil-immersion objective. The fine network of rod cell processes, particularly the finer side-arm or rectangular dendrites, is so abundant, as seen in these "colloidal" preparations, that a continuous hypertrophic network seems to have been formed. These finer processes are so delicate that it is only with the same principles as those used for staining spirochetes that they can be shown. Therefore, the method proves conclusively that the dendrite processes of the Hortega cells are actually part of their morphology, and removes the doubt cited by Kubie that "granules and spicules of silver along the course of processes are true protoplasmic structures." [5]

In conclusion, let me again emphasize that the technician read Kubie's articles to learn the theory of all silver impregnation. For this reason I have presented the foregoing outline from the dynamic standpoint, that those who try it may know what they are about, and that failures should not necessarily be attributed to the technic. I do not present this method as final. Such an attempt would be too empiric. What I have given is a broad technical theory, the practical application of which depends on the deftness of the technician in the juggling of variables. I furthermore advise that the first experiments be made on the cortex from a patient with dementia paralytica. In the cere-

bellum, the rod cells are more difficult to stain. In fact, I have had better results on such cerebellar tissue with the use of extremely dilute formaldehyde: 1 drop up to 1 per cent and conversely devised, by the addition of 1 drop up to 1 cc. of the dilute mastic medium without the 10 cc. of formaldehyde, but keeping constant the acetone and pyridine.

It is further recommended that Kubie's silver solution be varied by dilutions of 0.25 and 0.5 per cent, the time factor being considered proportionately; and also that the formaldehyde of the developer be varied from 2.5 up to 10 per cent, since the proper colloidal development of any individual tissue depends on the right proportion of those two substances.

Finally, if this dynamic technic seems too complicated over the usual static recipes, its one virtue lies in its ability to show more of the protoplasmic structure of pathologic Hortega cells than any method yet devised.[6] I have also had some good results with tissue fixed in plain formaldehyde.

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6. Kanzler, R.: Eine Modification der Darstellung der Hortega-Zellen für Formalinmaterial, Ztschr. f. d. ges. Neurol. & Psychiat. **122**:416, 1929.

HYDROCEPHALUS

STUDIES OF THE PATHOLOGY AND PATHOGENESIS, WITH REMARKS ON THE CEREBROSPINAL FLUID *

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CHICAGO

Studies of the pathology of hydrocephalus, with few exceptions,[1] have pertained to the condition of the ventricles and subarachnoid spaces. In general, they have been anatomic rather than histologic. In this article I shall attempt to describe the most important microscopic central nerve changes in this morbid condition and the conclusions that may be warranted from such observations as to some causes of the excessive accumulation of cerebrospinal fluid in the cerebral cavities and the mode of its absorption. Twelve cases of hydrocephalus have been studied, all of the so-called communicating type. In this, Dandy and Blackfan[2] included cases in which the communication between the ventricles remains open, the obstruction taking place in the subarachnoid space. If the ventricular communication is obstructed, by a tumor, for instance, the hydrocephalus is noncommunicating.

METHODS OF STUDY

Blocks from various portions of the brain, including the leptomeninges and the dura, when this was attached, were studied in celloidin, paraffin and frozen sections. Especial attention was paid to the

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* Read at the Fifty-Sixth Annual Meeting of the American Neurological Association, Atlantic City, N. J., June 9, 1930.

* From the Pathologic Laboratories of the Research and Educational Hospitals, University of Illinois and the State Psychopathic Institute.

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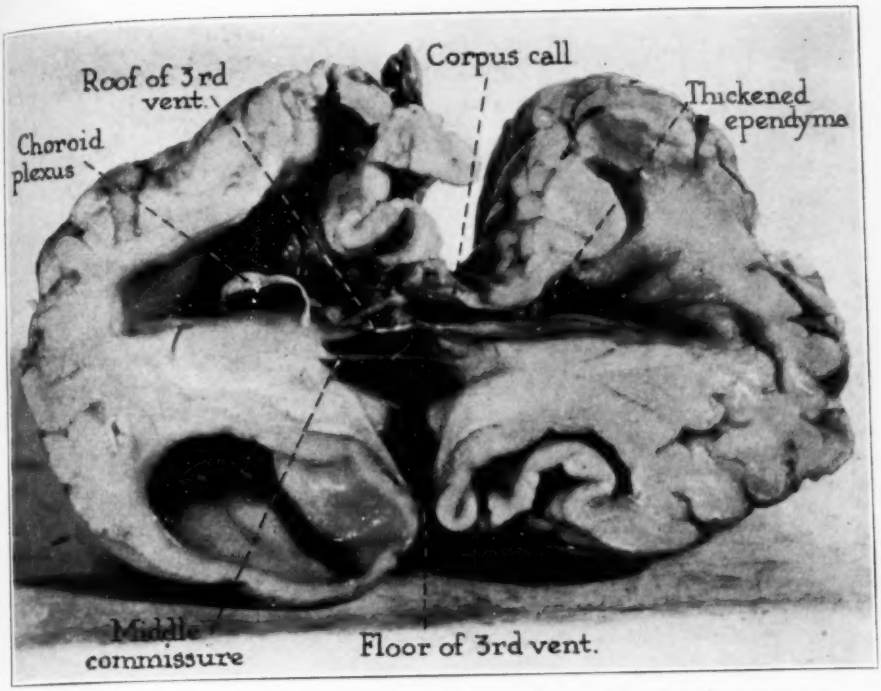

Fig. 1.—The lateral ventricles, like the brain, are collapsed and appear narrowed.

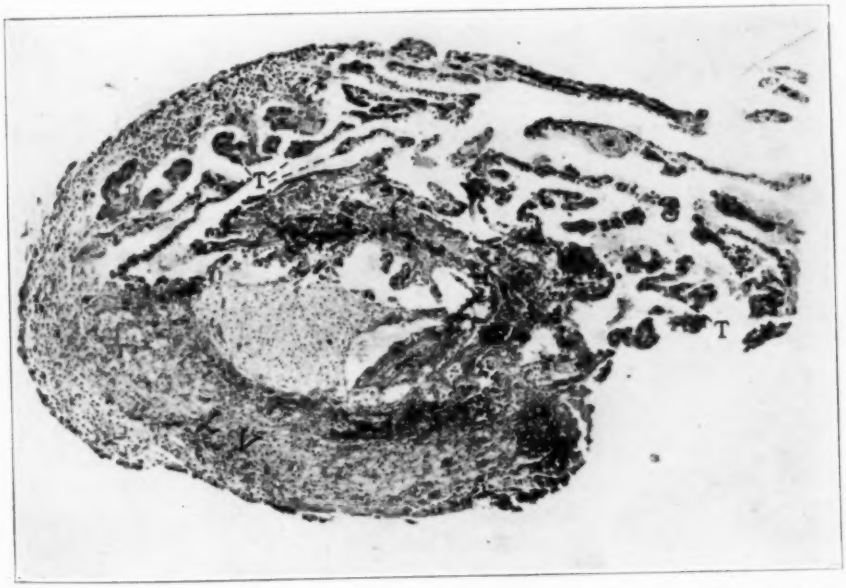

Fig. 2.—The choroid plexus is encapsulated in the wall of the lateral ventricle (*LV*); its tufts (*T T*) are sparse and thin. Other explanations will be found in the text. Van Gieson; × 65.

cerebral subarachnoid and the perineural spaces of the cranial nerves and the choroid plexus. The latter were studied mainly in paraffin sections, and the observations were contrasted with those from non-hydrocephalus cases. To avoid repetition, one case will be described in some detail; concerning the rest only the most important features will be mentioned.

Fig. 3.—Ossified dura mater. Toluidine blue; × 65.

REPORT OF CASES

An infant, born by a normal delivery at the Grant Hospital of Chicago on March 13, 1927, became on the next day cyanotic, developed convulsions and a temperature of 103 F. In about nine days, the infant apparently recovered and was discharged in a good condition. When readmitted on April 26, it showed a marked hydrocephalus. The main clinical signs and symptoms that developed subsequently were spasticity, apathy, nystagmus and malnutrition. The mouth, the shins and the cranial nerves, including the pupils, were normal. The spinal

fluid had a specific gravity of 1.006; the Pandy and Ross Jones tests were strongly positive; the Wassermann and Kahn reactions were 4 plus and the Lange colloidal curve was 666554321000; the sugar content was 22.2 mg. per hundred cubic centimeters. A Wassermann test with the mother's blood gave a negative reaction. The child grew steadily worse and died on June 30, aged 3 months and 17 days.

Necropsy (by Dr. Brown).—Examination revealed a greatly thickened dura over the longitudinal sinus; the pia-arachnoid was apparently normal over the

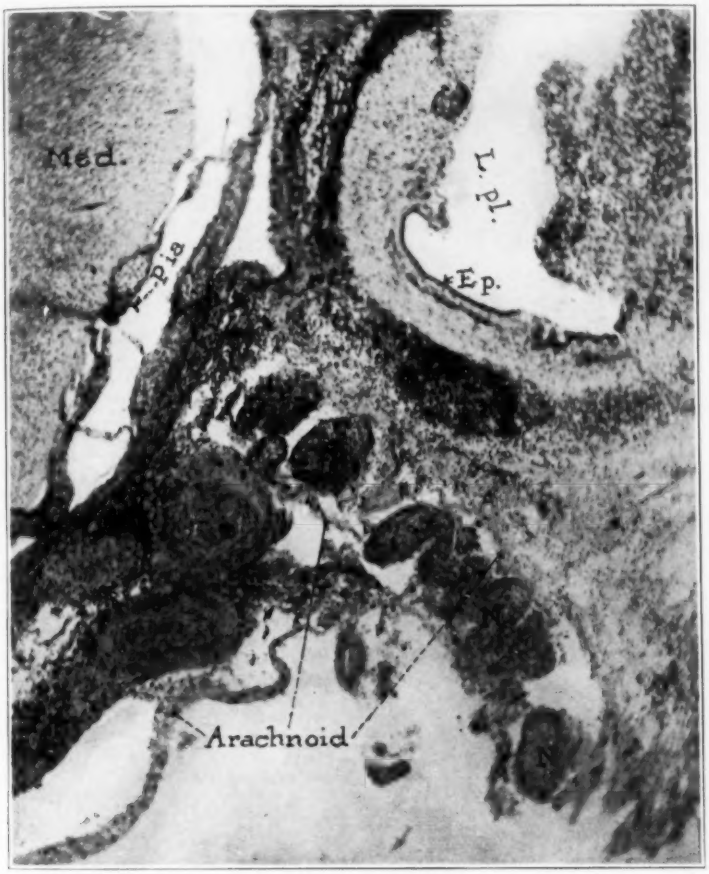

Fig. 4.—The subarachnoid space around the lateral surface of the medulla above the inferior olivary body. Between the infiltrated pia and the arachnoid membrane (below) is the subarachnoid space. *N N* and *N* indicate bundles of the ninth nerve surrounded by the perineural space and the arachnoid; *L. pl.*, the area occupied by the lateral (extraventricular) choroid plexus (pictured in fig. 16); *Ep.*, extraventricular ependyma of the fourth ventricle. Other explanations will be found in the text. Van Gieson; × 57.

convexity of the brain, but was opaque and thickened over its base; the ventricles were dilated (fig. 1) and the floor of the third ventricle bulged; the ependymal lining of the lateral ventricles was thickened, and the foramina of Monro were

greatly enlarged. The choroid plexus was sclerosed and atrophied; in the lateral and fourth ventricles it was adherent to the floor, as if buried in the parenchyma, and reduced to a thin connective tissue strand (fig. 1). In the third ventricle, a few tufts were present over the tela; the sylvian aqueduct was patent, and one of the Luschka's recesses was obstructed by an inflammatory focus.

Microscopic Observations.—The choroid plexus appeared encapsulated; its tufts were thin and tender (fig. 2), and were represented by a single row of epithelial cells; their nuclei stained well, but the cytoplasm in some was granular, disrupted and torn, and exhibited large vacuoles; in some it was dustlike and the centrally located nuclei were pyknotic. The blood vessels of the tufts were in

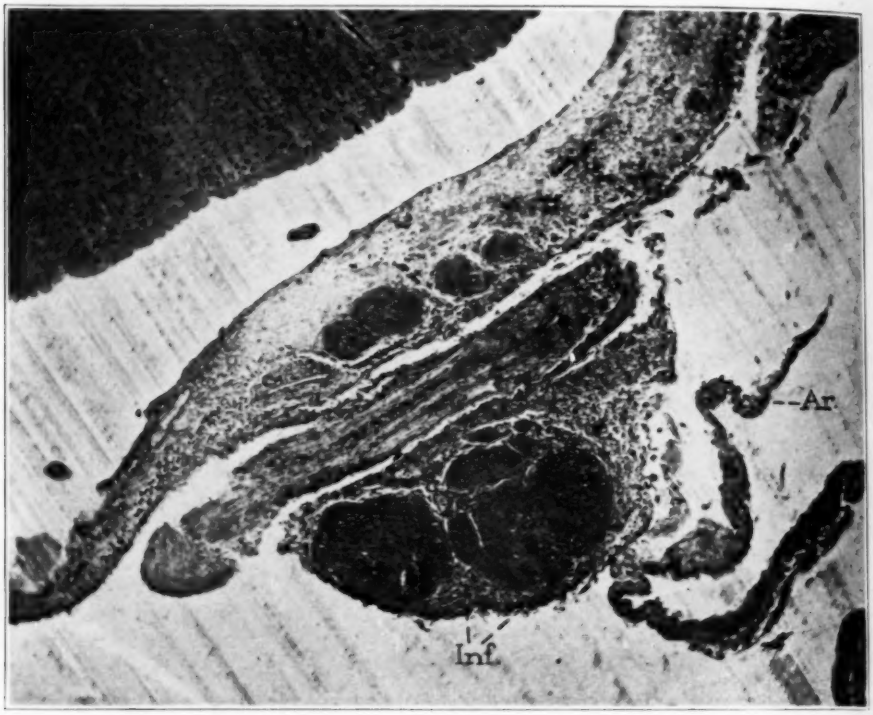

Fig. 5.—Nerve bundles enveloped by infiltrated and obstructed perineural spaces (*Inf.*) and embedded in an infiltrated subarachnoid space. *Ar.* indicates the arachnoid. Toluidine blue; × 57.

the form of thin capillaries and in many tufts they were lacking. The stroma of the choroid plexus was hyperplastic and fibrous and in general resembled a connective tissue scar. Covered by the tender tufts, it contained numerous lymphocytes, plasma cells, macrophages packed with blood pigment and many gitter cells filled with lipoids.

In contrast to such greatly changed intraventricular choroid plexuses, the extraventricular portion located in the lateral cistern was well preserved.

The meninges were markedly changed. The dura was thickened and hyperplastic; in some places it was ossified (fig. 3) and in some it was covered by

lymphocytes. Neither lacunae nor pacchionian bodies were here in evidence. The pia-arachnoid and the subarachnoid spaces showed an enormous infiltration with hematogenous elements. Over the convexity the subarachnoid space was dilated, but at the base, mainly around the pons and medulla, it appeared practically obliterated (fig. 4). Some cranial nerves were consequently embedded in vast accumulations of infiltrating cells and their perineural spaces appeared obstructed (fig. 5). The impression, in some instances, was that cranial nerves, the meninges and the cell infiltrations formed one mass (fig. 4). In other instances, again, the perineural

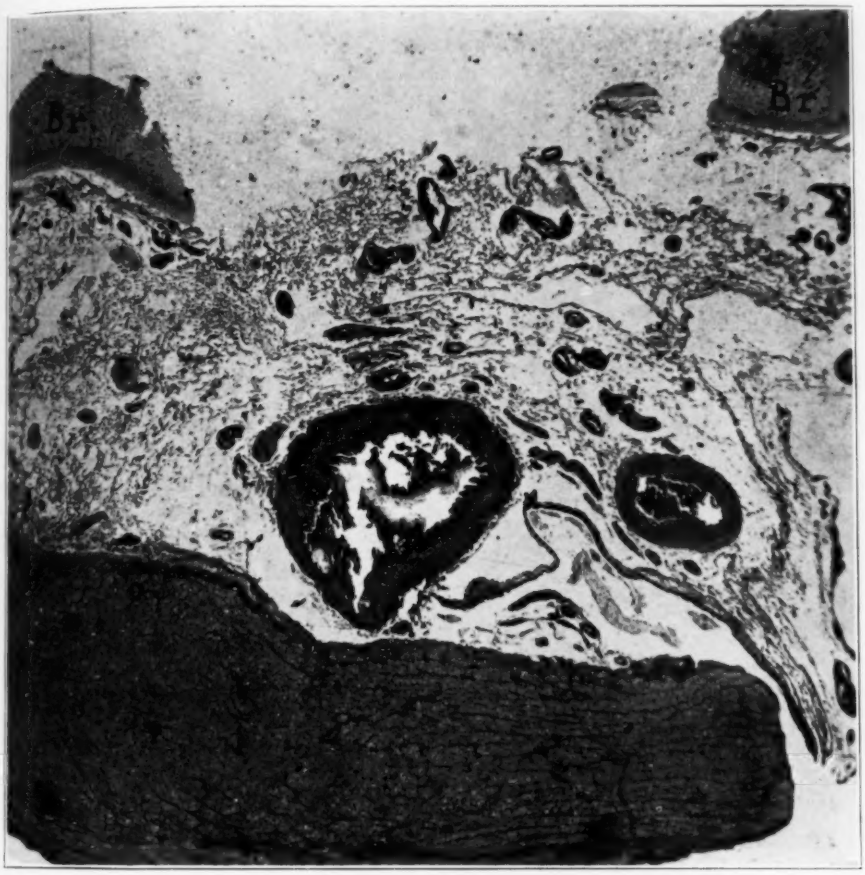

Fig. 6.—Distended subarachnoid or perineural space between the optic nerve in the lower half of the picture and brain tissue (*Br.*); × 21.

spaces were not obstructed but enormously dilated, as pictured in figure 6. The perineural space here (around the optic nerve) was not only greatly dilated but also infiltrated. A similar condition was also present around the olfactory nerve.

The parenchyma of the brain was spongy or areolar; the ganglion cells were vacuolated, and often they were shrunken and sclerosed. The walls of the lateral ventricles, the ependyma and the subependymal areas, especially around the fourth

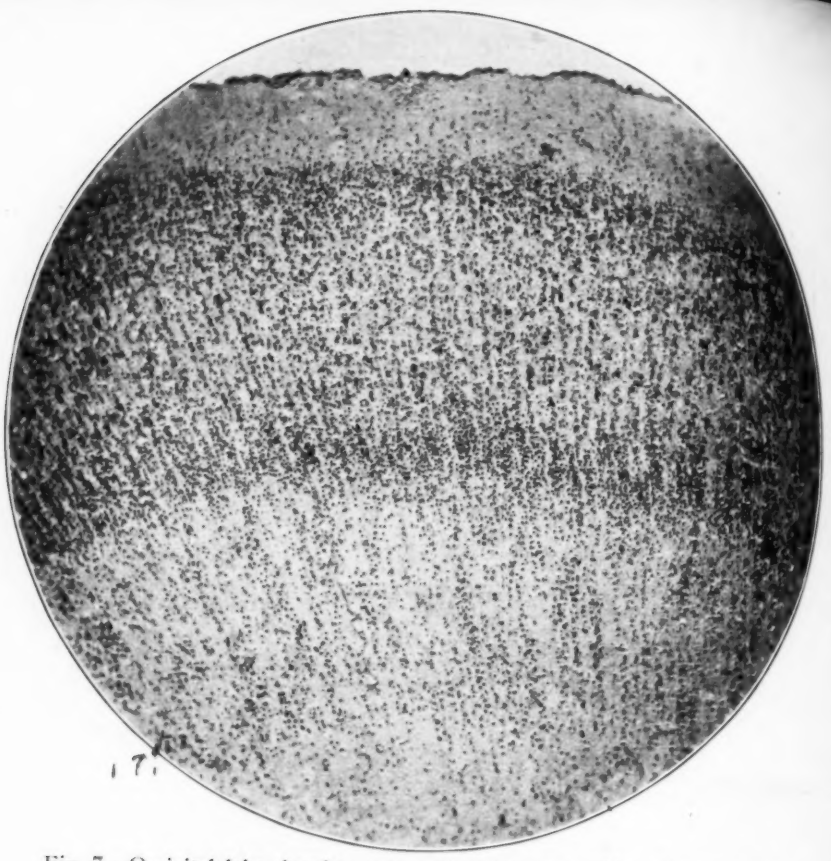

Fig. 7.—Occipital lobe showing a normal architecture. Van Gieson; × 71.

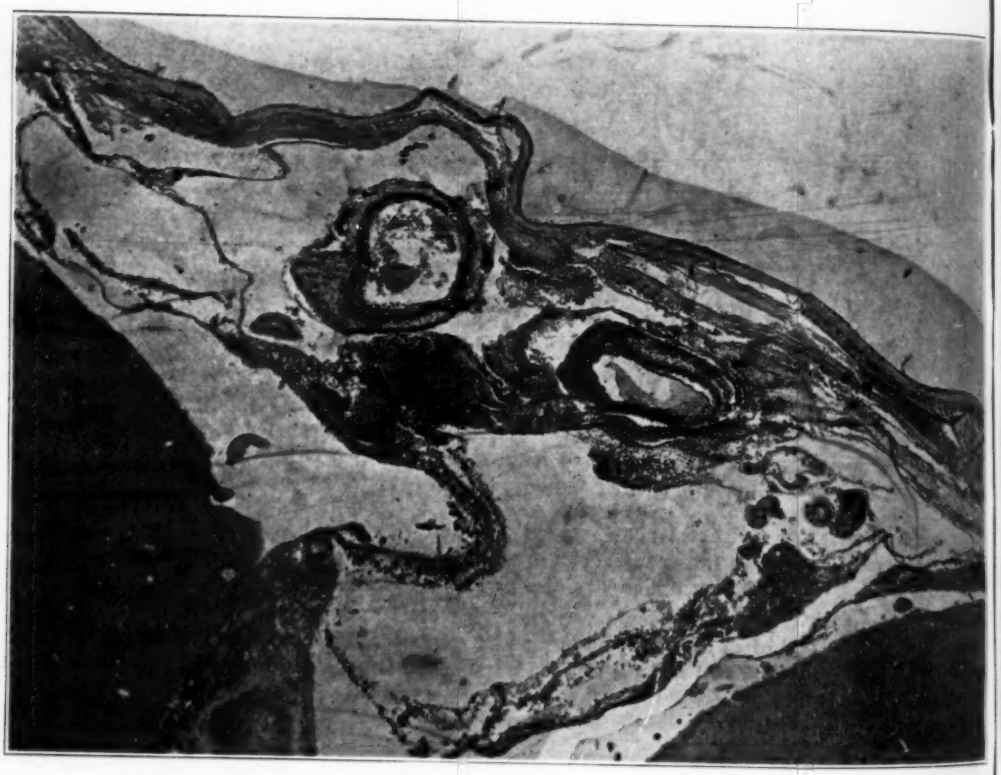

Fig. 8.—Distended subarachnoid space over the motor area. Van Gieson; reduced from × 30.

ventricle, were markedly infiltrated; often they were disintegrated and the blood vessels showed a marked perivascular infiltration with lymphocytes and plasma cells.

The anatomic changes, which in this case may be summed up as syphilitic meningo-encephalitis, with involvement of both the convexity and the base of the brain, comprised among other features: distention

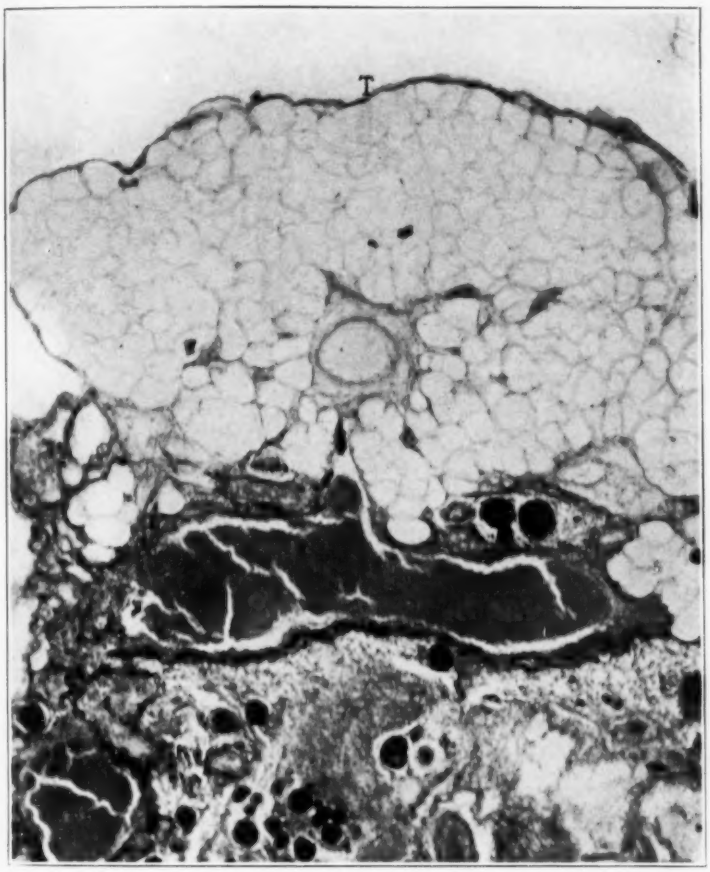

Fig. 9.—A vacuolated portion of the choroid plexus bordered above by a thin layer of tuft cells (*T*); in other parts (not in the picture) the choroid plexus was well preserved; in others it was sclerosed. The round black bodies are corpora arenacea. Van Gieson; × 57.

of the ventricles and the subarachnoid space of the convexity; obliteration of the basal subarachnoid space and the perineural spaces of some cerebral nerves; sclerosis of the choroid plexus and their adhesion to the cerebral parenchyma; edema and diffuse rarefaction of the cerebral substance and protrusion of the floor of the third ventricle.

Other Cases.—The changes outlined were fairly constant in the rest of the cases. In some they were even more marked, while in some additional features were noted. For instance, the cortex was invariably thinned, yet its architecture was preserved. Figure 7 shows the occipital lobe from another case of an enormous hydrocephalus caused by syphilitic meningitis. The latter was intense around the pons and medulla, but the subarachnoid space over the convexity was considerably distended (fig. 9); the perineural spaces of some cranial nerves

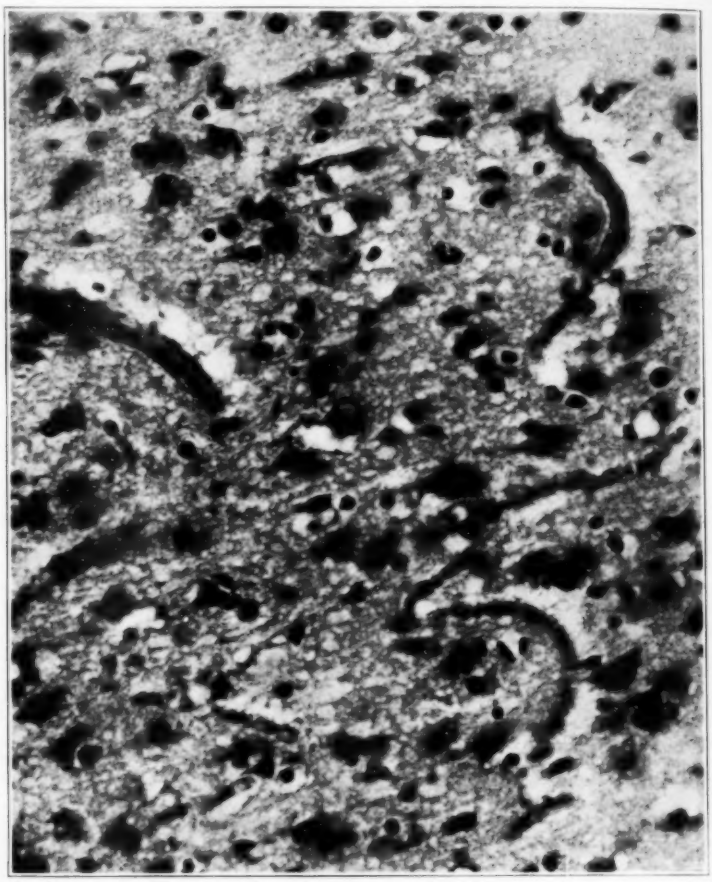

Fig. 10.—Rarefaction of brain tissue. Van Gieson; × 420.

were infiltrated and the choroid plexus appeared even more changed than in the case detailed. It was like a mere thread adherent to the floor of the lateral ventricles; in the fourth ventricle it was preserved much better.

In a third case, the choroid plexus, which was also firmly adherent to the ventricular wall, appeared vacuolated (fig. 9) and infiltrated in some portions; the subarachnoid space was excessively dilated and the cortex presented numerous areas of rarefaction (fig. 10). These were especially in evidence in a case of a hydrocephalus in which the hemispheres were represented by large empty bags.

Fig. 11.—Hyperplastic pacchionian bodies (*Pach.*), slightly infiltrated; the white space around each pacchionian body is the subdural space; the dura (*D*) shows markedly a cellular infiltration. *Ar.* indicates the arachnoid membrane. Reduced from × 57.

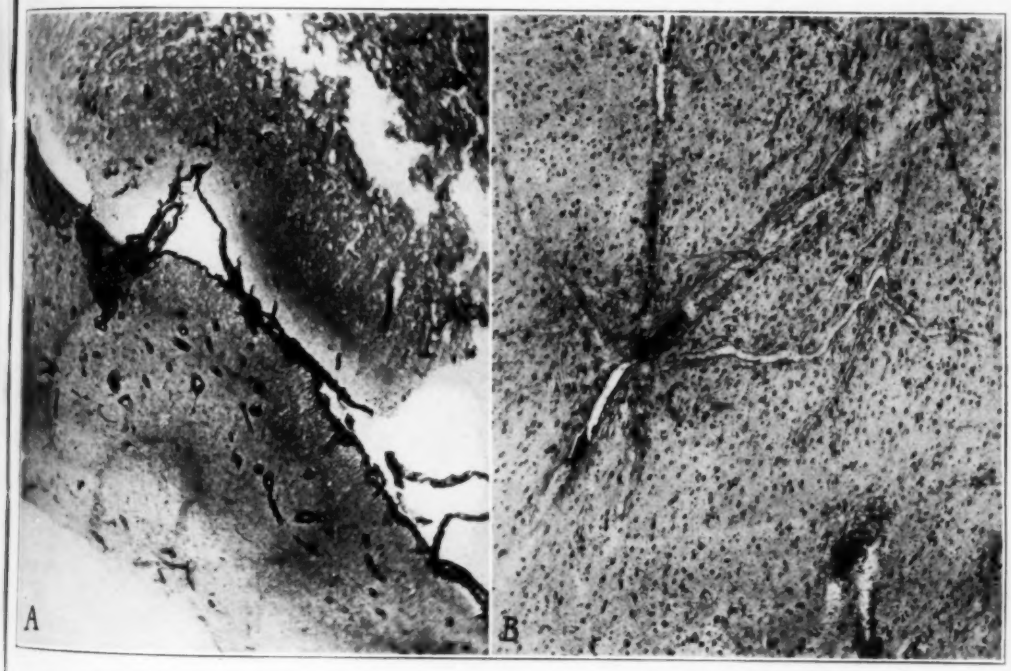

Fig. 12.—Blood vessels in the cortex; *A*, stained by Perdrau-Davidoff's method; reduced from ×100; *B*, stained by Cajal stain; reduced from × 65. The latter also shows the massing of the astrocytes around the blood vessels.

Fig. 13.—Lipoids in the white substance of the occipital lobe. Herxheimer stain; × 140.

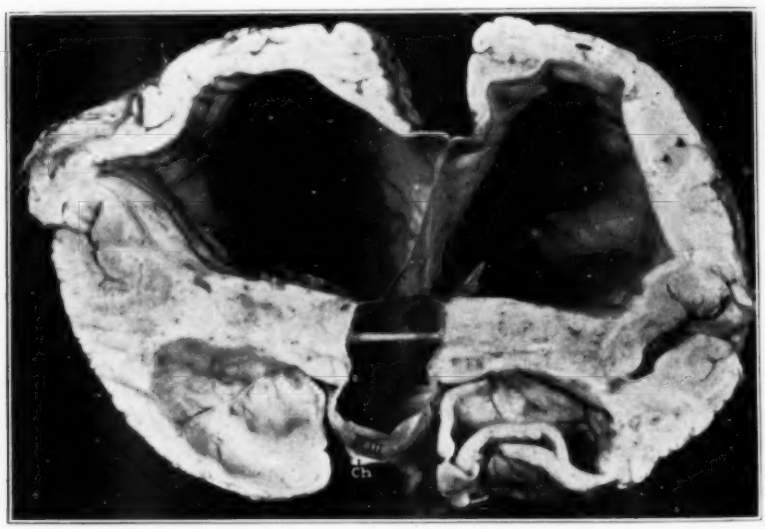

Fig. 14.—Macroscopic picture of a protruded floor of the third ventricle (*Fl. III*). *Ch* indicates the optic chiasm. Compare with figure 1, in which the protrusion of the floor is far less in evidence.

The gyri were narrow and numerous; the brain substance, especially the walls of the lateral ventricles, were thinned, the basal portions having been represented by thin, transparent membranes. The dura, the pia and the ependymal lining of the ventricles were thickened. The choroid plexus, as a narrow ribbon, was adherent to the floor of the inferior horn of the lateral ventricles, while no choroid plexus could be found in their frontal portion. The cerebellum was soft and collapsed; the third and fourth ventricles were distended. Microscopic examina-

Fig. 15.—Dilatation of the subarachnoid space around the pons (so-called external hydrocephalus). Toluidine blue; × 34.

tion showed a fibrous, hyperplastic leptomeningitis, and hyperplasia and infiltration of the pacchionian bodies (fig. 11), with hematogenous cells and macrophages which filled the connective tissue meshes of these avascular bodies. The brain tissue was markedly rarefied, especially around the ganglion cells and blood vessels, the latter of which were abundant. Some were thrombosed and their walls were hyperplastic (fig. 12*A*). In the pons the rarefaction was much milder; the ganglion cells here were much less involved and the subarachnoid space was

greatly dilated. The tuft cells of the choroid plexus were granular and their nuclei were pyknotic. The glia exhibited mild proliferative phenomena; the astrocytes were increased in size; their processes were often elongated and were densely gathered for the most part around the blood vessels (fig. 12 *B*); the oligodendroglia was swollen, while the microglia cells, especially in the white substance, contained numerous drops of lipoids (fig. 13). Lipoid substances were also present in the thinned corpus callosum, and were rather abundant in the subependymal areas and within the perivascular spaces of the blood vessels. In general, the brain appeared as if bathed in fluids which permeated the cerebral parenchyma, the ventricles and the subarachnoid spaces. It was in a hydropic condition, which was most likely the cause of its marked rarefaction.

In another case, the rarefaction was comparatively mild. Here the choroid plexus of the lateral ventricles was adherent to the underlying caudate nucleus; one of Luschka's foramina was obliterated and the pia was thickened and infiltrated, but the subarachnoid space was dilated.

A noteworthy feature, occasionally observed in hydrocephalus, was obliteration of the cisterns by a protruded base of the brain. For instance, in the case pictured in figure 14, the cerebral ventricles were considerably distended, the convolutions were flattened and the sulci were obliterated. The walls of the lateral ventricles and the middle commissure were thinned; the third ventricle was markedly dilated and its floor protruded. It pressed on the chiasm (*ch.*), obliterating the subjacent cistern, while the compressed optic thalami appeared excavated and the foramina of Monro enormously distended. In other cases again, the basilar subarachnoid spaces were enormously dilated (fig. 15), while others, as pointed out, were obliterated. The microglia cells were not more numerous than normally, but they were hypertrophied and their processes were abundant. Hypertrophied microglia was present also in the corpus callosum which was, as in other cases, greatly thinned and exhibited an accumulation of lipoid substances. The choroid plexus was adherent to and partly buried in the floor of the ventricles.

COMMENT

The observations recorded justify a conclusion that changes in hydrocephalus are present in the meninges, ventricles and subarachnoid space, including the cisterns, in the brain substance itself and the perineural spaces of some cranial nerves; the choroid plexus may be sclerosed and atrophied. Some changes—in the meninges, the choroid plexuses, the subarachnoid and the perineural nerve spaces—may be considered primary; the rest—distention of the cerebral ventricles, the subarachnoid space of the convexity and of some parts of the base and of the tissue spaces of the brain, protrusion of the floor of the third ventricle with the consequent obliteration of the subjacent cistern, the changes in the ganglion and glia cells—may be classified as secondary.

Striking as is the hydrocephalus itself, even more so is the sclerosis of the choroid plexus and the distention of the subarachnoid space. In hydrocephalus, there is thus an excessive accumulation of cerebrospinal fluid often in the presence of defectiveness or deficiency of the

structure that is supposed to produce it. Though noted by a number of observers (Anton,[3] Claisse and Levi,[4] Haushalter and Thiry,[5] d'Astros,[6] Burr and McCarthy,[7] White,[8] Spiller,[9] Orton [10] and Dott [11]), the causal relationship was ignored or given a hypothetic interpretation. Claisse and Levi, for instance, assumed that the cause of the excessive accumulation of fluid lay in the hypertrophy of the choroid plexus; Henle [12] and Gladstone and Dunlop,[13] in its inflammation, and White, in the circulatory disturbances of the choroid plexus, while Dott, who saw the choroid plexus in vivo in a lateral ventricle buried in scar tissue, stated that its appearance suggested that "it must be practically functionless"; yet the ventricle which harbored this "functionless" choroid plexus was overfilled with fluid, while the opposite ventricle where the choroid plexus was "actively secreting" showed no hydrocephalus. Something similar was present in the case of Winkelman and Eckel.[14]

These observations, like those here recorded, show that large accumulations of fluid may exist in the presence of a deficient or a defective choroid plexus. As it is defective and thus incapable of functioning properly, it cannot be expected to produce such large amounts of cerebrospinal fluid as are seen in hydrocephalus. For this reason an assumption is permissible that the cerebrospinal fluid is not the product of the choroid plexus. The condition that existed

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3. Anton, G.: Zur Anatomie des Hydrocephalus und des Gehirndruckes, Med. Jahrb. **3**:125, 1888.

4. Claisse, P., and Levi, C.: Étude histologique d'un cas d'hydrocéphalie interne, Bull. Soc. anat. de Paris **72**:264 (March) 1897.

5. Haushalter, P., and Thirty, C.: Étude sur l'hydrocéphalie, Rev. de méd. **17**:624, 1897.

6. d'Astros, Leon: Les hydrocéphalies, Paris, G. Steinheil, 1898.

7. Burr, C., and McCarthy, D. J.: Acute Internal Hydrocephalus: A Clinical and Pathological Study, J. Exper. Med. **5**:195, 1900.

8. White, W. C.: A Case of Idiopathic Internal Hydrocephalus with Recurrent Hemiplegic Attacks, Am. J. Insan. **58**:503, 1902.

9. Spiller, William: Two Cases of Partial Internal Hydrocephalus from Closure of the Intraventricular Passages, Am. J. M. Sc. **124**:44 (July) 1902.

10. Orton, S. T.: A Pathological Study of a Case of Hydrocephalus, Am. J. Insan. **65**:228 (Oct.) 1908.

11. Dott, N. M.: A Case of Left Unilateral Hydrocephalus in an Infant: Operation; Cure, Brain **50**:548, 1927.

12. Henle, A.: Beitrag zur Pathologie und Therapie der Hydrocephalus, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **1**:264, 1896.

13. Gladstone, R. J., and Dunlop, H. A.: A Case of Hydrocephalus in an Infant with Comments on the Secretion, Circulation and Absorption of the Cerebro-Spinal Fluid, J. Anat. **61**:360 (April) 1927.

14. Winkelman, N. W., and Eckel, J L.: Pathology and Pathogenesis of Unilateral Internal Hydrocephalus, Arch. Neurol. & Psychiat. **12**:187 (Aug.) 1922.

in some of my cases was altogether analogous to that produced experimentally by Dandy.[15] By removing the choroid plexus through an extensive incision over the cortex and blocking the sylvian aqueduct and one foramen of Monro, the edges of which had been scarified, Dandy obtained an obliteration of the corresponding lateral ventricle with practically no fluid in it. However, he did not give the histologic picture of the walls of the obliterated ventricle, and he did not mention the reactive phenomena which undoubtedly did take place as the result of the surgical manipulations. The latter, not the extirpation of the choroid plexus, were most likely responsible for the obliterated ventricle, for in the absence of reactive phenomena the choroid plexus may be atrophied and sclerosed, yet the ventricles may be greatly dilated and distended with cerebrospinal fluid. It is hardly conceivable that such atrophied structures can become hyperactive, and it should be conceded that facts furnished by pathologic studies strongly favor the view that the function of the choroid plexus is not the elaboration of the cerebrospinal fluid, but its absorption. The latter should not be understood in the sense that it absorbs the spinal fluid in toto (Wüllenweber,[16] Klestadt[17]), including the watery part. The absorption activity is confined to some constitutents of the spinal fluid. This fluid, whether a dialysate or some other biochemical product, represents the tissue fluids of the brain and spinal cord loaded with waste products. These must be removed, purified as it were, before the spinal fluid is absorbed. As shown elsewhere,[18] the tuft cells of the choroid plexus pick up from the cerebrospinal fluid the waste products, such as lipoids, prelipoids and other substances, and render the fluid not only less harmful but also absorbable. This function is performed for the fluids of the lateral, third and fourth ventricles by their choroid plexuses; for the fluids of the subarachnoid spaces, it is performed by the extraventricular choroid plexus, which is situated at the base of the brain outside the medulla (fig. 16), in the lateral cistern. This extends over the whole lateral part of the pons and is bordered above by a membrane extending from the upper margin

15. Dandy, W. E.: Experimental Hydrocephalus, Tr. Am. S. A. **37**:397, 1919.

16. Wüllenweber, G.: Ueber die Funktion des Plexus choroideus und die Entstehung des Hydrocephalus internus, Ztschr. f. d. ges. Neurol. u. Psychiat. **88**:208, 1924.

17. Klestadt, B.: Experimentelle Untersuchungen über die resorptive Funktion des Epithels des Plexus choroideus und des Ependyms der Seitenventrikel, Centralbl. f. allg. Path. u. path. Anat. **26**:161, 1915.

18. Hassin, G. B.: Effect of Organic Brain and Spinal Cord Changes on the Subarachnoid Space, Choroid Plexus and Cerebrospinal Fluid, Arch. Neurol. & Psychiat. **14**:468 (Oct.) 1925.

of the pons to the root of the fifth nerve. Below and laterally, the lateral cisterns are, in the region of the flocculus, connected with the cisterna magna (Boss [19]). In short, this portion of the choroid plexus must be traversed by the spinal fluid on its way from the convexity to the basilar cisterns for further elimination through the perineural nerve spaces. One may say that the function of the choroid plexus, as alluded

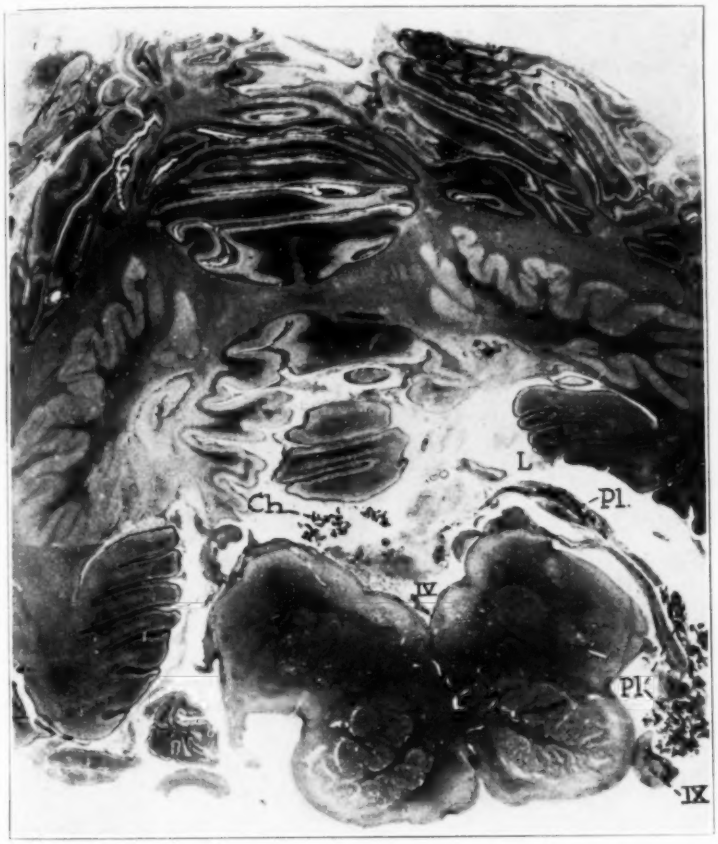

Fig. 16.—Extraventricular choroid plexus at *Pl*. *L* is Luschka's recess. The fourth ventricle (*IV*) is bordered above by the tela and the choroid plexus (*Ch*). *IX* indicates the glossopharyngeal nerve. Van Gieson; × 6.

to by Bland-Sutton, much resembles that of the kidneys which when diseased cannot prevent an accumulation in the blood of harmful toxic

19. Boss, Leo: Topographische Anatomie der Subarachnoidalräume an der Gehirnbasis, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **115**:64, 1926.

20. Bland-Sutton, J.: The Lateral Recesses of the Fourth Ventricle; Their Relation to Certain Cysts and Tumors of the Cerebellum, and to Occipital Meningocele, Brain **9**:352, 1887.

substances. However, the choroid plexus does not have to be damaged in every case of hydrocephalus; in some cases it is fairly well preserved, as in the case of Homen.[21]

Another noteworthy feature is the considerable distention of the subarachnoid spaces, especially over the convexity (fig. 8), and of those portions of the base (fig. 15) which were not obstructed by meningitis. According to popular teaching, the contents of the subarachnoid spaces come from the choroid plexuses of the lateral, third and fourth ventricles. From these it travels through the foramina of Monro, third ventricle, sylvian aqueduct, fourth ventricle, the foramina of Luschka, and possibly of Magendie, to the base of the brain. Here it fills the meshes of the subarachnoid space and the cisterns. Then it travels upward to the subarachnoid space of the convexity, presumably to be discharged through the pacchionian bodies into the venous sinuses. In case any of the foregoing pathways are obstructed, it is reasonable to expect that the ventricular contents would fail to reach their goal—the subarachnoid space of the convexity.

Deprived, as it were, of the source of fluid supply and compressed by a distended hydropic brain, the subarachnoid spaces, including the pacchionian bodies, should appear collapsed or obliterated. Globus[22] mentioned such a condition in his cases. Evidently basing his views on the embryologic studies of Weed,[23] he interpreted this condition as an embryologic defect of the pia which formed together with the arachnoid a solid mass of mesenchymal tissue as found in the primitive meninx. Globus admitted that such an embryonic defect was limited to certain areas, that inflammatory phenomena were absent throughout and that many subarachnoid spaces were open.

As a rule, large as the hydrocephalus may be, neither the subarachnoid spaces nor their derivatives, the pacchionian bodies, are obliterated, but show an abundance of connective tissue and cells. As pointed out elsewhere,[24] their structure resembles that of the arachnoid membrane, and, as figure 11 shows, they exhibit no adhesions to the dura from which they are separated. On the other hand, in a number of my cases the prolongations of the subarachnoid spaces over the cranial nerves (perineural spaces) were greatly distended and packed

21. Homen, E.: Seltene Sektionsfälle, Arb. a. d. path. Inst. zu Helsingfors **1**:367, 1905.

22. Globus, J.: Communicating Hydrocephalus and So-Called Idiopathic Hydrocephalus, Am. J. Dis. Child. **36**:680 (Oct.) 1928.

23. Weed, L. H.: The Development of the Cerebrospinal Spaces in Pig and Man, Contrib. Embryol., 1917, vol. 5, no. 14.

24. Hassin, G. B.: Villi (Pacchionian Bodies) of the Spinal Arachnoid, Arch. Neurol. & Psychiat. **23**:65 (Jan.) 1930.

with hematogenous and other cells (figs. 4 and 6). It is reasonable to assume that inflammation or other factors may cause obstructions not only of the subarachnoid but also of a number of perineural nerve spaces, which should reflect on the condition of the subarachnoid spaces. Their draining, or the discharge of their contents, may become interfered with and ultimately result in accumulation or stagnation of fluid in the subarachnoid space and the ventricles—in short, in the formation of hydrocephalus. Such an important additional factor, obstruction of the perineural nerve spaces, should be borne in mind when the blocking of the subarachnoid space itself is not manifest or is incomplete, that is, in obscure cases of so-called idiopathic hydrocephalus. Such a form, which is frequently diagnosed even now whenever the cause of the accumulation of fluid cannot be ascertained, should no longer be recognized.

PATHOGENESIS OF HYDROCEPHALUS

The patency of the subarachnoid space and its prolongations over the cranial nerves is essential to the so-called circulation of the cerebrospinal fluid. Occlusion of the foregoing spaces causes an accumulation of fluids not only in the ventricles of the brain, but in its parenchyma and the subarachnoid spaces, especially over the convexity. The question is, where do such excessive amounts of fluids come from? Assuming that the cerebrospinal fluid is a product of the choroid plexus, it is easy to understand its accumulation in the ventricles when its outflow is prevented (by a severe basilar meningitis, for instance). On the basis of this theory, it is not easy to understand why it should accumulate also in the brain tissues, where it causes their rarefaction, or in the subarachnoid spaces, where it causes their distention (figs. 8 and 15). On the other hand, assuming that the cerebrospinal fluid is derived from the tissue fluids of the brain itself, the foregoing incompatibilities can be explained satisfactorily. As emphasized in a number of contributions,[25] the tissue fluids of the brain, loaded with waste products, are discharged through the perivascular spaces, partly into the ventricles and partly into the subarachnoid spaces. One may say that the parenchymatous tissue spaces with the perivascular and subarachnoid spaces and the cerebral ventricles form one system. After the tissue fluids have been discharged into the ventricles and the subarachnoid spaces, they are ultimately accumulated in the cisterns at the base of the brain. The ventricular portion reaches them by the way of the recesses of Luschka; the fluid from the subarachnoid

25. Hassin G. B.: These are referred to in the paper, Notes on the Nature and Origin of the Cerebro-Spinal Fluid, J. Nerv. & Ment. Dis. **59**:113, 1924; footnote 18.

spaces reaches them by flowing downward (not upward, in an opposite direction, as maintained by the advocates of the secretion theory). From the cisterns and the subarachnoid spaces the fluid is carried to the periphery along their prolongations over the cranial nerves or the spinal nerve roots; according to Cornelia de Lange,[26] about one fifth of the fluid absorption occurs along the latter. Of course, it should be understood that not all the perineural spaces become obstructed simultaneously, or over their entire length. Many perineural spaces undoubtedly continue functioning and thus contribute to the escape of the cerebrospinal fluid to the periphery and indirectly to the draining of the tissue fluids of the brain. As the perineural nerve spaces are in communication with the epineural spaces, which are rich in blood vessels, it is proper to assume that the final escape takes place through the latter. The foregoing considerations find support in the observations of Magendie[27] and the experiments of Key and Retzius[28] and those of Weed.[29] The latter considered the perineural pathways as "merely a stage in the mechanism of lymphatic drainage" as a "phase of the accessory system of absorption for the cerebrospinal fluid," while Key and Retzius followed up the fluids injected into the subdural and subarachnoid spaces to the periphery. On the other hand, Magendie showed that the cranial nerves are "bathed in and enveloped by the cerebrospinal fluid" into which "the nerves plunge at their origin and which accompanies them down to their exit through the foramina of the cranium and the vertebral column." It is possible that the perineural spaces of the twenty-four cranial nerves suffice for the effective escape of the spinal fluid from the brain without any participation or assistance on the part of the pacchionian bodies.[24] It is probable that the escape is greatly assisted by the expansions and contractions of the brain by which the fluid is squeezed out, spongelike, as it were, into the perineural spaces of the cranial and spinal nerves for final elimination to the periphery. In case the fluid, during its course, meets with an obstruction (in the ventricles, sylvian aqueduct, the recesses of Luschka, the subarachnoid spaces and the perineural nerve spaces), it will accumulate cephalad to the obstruction, that is, in both the ventricles and the subarachnoid spaces by which it is drained.

26. Cornelia de Lange: Klinische und pathologisch-anatomische Mitteilungen über Hydrocephalus chronicus congenitus und acquisitus, Ztschr. f. d. ges. Neurol. u. Psychiat. **120**:433, 1929.

27. Magendie, F.: Recherches physiologiques et cliniques sur le liquide céphalo-rachidien ou cérébrospinal, Paris, Librairie médicale de Meguignon, Marvis Fils, 1842.

28. Key, A., and Retzius, G.: Studien in der Anatomie des Nervensystems und des Bindegewebes, Stockholm, P. A. Norstedt & Söner, 1876, vol. 2, p. 9.

29. Weed, L. H.: Studies in Cerebro-Spinal Fluid, J. M. Research **31**:81, 1914.

The dilatation of the subarachnoid space is associated with hyperplasia of the dura, the pia-arachnoid and its derivatives—villi and pacchionian bodies; marked atrophy of the cortical tissue is not in evidence. It is probably prevented by the compensatory function of the cerebral meninges. The stagnant fluid, rich in catabolic products, provokes in them, as well as in the pacchionian bodies, reactive phenomena, such as hyperplasia of connective tissue cells and even ossification (fig. 3). These phenomena are thus secondary and not primary conditions as one may gather from the studies of Temple Fay,[30] Bateman[31] and Winkelman and Fay.[32] The distended subarachnoid space must of necessity reflect also on the condition of the brain tissues. Filled with tissue fluids which, like the ventricles, it constantly receives from the brain, the subarachnoid space ultimately becomes, as it were, brim full and unable to accommodate more. The result is that the fluid is kept back within the brain tissues, overflows them and causes their distention, rarefaction or edema; in advanced cases, it causes the breakdown of the white substance with its consequent atrophy. In general, in hydrocephalus not only the cerebral (ventricular) and the extracerebral (subarachnoid) cavities are overfilled with fluids, but also the spaces of the cerebral tissues themselves. The brain with its appendages, bathed in its own tissue fluids, is in a state of general hydrops. In many features it is analogous to one produced by intravenous injections of hypotonic salt solutions, which have been so well studied by Ferraro.[33]

The outstanding component of the hydropic condition, whether in a human brain or produced experimentally, is the hydrocephalus. It attracts the principal attention, to the detriment of other components (the rarefaction and edema of the subarachnoid space) which, though less conspicuous, are probably more important. In some cases, they may be the outstanding phenomena and overshadow the hydrocephalus itself. I observed two such cases. In one case, that of a child, aged 6, the pathologic conditions were a leptomeningitis with a moderate infiltration with polymorphonuclear cells, lymphocytes, polyblasts and macrophages, all especially in evidence at the base around the occipital lobe.

30. Fay, Temple: Some Factors in the "Mechanical Theory of Epilepsy," with Especial Reference to the Influence of Fluid, and Its Control in the Treatment of Certain Cases, Am. J. Psychiat. **8**:783 (March) 1929; Generalized Pressure Atrophy of the Brain, Secondary to Traumatic and Pathologic Involvement of Pacchionian Bodies, J. A. M. A. **94**:245 (Jan. 25) 1930.

31. Bateman, J. F.: Meningitis with Special Reference to the Rôle of the Pacchionian Bodies, Ohio M. J. **25**:970 (Dec.) 1929.

32. Winkelman, N. W., and Fay, Temple: The Pacchionian System: Histologic and Pathologic Changes with Particular Reference to the Idiopathic and Symptomatic Convulsive States, Arch. Neurol. & Psychiat. **23**:44 (Jan.) 1930.

33. Ferraro, A.: The Reaction of the Brain Tissue to Intravenous Injections of Hypotonic Solutions, J. Nerv. & Ment. Dis. **71**:129 (Feb.) 1930.

Here the pia-arachnoid was greatly distended, and the pia was much congested. The cerebral parenchyma showed signs of generalized edema with a marked glia reticulum in the subcortical layers. The cytoplasmic glia was prominent, and the oligodendroglia was swollen. The cerebral blood vessels were congested, and the capillaries appeared increased in numbers, but the lateral ventricles were practically normal. The common pathologic diagnosis, in this case, would be edema of the brain with an acute localized leptomeningitis; the proper diagnosis, however, would be hydrocephalus. Some features of the latter were present in another case in which distended ventricles were also absent. The conditions in these two cases might be termed partial or incomplete cerebral hydrops.

The hydrops (complete or partial) must, of necessity, exert a deleterious influence on the ganglion cells, the neuroglia and the nerve fibers. It may also be the cause of massive accumulation of lipoids in the white substance and of mental deficiency. This is rather common in advanced cases of hydrocephalus, notwithstanding the normal cortical lamination, the apparently normal morphology of the ganglion cells and nerve fibers and the absence of manifest cortical atrophy.

CONCLUSIONS

1. Hydrocephalus is a partial manifestation of a hydropic condition of the brain.

2. Other components—distention of the subarachnoid spaces and rarefaction of the cerebral tissues—are probably the more important and may be the principal manifestations of the hydrops of the brain.

3. The sole avenue of escape or absorption of the cerebrospinal fluid is from the subarachnoid spaces by way of their prolongations along the perineural spaces of the cranial nerves and spinal roots.

4. The escape of the fluid is probably much aided by the expanding and collapsing movements of the brain.

5. The escape may be prevented by the occlusion of the cisterns and the perineural spaces of the cerebral nerves (important factors in producing hydrocephalus).

6. As the choroid plexus is sclerosed and atrophied in many cases of hydrocephalus, it cannot be considered an elaborator of the cerebrospinal fluid.

7. The phenomena of hydrocephalus are identical with those observed in acute hydropic conditions of the brain produced by intravenous injections of hypotonic salt solutions.

8. Studies of the pathologic changes of hydrocephalus suggest interpretations of facts and conclusions which do not correspond with those obtained experimentally; in fact, they are altogether contradictory.

ABSTRACT OF DISCUSSION

DR. WALTER E. DANDY, Baltimore: There are, of course, two ways of arriving at conclusions about the circulation of cerebrospinal fluid: (1) the anatomic method as reported here by Dr. Hassin with his microscopic studies and (2) the experimental method.

From the microscopic pathology one has to make such a long chain of inferences to arrive at conclusions. That is a dangerous method, as there are so many weak links in the chain. It eventually has to satisfy a number of experiments which are more or less crucial.

The problem of hydrocephalus, I should say, was solved. You can prove by very simple experiments that cerebrospinal fluid is formed by the choroid plexuses, and you can prove just as conclusively that cerebrospinal fluid is absorbed not in the pacchionian granulations, which have nothing to do with it, but in the subarachnoid spaces over the brain. For example, after the foramen of Monro has been blocked hydrocephalus will invariably follow. On the other hand, if one foramen of Monro is blocked and the choroid plexus of this ventricle is removed at the same time, hydrocephalus will never follow. The conclusion, therefore, is inescapable that the choroid plexuses secrete cerebrospinal fluid.

That the pacchionian granulations have nothing to do with absorption of the cerebrospinal fluid is easily shown by merely separating with a spatula both cerebral hemispheres from all of the great venous sinuses, the longitudinal, transverse, and cavernous sinuses. This is easily done in a dog, and hydrocephalus will never result, although all of the so-called pacchionian granulations are separated beyond a distance permitting repair.

You can prove by experiment that the cranial nerves and the spinal nerves have no practical part in the absorption of the cerebrospinal fluid. For example, you can obliterate the cisterna under the midbrain by applying iodine to a very localized spot. The spaces around the cranial nerves, around the spinal nerves, are still open to absorb cerebrospinal fluid, but hydrocephalus will always develop. The crucial test of any theory is the induction of any particular type of hydrocephalus by specific methods, and all types of hydrocephalus can be reproduced by experiments on animals.

Furthermore, there are no cases of hydrocephalus in which one cannot demonstrate with the naked eye a very gross lesion which is causing an obstruction to the pathways for cerebrospinal fluid. There are no exceptions to that rule. This demonstration is not dependent on the personal equation for interpretation. Occlusions in the ventricular system are obvious; those in the subarachnoid space —the cisternal—can be just as graphically demonstrated by injection of a colored solution, such as india ink, into the spinal canal of a cadaver; it will always pass to the obstruction and not beyond.

All of our operative procedures are based on a precise localization of obstructions. And clinically, if need be, the block can be demonstrated just as easily as at necropsy. One can inject air, which will stop at the obstruction and be demonstrable in the roentgenogram, just as graphically as india ink is demonstrable at the block in the postmortem specimen. Such a clinical demonstration is no longer necessary or advisable, but it has been done on many occasions.

Dr. Hassin is certainly not justified in entering such tremendous iconoclastic conclusions from such slight evidence when all of the experimental, clinical and pathologic data are now so absolute and so perfectly correlated that the cause of hydrocephalus can easily be shown in every case.

Dr. Nathaniel Winkelman, Philadelphia: Dr. Hassin's point of view is not new to those who have followed his work. He believes that the finding of an internal hydrocephalus in association with an atrophic choroid plexus is further substantiation of his theory that the choroid activity could not be responsible for the ventricular dilatation. The fact that the change in the choroid plexus may have been secondary to pressure was not considered.

Dr. Dandy's work is also known to those who have followed the subject. He thinks that the elimination of the spinal fluid takes place over the cerebral surface. The fact that the flow is in the reverse direction as shown in intracortical hemorrhage or abscess is proof, to me, that this mechanism is incorrect.

Most recent investigators agree that the source of the cerebrospinal fluid is double, but that most of it undoubtedly comes from the choroid plexus, and Dr. Hassin has shown that there is also an outflow from the brain tissue through the perivascular spaces into the subarachnoid space. In the cases reported by Dr. Hassin with obstruction at the base, there is still an outpouring of material into the subarachnoid space, and in these cases the pacchionian bodies might be absolutely normal. The fact that they are normal could not be taken as evidence of lack of functional importance, for they would still have to filter through them the fluid that comes from the brain substance itself.

I am therefore still of the opinion that the choroid plexus plays the predominant rôle in the formation of spinal fluid, and that the pacchionian bodies serve as filters for its elimination.

Dr. George B. Hassin, Chicago: I know of the work of Dr. Dandy, and I know that if he were here he would object to my conclusions. His views are based on experimental work which he considers of greater importance than human pathology. Such an attitude is unpardonable, for however skilful the experiments may be, they cannot stand comparison with the experiments performed by nature. The changes occurring in hydrocephalus cannot be explained by Dandy's theory. He states that when one removes a choroid plexus the corresponding ventricle becomes obliterated, and it therefore contains no fluid. Yet in my cases hydrocephalus, that is, excessive accumulation of cerebrospinal fluid, was present, regardless of the fact that the choroid plexus was severely affected. By sticking a piece of meat into the foramen of Monro, Dandy, I believe, caused reactive phenomena in the corresponding ventricle, which became obstructed and thus prevented the accumulation of fluid which is normally discharged there from the brain by way of the cerebral perivascular spaces. In none of Dandy's contributions which I studied carefully is there any histologic description of the obstructed ventricles. My histopathologic studies of human material force me to conclusions different from those arrived at by experimental workers. Who is right, I do not know. I think that I am.

Dr. Weed's work on the cerebrospinal fluid is unquestionably classic. Weed himself showed that the cerebrospinal fluid is discharged by way of the perineural spaces of the cranial nerves, but he states that this mode of discharge is merely additional to the main one, which is through the pacchionian bodies. For this purpose, the fluid must flow from the base of the brain upward to reach a few insignificant structures which as a matter of fact possess no function. Like many others, Weed was greatly influenced by the work of Key and Retzius. They gave excellently stained pictures of these formations in connection with the discharge of spinal fluid and its flow. During the more than fifty years since their famous publication, a number of new facts have accumulated, and it seems that it is time to change their view.

DERMATONEUROMYOSITIS RESULTING IN SCLERODERMA *

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Scleroderma as a disease entity is scoffed at by most authorities. The general conclusion is that it belongs to the class of trophoneurotic and angiotrophoneurotic disturbances with the outstanding characteristic of a nutritional disturbance of the skin leading to atrophy, induration and other anomalies. Like most other angioneuroses and trophoneuroses, the condition is rare. Oppenheim,[1] Steiner [2] and others called attention to the relationship of the scleroderma syndrome to dermatomyositis and to this condition as a possible sequel of dermatomyositis. Hoover [3] described several cases of dermatomyositis, which, from his description, also had the characteristic skin changes of scleroderma. In the course of several years, a number of cases of dermatomyositis, dermatoneuromyositis and scleroderma have been seen at the Lakeside Hospital in Cleveland, St. Joseph's Mercy Hospital in Ann Arbor and the Neurological Institute of New York; attention was attracted by outstanding characteristics that these three conditions have in common and the apparent relationship that the first two bear to the later appearance of the sclerodermal syndrome.

Certain constant features accompanying or preceding the appearance of so-called scleroderma in the patients seen warrant the reporting of these cases, calling attention to the fact that a certain class of sclerodermal conditions are the result of dermatomyositis or a dermatoneuromyositis in which there develop trophic changes. Some have these changes and others have not. Reported cases of dermatomyositis and dermatoneuromyositis resulting in scleroderma are few in number. Rosenthal and Hoffman,[4] in 1924, reported a case and reviewed the cases reported by Lewy, Adler and Hanford, Levy and Dorn. They called attention to the fact that concomitant muscle, nerve and skin involvement is extraordinarily rare, and that in the literature the con-

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* From the Neurological Institute of New York.

1. Oppenheim: Text-Book of Nervous Diseases, Chicago Medical Book Company, 1920, vol. 1, pp. 544 and 546.

2. Steiner, in Osler: Modern Medicine, Philadelphia, Lea & Febiger, 1920, vol. 6, p. 787.

3. Hoover, in Tice: Practice of Medicine, Hagerstown, Md., 1924, vol. 6, p. 536.

4. Rosenthal and Hoffman: Klin. Wchnschr. **3**:115, 1924.

dition is described as polymyositis or neuromyositis. Levy's case was one with the onset and symptoms of primary acute polymyositis, atrophy, a roseola-like exanthem and later a purpuric eruption. Adler's and Hanford's cases were described as a neuromyositis; one showed a purpuric exanthem and the other, skin discoloration and permanent pigmentation. Levy-Dorn's case was a polymyositis and neuritis with pigmentation and skin changes of the involved parts. Rosenthal and Hoffman reported a case of dermatoneuromyositis resulting in scleroderma, with an onset similar to acute febrile muscular rheumatism and polyneuritis, associated with erythema and edema of the skin. As the patient recovered from the muscle and nerve involvements, changes in the skin progressed and a typical sclerodermic syndrome eventually developed. On the basis that scleroderma is regarded by many as a disturbance of internal secretions, the cases mentioned and several that I have seen would be difficult to explain. Articular involvements of a hypertrophic or atrophic nature, described as accompanying sclerodermal changes,[5] also lend evidence to the supposition that the disorder is of an infectious nature. Bier,[6] Dercum,[6] Cassirer,[6] Mueller[6] and Berloletti[6] found roentgen evidence of changes in the bones and joints of the extremities in 25 per cent of cases of scleroderma, with the wrists and phalanges most frequently affected. Bier and Dercum described bone and joint changes as frequently being the first symptom. Atwater also called attention to the bone and joint changes accompanying scleroderma.[7]

Recent literature[8] has shown a trend toward the view that scleroderma may be caused by some alteration in an internal secretion or some disturbance in balance between the various internal secretions, and thyroidal dysfunction has been particularly emphasized as a main factor. However, the response to specific treatment on such a basis is still inadequate; one certainly would expect more uniform results following such a plan of treatment.

Anatomically, the changes that have been described by all authors[9] who have written on the subject are fairly constant and uniform, but one does not know whether they are primary or secondary. The central nervous system is usually normal;[10] the changes found in isolated cases

5. Zadek: Med. Klin. **12**:1325, 1916.

6. Quoted by Leontjew: Ueber Veränderungen der Knochen und Gelenke bei Sklerodermie, Arch. f. klin. Chir. **28**:293, 1924.

7. Atwater: Am. J. M. Sc. **158**:29, 1919.

8. Wise: Nelson's Loose Leaf Living Medicine, New York, Thomas Nelson & Son, 1927, vol. 3, p. 333.

9. Curschmann: Text-Book on Nervous Diseases, Philadelphia, P. Blakiston's Son & Company, 1920, vol. 2, pp. 946-954. Oppenheim (footnote 1, vol. 2, p. 1335).

10. Osler: Systems of Medicine, Philadelphia, Lea & Febiger, vol. 6, pp. 665-675.

are not of etiologic significance. The peripheral nerves may be intact; the changes found—simple degeneration, parenchymatous neuritis or perineuritis—should be regarded as secondary phenomena. The sympathetic system is also intact. The histologic changes of the skin are very differently described, but they usually include sclerosis of the smaller arteries, endarteritis, with obliteration in places, and increase in the elastic fibers below the papillary layer of the skin, extending into the subcutaneous tissues. In the muscles, bones and ligaments changes of simple atrophic or partly secondary inflammatory nature are found. These changes are described by all authors and are found in all cases that come to autopsy. The analogy to myxedema, of which scleroderma is the skin antithesis, suggests strongly a thyroid disease. Lewin and Heller [11] and others regard the disease as depending on unknown changes in the trophic centers. The pathologic and anatomic observations do not conflict with the impression that acute infection or prolonged chronic infection or intoxication with resulting secondary changes may account for the various phenomena making up the so-called syndrome of scleroderma. The fact that the changes in the skin, muscles, nerves, bones and joints do not differ in essentials from those associated with chronic infections of these systems adds considerable weight to this etiologic point of view, and the careful analysis of cases of scleroderma in many instances bears this out. Hoffman and Rosenthal's case, which was observed from the onset to the complete development of a sclerodermal syndrome, stimulated my interest in this point of view. A dermatomyositis with an included neuritis or perineuritis of an infectious nature certainly would account for the observations in many cases.[12]

REPORT OF CASES

The series here reported comprise cases of dermatomyositis, dermatoneuromyositis and the sclerodermal syndrome, and only positive features of the histories and results of examination are given.

CASE 1.—M. S., a man, aged 41, with no occupation, entered the hospital because of swollen legs and a cough. Ten days previous to admission he had noticed edema of the legs, and had had a sore throat, chills, a cough and watery stools. His past history was unimportant.

Examination showed a slight elevation of temperature for two days. The pharynx was red and the tonsils were large. There was a slight enlargement of the left ventricle. There were diffuse rhonchi over the chest. There was some aortic widening, and the aortic second sound was accentuated. The legs, from the knees down, were very red and felt hot; the skin was edematous and pitted on

11. Lewin, George; and Heller, Julius: Die Sklerodermie, Berlin, A. Hirschwald, 1895.

12. Atwater (footnote 7). Wise (footnote 8). Hoover (footnote 3).

pressure. There was considerable pain on pressure about the external malleoli. There was some induration of the skin of both ankles and fixation of the skin to the subcutaneous tissues of the legs. Deep pressure over the muscles of the legs caused severe pain. The Wassermann reaction was negative. The basal metabolic rate was increased 20 per cent before the patient's discharge.

A course of salicylates was started, and the pain, tenderness, erythema and temperature subsided, but the induration and fixation remained and were present at the time of discharge several weeks later.

CASE 2.—M. E., a woman, aged 54, a housewife, had noticed swelling of the ankles and legs six months before admission to the hospital. Three months later, the swelling increased and at times the skin became inflamed and was accompanied by considerable pain and throbbing. The past history disclosed fainting spells and shortness of breath on exertion, which had been present for years.

Both legs were red, edematous and very tender to touch and pressure from the knees to and including the ankles. The skin, subcutaneous tissues and muscles were adherent. By auscultation marked crepitus could be heard over the muscles when they were activated. At this time the heart disclosed no murmurs; the blood pressure was 110 systolic and 75 diastolic.

The urine contained a faint trace of albumin. Roentgen examination of the tibias showed some osteoporosis. The Wassermann reaction of the blood was negative; the white blood cell count was 15,000.

The process subsided almost entirely in the course of several days without any therapy other than rest in bed. After she left the hospital, improvement continued but the patient later was again admitted to the hospital because of a myocardial insufficiency; there the edema of the leg developed, but no redness or tenderness was present. She died of myocardial insufficiency, the result of chronic rheumatic myocarditis.

CASE 3.—M. D., a woman, aged 24, a housewife, entered the hospital because of fainting spells. After the birth of a child, thirteen months previously, the patient had had "rheumatism" (swelling, weakness and pain of the arms and of the arm and shoulder joints). From the age of 13 to 18 she had been subject to frequent attacks of chorea and had had palpitation and precordial pain for years. She had had pneumonia five times and influenza in 1918. Ever since the chorea, she had been pale and also had noticed tingling at the finger tips. Her family history showed that her father had had rheumatism all his life.

Examination showed the following positive observations: The tonsils were large; the thyroid was symmetrically enlarged, and there was a loud venous hum over the jugulars; there was an impulse and impact over the conus, and a systolic and presystolic murmur over the apex of the heart; there was anemia; red blood cells, 3,616,000; hemoglobin, 50 per cent; the blood plasma was not icteric.

One week after the patient's admission, edema developed over the left pretibial region, with redness, heat and tenderness over the same region and also about the left ankle and left knee. There was a definite hyperesthesia of the skin of this area. The muscles of the peroneal group were very tender to pressure and the muscles of the calf were very painful when stretched, and tender to light pressure. The skin over the ankle and left knee joint was also very tender, but motion of the joints was not painful. After several days of salicylate therapy the acute process subsided.

CASE 4.—B. G., a man, aged 35, had had pharyngitis with fever, chills, headache and nausea two weeks before admission to the hospital. Later he had noticed pain and swelling about the right ankle. Four days after the pain in the ankle

appeared, he noticed a painful red swelling on the medial side of the thigh and subcutaneous hemorrhages at these points. There was a history of malaria in childhood.

Examination showed an area of redness, heat and induration on the medial surface of the right thigh. There was induration which extended down and involved the muscles. There was also induration, heat, swelling and tenderness over the right ankle joint, especially on the medial side, and movement of the joint caused some pain; some crepitus could be noticed on moving the joint. Both hands had old subcutaneous hemorrhages on the finger tips. The spleen was palpable. The temperature on the patient's admission was 38.5 C. (101.3 F.).

He was given a course of cinchophen up to the point of toxicity, and the acute processes subsided; the fixation and induration, however, did not.

Comment.—The four cases just mentioned showed skin changes as the result of an acute dermatomyositis, indistinguishable from the skin changes seen in scleroderma, and then occurred in the presence of, or as a consequence of, rheumatic infection. At the time of discharge from the hospital, the patients had definite skin residuals.

The following cases, with the exception of case 8, showed varying degrees of involvement of the skin, muscle, nerve and bone. All the patients gave a definite history of rheumatic infection, either preceding or accompanying the onset of the symptoms; they were considered as cases of dermatoneuromyositis presenting a typical scleroderma. Although they were not seen until typical scleroderma had developed, the past history and early description of the difficulty led to the belief that the infections were similar to those seen in the acute cases, but were chronic extensive processes of the same nature.

In case 8, however, the patient gave no history in any way suggestive of a rheumatic infection; apparently the process had started insidiously early in life.

Sclerodactylia may suggest a syringomyelia in some cases, but the absence of typical sensory and other characteristic changes is sufficient to differentiate the condition. Leontjewa called attention to the fact that all cases do not show the usual onset or progress of the condition, and that there are cases of trophic and angiotrophic disturbances which result in the typical picture and in different variations of it.

Case 5.—M. S., a woman, aged 45, a housewife, entered the hospital complaining of stiffness of the hands. Ten years previously she had had severe "rheumatism," and she had had some slight recurrent attacks since. About five years later, she had had attacks of redness, swelling and pain about the fingers and some stiffness in the hands and pain in the wrist. Since then, the stiffness in the hands had been growing worse, and she also had acute swellings at the finger tips. In the last two years she had noticed that the skin over the face had been tighter than usual, and since she first noticed the stiffness of the hands she had also noticed a gradual brownish-red discoloration of the overlying skin. Except for an attack of influenza in 1918, there were no other antecedent illnesses. She had had six children.

Examination showed the skin of the face, particularly about the mouth, to be adherent to the underlying tissues. The mouth appeared puckered and the skin of the face was dry, felt thickened and had a brownish-red (weather-beaten) appearance. There was pain in the left trapezius muscle. The heart and lungs were normal. There was an area of unblanched skin extending from the midline at the umbilicus to the costal margin and the anterior axillary line. The skin over the hands was thick and adherent to the underlying tissue. Movement of the fingers and wrist was much restricted. There was an inflamed fluctuant area at the tip of the right thumb which was very painful and sensitive. About the tendon sheaths there was some thickening of tissue, particularly of those of the extensors of the left hand, and marked crepitation could be felt when the patient moved the fingers. The skin over the forearms was thick and adherent and was

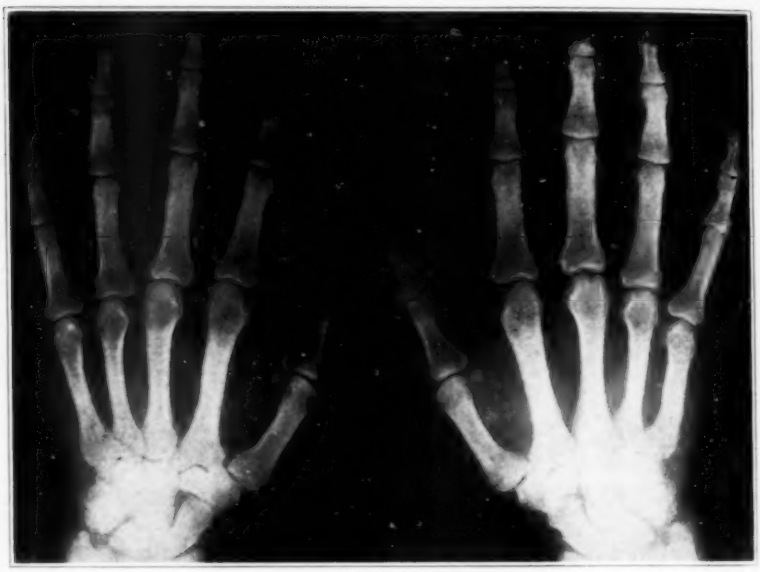

Fig. 1.—Atrophy and destruction of the terminal phalanges of all the fingers.

discolored a brownish red, the pigmentation being most marked on the extensor surfaces. The thickening of the skin extended above the elbow but was not as adherent; the patient complained of no disability above the elbows. The lower extremities were normal. The temperature at intervals rose to 38 C. (100.4 F.), and accompanying the rise in temperature there was much pain. Roentgen examination of the hands showed the distal ends of the phalanges of several digits to be absent (fig. 1). The changes were characteristic of scleroderma.

Case 6.—C. J., a woman, aged 39, a housewife, entered the hospital because of deformity of the hands and arms, loss of weight and indigestion. These symptoms had begun gradually about six years previously. She had noticed that the fingers felt hard; the skin over them had become tight, and later a gradual contraction of the hands and arms developed. The same process was noticed in the legs and feet, but to a lesser degree. She had also complained of pains in the shoulders, knees and hips, with drawing sensations in the extremities. The indigestion usually occurred after she had eaten greasy foods.

At the age of 15, the patient had had a severe attack of rheumatic fever, with redness, heat, pain and tenderness about the joints.

Examination showed that the skin over the extremities and face was drawn tense; it was glazed in appearance and pigmented brown in areas over the distal portions of the extremities. The skin was dry, and coarse hairs were still sparsely present over the lower limbs. The skin over the extremities was adherent to the underlying tissues. The face had a masklike expression; the lips were puckered so that furrows radiated from them. The skin of the neck was shrunken so that the sternocleidomastoids stood out. The skin over the arms was also tense, the hands being held in a claw posture. The fingers could not be extended nor could the hand or forearms be pronated or supinated. The arms could not be extended at the elbows; flexion of the hands was also much impeded. The legs could not be completely extended at the knees, and motion at the ankles was also markedly restricted in all directions. The digits, knee and finger joints were enlarged. There was marked contraction of the platysma, so that the neck could not be extended.

The contractures of the muscles and fixation of the skin and joints suggested a long-standing dermatoneuromyositis, more than simply scleroderma. The laboratory studies showed a basal metabolic rate of 25 per cent increase. Examination of the spinal fluid gave normal results.

CASE 7.—N. F., a woman, aged 35, a telephone operator, entered the hospital because of a tumor in the left breast. Three months before admission to the hospital she had had an illness in which the extremities had become swollen and the entire skin very red. She had been bedridden since that time and had grown very weak; the skin had become so contracted that it was difficult for her to move the arms, legs or neck. The condition began in both arms and legs at the same time, with marked redness and weakness, followed by swelling and then contraction of the skin.

Examination showed a masklike expression of the face. The skin everywhere was red, was firmly attached to the underlying tissues and lacked elasticity. There was a contracture of the arms and legs, so that she was unable to abduct either the thighs or the legs, and there was also marked contracture of the muscles of the neck so that motion was also markedly limited. The feet and ankles showed considerable pitting on pressure.

CASE 8.—T. H., a school-girl, aged 15, came to the hospital because of stiff fingers which she had first noticed at the age of 7. There was no history of an acute infection or anything suggestive of a rheumatic infection. She had had the common diseases of childhood. Her parents thought that she had always had the stiffness of the fingers, but she was little incapacitated by it. No pain, tenderness or swelling had been present. During cold weather the skin would become definitely blue.

Examination showed the skin of the face and over the whole body to have a smooth, glistening appearance. The palpebral fissures were narrowed, due to contraction of the lids, which were tense when palpated. The sternocleidomastoid muscles felt indurated and tense, the right more so than the left. The muscles throughout were of firmer consistency than one would expect in a person of this age. The skin everywhere was thickened and bound down to the subcutaneous tissue. The recti abdominis muscles were shortened and had lost much of their extensibility; in fact, extensibility of all the muscles was lessened. The laboratory observations were normal, one basal metabolic rate being 14 per cent decreased. Roentgen examination of the skull gave negative results. Roentgen examination

of both hands showed a slight general osteoporosis, and the distal phalanges of the right fifth finger showed some destruction of the distal end (fig. 2).

Case 9.—J. E., a woman, aged 35, a housewife, came to the hospital complaining of stiffness of the mouth and lips, dry skin and redness and numbness of the fingers. The onset had occurred about five years previously. Preceding that

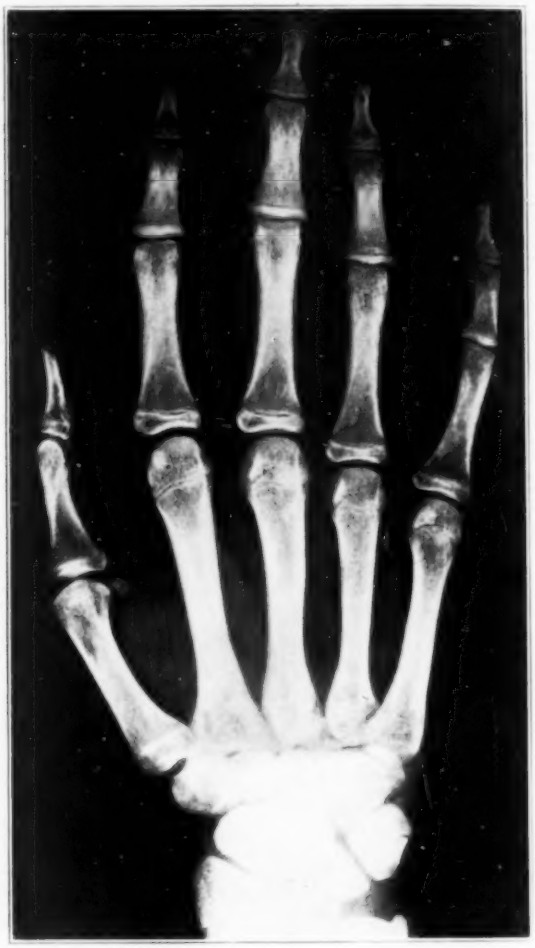

Fig. 2.—Generalized osteoporosis and destruction of the distal phalanx of the right fifth finger.

time, she had had an attack of severe pain and soreness about the back of the neck, both shoulders and in the upper arms, which she spoke of as severe rheumatism. Following this she noticed numbness in the fingers, particularly in cold air or water. This gradually involved all of the fingers, with redness and numbness of most of the hand. On exposure to a cold medium, the skin would frequently blanch. Subsequently, she noticed stiffness and dryness of the lips and that the mouth and tongue did not "feel right."

Examination showed the skin over the fingers and hands to be red, thin and glossy. Over the fingers it was difficult to pinch the skin into folds. The skin over the hands, wrists and part of the forearms was dry, coarse and somewhat fixed to the underlying tissue. The nose had a pinched appearance. The mouth was puckered and could not be opened widely; the mucosa of the lips, mouth and tongue was thin. There was also atrophy of the right side of the tongue. The right shoulder girdle muscles were smaller than the left, but there was no marked weakness.

About three months after the first examination, she had acute nasopharyngitis and bronchitis, for which she was confined to bed for a week. Accompanying and following this there was an accentuation of the complaints and a return of the pain across the shoulders, in the muscles of the arm and in the left wrist joints.

Case 10.—E. F., a woman, aged 35, about one and a half years before admission to the hospital had begun to notice a swelling over the palmar surface of the metacarpophalangeal joints of the right hand. There was no pain and no fever that the patient was aware of. The same swelling then appeared over the palmar surface of the joints of the left hand. This was followed by gradually increasing stiffness of the fingers and hands. Four months after the onset in the hands, similar swellings appeared over both feet and ascended as high as the knees, this likewise being followed by stiffness of the legs and feet. About six months after the onset, the swelling disappeared and was followed by an increasing tightness of the skin of the arms and legs, extending as high as the elbows and as high as the knees. With this there was considerable aching pain in the shoulders and the upper part of the back. The tightness and stiffness of the extremities increased gradually and she had considerable pain in the joints after the onset of the disorder.

Examination showed the skin to be tense, tight, dry and glistening over the extremities and adherent to the underlying tissues and muscles. The muscles of the legs and forearm were atrophied and of a brawny consistency. There was contracture of the fingers and arm muscles in a semiflexion at the elbows and phalangeal joints. The face of the skin and body in general was less involved, the face being pinched in appearance, with puckering about the mouth and thinning of the nose.

Roentgen examination of the bones and joints gave negative results, the deformity being due to muscular contractures. Laboratory tests, including Wassermann tests of the blood and spinal fluid, gave essentially negative results. The basal metabolic rate was minus 6.

Case 11.—E. M., a woman, aged 64, was admitted to the hospital because of stiffness of the face, jaws, arms and legs and puffiness of the eyelids and feet. About one year previously, she had noticed that the ankles had begun to swell. This had continued up to the time of examination. About six months previous to admission, the patient had begun to have severe pain in and about the jaws and neck. At first this was thought to be due to a tooth, and the tooth was extracted but there was no relief from the pain. Following the appearance of the pain in the face, the patient began to notice a feeling of stiffness of the face and jaws, so much so that she had difficulty in eating and chewing. At about the same time she began to have considerable pain in the shoulder regions and in the back as low as the waist. The hands and legs then began to be stiff and she began to have considerable aching pain in both arms. It became impossible for her to extend or flex the fingers completely or to stand erect. Because of this she had great difficulty in caring for herself. Accompanying the feeling of stiffness and pain,

the patient began to notice marked changes in the character of the skin over the face, arms and legs, the skin becoming hard, tense and reddish brown. Laughing and talking were difficult because of immobility of the face.

In her past history there was noted an attack of severe pain and aching about both knee joints lasting for six months. She had had the upper teeth extracted, and after three or four months the pains had disappeared. The family and marital history were unimportant.

Examination showed a dusky red-brown discoloration of the skin in general; it was most marked over the face and forearms. The eyelids were puffed. Everywhere the skin was tense and firm, and it was fixed to the underlying tissues; it had a brawny, indurated feel. The mouth was puckered and the nose pinched. There was some edema of the skin in the legs and feet. The heart and lungs were normal. The gait was slow, and the associated movements were lacking; the patient partly stopped because of inability to straighten the legs. There was a symmetrical atrophy of the muscles of the forearms and hands, with contracture and limitation of motion at the elbow, wrist and finger joints. Sensory examination showed a loss of vibratory sense in the lower extremities and pelvis; it was diminished in the spine, but acute in the upper extremities. Temperature and touch were diminished progressively from the elbows and knees downward. The reflexes were equal and present. Examination of the urine gave negative results.

Late in the course of the illness a typical herpes zoster eruption developed over the entire left arm, the area of herpetic lesions conforming almost identically with the area of marked skin change in that extremity.

In case 12, the onset is suggestive of Raynaud's disease, and belongs to the class that Cassirer has described as a scleroderma in which Raynaud's disease supervenes.

Case 12.—A. B., a man, aged 45, a carpenter, came to the hospital because of pain and coldness of the hands and ulceration of the finger-tips and elbows. In cold weather the pain was frequently so severe that he would not leave the house, while during warm or hot weather he noticed no discomfort. About a year before admission, he had noticed small ulcerations over the finger-tips. Preceding the appearance of the ulcers he had had a sensation of needle pricks over the area, and at intervals since then had had several ulcers over the finger-tips of both hands, which alternately healed and reappeared. About five months previously, he had noticed a feeling of tightness of the skin over the hands.

Three years previously, he had had attacks of aching pain about the arms; two years before he had had an attack of pain in both shoulders, in the muscles of the upper arm and in the legs, which had lasted for several weeks and had then disappeared. In the previous two years he had noticed diminishing strength and felt exhausted after the slightest exertion. He had lost about 15 pounds (6.8 Kg.) in weight.

Examination showed a dark complexion. The skin over the abdomen was a diffuse brown; the forearms were also brown over the extensor surfaces and about the wrist and finger joints. This brown pigmentation was not uniform, being interrupted by white patches. The skin over the face was not as freely movable as normal. The face was stiff; the mouth could not be opened widely, and the temporalis muscles were unusually firm. The recti abdominis muscles felt boardlike, and there was limited extension of the spine because of the fixation of the recti. There was lack of flexibility of both wrists; pronation of the hands and wrists was difficult, the skin being bound down to the underlying tissues; the

muscles of the forearm were hardened and of a fibrous consistency. Over the lower extremities the soleus and gastrocnemius showed a disparity on the two sides, the soleus being firmer and harder on the right. The feet and hands felt cold, and on being exposed to cool air became very blue. The tips of several of the fingers showed a marked thickening of the skin, with indolent, healed-over ulcers, which were very painful on pressure. The skin over the elbows showed the same thing. There were no other unusual observations, except hyperesthesia to cold over the involved parts; there were no other sensory disturbances.

The laboratory studies showed normal spinal fluid, and the Wassermann reaction of the blood was negative. The basal metabolic rate was 10 per cent and

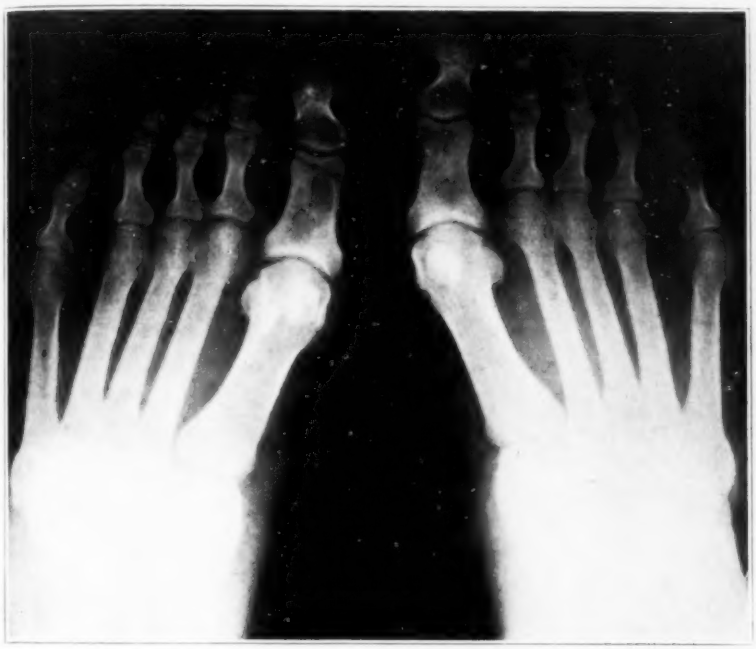

Fig. 3.—In addition to the same atrophy and destruction of the fingers as in patient in figure 1, this patient also showed similar changes in the toes.

4 per cent decreased, and a dextrose tolerance test showed a normal curve. Roentgen examination showed an atrophy and destruction of the terminal phalanges of all the fingers and toes. Roentgen examination of the skull and other bones showed no changes (fig. 3).

Comment.—In case 12 there was, as in the previous cases, also a suggestive history of rheumatic infection preceding the onset of the vasomotor and trophic phenomena. Cassirer made a division of these cases with marked vasomotor phenomena into: (1) instances of Raynaud's disease in the late stages of which trophic skin changes appear; (2) cases in which the condition starts with marked vasomotor changes which are gradually succeeded by a typical scleroderma, that is

not limited to the parts affected with the vasomotor phenomena; (3) rare cases in which, in a typical scleroderma, symptoms of Raynaud's disease with gangrene supervene.

SUMMARY

According to the observations made in the study of this series of cases, the most striking features are: 1. Rheumatic infection either precedes or accompanies the appearance of the scleroderma syndrome. 2. The concomitant skin, muscle, nerve, bone and other trophic changes seen in typical scleroderma are the result of rheumatic toxins in many instances. 3. The severest types, those in which there are concomitant skin, muscle and nerve changes, result in a classic sclerodermal syndrome, whereas in those in which skin and muscle changes only are associated trophic disturbances do not develop because of the absence of nerve lesions.

STUDIES IN EPILEPSY

XI. THE CALCIUM CONTENT OF THE BLOOD AND OF THE SPINAL FLUID *

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Among the convulsive disorders there is one, tetany, in which certain of the elements in the etiology are known. Tetany occurs in persons in whom there is loss or decreased function of the parathyroid glands, or it may arise in normal persons following prolonged hyperpnea. It is accompanied by a decrease in the ionized calcium and by an increase in the alkalinity of body fluids.

The question at once arises whether tetany and other convulsive disorders have features in common, and, if so, whether this holds therapeutic implications for patients commonly classed as epileptic. In pursuing this inquiry one wishes to know how commonly tetany and epilepsy are associated, whether distinctive symptoms or signs that are latent in one can be elicited in the other, whether measures that are effective in either preventing or producing tetany are of any influence in epilepsy, and whether laboratory examinations of epileptic patients reveal conditions suggestive of tetany. Lennox and Cobb [1] in their book presented the available information, so that there is no need to discuss the literature in detail in this paper.

It may be remarked that occasionally a person with tetany will also have attacks of generalized convulsions with loss of consciousness. Redlich [2] collected reports of seventy-two such cases. The association was clearest in the group in which the parathyroid glands had been removed at operation. Again, infants who have spasmophilia may later in life become epileptic or mentally deteriorated. This was true in sixteen of twenty-nine cases followed by Thom.[3] Conversely, in per-

* Submitted for publication, July 21, 1930.

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1. Lennox, W. G., and Cobb, S.: Epilepsy, Baltimore, Williams & Wilkins Company, 1928.

2. Redlich, E.: Tetanie und Epilepsie, Monatschr. f. Psychiat. u. Neurol. **30**:439, 1911.

3. Thom, D. A.: The Relation Between Infantile Convulsions and Chronic Convulsive Disorders of Later Life, Arch. Neurol. & Psychiat. **11**:664 (June) 1924.

sons with chronic epilepsy tetany may develop. These isolated case histories are interesting, but do not prove that the presence of tetany and of generalized convulsions in the same patient is more than a coincidence. Statistical studies would be needed to prove that attacks of grand or petit mal are more common in persons subject to tetany than in the general sick population.

As for clinical or pathologic evidence of tetany in persons called epileptic, Römer [4] found increased irritability of peripheral nerves in 16 per cent of 250 patients tested. Other scattered reports dealing either with increased nervous irritability or with disease of the parathyroid glands in epileptic patients are unconvincing because of the meagerness of the material. Likewise, treatment for epilepsy by means of calcium has been disappointing.

One is impressed with the negative quality of the evidence so far presented. Certainly in the two conditions one does not find the same portions of the nervous system involved. In tetany there is disturbed function of the peripheral nerves, and in epilepsy disturbed function of the central nervous tissues. We may, however, inquire whether there are not underlying conditions which account for the increased nervous irritability present in both tetany and epilepsy.

In support of this hypothesis there are experimental demonstrations that certain physicochemical changes in body fluids which produce tetany in parathyroidectomized animals tend to precipitate seizures in epileptic patients; such conditions are anoxemia, changes in ionic equilibrium and alkalosis. Apparently these conditions cause an increase in nervous irritability which manifests itself in various ways, depending on underlying but unknown individual tendencies. The importance of these invisible factors is illustrated by the following facts. One of the mysteries of epilepsy is the occurrence of seizures at times when the physical and chemical processes of the body are presumably in a state of equilibrium, such as during sleep. Tetany is equally mysterious in that parathyroidectomized animals have alternate periods of tetany and apparent well-being, though the pathologic process is presumably constantly present.

In view of the demonstration that alterations in the chemistry of body fluids, especially as regards acid-base balance and oxygenation of tissues, influence both tetany and epilepsy, it is important to determine whether persons with epilepsy show evidence of abnormality in their calcium metabolism. This could be done most convincingly not only by determination of the level of calcium in body fluids, but also by measurement of the calcium balance. It is possible, as in hyper-

4. Römer, Karl: Das Erksche Phänomen bei Epilepsie, Ztschr. f. d. ges. Neurol. u. Psychiat. **84**:1, 1923.

thyroidism, for the calcium level to be normal but the excretion excessive. Such detailed study of a group of epileptic patients has not been reported.

There have been a number of reports of the calcium values in the blood serum, but most of these deal with a few patients only. Bigwood [5] reported abnormally low measurements occurring in periods before seizures. His values were calculated for ionized calcium. Also the observed differences were hardly greater than the experimental error. Patterson [6] measured the amounts in the blood (serum?) of 100 patients, using the method of de Waard. The values lay between 9.65 and 16.97 mg. per hundred cubic centimeters of blood; 12 per cent were above 12.9 mg. Therefore, many of the measurements were abnormally high, due possibly to the method of analysis used.

In gathering data, one faces the difficulty that the decrease in calcium in tetany is principally in the ionized or diffusible portion, which one cannot measure directly. The usual methods of analysis measure only the total calcium. There is good evidence that the calcium of the spinal fluid is in the diffusible form. Therefore, by measuring both serum and spinal fluid calcium, one can gain an idea as to the level of ionized calcium in body fluids and the ratio of ionized to total calcium.

With this thought in mind, Marrack and Thacker [7] measured spinal fluid calcium in a small group of epileptic patients. Hamilton,[8] in seventeen patients, and Osnato and Killian,[9] in nineteen patients, measured serum as well as spinal fluid calcium. These various authors obtained results that were within the limits of normal. The last mentioned authors, however, begged the question by stating that patients who gave evidence of tetany on examination of the blood were excluded from the series. Naturally, only normal results could be expected of the patients that remained.

MATERIALS AND METHODS

The patients were in the wards or were outpatients in the neurological service of the Boston City Hospital. They were of various ages, of both sexes and unselected; i. e., all patients with seizures as a presenting symptom were included. Almost all these cases were of

5. Bigwood, E. J.: L'équilibre physico-chémique du sang dans l'épilepsie. L'ion calcium, Ann. de méd. **15**:24 and 119, 1924.

6. Patterson, H. A.: Some Observations on Blood Calcium Content in Epilepsy, Proc. A. Research in Nerv. & Ment. Dis., to be published.

7. Marrack, J., and Thacker, G.: State of Calcium in Body Fluids, Biochem. J. **20**:580, 1926.

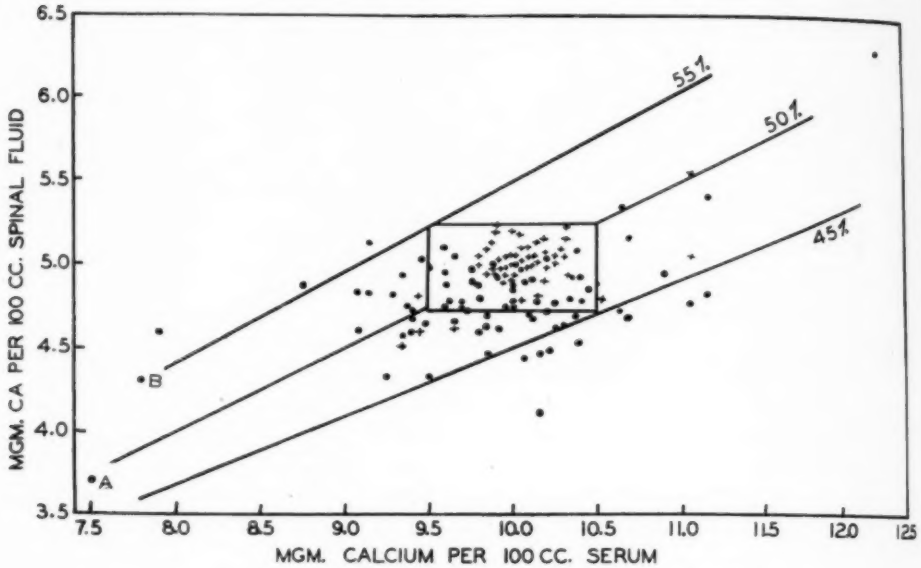
8. Hamilton, B.: Comparison of Concentrations of Inorganic Substances in Serum and Spinal Fluid, J. Biol. Chem. **65**:101, 1925.

9. Osnato, M., and Killian, J. A.: Biochemical Studies of the Blood and Spinal Fluid in Epilepsy, Brain **50**:581, 1927.

the group commonly called essential epilepsy. In a few instances adequate cause for the seizures (injury or tumor of the brain, etc.) could be demonstrated. Results of chemical examinations in these patients did not differ from the general results. One exception to this statement will be mentioned later. As a rule, the patients were not fasting. Blood was taken from an arm vein immediately after spinal fluid was withdrawn. Clark's [10] modification of the Kramer-Tisdall method was used.

RESULTS

Seventy-seven patients were examined. The results are shown graphically in the accompanying chart. Each dot represents the value



Measurements of calcium in blood serum and in spinal fluid. The abscissa represents milligrams of calcium per hundred cubic centimeters of serum; the ordinate represents the same for spinal fluid. The dots show measurements for 77 epileptic patients examined by us, and the crosses, those for 51 nonepileptic patients examined by Dr. Walter Bauer. The diagonal lines mark the points at which the spinal fluid values are, respectively, 45, 50 and 55 per cent of those of the serum. The rectangle shows rather strict limits for the normal. The dots marked *A* and *B* are measurements for one patient who had tetany; *A*, was made before, and *B* during treatment.

of the calcium in both serum and spinal fluid. The diagonal lines mark values of the spinal fluid which are respectively 55, 50 and 45 per cent of those of the serum. The small rectangle represents

10. Clark, E. P., and Collip, J. B.: A Study of the Tisdall Method for the Determination of Blood Serum Calcium with Suggested Modification, J. Biol. Chem. **63**:461, 1925.

strict limits of normal: between 9.5 and 10.5 mg. per hundred cubic centimeters of serum and between 4.75 and 5.25 mg. per hundred cubic centimeters of spinal fluid.

In any investigation of this kind, the question of normal values is all-important. The safest way is for one to gather his own normal measurements, using the same methods and materials as were used for patients. Dr. Walter Bauer gave us the data that he and Miss Houghton had collected from a group of patients at the Massachusetts General Hospital. They used both the Clark and the Fiske method. Fifty-one patients with miscellaneous neurologic conditions were examined, those with evidence of tetany or glandular disease being excluded. Dr. Bauer's measurements are represented in the chart by crosses. The average measurements for these two groups of patients were: for serum calcium: the epileptic group, 9.79 mg., and the nonepileptic group, 10.06 mg. per hundred cubic centimeters; for spinal fluid: the epileptic group, 4.75 mg., and the nonepileptic group, 5.19 mg. per hundred cubic centimeters. This agreement is close.

Inspection of the chart shows that there is a much wider scattering of dots representing epileptic patients than of crosses representing nonepileptic patients. Sixty per cent of the dots and only 14 per cent of the crosses lie outside the rectangle. As regards measurements for serum (indicated on the abscissa) 22 per cent of the epileptic patients had low, and 12 per cent had high, values; 34 per cent, therefore, were abnormal. Twelve per cent of Dr. Bauer's patients had abnormal serum values.

As for spinal fluid, 34 per cent of the epileptic patients gave values below, and 4 per cent values above, normal. Therefore 38 per cent were abnormal, as against 8 per cent of the nonepileptic patients. Thirteen per cent of the epileptic patients had a high value in either serum or spinal fluid, against 6 per cent of nonepileptic patients. Forty-three per cent of the patients with epilepsy had a low value in either the blood or the spinal fluid, against 8 per cent of nonepileptic patients. Analyzed in this way, it appears that in these epileptic patients there was an unusual scattering of values, the greatest variation from normal being found in low percentage of calcium in the spinal fluid.

As stated previously, we are more interested in the ratio of ionized to total calcium than in the absolute measurements. Therefore, we examined with special interest the relationship of the concentration of calcium in the spinal fluid to that in serum. If one accepts the normal measurements for calcium which have been named, the normal ratio lies between 45 and 55 per cent. Dr. Bauer's measurements all lay in this zone. In contrast, three (4 per cent) of the epileptic patients showed ratios above, and nine (12 per cent) ratios below, normal. Therefore 16 per cent were abnormal. Although the single measurements were scattered to an unusual degree, the average ratio for all

seventy-seven patients was 48 per cent, as against an average ratio of 51 per cent for the nonepileptic group. The average ratio for epileptic patients was therefore lower by 6 per cent, not a striking difference.

In the lower left-hand corner of the chart are two dots labeled *A* and *B*. These are measurements for a patient with tetany and convulsions. *A* indicates calcium values before, and *B* the values during, treatment. The case report of this patient is worth recording.

REPORT OF A CASE

History.—The patient was a man, aged 55, a Russian Jewish shoemaker, who entered the hospital in April, 1930, because of seizures. The diagnosis on entrance was epilepsy. The family history was negative as to seizures; the past history was not significant. For two months, the patient had been having grand mal convulsions with associated mental deterioration.

Physical Examination.—There were a coarse tremor of the face, tongue, hands and legs, retinal hemorrhages of the left eye without significant arterial changes, hyperactive tendon reflexes, slight incoordination and a positive Romberg sign. There was a bilateral Chvostek sign. Mechanical stimulation of the external popliteal, ulnar and median nerves caused a quick contraction of the appropriate muscles. Carpopedal spasm occurred one minute after the application of a tourniquet and after one and a half minutes of overventilation. A thumb flexor electrical response was obtained at 1.5 and a facial response at 2.5 ma. Five lumbar punctures were performed. In the three in which satisfactory pressure readings were obtained, the initial pressures were 245, 175 and 210 mm. of spinal fluid, respectively. The fluids were normal as to cell count, protein, Wassermann and colloidal gold tests. Roentgenograms of the skull were normal. Encephalograms showed the left ventricle to be smaller than the right. The blood pressure was normal; the temperature was subnormal (from 96 to 98.6 F.).

Course.—In the ward the patient became noisy, talked incoherently, and apparently was not in touch with his surroundings or aware of current events. He had several isolated generalized convulsions of the sort usually associated with epilepsy. When tetany appeared, several days after admission, he was given parathyroid extract subcutaneously (60 units a day), viosterol (4 cc. a day) and calcium lactate (6 Gm. a day). After twenty-four hours, there was improvement in the condition and in three or four days he was out of bed and conversing fairly intelligently. During the next two weeks, seizures did not recur, but he continued to show the emotional instability, euphoria, and somewhat clouded mental state that were present on his entrance to the hospital. The signs of tetany which were readily elicited before could be obtained only with difficulty.

Before the institution of treatment, the spinal fluid calcium measured 3.1 mg. and the serum calcium 7.5 mg. After a week of treatment, these measurements were 4.3 and 7.8 mg. At the end of another week, the serum values were still low, 7.4 mg. Before treatment, the carbon dioxide content of the arterial blood was 38.2 per cent by volume, the oxygen content 18.6 per cent by volume, the oxygen capacity 21.2 per cent by volume and the oxygen saturation 87.5 per cent. The oxygen saturation of blood from the femoral and internal jugular veins was the same, 50.7 per cent. After two weeks of treatment, the carbon dioxide content of the arterial blood was higher (45.3 per cent by volume), the oxygen content and capacity were lower (16.6 and 17.9 per cent by volume) and the oxygen

saturation higher (93 per cent). The oxygen saturation of venous blood from the internal jugular and femoral veins had increased to 53 and 62 per cent, respectively.

Comment.—This patient interested us because he was the first in a series of hundreds of epileptic patients whom we have studied who proved to have tetany. "Idiopathic," or "occupation" tetany, however, is frequently found in European cities. Frankl-Hochwart [11] encountered 528 cases in the 25 years preceding 1905; 47 per cent of these occurred in the months of March and April; 42 per cent of the patients were cobblers and 22 per cent tailors; 81 per cent were men. Tetany may occur in epidemic form. McCarrison [12] has described an epidemic in Himalayan valleys. Infection, food deficiency and ergot poisoning have been mentioned as etiologic factors. In our patient, we could obtain a history of none of these. We believed that he had Alzheimer's disease as the underlying pathologic process for the mental changes and convulsions and that the tetany was a complication.

If one regards epilepsy as a symptom, one should not expect any single abnormality to be present in all epileptic patients. A distinct abnormality either in the calcium content of the blood or spinal fluid or in the ratio of serum to spinal fluid calcium, if present in any of the patients, may be of etiologic significance.

The deviation from normal in the average measurements of calcium in the group reported is not great enough to attract attention. The abnormal distribution of measurements is much like the scattering of results in other physiologic and chemical measurements that have been made, such as basal metabolic rates, plasma bicarbonate, serum fibrin, blood sugar curves, spinal fluid chlorides, etc. These accumulated data suggest slight abnormalities in endocrine function, in ion equilibrium, or in the circulation of patients. These abnormalities may be of contributory importance in the precipitation of seizures.

SUMMARY

Measurements of serum and spinal fluid calcium were made in seventy-seven unselected patients who were subject to recurring convulsions. Average concentrations and average spinal fluid-serum ratios were within normal limits. As compared with measurements in nonepileptic patinets there was an abnormally wide distribution of values, with a special tendency toward low spinal fluid measurements. In one eighth of the patients the ratio of spinal fluid to serum calcium was less than 45 per cent. Only one of the patients had clinical tetany.

11. von Frankl-Hochwart: Die Tetanie der Erwachsenen, Vienna, A. Hölder, 1907; quoted in Falta-Myers: Endocrine Diseases, Philadelphia, P. Blakiston's Son & Company, 1923, p. 218.

12. McCarrison, R.: Epidemic Tetany in Gilgit Valley, Lancet **1**:1575 (June 10) 1911.

MULTIPLE SCLEROSIS AND CEREBELLOPONTILE ANGLE TUMOR

DIFFERENTIAL DIAGNOSIS *

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In 1920, our attention was directed to this subject by the postmortem observations in the case of a patient, aged 45, in which a diagnosis of multiple sclerosis had been made. This patient had been examined by competent observers, who had found bilateral nystagmus, marked speech defect, cerebellar incoordination, more marked on one side than the other, and general hyperreflexia. In particular there were no symptoms of a tumor of the brain, such as headache and vomiting, and examination of the eyegrounds gave no indication of any increase of intracranial pressure. The deafness was thought to be accounted for by an old disease of the middle ear. To the surprise of all concerned, at autopsy a cerebellopontile angle tumor was disclosed. Since, we have been on the alert for cases of this sort, and four cases of this type have come under our observation; in three an operation has been performed.

In a review of the literature on the differential diagnosis between tumors of the brain and multiple sclerosis, and in particular between tumors located in the cerebellopontile angle and multiple sclerosis, we have found a relatively small number of contributions. Eiselsberg and Ranzi [1] reported a case without choking of the disks that was apparently typical of an angle lesion; at the operation there was failure to find the tumor. At death, eleven months later, the typical pathologic changes of multiple sclerosis were found, with a circumscribed serous meningitis. Oppenheim [2] described a similar case, also without changes in the eyegrounds. This case eventually proved to be a typical multiple sclerosis. In 1921, Marburg [3] described two cases of suspected angle tumor. Choked disks were present in both cases. After long observation these

* Submitted for publication, June 12, 1930.

* From Temple University Medical School, University of Buffalo Medical School and the Laboratory of Neuropathology, Philadelphia General Hospital.

1. Eiselsberg and Ranzi: Arch. f. klin. Chir. **102**:311 (case 99) 1913.
2. Oppenheim, H.: Deutsche Ztschr. f. Nervenh. **52**:168, 1914.
3. Marburg, O.: Deutsche Ztschr. f. Nervenh. **68-69**:27, 1921.

cases were proved definitely to be multiple sclerosis. In the first case the patient, aged 26, for three years had shown right-sided ataxia, vestibular symptoms and bilateral nystagmus, with doubtful choking of the disks. Suddenly these symptoms improved, and eventually the case became one of typical multiple sclerosis. In the second case the patient, aged 33, had right-sided headache, occasional vomiting and periodic attacks of clonic jerking on the right side, associated with incontinence. One year later, there was tinnitus; then bilateral choked disks, with normal vestibular reactions. The abdominal reflexes were diminished, and the deep reflexes were increased. It was thought that these symptoms were indicative of a right-sided angle tumor. Suddenly, however, the symptoms changed, the disks became atrophic, all ataxia disappeared, and at the end of six months there was a complete picture of multiple sclerosis.

In 1927, Meusberger [4] described a case which showed marked intermittent symptoms over a period of fourteen years. It began with dizziness, then vomiting and then an attack of right-sided weakness, which, after a while, almost entirely cleared up. Nine years later, headache developed; then weakness of the seventh nerve on the right side appeared and disappeared, only to reappear again. In the tenth year, a tetraplegia set in, lasting only a few days. Following this, double vision, bilateral and vertical nystagmus and weakness of the right side of the face again appeared. There were reduced sensation of the cornea and diminished abdominal reflexes with a double Babinski sign and bilateral ataxia. The right ear showed diminished hearing. There was a disturbance of speech. Sudden death followed a convulsion. Autopsy revealed a right cerebellopontile angle tumor, which microscopically was diagnosed fibrosarcoma.

REPORT OF CASES

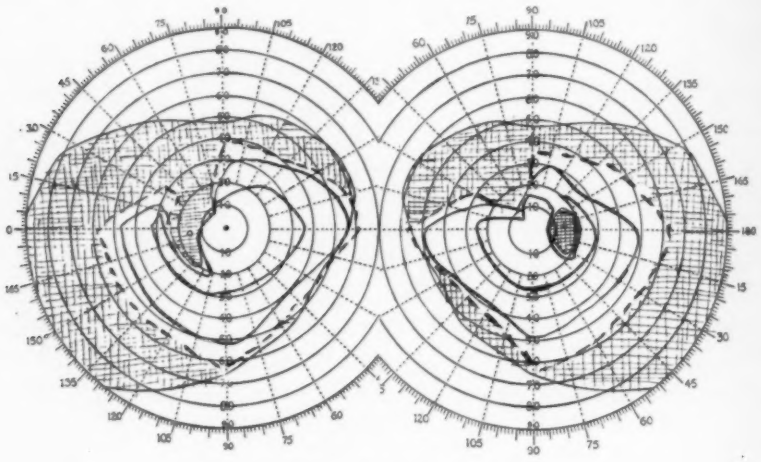
CASE 1.—*Clinical History.*—W. K., a man, aged 37, single, a tool maker, was sent by us from Buffalo to the University of Pennsylvania Hospital, service of Dr. Spiller, on May 26, 1929. He had been well until about October, 1927, when continuous ringing in the left ear began. An otologist informed him that his hearing was rapidly diminishing in that ear. In the spring of 1928, he noted that he staggered to either side as he walked. His knees frequently gave way, and he fell. In the summer of 1928, he went to a sanitarium, where he was given a strict diet. While there his hands began to tremble so that he could scarcely write. Both staggering and difficulty in writing increased until he became unable to walk in the dark or with the eyes closed. In the autumn of 1928, the trembling of the hands greatly improved. As this improvement began, an impediment of speech occurred; it was associated with difficulty in swallowing, so that fluids would choke him. The difficulty of speech constantly increased, but the ability to swallow improved. It soon became practically impossible for him to go up and

4. Meusberger, K.: Wien. klin. Wchnschr. **40**:1313, 1927.

down steps. Vision became poor at this time. At no time during the illness was there pain; in particular, headache was not complained of. There was no nausea and no vomiting.

With the exception of measles in childhood, he had had no illness; he said that he had not had venereal disease, severe fever, operations or accidents. There was no family history of nervous or mental diseases.

Examination.—On admission the blood pressure was 124 systolic and 80 diastolic. He was well built, intelligent and cooperative, but emotionally unstable. Station and gait were incoordinate, and this was increased when he closed his eyes. Speech was thick and indistinct. There was marked impairment of associated upward movement of the eyeballs, with slight impairment of the left lateral associated movements. Nystagmus was present in all directions. The finger-to-nose and heel-to-knee tests showed dysmetria, which was more marked on the left. Diadokokinesis tests were imperfectly performed on both sides, but



Visual fields of patient in case 1.

were more defective on the left. A very slight facial weakness was observed on the left side. The deep reflexes were all considerably exaggerated. There was neither clonus nor a Babinski sign. The abdominal and cremasteric reflexes were absent. Sensation of all forms was intact. Particular attention was paid to vibration and muscle joint, both of which were normal.

The eyes were examined by Dr. I. J. Koenig. The pupils were normal. Visual acuity was reduced to 20/50. There was bilateral choked disk. The uppermost point of elevation in the right eye measured 6 diopters; in the left eye, 4 diopters. There was intermittent blurring along the course of the veins. No central scotoma was found for color or form. The blind spot showed enlargement coinciding with the swelling about the disk.

A roentgen examination of the skull was made by Dr. E. Koenig. There was reduction in the density of the posterior clinoid processes. The ears were examined by Dr. Lewis Fisher. There were: (1) total loss of function of the left eighth nerve, both cochlear and vestibular branches; (2) loss of function of the right vertical semicircular canals, with preservation of hearing; (3) production of

marked oblique, almost vertical, nystagmus by the right horizontal canal; (4) poor movements of the pelvic girdle.

The visual fields were examined by Dr. Holloway. These showed a marked bitemporal contraction with homonymous deep cutting in the upper left quadrant of each field (see accompanying illustration).

Laboratory Examination: The blood showed: hemoglobin, 93 per cent; red cells, 5,200,000; white cells, 6,700; differential count: polymorphonuclear cells, 65 per cent; small lymphocytes, 30; large mononuclears, 3; eosinophils, 2 per cent. The urine had a specific gravity of 1.019; it was acid; there were no albumin, sugar or casts. The Wassermann reactions of the blood and spinal fluid were negative. The blood urea nitrogen was 16.4 mg. per hundred cubic centimeters; sugar, 100 mg. per hundred cubic centimeters.

Comment.—In the diagnosis in this case it was difficult to explain the paresis of upward associated and left lateral associated ocular movements. The former is usually suggestive of a lesion in the region of the corpora quadrigemina. These observations could be explained by an internal hydrocephalus, which was probably present. Great distention of the aqueduct may have interfered with the function of the ocular nuclei. In explanation of the bitemporal contraction of the visual fields one could assume that with an internal hydrocephalus of any degree the floor of the third ventricle is at times pushed downward, forming what may be taken for a large cyst, which presses on the inner fibers of the optic tracts. The homonymous cutting of the left upper quadrant could be explained by the involvement of the lower outer fibers of the right optic tract from pressure by the floor of the third ventricle.

While an absolute differential diagnosis in this case was not certain and lay between angle tumor and multiple sclerosis, the positive Bárány tests and the condition of the eyes made the diagnosis of angle tumor probable.

Operation and Course.—On June 14, the patient was operated on at the clinic of Dr. Frazier, by Dr. Grant. A large angle tumor was found on the left side. It was difficult to remove owing to severe hemorrhage, and the patient died. It was observed at the operation that the ventricular system was greatly distended. The tumor proved to be large and encapsulated, typical of an acoustic neuroma.

CASE 2.—*Clinical History.*—N. W., a white woman, aged 47, single, a nurse, was admitted to Temple University Hospital on Aug. 21, 1929. The family history was not of importance. She had had acute myocarditis ten years before admission, for which she had been in a hospital seven weeks. The present illness began one year previous to admission. She complained of a peculiar dizzy feeling while driving an automobile. Since that time she had had the sensation of falling toward the left when walking. There were transient feelings of numbness in the arms and legs at times.

Examination.—On admission she was well nourished. The pupils were of medium size, round and equal and reacted normally to light and in accommodation. There was marked bilateral and vertical nystagmus. There was slight ptosis of the left lid, and she complained of diplopia. There was some weakness of the left lower facial muscles. There was ataxia of the gait, which was more marked on the left side. The knee and ankle reflexes were prompt, with a suspicion of a Babinski sign on the left. The abdominal reflexes were absent. There were dysmetria and adiadokokinesis, which were more marked on the left side. Later, an examination of the eye revealed some rotatory nystagmus on fixation and in all fields of rotation. The eyegrounds showed choking of the disks, the right being 3.5 diopters, with some thickening of the vessels but no

hemorrhages, and the left disk 4.5 diopters. There were slight involvement of the left sixth nerve, slight ptosis of the left lid, slight weakness of the left lower part of the face and anesthesia of the left cornea.

As the case progressed there was peripapillary swelling of the disks. On August 26, it was noted that there was slight involvement of the left ninth nerve and that the patient was deaf in the left ear, with marked slurring of speech and marked ataxia on finger-to-nose and heel-to-knee tests, which was more marked on the left side. On this date all the tendon reflexes were present; they were equal and normal. There was no Babinski sign. The vibratory sense was normal. The Bárány test, made by Dr. Winston, showed: (1) upward spontaneous nystagmus; (2) disproportion between the duration of nystagmus and vertigo; (3) nonfunctioning of the right vertical semicircular canals, with good function of the right horizontal semicircular canal in the presence of good hearing in that ear; (4) nonfunctioning of the left ear in all portions (cochlear and vestibular).

Laboratory Report: The urine on several occasions was normal. The blood showed: hemoglobin, 80 per cent; red cells, 4,340,000; white cells, 6,200; a differential count revealed: polymorphonuclear cells, 56; small lymphocytes, 35; large lymphocytes, 4; eosinophils, 4 and basophils, 1. The Wassermann reaction of the blood was negative by the Kolmer and Kahn methods. The blood contained 95 mg. of sugar, 29.4 mg. of nonprotein nitrogen, 4 mg. of uric acid and 1.4 mg. of creatinine per hundred cubic centimeters.

Comment.—When these observations were put together, it was thought that the diagnosis lay between multiple sclerosis and a left cerebellopontile angle tumor. The preponderance of symptoms favored a tumor.

Operation and Course.—Dr. Temple Fay performed a two-stage suboccipital craniotomy, the first stage on Sept. 17, 1929, and the second on September 20. He found a tumor involving the left cerebellopontile angle. It was firm, red and encapsulated. Considerable shock was associated with the removal of the tumor, and the patient did not rally. She died on September 21.

Microscopic Examination.—The tumor was growing from the sheath of the eighth nerve. The cells were fibroblasts with oval nuclei, in places giving an appearance as if the cells had processes not unlike glia cells. The arrangement was in columns and not in palisades. The absence of nerve filaments within the tumor and the presence of considerable degeneration eliminated this from the so-called neurofibromas; the tumor in every way corresponded to a perineural fibroblastoma.

CASE 3.—*Clinical History.*—C. I., an Italian woman, aged 24, married, with one living child, consulted us on May 24, 1928, because of the sudden onset of slight weakness and numbness of the right side of the body, associated with diminished hearing in the left ear; all of this followed suddenly the death of her child, aged 8 months, from pneumonia, one week before. She stated that previous to this she had been perfectly well; with the exception of a few childhood diseases, she had never been ill.

Examination.—The patient appeared healthy and weighed 140 pounds (63.5 Kg.). The blood pressure was 120 systolic and 70 diastolic. The pupils were of medium size, round, equal and reacted to light and in accommodation. There was moderate nystagmus on looking to either side. The eyegrounds were normal. Hearing was much reduced in the right ear. The deep reflexes were present, equal and overactive. There were no abnormal toe signs. With the feet together there was some slight swaying, which became more marked when the eyes were closed. There was slight incoordination in the finger-to-nose test on the right side; there was no incoordination in the heel-to-knee test on either side. Vibration, localiza-

tion and spot senses were normal. She was very excitable, crying and laughing in rapid succession; this was explained largely by the recent death of the child. She complained of some nausea but no headache.

Course.—On June 5, all the symptoms on the right side were much improved. The strength had returned nearly to normal, and the subjective numbness had entirely disappeared. She was much more calm and composed. The incoordination of the right hand had disappeared, but the bilateral nystagmus was still present, and was about equal in each direction. The hearing in the right ear remained about the same. The eyegrounds were normal. Following this she went into the country, remaining until the autumn.

On September 1, she complained of some slight headaches on the right side. She explained that she was four months pregnant. The nystagmus and partial deafness of the right ear were still present.

On May 20, 1929, she stated that she had given birth to a full-term child on Feb. 17, 1929, and that labor was normal. In April she had observed for the first time that she began to stumble in walking, that she was growing restless and things annoyed her, and that she felt jumpy and "twitched all over" at times. There was bilateral nystagmus. The pupils were equal and reacted normally. At this time she presented choked disks, the swelling being about equal, 4 diopters in both eyes. There were tiny hemorrhages in both eyes. At times double vision was present. The outstretched fingers of both hands showed tremor and moderate incoordination in the finger-to-nose test. This was slightly more marked on the right side. There was incoordination in the heel-to-knee test, more pronounced on the right side. Diadokokinesis tests were poorly done on both sides, especially on the right. She again complained of weakness of the right side, but we were unable to demonstrate this by any objective tests. She was emotionally unstable. The tendon reflexes of both upper and lower extremities were present, equal and active. There was no clonus and no Babinski sign. There was moderate ataxia of the gait, which was more marked on the right side. There was complete deafness in the right ear.

Laboratory Studies: The urine showed: specific gravity, 1.010, acid, of amber color and with negative albumin and sugar tests; microscopically, it was normal. The blood showed: Wassermann reaction, negative; hemoglobin, 82 per cent; red cells, 4,200,000; white cells, 8,000; a differential count revealed: 70 per cent polymorphonuclear cells, 24 small lymphocytes, 5 large mononuclears, and 1 per cent eosinophils.

On February 24, vision was much reduced in both eyes. There were bilateral choked disks, with beginning pallor. There was dysmetria, especially of the right hand. Diadokokinesis tests were poorly performed on both sides, but more so on the right. There was bilateral and vertical nystagmus. The gait was markedly ataxic in all directions, but showed more deviation to the right. While the tendon reflexes were active, there was no Babinski sign. The abdominal reflexes were absent. There was a marked Romberg sign. There was no weakness of the facial muscles. Ataxia of speech and complete deafness of the right ear, with beginning diminution of hearing of the left ear, were noted. There was no complaint of double vision. Bárány tests showed: (1) total loss of function of the right eighth nerve, both cochlear and vestibular divisions; (2) diminished hearing in the left ear with loss of function of the vertical semicircular canal; (3) a slight vertical nystagmus of short duration produced by the left horizontal canal; (4) very poor pelvic movements. The eye fields showed a right homonymous cutting of about 30 degrees.

Comment.—During the first year of observation of this patient, the variability in the clinical picture made us consider multiple sclerosis as a possibility. The fully developed case, with choking of the disks and other evidences of increased intracranial pressure, pointed to the right angle as the seat of the lesion. The patient refused an operation. The Bárány examination in particular points to the correctness of this localization.

CASE 4.—*Clinical History.*—C. MacN., aged 27, was admitted to Temple University Hospital on Feb. 23, 1930, because of inability to walk. She had been under our care since 1927. The condition began in October, 1926, with vomiting and headache. She had had a peripheral left facial palsy twelve years before, but it had disappeared completely until one year before the first examination. She had also had a slight decrease of hearing in the left ear, but had had no tinnitus. A slight difficulty in gait began shortly after the onset. She had had a "nervous breakdown" in 1925, with marked sleepiness, blurring of vision and then diplopia. In April, 1925, she was in bed for one week, during which she slept the entire time.

Examination.—When studied in 1927, there was marked nystagmus in all directions, with weakness of the left external rectus muscle. The pupils were normal. A choked disk of 4 diopters was present in each eye. There was complete palsy of the muscles of mastication on the left. Sensation of the left side of the face showed a slight decrease, with corneal anesthesia. Hearing was slightly defective on the left. A peripheral left facial palsy was evident. Speech showed definite incoordination. The other cranial nerves were normal.

In the upper limbs the reflexes were exaggerated on the right side without a Hoffman sign and without sensory changes. The finger-to-nose test showed marked ataxia, especially on the left. Adiadokokinesis was marked on the left. The Holmes rebound phenomenon was evident on the left. There were no atrophies and no deformities.

There was a marked cerebellar gait, with deviation to either side. The reflexes in the lower limbs were much exaggerated, especially on the left. There was neither clonus nor a Babinski sign. Muscle position and vibration sensations were normal. Incoordination was present in the movements of the limbs.

The Bárány examination excluded lesions of either cerebellopontile angle. The conclusions were in favor of a lesion either in the left side of the cerebellum or in the pons.

The spinal fluid pressure was 12 mm. of mercury. The fluid showed no abnormalities.

Operation.—On Aug. 14, 1928, the patient was operated on by Dr. Francis Grant, who was able to explore the left cerebellopontile angle, but failed to find evidence of a tumor; extensive search was impossible, however, because of respiratory collapse.

Reexamination.—On admission to Temple University Hospital on Feb. 23, 1930, it was found that the condition had improved since the operation. Headache and vomiting occurred infrequently. Vision had improved, and there was less choking of the disks (from 1.5 to 2 diopters). The gait, however, had become worse.

The neurologic condition, however, had not changed, except that the cerebellar incoordination had increased, and the reflexes on the right were more active than on the left. A Bárány examination at this time again excluded both angles, and Dr. Winston believed that there was a lesion of the brain stem. Roentgenographic examination gave negative results. The eye fields were not taken.

Diagnosis.—In view of the observations recorded a diagnosis was made of a gross lesion on the left lateral surface of the pons, just above the angle, and surgical intervention was advised.

Second Operation.—On March 13, Dr. Temple Fay operated through a left lateral exposure and found a cystic tumor on the lateral surface of the pons on the left side, partly above and partly below the tentorium. Practically complete removal of the tumor was possible.

Course.—Convalescence was uneventful. The patient is still under observation. She is improving daily. Except for a corneal ulceration because of the involvement of the fifth nerve, there has been a good operative result.

COMMENT

In this country, multiple sclerosis is much less common than in Europe, and one should be cautious in the diagnosis of this disease. It has long been recognized that various conditions may simulate multiple sclerosis or be simulated by it. Not sufficient stress has been laid, however, on the similarity between angle tumor and multiple sclerosis, four cases of which are here reported.

Besides Marburg's [3] and Meusberger's [4] contributions, only an occasional report of this similarity and difficulty in differentiation appears in the literature. With the advent of the Bárány caloric test, less difficulty should be experienced in the differentiation. It is mentioned by Oppenheim [2] and others that choking of the disks may occur in multiple sclerosis, but it is usually not marked and is of rather short duration, rapidly leading to atrophy. In 1921, Ward Holden [5] stated before the Research Society in Nervous and Mental Diseases that he had never seen anything that he would call optic neuritis or papilledema in multiple sclerosis. On the contrary, Frank,[6] Uhthoff,[7] Rosenfeld,[8] Müller [9] and others have stated that, while it rarely occurs, they have seen it; it never lasts long, and they rarely found hemorrhage or exudate associated with it. Defects in the visual fields and scotoma, while not common in angle tumors, occur oftener in multiple sclerosis. That they may be present with an angle tumor is shown in our cases 1 and 3. Our explanation for this phenomenon is the protrusion of the floor of the third ventricle downward to press on the optic tracts, as a result of the great increase of intracranial pressure. It is recognized that in any increase of intracranial pressure, dilatation is most marked in the weakest portion of the ventricular system, the floor of the third ventricle. This well explains the partial homonymous hemianopia present in our cases, as well as the

5. Holden, Ward: Multiple Sclerosis, New York, Paul B. Hoeber, Inc., 1922, p. 108.

6. Frank: Deutsche Ztschr. f. Nervenh. **14**:167, 1898.

7. Uhthoff, in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1903, vol. 2, p. 337.

8. Rosenfeld: Neurol. Centralbl. **22**:702, 1903.

9. Müller: Thesis, Jena, 1904.

erosion of the clinoid processes, which might be misinterpreted as a pituitary result.

A Bárány examination is one of the most important single aids in the differentiation between multiple sclerosis and angle tumor. In the presence of total loss of function of the cochlear and vestibular divisions of the eighth nerve, plus loss of function of the vertical semicircular canals of the opposite side, on which hearing is well preserved, and with the production of nystagmus by stimulating the opposite horizontal canal, we believe that it is safe to make a diagnosis of a mass in the cerebellopontile angle on the side in which there is total loss of nerve function. It has been our experience that in several cases the Bárány observations have made the diagnosis of an angle tumor possible before the neurologic signs of this condition were evident. Bilaterality of the symptoms in tumor of one angle has been often commented on. This could be accounted for by distortion of the basis pontis by pressure from the tumor, or even as the result of a general increase of intracranial pressure. The absence of abdominal reflexes is supposedly of great value in the diagnosis of multiple sclerosis, but in women who have borne children or in others who are obese this sign is of doubtful value; in the presence of bilateral symptoms of the pyramidal tract the absence of abdominal reflexes is the rule. The involvement of the cranial nerves on one side, especially the fifth, seventh and eighth, is usually considered suggestive of an angle lesion. It does not infrequently happen, however, that the fifth and seventh nerves remain uninvolved. In multiple sclerosis the involvement of the cranial nerves is less orderly.

Vibration sensation has come to be considered one of the most important of the diagnostic features in multiple sclerosis. It is considered by Mann [10] and his associate, Max Lamm,[11] to be as important, and even more so, as the loss of abdominal reflexes, some change in vibratory sense having been found in thirty-two of thirty-seven cases of their series. Of the five cases in which vibratory sense was normal, three were in a state of remission, and in two the diagnosis was doubtful. They substantiate in this way the observations of Claude and Eggers,[12] who found a marked change in this sensation in twenty of twenty-two cases. Mann expressed the belief that because it is a more complicated sensation than muscle, position or joint motion, the patches of degeneration of the posterior column, while they may allow the coarser sensations to pass through as a result of the integrity of some axis cylinders, do not allow the complicated vibratory sensory fibers to ascend. A simpler

10. Mann, L.: Ztschr. f. d. ges. Neurol. u. Psychiat. **104**:443, 1926.
11. Lamm, Max: Inaug. Diss., Breslau, 1925.
12. Claude and Eggers: Rev. neurol. **14**:276, 1906.

explanation that occurs to us is that the test for vibration can be made more delicate than the gross determination of muscle position or joint motion. Our experience in the last ten cases of multiple sclerosis has shown that there was vibratory sense disturbance in all.

Variability of the symptomatology is one of the great characteristics of multiple sclerosis. One does not picture a tumor with remissions as a usual rule. It is rather interesting that in case 3 the patient has had partial remission; still there is no doubt that she has an angle tumor. Even in case 1, there was improvement in some of the symptoms extending over a considerable time. It is well known that in true multiple sclerosis there may be no variability of symptoms, which may again befog the issue. While the symptoms in tumor of the brain usually fit in with known anatomic localization, it is characteristic of multiple sclerosis to have disseminated irregular lesions and symptoms. It is possible, as in our case 1, particularly, to have symptoms that are the result of secondary internal hydrocephalus. We refer to the condition of the eye in this case, in which as a result of the internal hydrocephalus there must have been such intense dilatation of the third ventricle that the optic fibers were comprised. This we have seen in other cases of tumors of the posterior fossa.

The classic general symptoms of tumor of the brain are supposedly characteristic and should differentiate tumor from nonexpansile lesions, such as multiple sclerosis. It is to be noted that in case 1, particularly, the general symptoms were absent and this was certainly in favor of a non-neoplastic condition.

SUMMARY

In the four cases herein reported stress has been laid on the similarity of many of the clinical features of multiple sclerosis and cerebellopontile angle tumor. It goes without saying that typical cases of either condition show distinctive signs and can readily be diagnosed. On the other hand, borderline cases occur, and it is this group with which we are concerned. As has been pointed out, the clinical pictures of these two conditions may be so confusing that any aid in their differential diagnosis is welcome and desirable. While the clinical examination in every case should be as thorough as possible, certain symptoms have given us more aid than others. A history of a remission of the symptoms is in favor of multiple sclerosis. The presence of choking of the disks is significant and usually is in favor of a tumor. The unilaterality of the symptoms usually points to a tumor. A normal vibratory perception speaks in favor of a tumor. The one most important differential observation is that obtained by the Bárány test. A carefully done Bárány test will

either affirm or exclude an angle neoplasm except in instances, like case 4, in which the tumor was slightly higher than the usual situation.

CONCLUSIONS

1. Symptoms of tumors of the cerebellopontile angle and of multiple sclerosis may be so similar at the bedside that their differential diagnosis may be in doubt.

2. The Bárány test gives the most important single observation in the differential diagnosis.

3. Other symptoms, such as choking of the disks, progression without remission and the unilaterality of symptoms, all speak in favor of an angle tumor.

4. Disturbances of vibration sense and the absence of abdominal reflexes occur more commonly in multiple sclerosis than in cerebellopontile angle tumors.

5. The differential diagnosis is extremely important because of the good therapeutic result in the case of an angle tumor when an operation is performed early by a competent surgeon.

A CLINICAL STUDY OF THE DIFFERENTIATION OF CERTAIN PONTILE TUMORS FROM ACOUSTIC TUMORS *

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The clinical recognition of a case of acoustic neuroma as a rule presents no special difficulties. The well established syndrome with progression of symptoms which follow a definite chronology, together with the usually typical objective neurologic observations, was vividly and accurately brought out by Cushing [1] in his monograph on this subject in 1916. There are, however, some cases of verified acoustic tumors in which the history or neurologic signs do not conform entirely to the rule, and there are other cases of tumor in this general neighborhood giving symptoms so nearly like those of acoustic tumors as to cause confusion and error in diagnosis. Certain tumors of the pons fall into the latter group, and in looking up the records it appears that not infrequently these patients have been subjected to a suboccipital exploration which nearly always would have been inadvisable if a correct preoperative diagnosis had been made.[2] It is true, as in one of the cases to be reported, that occasionally a pontile growth that extends to a considerable degree into the lateral recess may be benefited by operation, but such cases must be exceptional. The present series has been studied with a view to bringing out, if possible, the differentiating features in certain pontile tumors that simulated acoustic growths. It by no means includes all patients with tumors of the pons coming to the clinic, of which there have been twenty-five verified cases. It is confined to those in which the diagnosis was so doubtful that an exploratory operation was performed on the assumption that the patient probably had an acoustic tumor.

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1. Cushing, Harvey: Tumors of the Nervus Acusticus, Philadelphia, W. B. Saunders Company, 1917.

2. Horrax, Gilbert: Differential Diagnosis of Tumors Primarily Pineal and Pontile, Arch. Neurol. & Psychiat. **17**:183 (Feb.) 1927..

REPORT OF CASES

There are eight cases which fall into the foregoing category, and they will be presented in the chronologic order of their appearance at the Peter Bent Brigham Hospital.

CASE 1.—*Deafness in right ear for one and a half years; staggering gait and general pressure symptoms; weakness of right side; dysarthria and dysphagia; negative exploratory operation.*

History.—C. E. P., a woman, aged 48, was admitted to the hospital on May 28, 1915, complaining of dizziness, unsteady gait and headache. She was referred by Dr. Keator of Roxbury, N. Y. One brother and one uncle had died of tuberculosis. She had had chronic otorrhea of the right side since the age of 11. In November, 1913, she had had a mastoid operation, and since that time had been deaf in the right ear. Dizziness had been present since August, 1913. For four months previous to admission, she had been unsteady in walking, not being able to walk alone during the last six weeks. For six weeks also, she had had occipital headaches accompanied by vomiting, and for four weeks weakness of the right arm and leg, dysarthria and dysphagia. Tinnitus in the left ear had been present for three weeks.

Physical Examination.—The pupils were unequal, the left being greater than the right. There was a low grade of choked disks, the process being somewhat more marked on the left. Hypesthesia of the right side of the face was present, with an absent corneal reflex on this side. She showed a facial weakness on the right side, peripheral in type, and the right ear was deaf. Caloric responses were normal on both sides. Both vertical and lateral nystagmus was present, the latter being coarser to the right. There was slight ataxia of both arms, greater on the right, adiadokokinesia of the right hand and a coarse tremor of both outstretched arms. Romberg's sign was positive, and she had an unsteady gait, walking with a broad base with deviation to the right. Dysarthria and dysphagia were well marked.

Diagnosis and Operation.—The preoperative impression of this patient was that she had a tumor of the right cerebellopontile angle. On June 2, 1915, a suboccipital exploration was made by Dr. Cushing. This disclosed what at the time was taken to be a cyst of the right lateral recess. She was discharged as improved on June 23, 1915.

Course.—On July 15, 1916, she reentered the hospital. She had improved for about six months, after which symptoms of increasing intracranial pressure developed. On July 20, 1916, the old operative area was reexplored. A large cyst of the right cerebellar hemisphere was evacuated and its lining treated with formaldehyde. She survived this operation only three days.

Necropsy.—The right cerebellopontile angle was filled with a large cystic tumor which extended from the right cerebellar hemisphere, the brachium pontis and a portion of the pars dorsalis of the right side of the pons. Histologic sections showed the tumor to be a fibrillary astrocytoma.

Comment.—In discussing this case it must be kept in mind that fifteen years have elapsed since the patient appeared at the clinic. At the time of her entry, the definite syndrome of acoustic tumor had not been established, and probably the best diagnosis that was then possible was the one made before the operation, namely, a tumor of the right

cerebellopontile angle. Whether the deafness was due to the old otitis media, or whether it was the result of the new growth, was an open question then and would be now, except for the fact that at the present time more stress would be laid on the caloric responses, which we now believe would have ruled out almost certainly an acoustic growth. Admitting, however, that the deafness was thought to be due to tumor and not to the old infection of the ear, this and the remaining chronology was sufficiently typical to make an acoustic tumor the probable diagnosis. One set of facts, nevertheless, should be emphasized. The patient had advanced symptoms, including involvement of several cranial nerves, together with dysarthria and dysphagia—all in the presence of an extremely low and early choking of the left disk, while the right was practically normal. This state of affairs would be almost impossible with an acoustic tumor, whereas it is frequently found in growths involving the pons, cerebellum and adjacent cerebellopontile angle.

CASE 2.—*Progressive deafness in the left ear associated with twitching of the left side of the face; subsequent development of pressure symptoms and staggering; generalized neurofibromatosis; negative suboccipital exploration.*

History.—E. T., a woman, aged 29, was referred to the hospital by Dr. J. H. McCain of Arcola, Ill., on June 2, 1919. For two years the patient had had persistent slight diplopia on looking to the left. One and one-half years before entry, she began to get deaf in the left ear. This deafness became gradually worse and at the time of admission was complete. It was not associated with tinnitus. She complained also of slight deafness in the right ear. About the time of the onset of deafness, she developed a left blepharospasm, which spread gradually to include the whole left side of the face. For one year, she had progressive difficulty in speech and in swallowing. During the year before entry, she had also become conscious of some numbness of the left side of the face, and had had suboccipital headaches with occasional vomiting. Six weeks before admission, she had an attack of influenza. After this illness she began to be unsteady on her feet, and noted some weakness of the right arm and leg.

Physical Examination.—There were numerous fibroma mollusca over the body. She had a left abducens palsy and nystagmus to both right and left, the slower jerks being to the left. There was a spasmodic left facial tic, involving the whole face and eyelids. The left eye showed slight enophthalmos, which may have been a congenital anomaly. A low grade of choked disk was present bilaterally. The left trigeminal nerve showed definite motor weakness with atrophy of the left masseter. There was loss of air conduction in the left ear. Dysarthria and dysphagia were well marked; both arms were ataxic; Romberg's sign was present, and the gait was staggering with deviation to the left. She had some questionable weakness of the right arm as compared with the left, and the deep reflexes were greater on the right than on the left side. Babinski's and Oppenheim's signs were positive on the right. Both legs showed marked hypotonia and ataxia.

Diagnosis and Operation.—The preoperative impression was that this patient had a left acoustic neuroma, with the possibility that she might have multiple tumors associated with Recklinghausen's disease.

On June 5, 1919, Dr. Cushing explored the subtentorial region. The patient took the anesthetic badly and great respiratory embarrassment was present

throughout the procedure. No tumor was disclosed. At the end of the operation it was necessary to resort to artificial respiration, and this was continued for four or five hours thereafter, but without avail.

Necropsy.—There was a large tumor which replaced the greater part of the left side of the pons and the left cerebellar hemisphere and extended into the left cerebellopontile angle. The fifth to the twelfth cranial nerves on the left side, and the sixth nerve on the right side were embedded in the tumor.

Histologic examination showed that this tumor could be classified as a unipolar spongioblastoma.

Comment.—This patient presented a most difficult problem for differential diagnosis, and had she been seen at the present time instead of ten years ago, it is doubtful whether the differentiation could be made with certainty. Although there are certain features in the record which would make one suspicious of something other than an acoustic growth, nevertheless, when confronted with such a case clinically the correct diagnosis depends greatly on where the chief emphasis is laid. In coming to a conclusion regarding this woman, it appears that the diplopia, which somewhat preceded the deafness, was discounted, and yet it was no doubt a point on which more dependence should have been placed. The remainder of the symptoms were correct for the chronology of events in an acoustic tumor, although dysarthria and dysphagia were prominent rather earlier than usual. Neurologic examination revealed conditions perfectly consistent with an acoustic tumor, but again it is unusual that the motor portion of the trigeminal nerve should have been affected with no sensory involvement. The questionable weakness of the right arm, and more greatly exaggerated reflexes on the right side may well have been underemphasized in the presence of more prominent symptoms. The striking thing, as one looks at the case as a whole, is exactly the state of affairs which was brought out in discussing case 1, namely, advanced evidences of involvement of the cranial nerve, together with marked ataxia, hypotonia and incoordination, i. e., "cerebellar signs," in a patient whose fundi showed only the earliest appearance of choking. On this situation, as well as the history of diplopia antedating the onset of deafness, it is evident in retrospect that attention should have been focused.

The next case should have given no difficulty in differentiation if sufficient emphasis had been placed on the chronology of symptoms.

CASE 3.—*History.*—F. N., a man, aged 41, was referred by Dr. C. L. McDonald of Cleveland with the diagnosis of acoustic tumor. The patient was admitted to the hospital on March 2, 1920, complaining of "dizziness, blurred and double vision." His family and past history were irrelevant. His present illness had begun two and one-half years previously with the onset of dizziness, unsteadiness and weakness, together with slight frontal headaches. Six months later, tinnitus developed in both ears, which was more marked in the left. About the same time he also noted a disturbance of speech, characterized by indistinctness. For one and one-half years he had had double and multiple vision and

numbness of the left side of the face. Eight months previous to admission, he had noticed weakness of the left side of the face and difficulty in swallowing. The last symptom to develop was deafness of the left ear, this being present from four to six months before entry. It may be noted, also, that he had recently had attacks of hiccupping.

Physical Examination.—There was hypesthesia of the left side of the face with anesthesia of the left cornea and somewhat lessened sensation of the right cornea also. He had a left abducens palsy, a left facial paralysis, peripheral in type, deafness of the left ear and hypesthesia of the pharynx on both sides with loss of the gag reflex. Nystagmus was present, both to the right and to the left, upward and downward. The cerebellar symptomatology included ataxia of the left arm and of both legs, generalized hypotonia, positive Romberg's sign and a staggering gait. All the deep reflexes were exaggerated on the left side with a positive Babinski sign on the left. The fundi were normal. Caloric responses showed total loss of normal reactions.

Clinical Diagnosis.—The general preoperative impression of this case was that it was probably an acoustic tumor, although a diffuse lesion of the pons was considered because of the Bárány reactions.

Operation.—On March 24, 1920, a suboccipital exploration was performed by Dr. Cushing, the note stating "for presumed acoustic tumor." A small tumor projecting into the porus acusticus was disclosed, and this nodule was partially removed.

On April 30, 1920, the patient was discharged unimproved. He died on Sept. 8, 1920.

Necropsy.—A dense tumor replaced the left side of the pons and the cerebellar hemisphere and projected into the left cerebellopontile angle.

Histologic examination showed that most of the cells were spongioblasts and a diagnosis of a unipolar spongioblastoma was made.

Comment.—No doubt the single, outstanding fact against the diagnosis of an acoustic tumor in this patient was the late development of deafness. It was, in fact, the last of a long train of symptoms which had come on during the course of two and one-half years. Furthermore, as in cases 1 and 2, cranial nerve palsies as well as cerebellar symptoms were present to a marked degree, yet the patient had only slight headaches and the fundi were normal. It is possible that his statements as to when hearing became impaired may have been discounted, but this discrepancy together with the lack of pressure symptoms in an advanced lesion should have been significant.

The next patient again showed an atypical chronology of symptoms, along with certain other features which have been pointed out in the three previous cases.

Case 4.—A. St. J., a man, aged 43, was referred to the clinic by Dr. P. E. de Bousquet of Montreal. He complained of "giddiness, deafness and facial paralysis." The only noteworthy fact in the family or past history was that he had suffered a severe injury to the back of the head by an iron bar four years before admission. No other details of this injury were given.

The symptoms, in the order of their appearance, were as follows: In November, 1922, his friends had noticed that he was unsteady in walking, and in

particular that he lurched to the right. In January, 1923, he complained of soreness of the right side of the tongue, as if he had smoked too much. This condition persisted. In February, 1923, he noticed ringing in the right ear and in the same month he began to be dizzy. In March, 1923, deafness of the right ear was first noted. In April, 1923, he had intermittent twitching of the right side of the face, followed soon by right facial weakness. In June and July, 1923, he began to have difficulty in talking and in swallowing, and also noticed some slight headache.

Neurologic Examination.—The following positive features were noted: nystagmus, spontaneous and horizontal, coarser to the right; corneal reflex absent on the right; right abducens palsy; loss of taste on the right side of the tongue; right lower facial weakness; deafness of the right ear; weakness of the right palate; weakness and atrophy of the right side of the tongue; marked cerebellar ataxia with positive Romberg sign, the tendency being for him to fall backward (retropulsion); weakness of the left arm and the left leg with increased deep reflexes on this side; absent lower abdominal reflex on the left side; left hemihypesthesia. The fundi were normal, and roentgenograms of the skull were normal.

Clinical Diagnosis.—The preoperative impression of this patient's condition was that he probably had an acoustic tumor, although a meningioma of the cerebellopontile angle was suggested.

Operation.—On Aug. 17, 1923, Dr. Cushing performed a suboccipital exploration. A tumor of the right lateral recess was exposed and partially removed. On August 19, the patient died without regaining consciousness.

Necropsy.—There was a large, firm, yellowish white invasive tumor replacing the entire right side of the pons, the right cerebellar hemisphere and filling in the right cerebellopontile angle. From histologic sections, the tumor was classified as a protoplasmic astrocytoma.

Comment.—In view of this patient's definite cranial nerve palsies on the right side, coupled with weakness and hypesthesia of the left arm and leg, it is almost impossible to see why the diagnosis of an acoustic tumor could have been entertained. In addition, deafness appeared late in the chronology of the symptoms, and again it is to be noted that the fundi were normal in the presence of advanced neurologic signs, a condition which could hardly be obtained in an acoustic growth, as the latter would almost certainly have produced internal hydrocephalus and marked choking.

On the other hand, it is to be borne in mind that records do not always give the correct clinical picture in a case. In this instance, weakness of the left arm and leg, together with the sensory changes and increased deep reflexes on that side, may have been so slight that they were considered questionable by some observers, leaving the deafness as well as the trigeminal and facial involvement on the right to dominate the picture, along with the marked cerebellar manifestations. In any event, however, the chronology of symptoms was not typical of an acoustic tumor, and, as has been stated, there was no evidence of increased intracranial pressure from the fundi.

If the diagnostic error in the case just recorded seemed hardly warranted, in the next case it is difficult to see how any other preoperative diagnosis than that of an acoustic tumor could have been made.

CASE 5.—H. S. L., a man, aged 33, was referred by Dr. J. H. Pratt of Boston, and was admitted to the hospital on Feb. 18, 1924, complaining of "deafness in the right ear and headache." His family and past history were irrelevant. He had first noticed some difficulty in hearing with the right ear in 1915, and this deafness had steadily increased until it became complete in 1920. In July, 1922, he began to have headaches, and these became extremely severe in August, 1922, being increased greatly by straining and by exercise. In December, 1923, he noticed some haziness of vision and numbness of the lower part of the right side of the face. During December, 1923, and January, 1924, he became weak and somewhat unsteady on his feet.

Neurologic Examination.—The following points were discovered: bilateral choked disks of from 3 to 5 diopters; weakness of the right abducens; right trigeminal hypesthesia with sluggish corneal reflex; loss of taste on the right side of the tongue, both anteriorly and posteriorly; complete deafness of the right ear; a Bárány test showed complete absence of responses from the right ear; nystagmus on looking upward, but none laterally; slight twitching of the right side of the face; staggering gait on a wide base, with a tendency to deviate a little to the right; positive Romberg sign; neurofibromatosis of the skin.

Clinical Diagnosis.—The preoperative consensus was that the patient had an acoustic neuroma.

Operation.—On Feb. 26, 1924, Dr. Cushing performed a suboccipital exploration. A soft glioma of the right cerebellopontile angle was exposed and fragmentarily removed.

Course.—The patient was discharged on March 18, 1924, slightly improved. Examination of the tissue which was removed at operation was reported as an astrocytoma.

For two years this man did very well. He had a course of deep roentgen therapy and returned to the practice of law in September, 1924, continuing in his profession until April, 1926, when he was again admitted to the hospital because of a return of the former symptoms. Examination at this time showed similar observations to those at the first entrance except that the fundi now revealed secondary atrophy without choking. The suboccipital decompression was full and tense.

On April 12, 1926, Dr. Cushing reexposed the cerebellar region and partially removed some of the tough tumor plastered throughout the right lateral recess. He was discharged on April 27 with the gait somewhat improved, but the other neurologic signs about the same.

Death occurred over a year later, on June 8, 1927, after a period of several months improvement.

Necropsy.—A large soft, cystic tumor replaced the greater part of the right hemisphere of the cerebellum, the right brachium pontis and the right basilar portion of the pons.

The tumor was composed mostly of protoplasmic astrocytes and was classified as a protoplasmic astrocytoma.

Comment.—This is the one outstanding case in the series in which operation was distinctly worth while. After a suboccipital decompression, with only fragmentary removal of that portion of tumor found in the lateral recess, the patient returned to his profession of law within eight months, and practiced until six weeks before a second admission to the hospital, some two years subsequent to the first entrance. The fact that he had a course of deep roentgen treatments may have influenced his convalescence favorably also, but it is doubtful whether it would have done nearly as much without an operation.

Our interest in the case, however, so far as the present communication is concerned, rests primarily on the diagnostic differentiation from an acoustic tumor. In favor of this type of lesion, it should be pointed out that deafness was the first symptom, preceding all others by seven years, and although the patient never had tinnitus, nevertheless this is not always present in acoustic growths. The remaining chronology might well have served for a neuroma of the eighth nerve, although it is unusual in patients with either pontile or acoustic growths to have general pressure symptoms antedate those attributable to pressure on cranial nerves or cerebellar tracts. Other points favoring an acoustic tumor were the complete lack of response from the right labyrinth and the generalized neurofibromatosis. In the face of these observations it is more than doubtful whether any diagnosis other than that of an acoustic tumor could have been entertained, either when the patient was seen clinically or in a review of the record. One point, however, should be mentioned, namely, the presence of vertical nystagmus, and the total lack of this condition in either lateral direction. There is also Dr. Cushing's observation that "the patient seemed to me to be in a little better physical condition than most cases (acoustic) with advanced trouble"—a statement which undoubtedly comes within the category of a surgical inspiration.

A year and a half elapsed before the next patient with a lesion of the type under discussion entered the clinic. The chronology of symptoms in this instance was fairly typical of an acoustic growth, but deafness was incomplete and the fundi were normal, although cranial nerve palsies were advanced.

CASE 6.—*History.*—A. E. A., a boy, aged 17, was admitted to the Peter Bent Brigham Hospital on July 19, 1926, complaining of "right facial paralysis." He was referred by Dr. H. N. Williams of Providence, R. I. Nothing relevant to the present illness was to be found in the family or past history. The present trouble had started with the rapid onset of deafness in the right ear three and one-half years previously. No history of tinnitus could be obtained. Paralysis of the right side of the face came on rapidly a year and one-half later, or two years before admission. Shortly thereafter he began to have difficulty in swallowing and in talking. For one year he had had some weakness, unsteadiness and clumsiness of the right arm and leg, together with staggering and

stumbling in walking. Twitching of the right side of the face had been present occasionally for nine months, and for about this length of time he had had slight headaches. Numbness of the right side of the face and right arm was noticed two weeks before admission.

Neurologic Examination.—The following signs were found: hyperemic fundi with possibly slight secondary atrophy of the disks; nystagmus to the right, left and upward; hypesthesia of the right side of the face to touch, pain and temperature, with absent corneal reflex; complete right facial paralysis of peripheral type; loss of taste over the whole of the right side of the tongue; partial deafness of the right ear; caloric responses showed inactive labyrinths on both sides; absent pharyngeal reflex on the right side, with paralysis of the soft palate on the right; exaggerated deep reflexes throughout on the right side; positive Babinski's and Oppenheim's sign on the right side; hypotonia and ataxia of the right arm and leg, with adiadokokinesia of the right hand; hypesthesia of the whole right side and some weakness in the grip of the right hand as compared to the left.

Clinical Diagnosis.—Although the preoperative impressions concerning this patient's lesion were doubtful, nevertheless an acoustic tumor was thought to be fairly probable.

Operation.—On July 27, 1926, a suboccipital exploration was carried out and disclosed an enormous tumor situated at the right side of the medulla.

The patient survived this procedure only twenty-four hours.

Necropsy.—There was a firm tumor replacing the right side of the pons and the greater part of the right cerebellar hemisphere. The tumor extended into the right cerebellopontile angle. The fifth, seventh and eighth cranial nerves on the right side were embedded in the tumor.

Histologic study showed that the tumor was a fibrillary astrocytoma.

Comment.—Here again was a patient with deafness preceding all other symptoms. Tinnitus was absent, as in case 5. The remaining symptomatology might well have been that of an acoustic tumor, although attention should be called to the fact that trigeminal involvement subjectively came after other cranial nerve symptoms, which is perhaps unusual. Objectively, it was found that deafness was incomplete. This again is extremely unusual in an acoustic tumor. Furthermore, the absence of caloric responses from either ear should have aroused great suspicion. Hypesthesia of the right side with a positive Babinski sign could have been due to pressure on the brain stem by any type of growth in this region, but once more the presence of advanced cranial nerve and cerebellar involvement in a patient with normal fundi should be taken as the keynote.

Another interval of over a year passed by before the seventh of these patients presented himself for diagnosis. In this case deafness had been coming on over an extraordinarily long period, and was followed by the usual cranial nerve, cerebellar and, finally, general pressure symptoms.

CASE 7.—*History.*—G. H. G., a man, aged 42, was referred on Sept. 16, 1927, by Dr. J. T. Watkins of Detroit complaining of "deafness in the right ear, staggering, nausea and vomiting."

The family and past history were irrelevant. The patient first had noticed beginning deafness in the right ear nineteen years previously. This had increased progressively, but was not yet complete. He had never had tinnitus. Two and one-half years before admission, he had first been aware of some numbness around the back teeth. This feeling gradually spread throughout the upper jaw, then into the lower jaw and finally over the whole face. Twitching of the right side of the face also started about two and one-half years ago, beginning at the corner of the right eye and spreading from there to the whole cheek. Two years before entrance, he noticed dizziness and some unsteadiness of gait. The latter increased to the point of staggering, involving especially the right leg. For something less than two years he had had at intervals sudden attacks of projectile vomiting. For a year and a half diplopia had been present constantly, and for a year the speech had become thick and slurred. Difficulty in swallowing came on about six months before admission, and finally during the three months previous to entrance he had had headaches on straining.

Neurologic Examination.—There were revealed the following positive features: nystagmus to the right and left, slower to the right; rotary vertical nystagmus; right abducens palsy; hypesthesia of the right trigeminal over all three divisions with loss of the corneal reflex, and impairment of the motor fifth on the right; right facial paralysis of peripheral type; incomplete deafness of the right ear with caloric responses only partial; deviation of the soft palate to the left; deviation of the tongue to the right with absence of taste on the posterior one third of the tongue; paresis of the right sternocleidomastoid and trapezius muscles; marked ataxia and hypermetria of the right leg but very little incoordination in the arms; positive Romberg's sign with falling to the right; staggering gait; cerebellar dysarthria; the fundi were somewhat hyperemic but were placed within normal limits.

Clinical Diagnosis.—The preoperative consensus was that this patient had either an acoustic tumor or a meningioma of the right cerebellopontile angle.

Operation.—On Sept. 29, 1927, Dr. Cushing explored the suboccipital region and disclosed a tumor of the pons involving the medulla.

The patient survived two months, but died on November 29 from a pulmonary abscess.

Necropsy.—The right side of the pons was asymmetrically enlarged by a dense tumor which extended into the right cerebellopontile fossa. The fifth to the eleventh cranial nerves on the right side were distorted and out of their normal positions. Coronal sections of the brain stem showed a dense, grayish white, finely multicystic tumor, which replaced a portion of both the dorsal and basilar portion of the right side of the pons, the brachium pontis and the right cerebellar hemisphere. The fourth ventricle was distorted by the tumor.

Histologic sections showed the tumor to be a fibrillary astrocytoma.

Comment.—Once more the striking feature in reviewing this case is the absence of choked disks in the presence of extremely marked cranial nerve palsies, including not only the fifth, seventh and eighth, but the ninth, tenth, eleventh and twelfth as well. Furthermore, there was advanced cerebellar incoordination. It is almost impossible to picture such a situation with an acoustic tumor without internal hydrocephalus and high choked disks. Nevertheless, the chronology of symptoms was perfectly consistent with that which is seen in acoustic tumors.

It is, however, inconceivable that a story of deafness starting nineteen years previously should not have become complete if due to an acoustic growth, but it is equally difficult to explain such a long period of impaired hearing on the basis that it was caused by a pontile glioma. In other words, in view of the type of growth which we now know this patient harbored, it is probable that the originally beginning deafness was due to some other cause. This, however, could hardly have been surmised at the time.

Our final patient entered the clinic one year ago, and although tinnitus was the earliest symptom, deafness did not come on until very late in the illness.

Case 8.—*History.*—P. H. W., a woman, aged 37, was admitted to the hospital on April 1, 1929, complaining of dizziness and vomiting.

The family and past histories were irrelevant. The present illness began four years before admission with "buzzing" in the left ear, and had been constant since that time. For two years she had had occipital headaches and these had increased in severity during the past year. Four months previous to entrance, she had begun to stagger in walking, and during the two months before coming to the hospital she had been confined to bed because of extreme dizziness and vomiting. Deafness in the left ear had been present for only one month so far as she was aware, and also for a month she had had tinnitus in the right ear as well as the left. For a week she had had some numbness of the left cheek.

Neurologic Examination.—In the fundi the disks were hyperemic with blurred margins, and there was a measurable elevation of from 0.5 to 1 diopter in each eye. Both pupils reacted only slightly to light. There was persistent nystagmus to the right and the left, as well as rotary vertical nystagmus. Hyperesthesia of the left side of the face was present, but both corneal reflexes were absent. The left ear was deaf. Caloric responses showed that both labyrinths were inactive. She was unable to get out of bed to make the Romberg test and for us to study the gait, but there was marked ataxia and dysmetria of both arms, the left being worse than the right. There was questionable astereognosis of the left hand, and a little twitching of the right hand in two seizures with loss of consciousness which occurred the day before operation.

Operations.—On April 3, 1929, a suboccipital exploration was started, but on finding no dilatation of the ventricles early in the procedure the operation was abandoned, and ventriculograms were made. The latter proved unsatisfactory. On the following day the suboccipital exploration was carried out, disclosing a tumor at the left side of the pons invading the cerebellum.

The patient declined rather rapidly and died twelve days after the operation.

Necropsy.—Coronal sections showed a soft, partially necrotic, grayish white tumor infiltrating the left pars dorsalis of the pons, the left brachium pontis, the left cerebellar hemisphere and the left side of the dorsum of the medulla oblongata. It extended into and partially occluded the fourth ventricle. The right fifth cranial nerve was surrounded by tumor. The right acoustic nerve was not seen in the tumor mass.

The gross appearance of this large, soft, necrotic tumor resembled that of the rapidly growing type of glioma. Histologic sections showed it to be a glioblastoma multiforme.

Comment.—There were several features about this case which would make the diagnosis of an acoustic tumor doubtful, the most important among these being the late development of deafness. In addition, the caloric responses showed that both labyrinths were inactive, and as further evidence of a bilateral process, both corneal reflexes were absent. Another unusual observation for an acoustic growth was the inactivity of both pupils to light. The fundi in this patient did present a slight degree of choking, but the cranial nerve palsies, vomiting and ataxic symptoms were out of all proportion to the amount of pressure as judged by her disks.

COMMENT

In a review of the series as a whole it is at once apparent that the chronology of symptoms plays a leading part in the differentiation of pontile from acoustic growths. Cushing [3] gives the usual chronology in the latter type of tumors as follows: "first, the auditory and labyrinthine manifestations; second, the occipito-frontal pains with suboccipital discomforts; third, the incoordination and instability of cerebellar origin; fourth, the evidences of involvement of adjacent cerebral nerves; fifth, the indications of an increase in intracranial tension with a choked disc and its consequences; sixth, dysarthria, dysphagia and finally cerebellar crises and respiratory difficulties."

Bearing in mind this general sequence of events, we may take up the same symptoms in this pontile series in order to see where it differs. Deafness was the first thing complained of by four of the eight patients, but in one case there was some question as to whether it was not due to an old mastoid operation. In one other patient tinnitus came first, but in this patient deafness was next to the last symptom to appear. In the other three patients auditory symptoms were second once, fifth once and last once. Patients with pontile tumors apparently do not complain, as do those with acoustic growths, of suboccipital discomforts, which are the second group in the acoustic symptomatology. This in itself then may be taken as a differentiating point. The third manifestations are those having to do with instability, and in our pontile series unsteadiness came first in one patient, second in two, third in one, fifth in three and not at all in one. The fifth group of symptoms, involvement of other cranial nerves, came second in three patients, fourth in two, fifth in one, last in one and not at all in one. Evidences of increased intracranial pressure, the fifth group in acoustic cases, came second in two of our patients, third in two and last or next to last in the other four. As in the acoustic, so in the pontile, series dysarthria and dysphagia tended to come toward the end of the list of symptoms.

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3. Cushing, Harvey (footnote 1, p. 176).

What may be gleaned from this recital, then, is that whereas cases of acoustic tumors tend to follow a fairly definite order in the symptoms which develop, those of pontile growths do not follow any set order and, in particular, there is by no means so great a tendency for deafness to be the primary complaint. On the other hand, if deafness is the first symptom, and therefore confusion with an acoustic growth is likely to arise, the remaining chronology of symptoms should be scanned with exceeding care to see if it follows the usual acoustic order.

Another feature of great interest in differentiation is the degree of deafness in the two groups of patients, together with their response to the Bárány tests. It may be taken as the rule that patients harboring acoustic tumors are in almost every instance completely deaf on the side involved, and even if deafness is incomplete, irrigation of the affected ear gives no labyrinthine response. In the eight pontile cases here recorded, deafness was complete in five instances and partial in three. As to caloric reactions, these were carried out in only six of the eight patients. Of these there was no response to irrigation of either ear in three cases, a partial response on either side in one case, a normal response on both sides in the fifth, and in the sixth no response on the side of the affected ear. It would seem then that in these criteria we have perhaps our most useful clinical data for the differentiation of these two groups of cases, as only one pontile case showed the usual acoustic tumor reaction.

A third point, which already has been mentioned several times in the individual comments, relates to the appearance of the fundi. It is a striking fact that in seven of the eight cases the optic disks were either normal or showed only the earliest type of choking with a barely measurable elevation, and yet all of these patients had marked involvement of the cranial nerves together with advanced cerebellar symptomatology. Such a state of affairs must at least be a great exception in acoustic cases, because by the time such a tumor has grown to a sufficient size to cause this type of advanced neighborhood difficulty, it would almost certainly have begun to occlude the cerebrospinal fluid pathway, causing internal hydrocephalus and consequent papilledema.

Two other points should at least have passing mention. The first of these is that six of these pontile cases showed vertical nystagmus, usually in addition to lateral nystagmus, and one further case is recorded as showing "spontaneous and lateral nystagmus," in which "spontaneous" almost certainly means the vertical component. In the only other case, nystagmus was confined to the lateral movements of the eye. The other point concerns weakness of the arm and leg of one side. This feature was recorded as being present in three patients, and in one other there was said to be some questionable weakness of one arm. Certainly if such recorded weakness had been outspoken it would

have militated definitely against the diagnosis of an acoustic growth, and we can therefore only surmise that it was in all probability an observation on which sufficient stress was not laid, or perhaps was confused with the coexisting ataxia and hypotonia.

The pathology of these lesions is of interest for, although previously they were classified as pontile gliomas, it was found on further study that each of the tumors involved the cerebellar hemisphere, the brachium pontis and the adjacent pons. Furthermore, each of the tumors extended into and almost completely filled the adjacent cerebellopontile angle. The cranial nerves on the side of the lesion, especially the fifth, seventh and eighth, were either embedded in tumor, distorted in their course or were not found. The porus acusticus was examined in each case but found to be unchanged. While it was impossible to tell where these lesions had their origin, in certain cases the lesion appeared to be more extensive in the cerebellar hemisphere, and to have extended to the pons from the brachium pontis.

Histologic examination showed that seven of the tumors were gliomas of the slowly growing type, and but one was of the rapidly growing type classified in this clinic as a glioblastoma multiforme. The seven slowly growing tumors were either astrocytomas or unipolar spongioblastomas.

ABSTRACT OF DISCUSSION

Dr. Ernest Sachs, St. Louis: Dr. Horrax alluded to a very important point in his last sentences. The differentiation between tumors in the substance of the pons and tumors of the acoustic nerve is sometimes very difficult. One symptom has proved of particular value in this differentiation, namely, that in tumors in the substance of the pons the symptoms at a very early stage become bilateral, while this practically never is true of tumors of the acoustic nerve. Of course, it is easy to see why this should be so; the structures in the pons are so closely packed together that it is almost impossible to conceive of a lesion on one side of the pons not affecting the other.

BASAL METABOLIC RATE IN UNTREATED AND TREATED PATIENTS WITH EPILEPSY*

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The problem of the consumption of oxygen in so-called "idiopathic" epilepsy has interested a relatively limited number of workers. The paucity of recorded material may be due to the fact that institutionalized patients with epilepsy were the main subjects for the test; since the majority were psychotic, not all of them were suited for the study. In my own study I found it necessary to discard about fifty readings which did not represent the actual basal metabolic rate, because the patients were too psychotic to observe the necessary state of complete relaxation during the test. I made several attempts to obtain readings on these psychotic patients, but the effort had finally to be given up.

I have tabulated the observations in the two sexes separately to permit an easier comparison of possible deviations in both groups. An attempt was also made to correlate the metabolic rate with the age of the patients, and therefore the ages are given in the tables along with the basal metabolic readings. In this connection it must be pointed out that although some of the patients were of advanced age when the tests were made, they all belong to the "idiopathic" group of epilepsy. No cases with definite symptomatic epilepsy, that is, with obvious involvement of the central nervous system, like arteriosclerosis or tumor of the brain, were used, as the purpose of this study is to establish the consumption of oxygen only in the idiopathic variety.

As no contributions on the effect of sedative treatment on the basal metabolism are reported in the literature, I thought it would be of interest to compare the observations in different groups: untreated, treated with phenobarbital and treated with bromide, and finally to compare the metabolic readings previous to a course of treatment with bromide with those taken during the treatment.

METHODS

The usual precautions were taken to obtain basal conditions, the test being performed at least twelve hours after the last meal and after one hour of rest in bed immediately before the test, which was made in all cases at 9 a. m.

The measurements were made by means of the Benedict graphic metabolism apparatus, and the normal standards of Aub and Dubois were used.

Dealing with psychotic patients, it was of importance to gain their confidence in order to avoid excitement; it was therefore necessary for me to perform all the tests myself.

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BASAL METABOLIC RATE IN UNTREATED WOMEN AND MEN WITH EPILEPSY

The group of untreated patients subjected to the basal metabolism test consisted of fifty with epilepsy, twenty-nine women and twenty-one men. Some of them had recently been admitted to the Manhattan State Hospital; others had been inmates in the hospital for some time, but all treatment had been discontinued for periods of from three months to a year previous to the test.

TABLE 1.—*Basal Metabolic Rate in Untreated Epileptic Women*

Name	Metabolic Rate, Per Cent	Age
1. A. B.	+29.0	32
2. N. D.	+18.0	65
3. A. A.	+ 8.0	64
4. H. L.	+ 7.4	42
5. E. B.	+ 3.0	41
6. K. Z.	+ 1.4	39
7. M. N.	+ 1.0	27
8. F. R.	+ 0.3	40
9. M. G.	+ 0.05	19
10. J. L.	— 0.3	38
11. Y. G.	— 2.0	39
12. R. W.	— 2.1	67
13. M. L.	— 2.8	39
14. M. D.	— 3.7	49
15. P. P.	— 5.0	32
16. E. P.	— 5.0	37
17. G. T.	— 5.0	34
18. M. C.	— 5.4	24
19. M. G.	— 6.0	48
20. F. B.	— 7.0	36
21. M. M.	— 7.0	25
22. A. S.	— 9.0	36
23. S. B.	—10.0	26
24. L. C.	—10.0	29
25. A. S.	—11.0	39
26. M. C.	—14.0	50
27. M. P.	—15.0	34
28. B. M.	—17.0	19
29. C. B.	—22.0	43

Above	+10 per cent	6.89 per cent
Between	+10 per cent and —10 per cent	75.86 per cent
Below	—10 per cent	17.24 per cent
Total	— Readings	68.96 per cent

If one considers the sexes separately, one sees (tables 1 and 2) that in the group of untreated women there were 6.89 per cent with readings above + 10, 75.86 per cent with readings between + 10 and — 10, and 17.24 per cent with readings below — 10. In the group of untreated men patients there is a slightly higher percentage of readings above + 10 (9.52 per cent) and a slightly lower percentage of readings below — 10 (14.28 per cent); the percentage of normal readings between + 10 and —10 is somewhat higher than in the female group (76.19 per cent). The total of minus readings in the female group was higher than the total of minus readings in the male group, in the former being 68.96 per cent, in the latter 47.61 per cent. In a few instances unusually high or low basal rates were found in both sexes.

Comparing these observations with those reported in the literature, a few workers have obtained a greater number of readings below the normal end of the minus scale, that is, below —10. In 133 patients Lennox and Wright [1] found 31 per cent of the measurements outside the normal zone of 10 per cent above or below; 23 per cent of these were below 10; 12 per cent were below 15. In 68 per cent the reading was between + or —10, a somewhat lower percentage of normal readings than I obtained; the readings below —10 were somewhat greater than in my cases. Boothby and Sandiford [2] found 23 per cent of 22 cases —10 or lower.

TABLE 2.—*Basal Metabolic Rate in Untreated Epileptic Men*

Name	Metabolic Rate, Per Cent	Age
1. C. R.	+21.0	18
2. H. S.	+14.0	54
3. M. P.	+10.0	48
4. F. M.	+ 9.0	27
5. W. K.	+ 6.3	73
6. F. P.	+ 5.8	23
7. E. B.	+ 4.0	20
8. A. G.	+ 3.0	35
9. S. W.	+ 1.6	40
10. F. O.	+ 1.5	50
11. J. P.	+ 0.47	41
12. R. P.	— 3.0	25
13. E. P.	— 5.0	21
14. D. T.	— 6.9	20
15. H. P.	— 7.0	33
16. J. N.	— 8.0	22
17. F. Z.	— 8.0	37
18. L. T.	— 8.0	43
19. H. C.	—16.0	48
20. N. K.	—26.0	35
21. W. C.	—29.0	36

Above	+10 per cent	9.52 per cent
Between	+10 per cent and —10 per cent	76.19 per cent
Below	—10 per cent	14.28 per cent
Total	—Readings	47.61 per cent

As these authors were among the few who attempted to establish definite proportions, I reproduce their table:

		Per Cent
Below	—20	9.1
	—20 to —16	4.6
	—15 to —11	9.1
	—10 to +10	77.3
	+11 to +15	0.0
	+16 to +20	0.0
Above	+20	0.0
	—15 to +15	86.4

1. Lennox, W. G., and Wright, L. H.: Studies in Epilepsy: The Basal Metabolism, Arch. Neurol. & Psychiat. **20**:764 (Oct.) 1928. Lennox, W. G., and Cobb, S.: Epilepsy, Baltimore, Williams & Wilkins Company, 1928.

2. Boothby, W. M., and Sandiford, I.: Summary of Basal Metabolism Data on 8,614 Subjects with Especial Reference to Normal Standards for the Estimation of the Basal Metabolic Rate, J. Biol. Chem. **54**:783, 1922.

It will be seen that the number of normal results of Boothby and Sandiford correspond to mine, but they did not have any readings above + 10; their percentage of readings below — 10 is greater than in my study. Davis,[3] in a recent study of forty patients, came to the conclusion that patients who are subject to convulsive seizures tend to have a basal metabolism lower than normal. His figures, however, give the following percentages: 60 per cent with normal readings, 5 per cent above + 10 and 35 per cent below — 10. Here again the percentage with lower than normal readings is almost twice as high as in my cases. On the other hand, Frisch,[4] in a study of forty patients, found positive values in the majority. He gave actual figures, however, for only six patients, of whom five had readings more than 10 per cent above normal. Bowman and Fry,[5] in fifteen cases, found the readings mostly within normal limits, with a tendency toward minus readings and a mean of — 2. Nielson,[6] in eighteen patients, reported average readings of — 10. Talbot, Hendry and Moriarty,[7] in fifteen children with epilepsy, obtained rates between — 9 and + 45 with an average of + 15 per cent. The higher percentage of readings below — 10 in some of the reports may perhaps be explained by the possibility that these patients were receiving some kind of antispasmodic (sedative) treatment. No mention is made by any of these workers as to whether or not their patients were under some form of therapy at the time when the tests were made. As will be seen later, my observations of epileptic patients treated with phenobarbital or bromide gave a greater percentage of readings below — 10.

The age of the patients, as is seen from the tables, does not have any particular bearing on the basal metabolic rate; I found ages varying from 20 to 73 in the group of men with epilepsy, and from 19 to 67 in the group of women with epilepsy with normal basal metabolic readings. Equally varied ages are present in both sexes in the group with readings above + 10 and below — 10.

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3. Davis, T. R.: Basal Metabolism and Gastro-Intestinal Findings in Epileptic Patients, with a Note on Treatment, J. Nerv. & Ment. Dis. **70**:264, 1929.

4. Frisch, F.: Der respiratorische Gaswechsel der Epileptiker, Ztschr. f. d. ges. exper. Med. **56**:118, 1927.

5. Bowman, K. M., and Fry, C. C.: Basal Metabolism in Mental Disease, Arch. Neurol. & Psychiat. **14**:819 (Dec.) 1925.

6. Nielson, J. M.: Etiology of Epileptiform Convulsions, M. J. & Rec. **112**: 338, 1925.

7. Talbot, F. B.; Hendry, M., and Moriarty, M.: Basal Metabolism of Children with Idiopathic Epilepsy, Am. J. Dis. Child. **28**:419 (Oct.) 1924.

BASAL METABOLIC RATE IN EPILEPTIC PATIENTS TREATED WITH PHENOBARBITAL

In order to study the effect of phenobarbital on the basal metabolic rate, tests were made on a mixed group of sixteen patients (table 3) who had been receiving phenobarbital for periods of from one to nine years. In only one instance was there a rating above + 10; 50 per cent of the readings were normal—between + 10 and — 10; and 43.75 per cent were below — 10. In the normal range of metabolic rates there was a drop of 25 per cent in the number of treated patients as compared with untreated patients, and an increase in the percentage of readings

TABLE 3.—*Basal Metabolic Rate in Epileptic Patients Treated with Phenobarbital*

Name	Metabolic Rate, Per Cent
1. F. M.	+17.0
2. J. M.	+ 8.0
3. H. D.	+ 8.0
4. N. G.	+ 7.0
5. A. H.	+ 2.0
6. M. L.	+ 1.0
7. G. Z.	— 3.0
8. J. M.	— 9.0
9. K. C.	—10.0
10. A. P.	—11.0
11. M. R.	—12.0
12. W. H.	—14.0
13. A. B.	—15.0
14. E. M.	—16.0
15. J. B.	—18.0
16. R. B.	—26.0

Above	+10 per cent	6.25 per cent
Between	+10 per cent and —10 per cent	50.00 per cent
Below	—10 per cent	43.75 per cent
Total	—Readings	62.50 per cent

below — 10 (43.75 per cent as against 14.28 per cent in men and 17.24 per cent in women) as compared with untreated patients.

BASAL METABOLIC RATE IN EPILEPTIC PATIENTS TREATED WITH BROMIDE

Twenty-six women patients who had been treated with bromide for periods of from six months to three years were used in this group. Here also there was a reduction of the basal metabolic rate. In none of the cases (table 4) was there a basal metabolism of + 10 or above, the highest being + 8; 30.76 per cent were below — 10, as compared with 14.28 per cent in the untreated male group and 17.24 per cent in the untreated female group; 69.23 per cent were between — 10 and + 10, and the total number of minus readings was 73 per cent, or 10.5 per cent greater than in the group treated with phenobarbital.

BASAL METABOLIC RATE IN EPILEPTIC PATIENTS BEFORE AND DURING A COURSE OF TREATMENT WITH BROMIDE

For the purpose of a more accurate estimation of the influence of bromide on the basal metabolism, I selected twelve women patients who had been admitted in the past three years. None of them had received

TABLE 4.—*Basal Metabolic Rate in Epileptic Patients Treated with Bromide*

Name	Metabolic Rate, Per Cent
1. S. G.	+ 8.0
2. A. S.	+ 7.0
3. P. R.	+ 6.0
4. A. B.	+ 1.6
5. M. N.	+ 0.9
6. M. D.	+ 0.8
7. A. S.	+ 0.5
8. F. R.	— 0.04
9. S. B.	— 0.7
10. R. B.	— 0.7
11. G. T.	— 1.5
12. K. Z.	— 4.0
13. M. C.	— 5.0
14. M. B.	— 7.0
15. E. P.	— 8.0
16. A. S.	— 9.0
17. N. D.	—10.0
18. E. B.	—10.0
19. S. B.	—11.0
20. L. H.	—11.0
21. H. D.	—11.0
22. E. R.	—12.0
23. A. S.	—12.0
24. F. H.	—14.0
25. H. H.	—15.0
26. M. P.	—17.0

Above	+10 per cent	0.00 per cent
Between	+10 per cent and —10 per cent	69.23 per cent
Below	—10 per cent	30.76 per cent
Total	—Readings	73.00 per cent

TABLE 5.—*Basal Metabolic Rate in Epileptic Patients Before and During a Course of Treatment with Bromide*

Name	Before Treatment, Per Cent	During Treatment, Per Cent
1. A. B.	+29.0	+ 1.6
2. N. D.	+18.0	—10.0
3. E. B.	+ 3.0	—10.0
4. K. Z.	+ 1.4	— 4.0
5. M. N.	+ 1.0	— 0.9
6. F. R.	+ 0.3	— 0.04
7. M. D.	— 3.7	+ 0.8
8. G. T.	— 5.0	— 1.5
9. E. P.	— 5.0	— 8.0
10. A. S.	— 9.0	+ 0.5
11. S. B.	—10.0	—11.0
12. M. P.	—15.0	—17.0

any form of therapy for a month or longer during the period of observation, and a basal metabolism test was made on each during this period. They were then given bromide therapy, and after several months of treatment the basal metabolism was again measured. Of the twelve patients (table 5), seven showed a pronounced decrease in the metabolic rate. For instance, in case 1 in table 5 there was a drop from + 29 to

+1.6, and from + 18 to — 10 in case 2. In a few instances, cases 7, 8 and 10, there was a slight increase in the metabolic rate. However, if one compares the two columns in this table, one will see that there is only one instance with a reading above + 1 in the patients treated with bromide. Patients having a rate above 0 before treatment seemed to show a greater reduction of basal metabolism under bromide treatment than patients whose rate was already low.

SUMMARY

The basal metabolic rate in a group of fifty untreated epileptic patients (twenty-nine women and twenty-one men) was normal in a high percentage in both sexes; my observations show a relatively smaller group of patients with a rate below — 10 than the reports of some other workers; there is a higher percentage of below — 10 readings in the female group than in the male group.

No correlation could be established between the chronological age and the basal metabolic rate.

The basal metabolic rate in a group of sixteen epileptic patients treated with phenobarbital showed a considerable decrease in the percentage of normal readings and a threefold increase of the percentage of readings below — 10. I conclude that phenobarbital definitely decreases the basal metabolism.

The basal metabolic rate of a group of twenty-six patients treated with bromide showed no rate above the normal range, a decrease in the percentage within the normal range and an increase in the percentage of the lower readings.

A comparative study of basal metabolism in a group of twelve patients before treatment and during the course of treatment with bromide tends to show a decrease, with only a few exceptions. The decrease is marked in some instances.

OBSERVATIONS ON CEREBROSPINAL FLUID OBTAINED AT THE TIME OF THE INJECTION OF AIR

A PRELIMINARY REPORT *

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Massive quantities of cerebrospinal fluid obtained at the time of the insufflation of air into the lumbar subarachnoid space may or may not show variations in the commonly analyzed elements when they are subjected to fractional examination. The variations noted are limited almost entirely to the cell content. The results of analysis for the other fluid constituents were relatively uniform.

Because of the constant, rapidly appearing headache, stiff neck and fairly frequent subsequent nausea, vomiting and fever in persons who have submitted to encephalography, air is considered to be irritating to the meninges. Herrmann [1] tended to substantiate this idea by observing a marked pleocytosis (chiefly polymorphonuclear leukocytes) and a high albumin content in cerebrospinal fluid obtained as early as fourteen hours after spinal inflations were performed on three psychotic patients. Friedman, Snow and Kasanin [2] reported a similar cellular increase in the fluid from three of forty-seven patients who had had encephalographies performed. In the latter report, the intervals between the procedures and the subsequent spinal punctures were not given. Undoubtedly, air is a meningeal irritant and the reaction is rapid, but a record showing the condition of the cerebrospinal fluid withdrawn at the time of the injection of air has not been found in the literature. Such data form the basis of this report.

Fractional examinations were made on cerebrospinal fluid obtained in such a manner from adult patients who were under observation and treatment in the neurologic department of the State University of Iowa, Iowa City. In most instances a diagnostic lumbar puncture had been performed, usually several days preceding the injection of air, and these observations on the fluid are given for comparison (table 1). Only one relatively early follow-up puncture was done.

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* Submitted for publication, Dec. 20, 1928.

1. Herrmann, G.: Cerebrospinal Fluid After Inflation with Air, Med. Klin. **18**:1146 (Sept. 3) 1922.

2. Friedman, E. D.; Snow, W., and Kasanin, J. I.: Experiences with Encephalography via Lumbar Route, Arch. Neurol. & Psychiat. **19**:762 (May) 1928.

TABLE 1.—*Routine Analyses of Lumbar and Fractionated Fluids*

Case	Diagnosis	Date	Procedure	Fluid Character	Cells per C.Mm.		Globulin Content	Meyers Reaction	Wassermann Reaction
					Lymphocytes	Polymorphonuclears			
1	Migraine	11/23/27	Lumbar puncture	Clear	3	0	Trace	0	0
		12/13/27	Encephalography						
			Sample 1	Clear	1	0	0	0	..
			Sample 6	Clear	15	2	0	0	..
		2/17/28	Lumbar puncture	Clear	1	0	1 plus	0	0
		7/ 5/28	Lumbar puncture	Clear	1	0	0	0	0
2	Migraine	11/22/27	Lumbar puncture	Clear	2	0	Trace	0	0
		11/29/27	Encephalography						
			Sample 1	Clear	1	0	Trace	0	0
			Sample 2	Clear	3	0	Trace	0	..
			Sample 3	Clear	2	0	Trace	0	..
			Sample 4	Clear	9	0	Trace	0	..
			Sample 5	Clear	13	1	Trace	0	..
			Sample 6	Clear	19	3	Trace	0	..
3	Migraine	8/29/28	Lumbar puncture	Clear	2	0	0	0	0
		9/15/28	Lumbar puncture	Clear	3	0	0	0	0
		9/18/28	Encephalography						
			Sample 1	Clear	2	0	0	0	..
			Sample 2	Clear	4	0	0	0	..
			Sample 3	Clear	4	0	0	0	..
			Sample 4	Clear	6	0	0	0	..
			Sample 5	Clear	7	0	0	0	..
			Sample 6	Clear	8	0	0	0	..
4	Idiopathic epilepsy	4/19/27	Lumbar puncture	Clear	0	0	0	0	0
		6/13/27	Encephalography						
			Sample 1	Clear	0	0	0	0	..
			Sample 5	Clear	6	0	0	0	..
5	Idiopathic epilepsy with mental deterioration	3/15/28	Lumbar puncture	Clear	0	0	0	0	0
		3/24/28	Encephalography						
			Sample 1	Clear	1	0	0	0	..
			Sample 2	Clear	4	0	0	0	..
			Sample 3	Moderately bloody	..	..	Trace	4 plus	..
			Sample 4	Bloody	..	..	..	4 plus	..
6	Idiopathic epilepsy with acute mania	6/30/28	Lumbar puncture	Clear	3	0	Trace	0	0
		7/13/28	Encephalography						
			Sample 1	Clear	0	0	0	0	..
			Sample 2	Clear	3	0	0	0	..
			Sample 3	Clear	12	0	0	0	..
			Sample 4	Clear	16	1	0	0	..
			Sample 5	Clear	23	2	0	0	..
7	Idiopathic epilepsy; vascular neurosyphilis; moron; blood Wassermann 4 plus	3/19/28	Lumbar puncture	Clear	4	0	1 plus	0	0
		4/16/28	Encephalography						
			Sample 1	Clear	1	0	Trace	0	0
			Sample 2	Clear	4	1	Trace	0	0
			Sample 3	Clear	5	1	Trace	0	0
			Sample 4	Clear	10	0	Trace	0	0
			Sample 5	Clear	9	0	Trace	0	0
			Sample 6	Clear	13	1	Trace	0	0
8	Chronic encephalitis; parkinsonian syndrome; diabetes insipidus syndrome	3/ 6/27	Lumbar puncture	Clear	1	0	0	0	0
		4/16/27	Encephalography						
			Sample 1	Clear	2	0	0	0	..
			Sample 6	Clear	8	0	1 plus	0	..
9	Acute epidemic encephalitis	6/17/27	Lumbar puncture	Clear	3	1	Trace	0	0
		6/28/27	Encephalography						
			Sample 1	Clear	4	0	Trace	0	..
			Sample 6	Clear	27	2	Trace	0	0
		7/ 2/27	Lumbar puncture	Clear	23	1	Trace	0	0
		11/ 1/27	Lumbar puncture	Clear	4	2	0	0	0
10	Acute epidemic encephalitis	11/ 4/27	Lumbar puncture	Clear	3	1	Trace	0	0
		11/22/27	Lumbar puncture	Clear	4	0	0	0	0
		12/ 9/27	Encephalography						
			Sample 1	Clear	11	0	Trace	0	..
			Sample 2	Clear	16	0	Trace	0	..
			Sample 3	Clear	19	0	Trace	0	..
			Sample 4	Clear	24	0	Trace	0	..
			Sample 5	Clear	34	0	Trace	0	..
			Sample 6	Clear	43	0	Trace	0	..

TABLE 1.—*Routine Analyses of Lumbar and Fractionated Fluids—Continued*

Case	Diagnosis	Date	Procedure	Fluid Character	Cells per C.Mm. Lymphocytes	Cells per C.Mm. Polymorphonuclears	Globulin Content	Meyers Reaction	Wassermann Reaction
11	Subacute cerebritis	3/19/27	Lumbar puncture	Clear	4	0	Trace	0	0
		4/ 2/27	Encephalography						
			Sample 1	Clear	4	0	Trace	..	..
			Sample 6	Clear	26	5	1 plus	0	..
12	Neurosyphilis; dementia paralytica	3/ 4/27	Lumbar puncture	Clear	60	9	1 plus	0	4 plus
		6/21/27	Lumbar puncture	Clear	14	0	Trace	0	4 plus
		12/ 3/27	Lumbar puncture	Clear	32	5	1 plus	0	4 plus
		4/ 9/27	Lumbar puncture	Clear	5	0	1 plus	0	4 plus
		9/22/27	Encephalography						
			Sample 1	Clear	11	0	1 plus	0	4 plus
			Sample 2	Clear	9	1	1 plus	0	4 plus
			Sample 3	Clear	13	2	1 plus	0	4 plus
			Sample 4	Clear	13	2	1 plus	0	4 plus
			Sample 5	Clear	45	4	1 plus	0	4 plus
			Sample 6	Clear	69	9	1 plus	0	4 plus
13	Brain tumor	3/18/27	Lumbar puncture	Clear	4	0	1 plus	0	0
		4/ 4/27	Encephalography						
			Sample 1	Clear	5	0	1 plus	0	..
			Sample 6	Clear	14	0	1 plus	0	..
14	Brain tumor	4/ 9/27	Lumbar puncture	Clear	8	0	1 plus	Trace	0
		4/13/27	Encephalography						
			Sample 1	1+ bloody	6	0	Trace	3 plus	..
			Sample 3	1+ bloody	9	9	1 plus	4 plus	..
15	Brain tumor	4/ 4/27	Lumbar puncture	Clear	5	0	2 plus	0	2 plus
		4/16/27	Encephalography						
			Sample 1	Clear	3	0	1 plus	0	0
			Sample 2	Clear	—	—	—	—	0
			Sample 3	Clear	—	—	—	—	0
			Sample 4	Clear	—	—	—	—	0
			Sample 5	Clear	—	—	—	—	0
			Sample 6	Clear	11	0	2 plus	0	0
16	Brain tumor	6/25/28	Lumbar puncture	Clear	2	1	Trace	0	0
		7/12/28	Encephalography						
			Sample 1	Clear	3	0	0	0	..
			Sample 2	Clear	9	1	0	0	..
			Sample 3	Clear	28	0	0	0	..
17	Brain tumor	8/28/28	Lumbar puncture	Clear	2	0	0	0	0
		9/21/28	Encephalography						
			Sample 1	Clear	1	0	0	0	..
			Sample 2	Clear	2	0	0	0	..
			Sample 3	Clear	2	0	0	0	..
			Sample 4	Clear	4	0	0	0	..
			Sample 5	Clear	5	0	0	0	..
			Sample 6	Clear	19	2	0	0	..
18	Brain tumor	3/10/27	Lumbar puncture	Clear	5	0	0	0	0
		4/ 1/27	Encephalography						
			Sample 1	Clear	4	0	Trace	0	..
			Sample 2	Slightly bloody	14	0	Trace	1 plus	..
			Sample 3	Bloody	—	—	—	4 plus	..
		4/23/27	Encephalography						
			Sample 1	Clear	5	0	Trace	0	..
			Sample 5	Clear	7	1	Trace	0	..
19	Brain tumor	3/15/28	Lumbar puncture	Clear	3	0	2 plus	0	0
		3/24/28	Encephalography						
			Sample 1	Clear	3	0	2 plus	0	..
			Sample 2	Clear	6	1	2 plus	Trace	..
			Sample 3	Clear	6	0	3 plus	0	..
			Sample 4	Clear	10	3	3 plus	0	..
			Sample 5	Clear	8	3	3 plus	0	..
			Sample 6	Clear	14	4	3 plus	0	..
20	Brain tumor	1/11/28	Encephalography						
			Sample 1	Clear	4	0	1 plus	0	..
			Sample 2	Clear	3	0	0	0	..
			Sample 3	Clear	5	0	0	0	..
			Sample 4	Clear	4	0	0	0	..
			Sample 5	Clear	5	0	0	0	..
			Sample 6	Clear	15	0	1 plus	0	..

Twenty-one diagnostic or therapeutic encephalographic procedures were performed on twenty patients who presented cerebral disorders (table 1). Six of these had functional and fourteen had organic cerebral disturbances. The functional disorders were cases of either severe idiopathic epilepsy or migraine. Eight intracranial tumors and six cerebral inflammatory lesions comprised the organic group. The inflammatory reactions were due to subacute cerebritis, acute or chronic epidemic encephalitis and vascular or parenchymatous neurosyphilis. The syphilitic patients had received 9 and 28.8 Gm. of neoarsphenamine, respectively; both had been given the last intravenous treatment at least three months before the fractional analyses were made. Although four cases

TABLE 2.—*Data on Encephalographic Procedure*

Case	Anesthesia	Posture	Amount of Fluid Out	No. of Samples	Amount of Air In	Time, Minutes	X-Ray Film Shows Air In: Ventricles	X-Ray Film Shows Air In: Cerebral Subarachnoid Spaces
1	Ethylene	Erect	90 cc.	6	80 cc.	5–10	Yes-Lateral	Yes
2	Ethylene	Erect	100 cc.	6	90 cc.	5–10	Yes-Lateral	Yes
3	Ethylene	Erect	100 cc.	6	90 cc.	5–10	Yes-Lateral	Yes
4	Ethylene	120	88 cc.	5	80 cc.	5–10	Yes-Lateral	Yes
5	Ethylene	Erect	60 cc.	4	45 cc.	5–10	Yes-Lateral	Yes
6	Ethylene	Erect	80 cc.	5	75 cc.	5–10	Yes-Lateral	Yes
7	Ethylene	Erect	105 cc.	6	95 cc.	5–10	Yes-Lateral	Yes
8	Ethylene	120	90 cc.	6	90 cc.	5–10	Yes-Lateral	Yes
9	Ethylene	Erect	100 cc.	6	100 cc.	5–10	Yes-Lateral	Yes
10	Ethylene	Erect	100 cc.	6	90 cc.	5–10	Yes-Lateral	Yes
11	Ether	Erect	95 cc.	6	102 cc.	5–10	Yes-Lateral	Yes
12	Local	165	120 cc.	6	110 cc.	5–10	No	Yes
13	Nitrous oxide	135	90 cc.	6	100 cc.	5–10	Yes to third	Yes
14	Ether	Erect	40 cc.	3	45 cc.	5–10	No	Yes
15	Ether	Erect	84 cc.	6	80 cc.	5–10	No	Yes
16	Ether	Erect	45 cc.	3	40 cc.	5–10	No	Yes
17	Local	135	110 cc.	6	100 cc.	40	Yes-Lateral	Yes
18	Ether	Erect	45 cc.	3	55 cc.	5–10	Yes-Lateral	Yes
18	Ether	Erect	74 cc.	5	80 cc.	5–10	No	Yes
19	Ethylene	Erect	100 cc.	6	90 cc.	5–10	No	Yes
20	Ethylene	Erect	100 cc.	6	90 cc.	5–10	Yes to third	Yes

of tumor and one of acute epidemic encephalitis presented swollen optic nerve heads, there were no complications or deaths from the injection of air.

METHODS

For the encephalographic procedures a variety of anesthesias and postures were used (table 2). The punctures were made between the third and fourth lumbar vertebrae. In all but one instance the withdrawal of the fluid and the injection of air took between five and ten minutes. For this one insufflation, forty minutes were consumed. The fluid was caught in medicine glasses in samples of approximately 15 cc. each, given consecutive numbers as withdrawn, and an equivalent amount of filtered air was injected after each specimen was obtained This process was continued until it was thought that the exchange was sufficient, air bubbled back through the needle or the fluid became increasingly bloody. From 80 to 110 cc. of air was injected in sixteen cases. Air began to return without apparent cause at 40, 45 and 74 cc. in cases 14 and 16, and during the second procedure in case 18.

The fluid was increasingly bloody in two instances (case 5 and the first procedure in case 18). In the majority of cases from 5 to 10 cc. less air was injected than fluid withdrawn, and whenever the converse condition was present an attempt was made to equalize the exchange by allowing air to escape. Roentgen examination revealed air in the third ventricle of two patients, in the lateral ventricles of thirteen and in the cerebral subarachnoid spaces of all of the patients. It would be impossible to state definitely the area from which each sample was obtained. The first specimen from every patient undoubtedly was from the spinal space, but there probably was a mixture of fluid from the cerebral and spinal subarachnoid spaces and the cisterna and at times from the ventricles in the late samples. The later fluid very likely was chiefly from the cerebral spaces and cavities.

The analyses done as a routine procedure were made on either the first and last or all of the samples, within thirty minutes after the fluid was obtained. A solution of gentian violet and glacial acetic acid was used as a stain for the total and differential cell counts and Noguchi's test for the globulin content. The reaction to Meyer's reagent and hydrogen dioxide was observed on each analyzed specimen to prevent error from occult blood. In cases indicating it, the Wassermann test was carried out on the fractioned samples. Few examinations were made for the other constituents. In some instances the sugar, total protein, uric acid and creatinine contents and the reactions to the Lange test were noted serially.

TABLE 3.—*Comparison of the Highest Cell Count in Each Series with the Respective Diagnoses*

Total Cells per C.Mm.			
0 to 9	10 to 19	20 to 29	Above 30
4 Idiopathic epilepsy	11 Brain tumor	21 Brain tumor	31 Subacute cerebritis
6 Idiopathic epilepsy	14 Brain tumor	22 Migraine	43 Acute epidemic encephalitis
8 Migraine	14 Brain tumor	25 Idiopathic epilepsy	78 Neurosyphilis-dementia paralytica
8 Chronic epidemic encephalitis	14 Neurosyphilis-vascular	28 Brain tumor	
8 Brain tumor	15 Brain tumor	29 Acute epidemic encephalitis	
	17 Migraine		
	18 Brain tumor		
	18 Brain tumor		

RESULTS

The last sample of fluid in every case showed an increase in the cell content. When counts were made on every specimen, the cellular increases were present, though usually slight, until the last one or two samples were reached. The results of the counts on the last 15 or 30 cc. of cerebrospinal fluid obtained were remarkable, because they were always relatively high.

After grouping the cases according to the greatest count of each series, it was found that five remained below 10, thirteen ranged between 10 and 30, and three were above 30 cells per cubic millimeter (table 3). The ratios between the first and last counts were greatest in the functional disorders and least in the fluid from patients with acute or subacute cerebral inflammatory lesions. However, the highest cell counts were noted in the last samples from the patients with such inflam-

matory processes. Especially interesting was the high cell count of the last specimen from the patient with neurosyphilis of the dementia paralytica type. The mononuclear cells predominated throughout. Polymorphonuclear leukocytes appeared late if at all. There were at least three predominant sources for these cells: (1) those normally present in the fluid before the injections, (2) those due to the immediate pia-arachnoid and perivascular tissue responses and (3) those which may have been present as an exudate before the procedures.

A slight rise in the globulin content was noted in the later samples in five cases. One of these increases undoubtedly was due to the mixture of blood with the fluid. The other differences were noted in cases of tumor of the brain (two), subacute cerebritis and chronic epidemic encephalitis. The other fluids had the globulin distributed uniformly. The reactions to the Wassermann tests were constant and checked with

TABLE 4.—*Extra-Routine Analyses of Fractionated Fluid* *

	Sugar Content				Uric Acid Content		Creatinine Content		Total Protein Content,
Sample	Case 6	Case 12	Case 16	Case 17	Case 6	Case 16	Case 6	Case 16	Case 19
1	91	62	108	103	1.22	1.30	0.5	0.5	44.2
2	84	56	101	82	1.16	1.23	0.5	0.5	44.0
3	77	62	95	88	1.13	1.27	0.5	0.5	45.8
4	71	63	...	79	1.12	....	0.5	...	44.3
5	68	54	...	95	1.11	....	0.5	...	42.1
6	69	60	...	100	1.02	....	0.5	...	44.1

* The results are expressed in mg. per hundred cubic centimeters of cerebrospinal fluid.

those of the diagnostic fluids with one exception, that being a 2 plus report on the diagnostic fluid in case 15. The subsequent negative serial reactions and the discovery at operation of a tumor of the brain, together with complete lack of other clinical evidence of syphilis in this patient, seem to prove conclusively that the positive report was false. With the exception of consistent decreases in the sugar content in case 6, there was very little fluctuation in the values of the other fluid constituents that were analyzed fractionally (table 4). The reactions to the Lange test noted in cases 7 and 12 were uniform.

COMMENT

It is realized that definite conclusions cannot be drawn from so limited a series of cases, but a few interesting facts and possibilities present themselves. It would seem that in these cerebral conditions the globulin, sugar, total protein, creatinine and uric acid contents and the syphilitic antibodies tended to be uniformly distributed through the cerebrospinal fluid and were not affected by the immediate responses of the meninges to this irritant. The increasing cell counts on the fractionated

samples show that the meningeal reactions to air were definite and prompt. The early cellular responses were predominantly lymphocytic, which was in distinct contrast to the known polymorphonuclear leukocyte exudates noted in fourteen (Herrmann) or more hours (Friedman) after the injection of air. In one instance (case 9, table 1) the lumbar fluid contents apparently were returning to normal eighty-four hours after the encephalography. This patient's meningeal syndrome had been moderate. A review of the maximum cell counts (table 3) might raise the question as to whether the air alone was responsible for such variable meningeal reactions. Other factors may have entered at times to cause, in part, some of these responses. The types of anesthesia and postures of the patients did not seem to have any particular bearing on the observations. The time factor was constant, with one exception (case 17, tables 1 and 2) in which the exchange consumed four times the time taken for the other processes, but this relatively prolonged contact of the meninges with air did not show conspicuous results in the number or type of cells produced. The normal and relatively normal meninges of the majority of these patients must have been unequally irritable and certainly, if the inflammatory lesions of the acute and subacute type were adjacent to the meninges, they could be expected to influence the immediate response.

Clinical Notes

THROMBOSIS OF THE CAROTID AND MIDDLE CEREBRAL ARTERIES WITH BILATERAL HEMORRHAGIC OPTIC NEURITIS*

S. A. Agatston, M.D., New York

History.—A. W., a man, aged 55, married, a brush maker, who was born in Austria, was admitted to the Montefiore Hospital on Feb. 6, 1930, with a right hemiplegia and aphasia. The family and past history was without significance. He had been suffering from an infection of the large toe on the right foot during July, 1929. In August, when the infection was beginning to clear up, he began to complain of daily spells of headache, dizziness and a feeling of nausea. Between August and September, he lost consciousness on three occasions. He would fall suddenly and would have to be picked up and brought home. Following these episodes the patient knew that he had been unconscious. He complained of dimness of vision, and black and white spots appeared before the eyes. There was no diplopia. One day in October he returned home from work, felt tired and went to bed; his son noticed that he was unable to move the right arm. Three days later he lost the power of speech, although he understood perfectly. About one week later ability to understand diminished. At this time he also began to suffer from weakness in the right lower extremity, but power was not as greatly impaired as in the right upper extremity. The patient was treated at various hospitals, but showed no improvement. There was a gradual progressive weakness of the right side. Speech became more and more impaired, and the patient was transferred from the Metropolitan Hospital to the Montefiore Hospital.

Physical Examination.—The heart was slightly enlarged to the left; the sounds were of good quality, and there were no murmurs. The blood pressure was 140 systolic and 88 diastolic. There was only a slight amount of vascular sclerosis. Rectal examination gave negative results. The large toe of the right foot showed signs of chronic infection.

Neurologic Examination.—The patient had a right hemiplegia with aphasia. The right upper extremity was markedly involved. The right lower extremity showed some impairment of motor power but the patient could flex and extend the limb. The aphasia was of both motor and sensory types. The deep reflexes were hyperactive on the right side; the abdominal and cremasteric reflexes were absent; a positive Babinski sign and other confirmatory signs were present. Sensation could not be tested accurately, but it appeared as if there was some sensory involvement of the right side. There was also a suspicion of a right homonomous hemianopia. The cranial nerves appeared to be uninvolved, except for a slight supranuclear facial palsy on the right side.

* Submitted for publication, July 2, 1930.

* From the Neurological and Ophthalmological Service of Montefiore Hospital.

* Read before the New York Academy of Medicine, Section of Ophthalmology, May 19, 1930.

There was a marked bilateral choked disk, with hemorrhages and thrombi of the retinal vessels. The swelling measured about 4 diopters in each eye. Following my examination of the fundus, I suggested that the choking was caused by venous distention and not by intracranial pressure.

Laboratory Data.—The Wassermann reaction of the blood was negative. All other laboratory data were negative except the x-ray picture of the skull which showed an area of destruction of the bone in the parietal region. Roentgen examination of the lungs gave negative results.

Operation.—The patient was transferred to the surgical division about one week after admission to the hospital and was operated on for a neoplasm of the brain in the left frontoparietal region. The vessels had a wormy appearance and contained no blood. The cortex had a soft, mushy appearance, but no neoplasm was found.

Postoperative Course.—At the end of the operation the patient had a convulsion of the right side and the left pupil appeared larger than the right. He never regained consciousness. The temperature rose to 106.2 F. The patient died on February 21.

Diagnosis.—The clinical diagnosis was tumor of the brain in the left frontoparietal region. The anatomic diagnosis was healed thrombosis of the left internal carotid artery and encephalomalacia.

Neuropathologic Report (Dr. Charles Davison).—Macroscopic Observations: The left hemisphere of the brain was smaller than the right. The portion involving the third frontal convolution and all of the temporal, motor and parietal convolutions, as well as part of the occipital lobes, were soft to the touch. There was a subarachnoid hemorrhage extending over the precentral, central and superior parietal areas (postoperative). Some of the vessels over the parietal lobes were free from blood. The left middle cerebral artery had an atheromatous plaque and was occluded. The rest of the vessels did not show atheromatous changes.

The brain seemed firm on being cut horizontally in the region of the lateral ventricle. The section had the appearance of softening with areas of hardening in the third frontal, motor and parietal regions. One nodule in the parietal area had the appearance of a tumor mass. Both ventricles were somewhat obliterated, the right more than the left. The section in the region of the diencephalon showed an area of destruction extending from the third frontal lobe to the occipital lobe. The left internal capsule and the lenticular nuclei were completely destroyed.

A piece of the upper cervical cord, as well as a whole slab of the brain, was embedded.

Microscopic Observations: A section of the brain from the area of softening stained with hematoxylin and eosin showed numerous compound granular corpuscles, some of which were multinucleated. Part of the tissue showed a reabsorption with an attempt at replacement by glial elements. The blood vessels showed endarteritic changes and in places were surrounded by varieties of glial elements. There was a marked pigmentation within the destroyed area. The microscopic diagnosis was cerebral arteriosclerosis, and thrombosis of the middle cerebral artery.

Ophthalmoscopic Diagnosis.—The optic neuritis differed from the ordinary choked disk by the moderate swelling with markedly dilated and thrombotic veins and numerous peripapillary hemorrhages. The ophthalmoscopic picture was suggestive of venous obstruction, especially as there was a rapid loss of vision. Autopsy (thrombosis of the carotid) proved that the optic neuritis was of vascular origin, having evidently been caused by secondary venous stasis and multiple thrombus formation.

News and Comment

NATIONAL COMMITTEE FOR MENTAL HYGIENE ANNOUNCES FELLOWSHIPS FOR TRAINING IN EXTRAMURAL PSYCHIATRY

The fellowships are designed to provide special training for physicians who have had previous hospital training in psychiatry but who wish to prepare themselves for extramural work in the fields of child guidance, delinquency, education, dependency and industry. The fellowships are open to physicians who are under 35 years of age and graduates of class A medical schools, and who have had at least one year of training in a hospital for mental disease maintaining satisfactory standards of clinical work and instruction. A longer period of hospital training is desirable. Applicants able to meet these requirements will not be required to take competitive written or oral examinations. Selections will be made on the basis of length and type of previous training in formal psychiatry, on general fitness for the work contemplated and, in most cases, on the results of a personal interview.

These fellowships cover a period of training approximately one year in length. During this training period trainees usually are assigned for three to four months' periods to such places as the Boston Psychopathic Hospital, Judge Baker Foundation, Boston, Institute for Juvenile Research, Chicago, and other places of a similar nature, as well as to various child guidance clinics located in Cleveland, Philadelphia and other cities. Assignments to these training centers are not definite, however, and assignment to any given place will depend on the availability of instruction at such place, as well as on the special needs of the trainee. Assignments are not made for more than three months in advance, and adherence to a fixed program made in advance for the year's training period is impossible. The fellowships carry stipends of from $2,000 to $2,500 for the twelve months' period.

Applications need not be filed within stated periods, but will be received at any time. In the case of successful applicants, arrangements will be made to begin work whenever mutually convenient to the applicant and to the director of the training center to which the applicant is first assigned. Applications or inquiries for further information should be sent to Dr. Frankwood E. Williams, Medical Director, National Committee for Mental Hygiene, 370 Seventh Avenue, New York.

ELEVENTH ANNUAL MEETING OF ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASE

The eleventh annual meeting of the Association for Research in Nervous and Mental Disease will be held on Monday and Tuesday, Dec. 29 and 30, 1930. The headquarters of the Association will be as usual at the Commodore Hotel, Forty-Second Street and Lexington Avenue, New York, and the sessions will be held in the rooms that have been engaged by the Association in the past. The meeting will consist of morning and afternoon sessions, the former being called to order at 9:15 a. m. and the latter at 2:30 p. m. The 1930 meeting will be devoted to a consideration of "Manic-Depressive Psychosis" and the program has been arranged by Dr. William A. White, Director of St. Elizabeth's Hospital, Washington, D. C.

The final program will be composed of papers selected from the following list of contributions: (1) historical: History of the Manic-Depressive Reaction Types, Smith Ely Jelliffe, M.D., New York. (2) constitution: Organic Constitution of the Cyclothymic, Walter Freeman, M.D., Washington, D. C.; Build in Manic-Depressive Psychosis, Horace Gray, M.D., San Francisco. (3) childhood reactions: Mental and Health Factors in Juvenile Cases of Manic-Depressive Psychoses,

Albert M. Barrett, M.D., Ann Arbor, Mich.; Personality Patterns in Children in Families with Manic-Depressive Psychosis in the Direct Line, Lawson G. Lowrey, M.D., New York; The Behavior Problem in Childhood, M. G. Peterman, M.D., Milwaukee; The Affective Psychoses in Children, J. Kasanin, M.D., Boston; The Rorschach Test in Manic-Depressive Psychosis, David M. Levy, M.D., New York. (4) etiology: A Study of Psychogenic Factors as Causative Agents in Manic-Depressive Attacks, Clarence A. Bonner, M.D., Hathorne, Mass. (5) pathology: Somatic Expressions in Manic-Depressive Psychoses, Nolan D. C. Lewis, M.D., Washington, D. C. (6) symptomatology: Studies on the Blood-Cerebrospinal Fluid Barrier in Manic-Depressive Psychosis, William Malamud, M.D., Iowa City, and David Rothschild, M.D., Foxborough, Mass.; Physico-Chemical Changes of the Blood Serum and Different Phases of the Manic-Depressive Psychosis, S. DeW. Ludlum, M.D., Gladwyne, Pa.; Contributions to the Laboratory Study of Manic-Depressive Psychosis: I. Basal Metabolism and the Specific Dynamic Factor of Protein; Indices of Water Metabolism; Acid-Base Equilibrium; Blood Sugar Curves as Affected by Emotion; Gastric Secretion and Mobility; Anthropometry, Clifford B. Farr, M.D., and Kenneth E. Appel, M.D., Philadelphia; II. Pharmacodynamic Tests with Special Reference to Histamine, Norvelle C. La Mar, M.D., and Harold D. Palmer, M.D., Philadelphia; III. Psychogalvanometer Readings, Edward M. Westburgh, M.D., and Elmer V. Eyman, M.D., Philadelphia; Discussion, Earl D. Bond, M.D., Philadelphia; Gastrointestinal Motor Functions in Manic-Depressive Psychoses: Roentgenologic Observations, George W. Henry, M.D., White Plains, N. Y.; Physiological Periodicities and Their Relation to Manic-Depressive Cycles, Curt P. Richter, Ph.D., Baltimore; Recurrence and Duration of Attacks in Manic-Depressive Psychoses, Horatio M. Pollock, Ph.D., Albany, N. Y.; The Motility in Manic-Depressive Psychosis, Paul Schilder, M.D., New York; Delusions and Hallucinations in Manic-Depressive Psychoses, Karl M. Bowman, M.D., Boston; An Empirical Determination of the Association of Symptoms in the Manic-Depressive Psychoses, Thomas V. Moore, M.D., Washington, D. C. (7) borderland symptomatology: The Significance of Schizoid Mechanisms in the Manic-Depressive Syndrome, Chester L. Carlisle, M.D., New York; Relation of Other Reaction Types to Manic-Depressive, Lucile Dooley, M.D., Washington, D. C. (8) mechanisms: Socialization and Compensation in Manic-Depressive Psychosis, Leland E. Hinsie, M.D., New York. (9) statistical: Manic-Depressive Psychoses Among Ex-Service Men, Philip B. Matz, M.D., and O. C. Willhite, M.D., Washington, D. C. (10) psychology: Personality and the Concept "Manic-Depressive Psychosis," G. E. Partridge, Ph.D., Towson, Md.; Intellectual Deterioration in Manic-Depressive Psychosis, George Van Ness Dearborn, M.D., New York; The Psychodynamics of Depressive Reactions to Parenthood, Gregory Zilboorg, M.D., White Plains, N. Y.; Psychoanalytic Sidelights on the Manic-Depressive Reactions, C. P. Oberndorf, M.D., and Monroe A. Meyer, M.D., New York. (11) ethnography: Manic-Depressive Reaction in the Negro, Nolan D. C. Lewis, M.D., and Elizabeth R. Vann, M.D., Washington, D. C. (12) prognosis: The Prognosis in Manic-Depressive Psychoses, Edward A. Strecker, M.D., Philadelphia; The Mechanisms and the Prognostic Aspects of the Manic-Depressive Schizophrenic Combination, Nolan D. C. Lewis, M.D., and Lois D. Hubbard, M.D., Washington, D. C. (13) medicolegal aspects: Manic-Depressive Psychoses: Their Medico-Legal Implications, Theophile Raphael, M.D., Detroit. (14) treatment: Treatment of Manic-Depressive Psychosis: Survey of the Literature, Leland E. Hinsie, M.D., and Siegfried E. Katz, M.D., New York; Results Obtained by the Use of Sodium Bromide in the Treatment of Depressions of the Manic-Depressive Type, William W. Wright, M.D., Utica, N. Y.; Solution of Manic-Depressive Psychosis Through Energy Direction, Anita M. Mühl, M.D., San Diego, Calif.; Psychotherapeutic Studies in Manic-Depressive Psychoses, Nolan D. C. Lewis, M.D., Washington, D. C.; Changes in Mood and in Mental and Motor Control in Normal Subjects Following Massive Doses of Bromides, Donald A. Laird, Ph.D., Hamilton, N. Y.; The Applicability of Psycho-Analysis to the Treatment of Depressive States, Bernard Glueck, M.D., New York.

Abstracts from Current Literature

SOME VIEW POINTS OF THE ACTUAL PROBLEM OF SCHIZOPHRENIA. TORSTEN LINDNER, Jahrb. f. Psychiat. u. Neurol. **47**:12, 1930.

According to Lindner, who is a physician in the neurologic clinic of the Serafimerlasarettet in Stockholm, the recent "psychologic interest in dementia praecox" that unquestionably is valuable for a thorough understanding of the psychic symptoms as well as for prognosis "has displaced all previously made observations, especially by Americans and the English," as to the rôle played by infections in the pathogenesis and clinical course of this psychosis. In this connection the author states that it is noteworthy that Wagner von Jauregg and his school still maintain the validity of the "infectious theory," namely, the significance of intestinal infection combined with endocrine factors in the pathogenesis of dementia praecox. (The two references to Wagner von Jauregg's views given by the author are: *Wien. klin. Wchnschr.*, 1896, and *Jahrb. f. Psychiat.*, 1902.)

Following this introduction, Lindner cites from the literature numerous theories and hypotheses as to the relationship between immunity, constitution, endocrine glands and schizophrenia. He apparently reaches no definite conclusions as to this relationship, but he utilizes the various theories for what may be considered the keynote of his paper, which he discusses under the heading: "liver, brain and schizophrenia." In this part, Lindner points out that as far back as the Middle Ages some relationship had been assumed between diseases of the liver and those of the mind. In 1892, Clippel (cited by Schrijver: *Ztschr. f. d. ges. Neurol. u. Psychiat.* **93**:472, 1924) found in many psychoses an intermittent urobilinuria; this was subsequently confirmed by numerous other observers, so that he attempted to establish a causal relationship between disturbances in liver function and psychoses. Cullere criticized Clippel's views and raised the question whether it was not more likely that the involvement of the liver and the psychoses were both due to a common cause. Owing to the fact that with the development of an organic disease of the liver in many patients suffering from a psychosis the latter condition remained unaffected, many observers were of the opinion that the two conditions had nothing to do with each other.

Lindner then admits that in spite of the fact that in a large number of cases of schizophrenia painstaking examinations of liver function had been made, as yet there is no method, qualitative or quantitative, for testing liver function that could be regarded scientifically as a positive test. He also believes that even if there were such a positive test, one would still be unable to meet Cullere's objections to Clippel's view (that the disease of the liver and the psychosis are coordinate and that the former is not the cause of the latter), nor would it be possible to determine whether the disturbed function of the liver was not due to the same changes in the brain that are also at the basis of the psychosis. Lindner recalls that Lingjaerde found a pathologic urobilinuria in schizophrenia in 60 per cent and in patients with agitated schizophrenia in 100 per cent of the cases, whereas in stationary and inactive states of the same psychosis, urobilinuria was found in only 20 per cent of cases. Lingjaerde also made numerous determinations of the bilirubin in the blood and bromsulphalein tests, the results of which also gave some support to the belief that in these cases one may be dealing with some condition of the liver. This observer, however, is conservative and states that in spite of all these observations he is not absolutely convinced that the tests of the liver function as carried out by him are indicative of disturbances of this function.

According to Lindner, experimental, as well as a large number of clinical, observations in some cases seem to point to severe diseases of the liver (sub-

chronic hepatic atrophy, liver metastases) as an undoubted cause of secondary cerebral involvement, such as striatal and lenticular degeneration. The lenticular degeneration and the changes in the liver found in the cerebral pseudoscleroses also seem to have some causal relationship to each other. Borberg recently (1927) published the report of a case of adenocarcinoma of the pancreas in which he found metastases in the liver and lenticular degeneration in the brain. Borberg considered the lenticular degeneration secondary to the metastases in the liver and Lindner is inclined to agree with this view, although he admits that the problem of the relationship between lesions in the liver and those in the brain is by no means solved. In this connection Lindner emphasizes the importance of recognizing that functional disturbances of the liver as determined by chemical tests are not necessarily an index of the extent and intensity of the pathologic changes in that organ. One can readily conceive, therefore, that in a given case of schizophrenia there may exist during life marked evidences of severe disturbances in liver function, whereas the macroscopic changes in the liver found at necropsy are relatively insignificant.

Among others, one of the most important functions of the liver is its detoxicating property. Lindner believes that if this is so there may result in liver dysfunction a diminution in the detoxicating property of this organ with a consequent increased invasion of the blood stream with poisonous products (peptones, histamines, aromatic amines, etc.) which may ultimately be carried to the brain (Büchler, Bonnhoeffer). Following this line of reasoning, he states that one could conceive that in the presence of slight disturbance of liver function or even with a relatively normal liver the latter may be so overwhelmingly flooded by an excess of poisonous products derived from the intestinal flora (increased protein decomposition) that filtration of these poisons becomes incomplete, and the toxic products in unfiltered form reach the blood stream and finally the brain. If this is so, the increased urobilinuria found in schizophrenic patients and in patients in agitated catatonic states could be readily explained by assuming that they may be partly due to a "central effect on the liver and intestinal functions." In partial support of the validity of this assumption, Lindner cites G. W. Henry, an American, who studied roentgenologically the intestinal motility in schizophrenic patients and found that in 70 per cent of cases with acute hallucinatory states the barium remained in the intestines more than five days. On the other hand, Lindner admits that while all this may be true, one must take into consideration that urobilinuria may also be partly an expression of a toxic anemia.

In addition to the factors discussed thus far, Lindner points out that the frequent occurrence of polyglandular disturbances, especially of the thyroid and suprarenal glands, in the schizophrenic psychoses must be more than a mere coincidence. He is inclined to attribute to these disturbances some rôle in the vicious circle at the basis of the pathogenesis of schizophrenia, which he would formulate as follows: dysfunction of the endocrine system plus dysfunction of the reticulo-endothelial system of the liver; the combined effect of these two factors giving rise to diminished oxidation, diminished detoxication, diminished hormonal detoxication and diminished uric acid formation with an eventual oversusceptibility of the organism to toxic products (peptones and histamine) and an abnormal production of aromatic amines. It is this dysfunction that, after a prolonged duration, may exert a secondary effect on the brain which gives rise to the schizophrenic symptoms.

Buscaino, an Italian, in 1924 and in 1925, described toxic degenerative changes in the liver (interstitial chronic hepatitis) in cases of dementia praecox in which he "thought" he found abnormal amines (histamines) in the urine during life. Buscaino also found certain histologic changes in the basal ganglions, similar to those found by Marcus, in Scandinavia. He believes that they are due to the abnormal amines circulating in the blood. Buscaino found these toxic amines not only in schizophrenia, but also in amentia, in the amyostatic symptom complex, postencephalitic states, typhoid and pneumonia and in febrile states with a temperature above 39 C. (102.2 F.); hence these toxic amines are not pathognomonic

for dementia praecox. While numerous competent observers failed to confirm Buscaino's observations, other equally competent observers found these toxic amines also in the urine of patients suffering from epilepsy and manic-depressive psychoses. To cast further discredit on Buscaino's observations, Lindner cites Bettziehe (*Deutsche med. Wchnschr.*, 1927) and Zamecki (*Zentralbl. f. d. ges. Neurol. u. Psychiat.*, 1928), both of whom claim that Buscaino is "ignorant of the most elementary principles of chemistry." Bowman, an American, has recently published in the *Journal of Nervous and Mental Disease*, systematic, biochemical investigations of the same type as Buscaino, but these, according to Linder, have yielded no better results than those published by Buscaino himself.

Lindner then reviews briefly the various investigations that have recently engaged the attention of numerous students of the problem of schizophrenia. Of these investigations, the most prominent are: studies of basal metabolism, sedimentation of red blood cells, lipoid metabolism, protein metabolism (Folin) and changes in the blood picture. The results of all these studies have been so contradictory that no definite conclusions can be drawn from them.

The next reference is made to two interesting publications that have recently appeared in the north: (1) a monograph on the pathology of dementia praecox by Paul Reiter and (2) a paper by Sven Stenberg on "Psychosis and the Blood Lipoids." Reiter's conception of the problem of schizophrenia confirms Lindner's views in many respects. Reiter's theory is: Dementia praecox is due to gastro-intestinal disturbances (as described by Cotton, Buscaino and others) associated with a congenital abnormal anlage of the liver and also perhaps a defective anlage of the gastro-intestinal canal, and that, owing to a lesion in the liver, the apparatus that normally protects the body against enterogenous poisons, these poisons invade the blood stream and finally affect the brain. According to Reiter, this theory "partly fills the gaps in our knowledge and partly agrees completely with our present knowledge."

In his contribution to Professor Justchenko's "Festschrift" in 1928, Lindner expressed his belief that in addition to purely constitutional, metabolic and pathologic-anatomic problems, schizophrenia also presents bacteriologic and immunologic problems. It was the recent investigations by Tokumitso on the relationship between hormones and immune bodies that suggested to Lindner the possibility of a relationship between the endocrine disturbances, so frequently observed in schizophrenia, and certain disturbances of immunity in the organism in this disease. He finds further support for this belief in Bogendörffer's investigations which point to the existence of a relationship between the functions of the central nervous system and the formation of immune bodies. The close contact between the central and vegetative nervous systems, on the one hand, and the endocrine system on the other, seems to Lindner to favor the hypothesis that a central disturbance of the vegetative centers in the midbrain could influence the formation of antitoxins (Tokumitso) against various bacteria, indirectly through the endocrine apparatus (by hormones), perhaps through some effect on the reticulo-endothelial system in the liver. According to Lindner, the experiments by Heilig and Hoff performed in Wagner von Jauregg's clinic in this direction also seem to show the plausibility of this hypothesis. At the same time, it must also be recognized that there must be some significance in the relationship between the endocrine apparatus and lipoid (cholesterol) metabolism to the general immunity of the body. Therefore disturbances of the central nervous system and of the endocrine glands, by affecting cholesterol metabolism, at the same time may be of some significance in the pathogenesis of schizophrenia. Lindner finds support for this hypothesis in Stenberg's investigations (1929) which demonstrated disturbances in the cholesterol metabolism in psychoses, and in Pende's (*Endokrinologie*, 1928) systematic clinical investigations which also showed a relationship between thyroid, brain and liver function.

Paul Reiter and Seyderhelm, as well as Lindner, see some features in dementia praecox that are somewhat analogous to pernicious anemia. They believe that there occurs in pernicious anemia an increased permeability in the intestinal mucous

membrane with an eventually diminished resistance or perhaps hereditary defect of the liver and a resulting tendency to resorption of toxic substances, which first affect secondarily the liver and its functions and then directly or indirectly the brain. They also believe that the the periodic variations in the functioning of the hepato-endocrine apparatus are probably the basis for the periodicity and remissions both in pernicious anemia and in dementia praecox.

It was on the basis of this analogy between dementia praecox and pernicious anemia and of Sato Akira's discovery of yakriton in the liver, a substance that has been shown to possess a detoxicating effect on histamine and other such products as Buscaino believes to bear a pathogenetic relationship to dementia praecox, that prompted Lindner, in 1927, to subject a number of patients with chronic and far advanced cases of schizophrenia to treatment with liver extract. The patients so treated showed schizophrenic confusional states, pathologic urobilinuria, icteric skin, increased sedimentation of the erythrocytes and marked secondary anemia; the clinical picture was also such that Lindner thought he was justified in assuming that in all of them he was dealing with a "chronic toxic process." While under liver treatment the patients were carefully studied somatically and psychically; marked improvement was noted in both spheres. (No further details of these cases are given in this communication.)

Owing to the marked improvement following treatment with liver observed by Lindner in these cases as well as in other diseases in which infections play a prominent rôle, and in secondary anemias, he proposes to employ liver extract in dementia praecox and in other toxic psychoses.

Comments by the Abstracter.—This rather "loosely" written paper, consisting of thirty-eight pages and containing ninety-six references to the literature, is the author's attempt to formulate a wholly somatic approach for the pathogenesis of dementia praecox. His formulation, which has been attempted previously along similar lines by numerous other writers, is not convincing. It is not convincing because it is not based on proved facts, but on theories and hypotheses that have never been fully substantiated by critical clinicians nor confirmed by competent physiologic chemists. Lindner's formulation is based principally on the theory of infection as elaborated by Cotton, Hunter, Graves, Smith and others, which, according to Lindner himself, has been disproved by Kopeloff, Kirby, Cheney, Wuth, Henderson and other reliable observers. Furthermore, Lindner attaches undue weight to the significance of disturbances of the endocrine glands as causative factors in dementia praecox. His evidence as to this is even less convincing than that offered by Mott in his formulation of the theory of gonadal inadequacy as an etiologic factor in this disease. Lindner presents no better reasons for the endocrine factor in his formulation than do most endocrinologists who attribute any and all etiologically obscure diseases to disturbances in the glands of internal secretion. Not unlike most endocrinologists, this author appears to make no critical distinctions between cause, effect and coincidence.

While it is true that the author's title to the paper is "Some Viewpoints, etc.," and therefore he should not be condemned for not discussing all points of view, he mentions practically every point of view but devotes only three or four lines to the psychologic point of view, and this in a manner as though it were of little if of any importance in any formulation of the pathogenesis of dementia praecox. Lindner seems to take no cognizance of the modern practically universal acceptance of the conception of this disease as depending on special personality and constitutional defects and on habit disorganization. He apparently disregards the well known fact that there may be cases of dementia praecox with some congenital or acquired physiologic inadequacy which contributes to the clinical picture of the disease and which may even show structural evidences of this inadequacy on the necropsy table. He also seems to forget that one is not justified in generalizing from cases of this type, because it is conceivable that a person may be incapacitated in his adaptation to his surroundings and to reality through some basic inadequacy of his somatic (metabolic) mechanism, but mostly through the inadequacy of the more complex mechanism of his personality.

In conclusion, however, I wish to state that in spite of my adverse criticism of the author's paper, his suggestion that patients with dementia praecox be subjected to treatment with liver extract is worthy of serious consideration. I feel that in any disease in which one is as helpless therapeutically as in dementia praecox any method of therapy that is not dangerous to life is worthy of trial, even though that particular form of therapy has no better basis for it than pure empiricism or analogy.

KESCHNER, New York.

INFECTION AND THE NERVOUS SYSTEM. W. SPIELMEYER, Ztschr. f. d. ges. Neurol. u. Psychiat. **123**:161, 1930.

Spielmeyer discusses in this paper the changes produced by known infections of the nervous system, and the application of this knowledge to special questions of neuropathology. One and the same infection can produce very different changes in the nervous system. In one case there may be regressive parenchymatous changes, in another, circulatory changes, and in a third, inflammatory changes. In the total complex of the inflammatory disease there are, besides the local reactive-exudative phenomena, also circulatory disturbances. In a case in which inflammation is the most important symptom circulatory disturbances with their consequences may exist independently, as well as true parenchymatous degeneration of the nerve substance. With regard to infection, Spielmeyer says that a very important and little known fact is that the reactive glial changes in infectious diseases are more or less independent of mesenchymal, leukocytic and lymphocytic infiltrations. The glia may take part in local reactions all alone. Spielmeyer shows glial foci in cases of trichinosis, endocarditis and staphylococcus infections. He calls these special glial reactions. They have a more or less negative significance, and their exact functional importance is not yet known. These foci must probably be looked on as a sign of a defense reaction, even when they occur without lymphohistiocytic infiltrations. In this sense these foci are signs of an inflammation, even though Spielmeyer has elsewhere warned against speaking of inflammation in the nervous system in the absence of exudative-infiltrative phenomena. This view is in accord with that of Fischer-Wasel, who groups the glia with the mesoderm as a part of the defense apparatus of the organism.

It cannot be decided anatomically whether a certain process is definitely infectious because many toxins may cause inflammations in the brain and cord, as, for example, carbon monoxide and the toxins of fungus formation. In the case of endogenous processes the question is particularly difficult, for the destruction of nerve tissue can produce an inflammatory reaction, a so-called reparative inflammation. This is not a primary inflammation, but a secondary, symptomatic inflammation. Who will say, however, whether the inflammatory process is primary or only the answer to destruction of nerve tissue and therefore secondary and reparative? How is it with multiple sclerosis? Spielmeyer himself has asserted the inflammatory character of multiple sclerosis. Were any one to assert that the inflammatory process is the result of a symptomatic response to the loss of cells and tissue, Spielmeyer could not deny this. But he could not demonstrate its infectious nature anatomically. Pette says: "According to the present status of our knowledge we must look on these facts—the presence of plasma cells in the vessel sheaths—as pathognomonic of the infectious character of a disease, that is, of the presence of a living exciting agent."

Is there any distinction in the changes in the nervous system due to visible and invisible (ultramicroscopic) agents? Pette says that in the ultramicroscopic cases first the parenchyma is diseased, then the glial reaction is very prominent, and the mesenchymal reaction is secondary. Spielmeyer says that there is no difference between the two. In poliomyelitis there is first a polymorphonuclear and lymphocytic reaction, followed by a glial reaction, and still later by glial polyblasts. On the other hand, in bacterial infections there may be a purely glial response to noxious agents.

Certain diseases have a predilection for the gray matter—poliomyelitis, epidemic encephalitis, Borna disease and lyssa. Other diseases involve the white matter chiefly. Pette says that the characteristic of the latter is that the causative agent has an affinity for certain parts of the nerve parenchyma, the myelin sheath. He gathers into this group the postvaccinal encephalitides and the encephalomyelitides. Spielmeyer points out, however, that in postvaccinal and measles encephalitis, not only is the myelin sheath involved, but also the entire nerve axis cylinder. The changes in these diseases differ from those in multiple sclerosis. One must differentiate between encephalomyelitis disseminata and acute multiple sclerosis. Not to be able to do so pathologically does not say that there is no difference between them.

The number of anatomic syndromes is very limited. Where an injury causes a pathologic increase in the living defense mechanism, the stromal reactions are fundamentally the same, regardless of whether it is the effect of a visible or invisible noxious agent, of infectious-toxic injuries, of poisons of an exogenous nature, or reactions to endogenous metabolic and degenerative products, for always the same defensive apparatus is called into function.

ALPERS, Philadelphia.

SERUM MENINGITIS. DOUGLAS GOLDMAN, Arch. Path. **9**:1027 (May) 1930.

The stimulus for undertaking the investigation here recorded was the occurrence of several cases of aseptic meningitis discovered at autopsy in patients who had received serum injections into the spinal theca. Most of the cases were instances of fatal tetanus, but a case of cerebral hemorrhage clinically resembling meningitis in a young adult is also on record. Interest in the subject has grown recently because the induction of aseptic meningitis has been found of use in the treatment for schizophrenia and other mental states (Carroll, Kubitschek and Carmichael). A further experimental study in laboratory animals, with a review of as much of the literature as was available, seemed to be of timely interest. Recently, Bagley reported experiments on the effect of blood in the cerebrospinal fluid. He found a definite microscopic cellular reaction to the presence of the blood, most marked at the site of the injection (subdural). His experiments, however, dealt with the more prolonged and severe types of bleeding into the subarachnoid space.

The results of these experiments are in agreement with those of other investigators. It was attempted to rule out all possibility of infectious meningitis by rigid adherence to aseptic technic and by culture of the cerebrospinal fluids obtained from the animals. It is hardly likely, therefore, that other than aseptic meningitis was present.

Various serums affected the meninges similarly. All called forth a polymorphonuclear reaction in the meninges and spinal fluid. The intensity of the reaction varied from one animal to another and seemed, within limits, not to be related to the amount of serum injected or other controllable factors. It is significant that spinal fluid leukocytosis is always associated with a cellular reaction in the meninges. The experiments with serums rather definitely indicated that the cell content of the cerebrospinal fluid tends to diminish more rapidly and sooner than the meningeal reaction. That second injections had such small effect in dogs is not surprising, since the dog is not very susceptible to anaphylaxis. The animals showed no unusual symptoms in cleancut experiments. Occasionally, it was noted that an animal was somewhat more active following injections of the serum, but usually the attendant was the only one who could note the difference in behavior. The serum caused no febrile reaction in the dogs. The effect of the injection of salt solution and cresol calls for no comment. The reaction to the reinjection of spinal fluid was so mild and transient that it is of little more than theoretical significance.

It is particularly in diseases such as tetanus or in diseases with meningeal symptoms but without actual meningitis that repeated intrathecal injection of large doses of serum may lead to confusion in diagnosis and at times to actual harm to

the patients so treated. Recognition of the effect of serum and other sterile fluids on the meninges will serve to dispel this confusion. In cases of infectious meningitis in which serum treatment is used, daily observation of the condition of the spinal fluid is necessary to regulate the therapy, for continued use of serum is not without harmful effects in the absence of an infection necessitating its use. There is, however, no contraindication to the use of therapeutic serums intrathecally when the hazard of the disease is greater than that of the serum which promises relief.

The author concludes that: Aseptic meningitis has received scant recognition in the textbooks and only occasional serious consideration in the literature. Intrathecal injections of serum produce spinal fluid leukocytosis and an accompanying aseptic meningitis with great regularity in experimental animals. This aseptic meningitis is of short duration and produces no permanent changes of any importance in the meninges or nervous system.

WINKELMAN, Philadelphia.

THE PSYCHOGALVANIC REACTIVITY IN NORMAL PERSONS AND IN VARIOUS PSYCHOPATHIC CONDITIONS. ORNULV ODEGAARD, Acta psychiat. et neurol. **5**:55, 1930.

The author studied the results of 598 psychogalvanic tests in 526 persons — 182 normal persons and 344 psychiatric patients. In this study the curve as a whole was taken as a sample of the emotional vegetative reactivity in general rather than as individual reactions to specific stimuli. The psychogalvanic reactivity is regarded as an interaction of two components: one (called negative) due to increased potential in the palm of the hand, another (called positive) due to increased potential on the back, and causing a deflection in the opposite direction. From the shape of the reactions (negative, biphasic and positive) the share of each component can be determined. One type of curve was adopted as standard, because it was obtained from 65 per cent of the normal persons. Its main characteristics were: (*a*) the reactions had a fairly high amplitude — at least three in each curve were more than 10 mm. high; (*b*) there were definite intervals of rest between the individual fluctuations of the string, so that the reactions were easily separated, and (*c*) both components of the reactivity were well represented.

The curves derived from patients differed from the normal in that several curves had a definitely decreased reactivity, with no reactions higher than 5 mm. — 14 per cent in the normal persons against 28 per cent in the patients. Many curves showed a continuous fluctuation back and forth of the string, with no intervals of rest to make possible a distinction between individual reactions (continuous curves) — 4 per cent in the normal persons against 37 per cent in the patients. Several curves showed a definite decrease of the positive component (as compared to the standard type), while the negative one had its normal strength. If the pathologic factors were pronounced, the curve was considered atypical. There were three types of atypical curves: (*a*) curves without any reactions; (*b*) all continuous curves, and (*c*) curves with no positive component, called stair-shaped because of their peculiar shape. Atypical curves were found in 8 per cent of the normal persons against 58 per cent of the patients.

The psychogalvanic reactivity was most disturbed in patients with toxic and organic psychoses, schizophrenia and manic excitement and less so in the patients with depression and neurosis, hysteria and constitutional disorders. Decreased reactivity was most marked in patients with organic psychosis and schizophrenia. Lability was most pronounced in those of the constitutional and manic groups and in patients with hysteria. In patients with depression, decreased reactivity was the most marked pathologic feature. The difference between the clinical disease entities was not sufficiently marked to give the method diagnostic value.

There were more outstanding differences when the cases were classified on the basis of specific symptoms rather than on clinical entities. Among the cases of depression and neurosis with anxiety and agitation there were 76 per cent

atypical curves, against 48 per cent in similar cases without these symptoms. In schizophrenic patients, catatonic and affective symptoms (apathy as well as increased tension) gave the most atypical reactivity, while paranoid symptoms (delusions, hallucinations and passivity feelings) had no influence. In hysteria the reactivity was most atypical in subacute cases with no sign of clinical improvement.

As these facts are in harmony with clinical experience, the author believes that the method is useful and reliable for the study of reactivity in different groups of nervous and mental diseases. Such studies will, on the one hand, give information of the manner in which the vegetative-emotional function is influenced by different mental states and attitudes. On the other hand, they will gradually make more clear the meaning of the different pathologic features of the psychogalvanic reactivity — their psychobiologic significance.

PEARSON, Philadelphia.

ENCEPHALOGRAPHIC OBSERVATIONS IN NEUROLOGIC AND PSYCHIATRIC CASES. E. MEYER, Arch. f. Psychiat. **89**:177, 1930.

The author discusses the results of encephalographies undertaken in 200 cases at his clinic. The technic employed was usually that of the lumbar or suboccipital routes. In some cases in which the filling of the ventricles could not be accomplished by these methods, ventricular puncture was resorted to. There were no untoward symptoms, and he believes that the method is perfectly safe provided the technic is well mastered. The greatest difficulty is encountered in reading the pictures obtained. He quotes a number of representative cases from the different fields that he studied.

In tumors of the brain, the injection of air is practically always helpful for diagnosis and localization. Some cases, except for general symptoms such as choked disks, headache and vomiting, in which there were no localizing signs, the pictures obtained after an injection of air were extremely useful in localizing the lesions. It is understood, of course, that this method can be used only in conjunction with clinical and serologic examinations and can serve only as a link in a chain of observations. In cases of inflammatory diseases, the injection of air is not as useful except in chronic diseases with consequent atrophy and dilatation of the ventricles.

Injection of air is particularly helpful in injuries of the head. The occurrence of dilated ventricles, unequal in size, and a drawing of the larger of the two lateral ventricles over toward the site of injury indicates definite atrophy and contraction of the tissues and offers diagnostic help. In cases in which symptoms develop following injury of the head, which cannot be differentiated from psychogenic symptoms and in which no neurogolic signs are present, the demonstration of such defects in the picture points to a definite organic lesion. He quotes cases in which the patients were thought to be either malingering or neurotic, in which encephalography subsequently proved the existence of organic disease. The same is true of cases of concussion, although here the defects are not as frequent. The author's experiences with epilepsy do not seem to coincide altogether with those of other authors who claim to have found encephalographic defects in the majority of cases. In patients with traumatic epilepsy, enlargements of the ventricles and irregularities of the periphery were found. In genuine epilepsy, however, these were rare and not definite.

In dementia paralytica, enlargement of the ventricles is common as also are irregularities in the periphery, which are especially marked in the temporal region. Cases of dementia paralytica that show marked signs of this type are not favorable for malarial therapy. Similar observations are reported in cases of senile dementia and of psychoses with arteriosclerosis. In the latter, the defects are rather isolated and limited to special regions. Chronic alcoholic patients, too, show enlargement of the ventricles but no definite involvement of the periphery.

In schizophrenic patients, some disturbances of the ventricular outline as well as occasional defects in the periphery were noted. These, however, were found just as frequently in fresh cases as they were in chronic cases.

The author concludes with the statement that further training and experience in the reading of the plates is necessary and for that purpose a more definite knowledge of the normal picture should be obtained.

MALAMUD, Iowa City.

RECIPROCAL INNERVATION OF THE EXTRA-OCULAR MUSCLES. FRANCIS HEED ADLER, Arch. Ophth. **3**:318 (March) 1930.

Sherrington's law of reciprocal innervation for muscle contraction is first discussed. The law, as stated by Fulton, is, "as contraction occurs or progresses in a muscle, for example an extensor, contractile activity diminishes pari passu in the antagonistic flexor." The law is based on innervation in that an excitatory stimulus to a muscle or a group of muscles is accompanied by an inhibitory stimulus to the antagonistic group. The experimental work done by Sherrington in 1893 to demonstrate the correctness of the law (as it applies to the ocular muscles as well) is explained in detail. It consisted in sectioning the third and fourth cranial nerves on one side in a monkey. Cortical stimulation for the causation of conjugate deviation developed a sharp external rotation of the normal eye while the opposite eye, with an operative third nerve ophthalmoplegia, also moved in slightly from its position of abduction. This must have been due alone to a relaxation of contraction of the external rectus of that eye. This normal contraction rotation of one eye with the accompanying relaxation in contractility of the opposite external rectus constitutes reciprocal innervation.

The literature of this function is then briefly reviewed. There is sufficient authoritative evidence of this nature to answer various writers who have doubted its existence. The experimental work of deKleijn, also included in the paper, demonstrated further that the phenomenon of relaxation is not of a mechanical or artificial nature due to hypertonicity in an unopposed muscle. DeKleijn's work was done through a study of nystagmus produced by labyrinthine stimulation in decerebrate rabbits.

Adler then presents two cases in detail to demonstrate the presence of this physiologic function of the extra-ocular muscles. Case 1 was one of bilateral third nerve internal and external ophthalmoplegia; case 2, of unilateral internal and external ophthalmoplegia. While positive evidence of paralysis of the fourth nerve as well could not be presented, these muscles, as the author states, are abductors and hence would play no part in the demonstration. As Adler states in his conclusions, "this inward movement of one eye in the presence of a complete paralysis of the third nerve when the fellow eye is abducted can be explained only by an inhibition of the contractile activity of one external rectus associated with the increase in contractile activity of the opposite antagonistic external rectus."

SPAETH, Philadelphia.

LATENCY OF AFTER-IMAGES AND INTERACTION BETWEEN THE TWO RETINO-CEREBRAL APPARATUSES IN MAN. R. S. CREED and R. D. HARDING, J. Physiol. **69**:423 (June) 1930.

Creed and Harding have investigated how the time relations of the negative after-image of a simple white object, seen uniocularly, might be modified by projecting the after-image on a screen presented to the other eye, and also the influence of certain other factors on the time relations, particularly that of the luminosity of the projection field. The technic used was that of Creed and Granit. It was found that after-images projected into complete darkness (black projection) have a longer latent period than those projected on a white screen. They also have a much more distinct halo and, with small disks, are more intensely black. With large disks, they sometimes appeared colored.

Binocular and uniocular after-images were found to have much the same latency when projected on a white surface. Binocular black projection images, on the other hand, appear earlier than right black projection or left black projection. In order to account for the early arrival of the binocular image, the authors assume that the uniocular processes reinforce one another (or are otherwise interacting). If the primary image is received by only one eye and the after-image is projected on a white screen presented only to the other eye (R → L and L → R), the latent period is longer than when the same eye is used throughout the experiment. The after-image in the former case has a bright halo, and at the moment of its arrival the whole screen appears to darken and remains so until it fades. It is therefore of the black projection type, and is in successful rivalry with the white field by which the now open eye is being stimulated.

The R → L image, however, appears earlier than the right black projection. Its arrival is therefore hastened by the formation of an image of the white screen in the left eye, and this indicates interaction at a subperceptual level between the retinocerebral apparatus of the two eyes.

It is suggested that an explanation may be sought for certain differences between R → L and L → R images, and between right and left black projection images, by reference to the habits of the observers in concentrating their attention on the field of view of one eye, rather than on that of the other. Further evidence of interaction is afforded by observations of the type binocular → R. The evidence already adduced for interaction between the two uniocular mechanisms is discussed in its relation to Sherrington's experiments on binocular flicker.

ALPERS, Philadelphia.

DISEASES OF THE ESOPHAGUS. ANGIONEUROTIC EDEMA, URTICARIA, SERUM DISEASE AND HERPES. CHEVALIER JACKSON, Arch. Otolaryng. **11**:397 (April) 1930.

The diseases mentioned in the title may all be due to a neurosis or a neuronosis. They are all somewhat similar to one another and may be reclassified in the future. Dr. Jackson reports the case of a patient who was referred for dysphagia and was unable to swallow solids. Firm, swollen, bleeding nodules were found in the esophagus. These disappeared with the lesions of other parts of the body. The lesions were unlike those of any of the lesions commonly encountered in the esophagus.

A patient with a history of urticaria and asthma was referred because of inability to swallow anything, even water. The esophagus was found completely closed, with white, nodular swelling of the walls. A second examination immediately after the first one showed a wheal. Reexamination in a week showed a normal esophagus.

A boy, aged 18, had complete obstruction of the esophagus following four days after the injection of a prophylactic dose of diphtheritic antitoxin. He had a typical urticaria of the chest with swelling of the tongue and enlargement of the glands in the neck and axilla. There was no difficulty in breathing. The larynx was normal but the thoracic esophagus was completely closed by extremely firm, white, nodular masses that seemed integral with the esophageal wall. There was no bleeding, erosion or ulceration.

A woman, aged 52, had a constant feeling of pressure, discomfort and burning in the midthoracic region, with pain and difficulty in swallowing solids and sometimes liquids, especially if hot. She had had recurrent attacks for three years. Examination showed chronic esophagitis and an ulceration that was diagnosed as peptic ulcer. It healed rapidly and subsequent esophagoscopy showed a normal condition. The symptoms returned and the patient was reexamined with the esophagoscope; lesions resembling herpes were seen. Later a herpetic eruption was noted on the body. Shortly after this, the esophagus was normal.

Dr. Jackson emphasizes the importance of looking into the esophagus when a patient has symptoms referable to it.

HUNTER, Philadelphia.

THE AMELIORATION OF MENTAL DISEASE BY INFLUENZA. KARL A. MENNINGER, J. A. M. A. **94**:630 (March 1) 1930.

Symptom manifestations of mental illness or attacks of mental disease may be (1) precipitated by somatic infectious disease, (2) aggravated by it or (3) ameliorated thereby. As to the latter, the literature affords ample evidence that melancholia, mania, confusional psychosis, hebephrenia, catatonia, paranoia, epilepsy, idiocy and dementia paralytica may be favorably affected by typhoid, typhus, recurrent fever, scarlet fever, rheumatic fever, smallpox, angina, pneumonia, pleurisy, abscesses, erysipelas, diphtheria, measles, cholera, malaria, osteomyelitis and influenza.

The paper is concerned with: (1) a review of the literature on influenzal amelioration of mental disease; (2) reports of cases; (3) an analysis of the cases, and (4) the theoretical implications of the phenomenon of amelioration ex rocentibus. The review of the literature covers cases from 1889 to the epidemic of 1918, and includes melancholia, chronic delusional psychoses, puerperal psychoses, schizophrenia, epilepsy, idiocy and manic-depressive psychoses. The author's own cases are six and include epilepsy with cure, epileptic psychosis with remission, epilepsy with improvement and remission, chronic mania with remission, migraine cured, and idiocy with improvement. Mennninger noted no improvement in cases of schizophrenia, psychoneurosis or melancholia. There were nine males, seven females, and two of sex unknown.

Considering theoretical implications, the author considers: 1. What are the mechanisms of the process? 2. What are the psychodynamics involved? 3. What is the essential nature of schizophrenia and particularly its reversibility. Under the first question he mentions the chemical theory or that wherein primary toxins are neutralized by secondary toxins. He then mentions the physical theory wherein the diseased neurons lying beside and inhibiting healthy ones are allowed uninhibited action through the paralyzing of the diseased neurons by further disease. He then discusses a vague humoral theory and discusses a catalytic theory at some length.

In discussing the second question, Menninger believes that the psychologic factors involved are identical with those involved in posttraumatic cures. There is a redistribution of libidinous streams.

CHAMBERS, Syracuse, N. Y.

CHRONIC SUBDURAL HEMATOMA IN INFANTS. DAVID SHERWOOD, Am. J. Dis. Child. **39**:980 (May) 1930.

Because the designation "pachymeningitis hemorrhagica interna" implies an infection, Sherwood rejects it as applicable to the subdural hematoma sometimes found in infants. The etiology is indeterminate, and infection and trauma seem conspicuous, at least as secondary factors. The author reports nine cases of this condition, and he suggests the possibility of a hemorrhagic diathesis, although in none of the cases could this be verified. Sherwood believes that any infant with convulsions and a large head should be considered a possible victim of subdural hematoma. Hemorrhages into the eyegrounds may help to confirm this suggestion, and positive observations on subdural tap should establish it. The tap should be performed with a hypodermic needle and syringe, and not with a lumbar puncture needle. The course is variable, ranging from a few weeks to half a year. The prognosis as to life is good, but infection and sequelae are constant possibilities. Feeblemindedness is the commonest of the serious sequels, and cranial nerve palsies, affecting especially the nerves to the eyeball musculature, are also frequent. The same subdural tap that is used for diagnosis is also therapeutically valuable. Operation should be postponed as long as the fontanels remain open enough to allow drainage by that route. After recovery from the hematoma, the danger of adhesions and arachnitis should be considered and neurosurgical advice sought as to the desirability of removing the membranes. General care, including attention to nutrition, is particularly necessary in cases

of this sort. The paper closes with the presentation of nine cases, in which eight were marked by convulsions, seven by a large head, and all by xanthochromic subdural tap. Eight of the patients are still living; five of them present sequelae.

DAVIDSON, Philadelphia.

PONTILE GLIOMAS. A PATHOLOGIC STUDY AND CLASSIFICATION OF TWENTY-FIVE CASES. RICHARD C. BUCKLEY, Arch. Path. **9**:779 (April) 1930.

In this article Buckley gives an excellent summary of gliomas of the pons and cerebellum. While he grouped them as pontile gliomas, only four were limited to the pons. The remaining twenty-one invaded the brachia, the cerebellum and the pontile angle. Ten of the twenty-five tumors were classified as glioblastoma multiforme, with an average duration of life of four months. These cases showed large necrotic tumors with many hemorrhages and cysts; the vessels and nerves were buried in the tumor. They differed in no way from the structure of similar tumors in the cerebrum. Fourteen of the gliomas were of slower growth and fitted in with the astrocytomas or unipolar spongioblastomas. The average duration of life was two years. Buckley found that the reaction of the tissue around the tumor differed in no great essential from that of the cerebrum. From the clinical standpoint, the frequency of involvement of the cranial nerves, especially the third, both at the onset of the illness and during the course of it, is noteworthy. What are called pseudocerebellar signs are attributed to involvement of the cerebellum directly. The author does not consider apparently the cerebellar fibers going through the pons as of sufficient importance to attach to them the significance which may be due them. It is interesting also that, in a goodly percentage of the cases, an increase of intracranial pressure was found. This has not been considered characteristic of pontile lesions. A typical summary of a case is as follows: "Rapid onset with paralysis of left sixth and seventh nerves. General pressure symptoms. Pseudocerebellar signs and symptoms. Suboccipital exploration yielding no definite evidence of a tumor. Death. Necropsy disclosed tumor of pons." "Onset with attacks of right internal squint, headache and spasm of muscles of left side of neck. Weakness of left side. Staggering gait. Dysarthria. Diplopia. Symptoms of general pressure. Diagnosis of tumor of pons. No operation. Death. Necropsy."

WINKELMAN, Philadelphia.

OBSERVATIONS ON THE RETINAL VESSELS OF FEEBLEMINDED CHILDREN. H. GAUSS, Arch. f. Psychiat. **88**:776, 1929.

The author reports the results of a study of the retinal vessels in 122 feebleminded children. This study was undertaken to check the reports by Jaensch and his co-workers of changes in these vessels as corresponding to deviations in the capillaries in other organs of feebleminded persons. Sixty-nine of the children were boys and fifty-three girls. The diagnoses made were: morons, forty-one and imbeciles, eighty-one. Three of the patients were low grade imbeciles and there were two cases of Mongolian idiocy.

The observations included photographic pictures of the retinal vessels, ophthalmoscopic examinations and examinations of the movements of the extra-ocular and intra-ocular muscles. Depending on the shape of the vessels, the children were divided into eight groups: (1) with normal vessels; (2) with slight variations from the normal; (3) with marked tortuosity of both arteries and veins; (4) with moderate tortuosity of the vessels; (5) with tortuosity of the arteries only; (6) with tortuosity of the arteries and slight deviation of the veins; (7) with tortuosity of the veins only, and (8) with tortuosity of the veins and slight deviations in the arteries. The results were as follows: forty-eight cases belong to group 1, forty-four cases to group 2, two cases to group 3, thirteen cases to group 4, five cases to group 5, two cases to group 6, seven cases to group 7 and one case to group 8.

The author comes to the following conclusions: In a comparative examination of the retinal vessels of normal and feebleminded persons, no definite differences between the two could be found. The feebleminded children showed 25 per cent of definite deviations as compared with 30 per cent of such deviations in normal persons. The individual deviations from normal found in the vessels were certainly acquired after birth in some cases, whereas in the others they were so slight that they could not be considered as significant of a special type of constitution in these children. The results of this study therefore contradict the reports of Jaensch and his co-workers. In accordance with observations made by a large number of investigators, deviations in other parts of the eyes were found frequently, such as errors in refraction, pseudoneuritis, strabismus, maldevelopments and pallor of the disks.

MALAMUD, Iowa City.

NORMAL AND MORBID ACTIVITY. EUGEN BLEULER, J. Nerv. & Ment. Dis. **71**:361 (April) 1930.

From a statement at the end of the article one infers that its aim is to examine the cooperation between the instincts and the intellect in human beings. Psychologically and biologically, the instinct is primary to the intellect, which serves as a means whereby the former gains its ends. The "morally ungifted" or constitutional psychopath differs from the normal person in a deficiency of instinctive impulses. These instinctive affective tendencies of man are greatly modified by chemical and toxic agents, and this is particularly true of those associated with the basal ganglia. In catatonia the affects are not entirely destroyed but are only hampered in some way, yet here, as well as in alteration of character occurring after encephalitis, one sees the modifiability of the instinctive make-up of man. The author dwells on the importance of the sex instinct, but considers that the inhibitions of the sex impulse are part of the instinct itself and not introduced by civilization. He considers the freudian division of ego, id and suger-ego as overschematic and unnecessary, and sees nothing in the "death impulse." He regards ethical qualities as hereditary "just like any impulse in the soul and the color of the hair." Morality and ethics are thus expressions of the higher instincts, which preserve both the race and the individual, particularly the former, and defects in these instincts can exist in acquired and inherited form. From this the author passes to the concept of the integration of personality, indicating that man's decisions are not accurate mathematical compromises but are reached as the result of the totality of all his impulses. When such harmonious integration in totality is incomplete there are ambivalence, the characteristic of schizophrenia, and capriciousness, which depends on incoordinate impulses. Ultimately, it is the instincts that determine aims and not logic, and through them one can understand the delusional trends of patients.

HART, Greenwich, Conn.

THE CLINICAL SYNDROME OF HYPERPARATHYROIDISM. DAVID P. BARR and HAROLD A. BULGER, Am. J. M. Sc. **179**:449 (April) 1930.

With the production of experimental hyperparathyroidism by Collip, its clinical interpretation became possible. Experimental studies indicate an inherent power of the parathyroid gland to increase in size under a great variety of stimuli. A total of seventy-four examples of pathologically enlarged parathyroid glands are considered, forty-five cases having previously been reported by Hoffheinz in 1925. Lesions of the bone have been present in 60 per cent of these cases; clinically, the most striking association is with the generalized form of ostitis fibrosa cystica (von Recklinghausen's disease) in which there is localized or generalized decalcification. Some interesting features associated with ostitis fibrosa are: marked calcium metabolic disturbance, metastatic calcification and functional muscular changes.

An interesting case of ostitis fibrosa cystica is described. At first, a diagnosis of giant cell sarcoma, bilateral nephrolithiasis and unexplained hypotonia of muscles and joints was entertained. A year later, a parathyroid tumor was

removed, tetany developed and calcium chloride had to be given intravenously. Another case, with autopsy observations, similar to von Recklinghausen's disease, is very enlightening. The most important differential diagnostic sign of hyperparathyroidism is hypercalcemia. The fact that increased urinary excretion of calcium, abnormal deposit of calcium in tissues and muscle weakness are found in experimental overdosage with parathormone; the discovery of parathyroid hyperplasia and tumors, and the fall in serum calcium which accompanies the removal of a parathyroid tumor substantiate the view that hypercalcemia of ostitis fibrosa cystica is due to hyperparathyroidism.

It cannot definitely be proved whether parathyroid hyperplasia is primary or secondary in ostitis fibrosa. In multiple myeloma, the probability is great that it is secondary to the bone changes. The importance of determining the calcium and phosphate content of the serum in cases of generalized bone disease becomes evident.

MICHAELS, Detroit.

THE EFFECT OF HEMOLYTIC TOXINS ON NERVOUS TISSUE. ARTHUR WEIL, Arch. Path. **9**:828 (April) 1930.

The hemolytic and myelolytic actions of saponin, sodium taurocholate, cobra venom and streptolysin were studied. It could be demonstrated that hemolytic toxins dissolve the myelin sheath of nerve fibers in the same way as they destroy the pellicles of red blood cells. The relation of the hemolytic effects to the myelolytic effects of the four toxins may vary. Compared with saponin, cobra venom and streptolysin are more myelolytic than hemolytic, in test tube experiments, while the action of sodium taurocholate is similar to that of saponin, though potentially stronger. The amount of saponin bound by 1 Gm. of spinal cord increases with the amount of saponin present. This phenomenon corresponds to the action of hemolytic amboceptors on red blood cells. The constants of Arrhenius' formula, calculated for the saponin-cord system, are relatively constant for final concentrations of more than 0.2 per cent.

The action of different lipids (lecithin, cephalin and galactolipids) on saponin was studied. It could be demonstrated that mixtures of equal parts of saponin and lipids did not reduce the hemolytic, myelolytic and toxic effects. Mixtures of one part saponin with five parts of the lipids were much less hemolytic. While the combination with lecithin was also reduced in its myelolytic and toxic action, however, the combination of saponin with five parts of galactolipids had the same myelolytic and toxic effect as the 1:1 combination, if high toxic doses were used. The inhibitory effect of lecithin or sodium taurocholate was much more pronounced than the effect on saponin. Five parts of lecithin added to one part of sodium taurocholate completely inhibited the latter's hemolytic, myelolytic and toxic action.

Mixtures of saponin with red blood cells were no longer hemolytic, and less myelolytic and toxic than the corresponding amount of saponin alone. Mixtures of saponin with emulsion of gray matter or emulsion of white matter of brains or emulsion of total brain substance were much less hemolytic and less myelolytic and toxic than saponin.

WINKELMAN, Philadelphia.

FUNCTIONAL DYSINSULINISM WITH EPILEPTIFORM SEIZURES; TREATMENT JOHANNES M. NIELSEN and ELMER L. EGGLESTON, J. A. M. A. **94**:860 (March 22) 1930.

Dysinsulinism due to organic pancreatic disease is now established as a cause of epileptiform seizures. The authors present in considerable detail three cases successfully treated, in which the dysinsulinism and resulting hypoglycemia and epileptiform seizures were functional. A rational classification for these cases is difficult to determine. The patients all present symptoms which might be called vagotonia, sympathicotonia, hyposuprarenalism or hyperinsulinism. It is unquestionably more than just a mere dysinsulinism.

Suprarenal extract, whether by hypodermic injection or orally, may produce either hyperglycemia or hypertension without causing an increase in both. One must look to the physiologic interaction of the glands of internal secretion to find a reason for this phenomenon. The pancreas, suprarenals, pituitary, thyroid and liver are certainly concerned in blood sugar regulation. Pituitary and thyroid disturbances are recognized; "hypoglycemic reactions" are not necessarily accompanied by hypoglycemia.

In three cases of "epilepsy" due to dysinsulinism or hyposuprarenalism, the patients have been treated by the authors with frequent feedings or with suprarenal gland, or with both, and the attacks of unconsciousness have ceased for twenty-two months, seven weeks, and eighteen months, respectively. Blood sugar, taken one morning during a starvation period, is not always a reliable index of the patient's low point, especially in cases of functional dysinsulinism. The blood sugar may rise or fall markedly in cases of functional dysinsulinism without any, at present, detectable signs. The degree of increase in blood pressure under epinephrine is no indication that the blood sugar has risen and vice versa. Frequent feeding alone may suffice in these cases, but the addition of orally administered suprarenal gland is a surer way. Another group of cases has thus been subtracted from the large unknown group of idiopathic epilepsy. The treatment here outlined had no effect in cases of idiopathic epilepsy.

CHAMBERS, Syracuse, N. Y.

TWENTY YEARS OF THE NATIONAL COMMITTEE FOR MENTAL HYGIENE. GEORGE K. PRATT, Ment. Hyg. **14**:399 (April) 1930.

In 1909, the National Committee for Mental Hygiene was founded, directing its first labors to the correction of gross abuses. From the first it was supported by scientific opinion, and not built on sentiment alone. Pratt divides its history into three phases, the antebellum period from 1909 to 1917, the military period to the end of the war, and a postbellum period covering its last ten or eleven years. The first phase was devoted largely to the collection of statistics. A directory of institutions devoted to the care of the insane was an early accomplishment of no mean value; during this period it began educating the public, and soon found itself a clearing house for information about commitment, facilities for treatment and similar matters. In 1912, Dr. Salmon, the first director of the committee, prepared the mental hygiene exhibit at the International Congress of Hygiene, bringing psychiatric prophylaxis, the Cinderella of hygiene, into a more appropriate dignity. The rapid abandonment of jails and almshouses as homes for persons with mental disease may be traced to the influence of this committee. In 1917, the committee closed its first phase by investigating the problem of criminal psychiatry with the establishment of the mental clinic at Sing Sing. During the war, the National Committee advised the army and navy as to the detection of the mentally unfit, and the diagnosis and treatment of the neuroses and psychoses of war. The third phase of the committee's work began with the end of the war, when attention was paid to the training of psychiatric personnel, both medical and nonmedical, and with the publication of the quarterly journal *Mental Hygiene*. The study of the mental mechanisms of children also represents a valuble contribution of this period. The crowning recognition of the labors of the committee came in May, 1930, with the calling of the First International Congress of Mental Hygiene; and with it came new inspiration and new hope of bringing light into the darkness of the problem of human madness.

DAVIDSON, Philadelphia.

LESIONS OF THE NERVOUS SYSTEM OF DOGS IN EXPERIMENTAL LEAD POISONING. E. K. EWSEROWA, Arch. f. Psychiat. **88**:752, 1929.

The author reports observations on experiments with five dogs. Lead was administered in the form of lead acetate, either mixed with the food or in the form of pills. The observations reported include those of a clinical nature as well as histologic examinations post mortem. Clinically, the effect showed itself

in: (1) Psychic changes such as excitement, irritability alternating with apathy and marked weakness. In some of the dogs a definite change in behavior was noted throughout the experiment. (2) In four dogs there were convulsions of a generalized nature, but in the fifth they were typical of cortical irritation. (3) Paralysis of the limbs and ataxia. (4) Emaciation, exhaustion and at times definite cachexia.

The duration of life following the beginning of the administration of the lead varied from thirty-two to two hundred and twenty-four days. The duration of life, as well as the degree and extent of symptoms, had no apparent relation to the amount of lead administered. The histologic changes can be classified under two headings: (1) a diffuse degenerative process in the visceral and somatic centers of the brain and spinal cord with special predilection for localization either in the deep cortical layers or in the tuber cinereum, and (2) cell proliferation of the walls of the blood vessels and the formation of foci in them. In one case a definite focus was found in the nerve tissue.

The author is of the opinion that the psychic manifestations, the convulsions, the paresis and the ataxia, were directly due to the diffuse degenerative processes in the brain, especially in the cortex. The metabolic changes, the exhaustion and the cachexia were probably associated with the lesions in the nuclei of the tuber. This, however, could not be considered as the only cause of these phenomena. In reviewing the literature, the author finds that the histologic changes observed in these experiments did not correspond entirely with those in the case of cats described by other authors. The most important deviations noted were the absence of ameboid neuroglial reactions and the occurrence of proliferative changes in the blood vessels.

MALAMUD, Iowa City.

THE INHERITANCE OF ACQUIRED CHARACTERS. G. SACHAROFF, Med.-Biol. J. (Moscow) **5**:5, 1929.

In mice of from 5 to 6 weeks of age the spleen was removed and after this careful whole and differential blood counts were made from time to time. In the splenectomized animals a leukocytosis develops which persists for from ten to twelve months. When the splenectomized animals were interbred, the F_1 generation also showed a leukocytosis, but less marked than in the parental generation. F_2, F_3 and F_4, on the other hand, showed a leukopenia. When the spleen was removed from both animals in the F_1 generation, the F_2 generation showed a tremendous variation in the number and type of cells. When animals were crossed in the F_2 generation, the F_3 generation showed a very marked leukopenia and the animals died within a very short time.

The author interprets this result as follows: The leukocytosis in the splenectomized animals is due to the overcompensation by the hematopoietic system. The persistence of the leukocytosis in the F_1 generation is due to the transmission of the tendency to overcompensation. However, the fundamental reaction after the splenectomy is leukopenia, and the F_2 generation gives the normal biologic reaction. Both the tendency to leukopenia and the tendency to overcompensation are transmitted in the germ plasm with the result that the extreme reaction to overcompensation spends itself very soon, and the fundamental biologic reaction comes to the surface. The fact that, when the mice of the F_1 generation were splenectomized and interbred, the F_2 generation showed a much greater leukopenia, is a proof of the author's contention, as shown by the formula that crossing DR with DR, one gets mostly DRs. It is significant that transmission of the acquired characters takes place through the mother.

KASANIN, Boston.

IS STUTTERING A MEDICAL PROBLEM? J. A. GLASSBURG, Arch. Otolaryng. **11**:430 (April) 1930.

Speech difficulties should be looked on as a medical subject and the cure supervised by physicians. The lack of instruction on speech disorders in medical colleges is to be deplored. Disorders of speech may be classified as (1) stuttering

and stammering and (2) defective phonation. The author considers stammering and stuttering as synonymous. Heredity is an important factor. These patients are hypersensitive and often have personality defects. Predisposing causes are found in the fact that they are introspective, shy, secretive, overanxious and absent-minded and have inferiority complexes. The exciting causes are mental shock and psychic trauma.

Psychoanalysts think of stuttering as the outward expression of an internal conflict. Stuttering may be defined as a spastic coordination neurosis based on a mental conflict. The patient should have a thorough physical, mental, vocal and phonetic examination, and a careful history should be taken, which should cover at least three generations and include the developmental period, previous illnesses, injuries, operations, habits, progress at school and the home and social environment. Physical causes must be eliminated. A chart for phonetic examination is submitted. In treatment, the first point is to neutralize the emotional obstacle. The patient must understand that the treatment will take a long time. Psychoanalysts may be employed to discover the conflict. Suggestions may give self-confidence; distraction may help; muscles must be relaxed; an effort must be made to make the stutterer less self-conscious by exercising systemic relaxation; proper control of breathing and efforts to exercise the tongue, lips and soft palate will all prove beneficial. As there is no primary disorder in stuttering, efforts to cure with phonetic instruction alone are harmful. However, bad habits of speech may be formed and these require reeducation along phonetic lines.

HUNTER, Philadelphia.

TRAUMATIC PNEUMOCEPHALUS. CARL W. RAND, Arch. Surg. **20**:935 (June) 1930.

Eight cases of traumatic pneumocephalus are reported in detail, with roentgenograms. Communications between a frontal sinus and the subarachnoid space was the cause of the pneumocephalus. In four instances a cerebrospinal rhinorrhea was present. Three patients died of meningitis; of these one was operated on but no rent in the dura was discovered through which air might have been admitted to the subarachnoid space. Of the remaining five cases, four showed dural tears that were repaired by suture alone or re-enforced by fascia lata and muscle transplants. The fifth patient recovered after elevation of the bony depression, the dural tear having been inadvertently repaired at the same time. Of interest is the fact that in only two of these cases did pneumocephalus develop immediately after the injury; in the remaining six cases it developed in from three to seven weeks later. Hence the author stresses that in a case of fracture of the skull communicating with an accessory nasal sinus there should be a careful observation for the possible development of pneumocephalus.

The author believes that "If pneumocephalus is demonstrated, whether the air is present in the frontal lobe, lateral ventricles or both, its removal should be attempted at once, together with closure of the dural rent. Signs of meningitis are often present, even in the absence of infecting organisms; consequently, they should not necessarily deter an attempt to correct the pneumocephalus. It may even be justifiable to proceed in certain cases in which infecting organisms have been demonstrated in the fluid. The prognosis in cases of pneumocephalus is probably better if operative rather than expectant treatment is used. Success depends largely on early recognition and prompt intervention."

MASSON, New York.

SCHILDER'S DISEASE (ENCEPHALITIS PERIAXIALIS DIFFUSA). BENJAMIN M. GASUL, Am. J. Dis. Child. **39**:595 (March) 1930.

After reporting a case of Schilder's disease, Gasul reviews the literature. He mentions Schilder's original paper, in which the condition was limited to diffuse subcortical involvement of the white matter, and shows the broadening of the

concept of the disease as newer studies were made. Pathologically, there are bilateral softenings in the white matter, in which the axons and myelin sheaths, especially the latter, are much affected. The astrocytes first proliferate, but subsequently break down, often leaving nothing but gitter cells as a residue of the cyto-architecture of the affected area. Clinically, the cases are characterized by changes in mental status and by gross sensory defects, especially blindness. The latter is of a cerebral type—that is, there are no disk or pupillary changes. Paralyses of one or more limbs are expected, and these are spastic. Males are more often the victims of Schilder's disease than females, and children make up the vast majority of the patients in the cases reported. The author reviews in tabular form the seventy-one cases reported between Schilder's paper in 1912 and the recent reports of Globus and Strauss in 1928 and Shelden and Doyle in 1929. In the case cited by Gasul, the clinical and pathologic observations are characteristic of the usual pattern of Schilder's disease, except that the occipital lobe was relatively free from involvement, the parietal lobe being the seat of most of the pathologic process. The author closes with a speculation as to whether the disease is degenerative or inflammatory, inclining to the former point of view, because he believes that the perivascular infiltration is comparable to the tissue reactions associated with noninflammatory softenings. He expresses appreciation of the term "progressive degenerative subcortical encephalopathy" synthesized by Globus and Strauss for this condition, but adheres to the older expression "Schilder's disease" as generally preferable.

DAVIDSON, Philadelphia.

THE PHENOMENON OF "RÉFLEXE PENDULAIRE" IN THE NORMAL KNEE JERK. SERGE MAIKOFF, Rev. Psychiat., Neurol. & Reflexol. (Liningrad) **4**:275, 1929.

In 1916, André Thomas described a special variation in the knee jerk in patients suffering from cerebellar disease, which he called "réflexe pendulaire." When a patient was allowed to sit on a high stool with the feet hanging down, stimulation of the quadriceps tendon resulted in a series of to and fro swayings of the leg, resembling the oscillations of a pendulum. This abnormal reflex was very striking in unilateral conditions of the cerebellum. The observations were later confirmed by Austregesilo (*Rev. neurol.*, 1927, no. 3). Both authors make only vague references to the occurrence of the reflex in normal people.

To check this phenomenon, the author investigated the reflex in normal people. A special high stool was designed, with automatic devices to release the reflex hammer so that it would strike the tendon with the same amount of force. The amplitude of the oscillations of the knee was registered by a reflexograph, making tracings on a smoked drum. One hundred and fifty students of the state school of physical education were used as subjects in this experiment; 113 were men and 37 women. It was endeavored to produce the same emotional state in all cases by suggesting to the subject the desire for conscious passivity.

The author found that the tendency toward pendular swaying of the leg, with several to and fro oscillations, is observed in most healthy people. More than six oscillations is probably pathologic. The normal number of oscillations of the leg when the knee jerk is tested is midway between the complete absence of oscillations observed in encephalitis and the large number of oscillations observed in cerebellar disease.

KASANIN, Boston.

CLASSIFICATION OF RETINITIS. ERNST FUCHS, Arch. Ophth. **3**:393 (April) 1930.

The usual classification of retinitis is by etiology and, as Fuchs states, no doubt this is best for clinical purposes. He has presented, however, another classification based wholly on an anatomic foundation in which the various forms of retinitis are considered according as they involve the first, second or third retinal neuron, the entire nervous path or the mesoblastic part of the retina. Further, the various disease conditions of these anatomic parts are subdivided according as they are congenital, acquired and primary or secondary.

Congenital conditions of the first neuron include albinism, pigmentary degeneration, retinitis albescens and tapetoretinal degeneration. Those of the second neuron are the gliomas. Those of the third neuron are the cases of Tay-Sachs' amaurotic idiocy. Those of the entire nervous mechanism include microphthalmus, coloboma, conus, albinism, total blindness and congenital amblyopia. The congenital defects of the mesoblastic part are almost wholly cases of angiomatosis retinae.

The acquired disease conditions are in like manner outlined as they fall within each anatomic subdivision. From the standpoint of a neurologic review of this great paper, those already mentioned and those connected with pathologic conditions of the mesoblastic elements are of most interest. The latter can be subdivided into disease conditions of the blood and of the walls of the vessels. The latter include spasm, arteriosclerosis, syphilis, tuberculosis, nephritis, hemorrhage and Berlin's edema. The author concludes: "while for better understanding the lesions of the different constituents and layers of the retina are dealt with separately, in reality the conditions are much more complicated and intricate. A determined obnoxious agent may act on the tissue in different ways at the same time and different tissues may be affected simultaneously by the same agent."

SPAETH, Philadelphia.

THE HISTOPATHOLOGY AND PATHOGENESIS OF THE WILSON PSEUDOSCLEROSIS GROUP. YUSHI UCHIMURA, Ztschr. f. d. ges. Neurol. u. Psychiat. **123**:679, 1930.

Uchimura found focal changes in the striatum in a case described as belonging to the Wilson pseudosclerosis group. The striatum and pallidum showed an increase in nuclei of a glial nature. These were of two types: (1) small cells filled with fat and (2) typical, large Alzheimer glia cells. The small ganglion cells in the striatum had disappeared. The large cells were present but were degenerated. In the putamen there was perivascular infiltration with fat-laden cells. The most striking feature of the case was an accumulation of fat-laden cells in the striatum, sharply demarcated from the surrounding tissue and without loss of tissue structure. There was a definite relation of these fatty cells to the myelinated fibers, particularly the radiary fibers of the putamen. The caudate nucleus was similarly involved. So also were the external capsule and anterior commissure. In the internal capsule was a focus of fatty cells involving the lowest portion of the capsule. This focus in the internal capsule was a part of the larger focus in the striatum. In all these areas, the accumulation of the fat-laden cells was much more pronounced along the myelinated fibers. The vessels showed only mild changes — mild vascular proliferation, and an increase in the adventitial silver fibrils and the cordons unitifs. The pia showed localized chronic inflammatory changes and the ganglion cells often showed a severe cell disease with an increase in the content of fat.

Fatty degeneration in the Wilson pseudosclerosis group has often been reported. The fatty degeneration in Uchimura's case however, was sharply localized and was without destruction of the network of tissue, indicating that it was a fresh process.

ALPERS, Philadelphia.

THE TREATMENT OF ACUTE EPIDEMIC POLIOMYELITIS BY THE INTRATHECAL ADMINISTRATION OF EPHEDRINE. M. B. BRAHDY and I. H. SCHEFFER, Arch. Int. Med. **45**:102 (Jan.) 1930.

On the theory that the pathology of infantile paralysis lies largely in the pressure on the nerve cells due to hyperemic edema, Brahdy and Scheffer suggest the local use of ephedrine because of its vasoconstrictor affect. They realize that some toxic action may occur, apart from the influence of pressure, but believe that even then the resistance of the cells can be maintained at a higher level by relief from the pressure on them. They recall that the intrathecal use of epinephrine based on the same principle was not followed by much success, but believe that the rapid and temporary action of that drug accounts for its failure.

Instead, they call on the slower and more lasting effect of ephedrine. A 1 per cent solution of ephedrine in spinal fluid is made by first using 50 mg. of ephedrine salt in sterile water (1 cc.), mixing it with spinal fluid (5 cc.) and allowing the mixture to reenter the subarachnoid space by gravitation. For study they selected twenty-nine cases of the bulbar form, all classed as severe and all considered as being prognostically bad. Ten patients received ephedrine as described; the other nineteen did not. The mortality was 50 per cent for the treated and 75 per cent for the untreated group. The authors recognize the unreliability of drawing conclusions from so few cases, but they are certain that benefit resulted from this method of treatment and present this series as a preliminary report. The physiologic basis for the treatment lies in the fact that ephedrine causes the vessels which it reaches to contract, thus reducing the blood supply to the part; this, in turn, means less hyperemia, therefore less swelling and consequently less pressure. If the nerve cell has not already been destroyed, relief from pressure should assist it to recover.

DAVIDSON, Newark, N. J.

THE HISTOGENESIS AND CHEMICAL COMPOSITION OF SENILE PLAQUES. B. HECHST, Arch. f. Psychiat. **88**:126, 1929.

The author reports the results of studies on the clinical composition and the manner of development of the senile plaques. He finds that the whole body of this structure, the nucleus as well as the surrounding area, can be demonstrated by all of the amyloid reactions (such as the iodine stains, methyl violet and kongo red). The amyloid nature of these bodies is furthermore made probable by their insolubility, their resistance to decomposition and various other characteristics peculiar to amyloid structures in other organs. The author is of the opinion that the amyloid substance that goes to make up these bodies originates in the central nervous system itself as a result of pathologic metabolism of the nerve cells. The primary step in the development of these bodies is probably the occurrence of dissolved amyloid in the form of crystals that collect around the smaller blood vessels. This collection of amyloid apparently behaves like a foreign body, irritating the microglia cells. The latter arrange themselves around the foreign body and gradually begin to break up. Hechst does not think, however, that the microglia cells play any primary rôle in the development of the plaque. Neither the apolar neuroglia nor macroglia play any part in the development of this structure. The latter, however, may form a ring around the plaque so as to attempt a defense of the rest of the tissues against the amyloid substance. It would seem that the Alzheimer fibril degenerations have no relationship to the amyloid substance, and therefore the author considers them as entirely unrelated to the senile plaques.

MALAMUD, Iowa City.

THE CONDITIONED REFLEXES IN CHILDREN WITH DECREASED RESPONSES TO STIMULI AND DIMINISHED INHIBITORY REACTIONS. A. WOLOWIK, Med.-Biol. J. (Moscow) **5**:110, 1929.

The work of Pavlov has shown that strong stimuli cause strong responses as well as strong inhibitory reactions. The disturbances in the delicate balance of the two have been described previously. In children with a predominant inhibitory mechanism, one gets hysterical manifestations, while children with dominant stimulatory responses develop reactions similar to those found in epidemic encephalitis. Children have been found who react very inadequately to very strong stimuli. Such a child was used as the subject in this experiment. He has been a patient in Krasnogorski's Clinic for several years, and was a dull, restless youngster, who previously had had a tuberculous infection of the bones with some sequelae.

In using the standard technic for elicitation of the conditioned reflexes, it was found that this child had a fundamental weakness in the development of

responses to strong stimuli and also a weakness in the formation of inhibitions. The stimulus in this case was cranberries, accompanied with the sounds of a metronome and an electric bell.

The author comes to the conclusion that the weakness in responses, as well as the formation of inhibitions, points to a fundamental weakness of the cortical processes. A weak cortex does not provide sufficient regulatory influence over processes arising in the lower centers. Special technic can increase the inhibitory reaction. The asthenic type of salivary glands shows in hyposecretion, which is not increased by administration of drugs such as pilocarpine.

KASANIN, Boston.

A FORM OF MYASTHENIA GRAVIS WITH CHANGES IN THE CENTRAL NERVOUS SYSTEM. DOUGLAS MCALPINE, Brain **52**:6 (April) 1929.

This article is based on a clinical and pathologic study of a case of myasthenia gravis in which certain changes in the central nervous system were found that have not previously been described. A woman, aged 23, developed dysarthria, which was followed in a month by diplopia and fatigability. These symptoms always were more marked during the evening or after exertion. During fifteen months in the hospital her condition gradually grew worse. Dysphagia, weakness of the neck muscles, paresis of the limbs and attacks of dyspnea became marked. Death occurred in a dyspneic attack.

The histologic observations were unusual. All of the visceral and endocrine organs were normal except the thymus gland which was markedly hypertrophied. No lymphorrhages were observed in the muscles examined. The cerebral cortex, subcortical white matter and basal ganglia were normal, apart from mucocytic degeneration which extensively involved the white matter. The motor nuclei in the brain stem were unaltered, except for slight changes in a few nerve cells ot the oculomotor nuclei. Many of the vessels of the spinal cord had a marked perivascular round cell infiltration. A slight degree of gliosis was present, particularly about the anterior horns.

Mucocytic degeneration is fully discussed. The literature on this interesting and still somewhat obscure topic is well reviewed, from Buscaino's original observation to Ferraro's recent critical review. A possible relationship of myasthenia gravis to epidemic encephalitis is indicated because of a similarity in the pathologic changes.

BECK, Buffalo, N. Y.

PAIN OF CENTRAL ORIGIN: A DISCUSSION OF SOME DISEASES OF THE CENTRAL NERVOUS SYSTEM IN WHICH PAIN IS A MAIN SYMPTOM. H. L. PARKER, Am. J. M. Sc. **179**:241 (Feb.) 1930.

Cases representing the different central sensory levels of the nervous system are reported. Traumatic lesions, especially of the cervical region, syringomyelia, and intramedullary and extramedullary tumors of the spinal cord are conditions of the spinal cord that are sometimes associated with central pain. In the medulla oblongata, the syndrome of thrombosis of the posterior-inferior cerebellar artery, syringobulbia and tumor of the fourth ventricle may be accompanied by central pain. Herpes involving the fifth cranial nerve, especially in its first and second divisions, is often associated with persistent, severe, constant and burning pain. An avulsion of the gasserian ganglion may be performed but without avail.

Since Dejerine and Roussy, in 1906, described the syndrome of the optic thalamus, numerous cases have been reported of a somewhat similar nature. Head ably elaborated and emphasized the excessive response to affective stimuli. An interesting case of a tumor involving the left precentral and postcentral gyri is described as having caused symptoms of a burning, aching sensation in the right side of the face that later passed into the right arm.

MICHAELS, Detroit.

A Case of Decerebrate Rigidity, with Autopsy. S. H. Epstein and P. I. Yakovlev, J. Neurol. & Psychopath. **10**:295 (April) 1930.

The authors give a historic discussion of decerebrate rigidity and report a case. The case is that of a boy, aged 8, who was born after a long and difficult labor and was cyanotic for six weeks. He had convulsions of two types from the time of birth until death. One type was a generalized tonic convulsion, while the other consisted of "crying spells" of a peculiar type. The first type was produced by excessive sensory stimulation — usually auditory — while the second was brought on by excessive manipulation. The four major points in the examination of the patient were: (1) attitude, (2) muscle tonus, (3) fits and (4) general and local motor reactions. These were similar to those described by other authors.

The pathologic examination showed various aplasias of the cerebrum and cerebellum, and an entire lack of the corpus callosum. Throughout the cerebrum, cerebellum and midbrain there was a paucity of ganglion cells, an increase of all glial elements and an increase of capillaries. The cortical layers of the cerebrum were not only deficient in ganglion cells, but the cellular architecture was greatly disturbed. The spinal cord showed little disturbance of the gray matter, but the corticospinal tracts were completely degenerated.

This case is of interest because it confirms clinically much experimental work on this subject.

Robinson, Kansas City, Mo.

Papillomas of the Choroid Plexus. William P. Van Wagenen, Arch. Surg. **20**:199 (Feb.) 1930.

Two cases of true papilloma of the choroid plexus of the left lateral ventricle are reported. One, in the case of an infant, aged 3 months, was uncovered at an operation, treated with deep roentgen-ray therapy and later removed in toto at a second stage operation. In the second case the tumor was encountered at autopsy; it showed one large growth with numerous small papillomas "seeded" throughout a portion of the ventricular system. The choroid plexus in the right ventricle was normal. Not only the author's case but some five or six other cases in the literature are examples of "seeding" of choroid plexus papillomas. This, van Wagenen considers not as an evidence of malignancy of the tumor, but rather as a probable accidental occurrence in which trauma may have played a considerable part.

A review of the literature revealed about forty-five cases; from them the author found that the favorite site for these tumors is in the fourth ventricle where 50 per cent of the recorded cases were found; 34.7 per cent were in the lateral ventricles, and 17.3 per cent in the third ventricle. By some curious coincidence, 93 per cent of tumors of the lateral ventricle have been found on the left side. Van Wagenen also found that the age incidence was greatest in the first decade and gradually diminished up to the sixth decade.

Masson, New York.

Regulation in the Growth of Transplanted Eyes. Victor C. Twitty, J. Exper. Zool. **55**:43 (Jan. 13) 1930.

Grafts were made, heteroplastically, between *Amblystoma punctatum* and *Amblystoma tigrinum*. The experiments demonstrated that the environment affects the growth of the eye, since it is possible, by grafting this organ in the proper direction, either to inhibit or to accelerate its growth relative to the growth of the host. If the host is the older the eye may be made to grow strikingly, while the growth of the host and the normal eye is prevented by very light feeding. If these animals are given no food, however, this tendency toward equalization does not appear. If the host is younger than the donor, the growth of the eye is inhibited even when the host is fed liberally and grows at a rapid rate. This condition can be explained by assuming that during the growth of the animal the eye and its organic environment normally undergo correlated, progressive changes

which regulate the gradual expression of the intrinsic growth capacity of the eye. Expression of the growth capacity of the eye is primarily a function of the relation between the physiologic conditions in the organ and in the environment provided by the host, rather than of mere growth in size of the latter. One is inclined to conclude that the potential size of the eye is largely determined by intrinsic factors, but that the expression or realization of this potentiality during the growth of the animal depends on its interaction with a gradually changing organic environment.

WYMAN, Boston.

SOME OBSERVATIONS ON EXPERIMENTALLY PRODUCED CONVULSIONS. F. H. PIKE, C. A. ELSBERG, W. S. MCCULLOCH and A. RIZZOLO, Am. J. Psychiat. **9**:259 (Sept.) 1929.

By injecting intravenously an alcoholic solution of absinth, Pike and his colleagues have been able to produce convulsions in cats. They discovered that with the motor apparatus intact the convulsions are clonic, but that if they ablate or injure the hemispheres, the motor phenomena become tonic. If given time to recover, the decorticated animal will give clonic convulsions again under absinth intoxication. The authors expressed the belief that this is due to the fact that other motor mechanisms, possibly those of the opposite side, have assumed the upper neuron motor function. It was not shock or inhibition which accounted for the clonic convulsions being replaced by tonic after removal of a hemisphere, but rather the fact that the cells through which the stimulus to clonic movement passed—namely, the Betz cells—were destroyed. Furthermore, this does not prove that the extrapyramidal tracts mediate tonic movements; it merely shows that this occurs if the cortex is injured. The authors conclude by expressing agreement with the belief of Hughlings Jackson that the motor mechanism acts together as a unit when the brain is intact.

DAVIDSON, Newark, N. J.

AN INTERPRETATION OF DEFECTS IN THE VISUAL FIELD. J. N. EVANS, Arch. Ophth. **3**:153 (Feb.) 1930.

This is the presentation of an extensive work done by the author in the study of angioscotometry, i. e., defects in the visual field due to the influence of the perivascular lymph spaces (retinal). The author first discusses in great detail and with a wealth of references from the literature the minute anatomic structure of the vessels and of the perivascular spaces, their presence and development, their relationship to the anatomy and physiology of the retina, and their relationship to the mechanism of venous stasis (posterior drainage mechanism), as it applies to the fluids of the eye, and hence "as it pertains to the interpretation of defects in the visual fields."

The author next discusses the formation and the mechanism of the production of scotoma. This is a logical continuation of the accepted facts and theories presented in the first part of the paper. The necessary technic is also stated.

The value of the detailed examination from a diagnostic standpoint lies in the influence of the angioscotoma on the mechanism of the defects of the central field (glaucoma, toxic conditions characterized by defects in the central field), to certain peripherally located blind areas, and to cases in which there is a known interruption of the conductivity of the fiber bundle, as by tumor pressure, along the optic pathways back of the eye.

SPAETH, Philadelphia.

TURNING TENDENCY AND CONJUGATE DEVIATION. O. OEDEGAARD and P. SCHILDER, J. Nerv. & Ment. Dis. **71**:260 (March) 1930.

The case is described of a man, aged 25, who complained of stiffness of the left side of the neck for five years. Attacks were observed in which loss of consciousness with general rigidity, in which turning of the head from side to side and bending of the body forward or to the right were present for eight months.

They were followed by confusion, disorientation, and auditory and visual hallucinations. Craniotomy and roentgen examination showed no evidence of tumor of the brain. Visual perception became impaired. Turning of the body from 90 to 180 degrees, beginning with the head, occurred most often while the patient was in the sitting posture. Apraxia and decreased reaction to pain stimuli in the arm were observed. During an epileptiform attack, the turning tendency was increased; afterward it decreased. The author considered the most probable diagnosis as a tumor and located it in field 19 of Brodmann, which Foerster has shown to be involved with conjugate deviation to the opposite side. The excitation in this case probably radiated from this center to neighboring centers by turning of the trunk. Rothfeld described a case with this tendency in which a tumor of the thalamus involving the occipital region was considered. In this case the author considered that the loss of pain appreciation was connected with a lesion of the neighboring supramarginal gyrus.

HART, Greenwich, Conn.

QUANTITATIVE PERIMETRY AND TUMORS OF THE TEMPORAL LOBE (CUSHING SCHOOL). A. KOUTSEFF, Rev. d'oto-neuro-opht. **7**:325 (May) 1929.

This article extols the merits of quantitative perimetry as practiced by Cushing, based on the publications of the latter. Since the writings of Cushing are available to American neurologists, it appears unnecessary to do more than repeat his conclusions: (1) The temporal lobe is frequently the seat of cerebral tumors—21 per cent of supratentorial tumors. (2) In fifty-nine cases of verified tumors of the temporal lobe, perimetry has been impossible in twenty cases. Of the thirty-nine remaining, homonymous hemianopias, total or in quadrant, were present in thirty-three (84 per cent). (3) In former times, uncinate gyrus crises were thought to be the most important symptoms of tumors of the temporal lobe; in this series these crises were noted in only twenty-four cases (40 per cent). (4) Visual hallucinations, often under the form of animated pictures appearing on the side opposite from the lesion, were frequent, occurring thirteen times (22 per cent). (5) Auditory symptoms were almost absent. (6) Errors of diagnosis are difficult to avoid: (*a*) when the tumor is localized in the occipital lobe in the presence of a total homonymous hemianopia and (*b*) when the cerebellar symptoms are marked and there is not a clear hemianopia in quadrant.

Although a minute examination of the visual fields is a long procedure, the trouble is justified by the important indications that are obtained.

DENNIS, Colorado Springs, Colo.

SOME EXPERIMENTS IN INTRACRANIAL PRESSURE IN MAN DURING SLEEP AND UNDER CERTAIN OTHER CONDITIONS. LEWIS STEVENSON, B. E. CHRISTENSEN and S. BERNARD WORTIS, Am. J. M. Sc. **178**:663 (Nov.) 1929.

Contrary to the prevalent theory that sleep is associated with a decrease in intracranial pressure along with cerebral anemia, the experiments reported in this article attempt to show that the intracranial pressure rises as the patient falls asleep, is maintained during sleep and falls on awakening. The apparatus consisted of one tambour bandaged over the brain hernia and another tambour registering on a smoked drum the changes of intracranial pressure due to pulse, breathing and sleep. Five patients with brain hernia were the subjects. In a patient with jacksonian epilepsy and a brain hernia, the fits ceased during gas-oxygen-ether and chloroform anesthesia, and there was a marked rise in the intracranial pressure.

Morphine increases intracranial pressure, whereas caffeine decreases it, therefore contraindicating the use of morphine for the relief of headache in brain tumor and indicating the use of caffeine to reduce intracranial tension during brain operations. Pituitary, epinephrine, ephedrine sulphate and scopolamine caused a delayed but prolonged rise in intracranial pressure. It is suggested that the sympathetic center in the brain which maintains vasomotor tone in the vessels

becomes periodically fatigued, resulting in a dilatation of the blood vessels of the brain as well as those of the periphery. With increase in brain volume there would be a consequent pulling apart of the neurons (diaschisis); the increased intracranial pressure would tend to alter the conductivity of the synapses.

MICHAELS, Detroit.

THE PATHOGENESIS OF TABES DORSALIS. O. GAGEL, Ztschr. f. d. ges. Neurol. u. Psychiat. **122**:423, 1929.

The earlier conception that tabes was due to a primary degeneration of the posterior columns has been opposed by the view that it is purely a secondary degeneration. The primary process lies in the posterior root and consists of a sclerosing granuloma with spirochete invasion. The customary seat of this process is in the Nageotte locus. Owing to this local syphilitic process there is an interruption of the axis cylinders in this location which leads to a secondary degeneration in the peripheral portion of the neuron, i. e., in the posterior columns. This conception is true of old cases of tabes. Spielmeyer has warned, however, that one must look on the changes in the posterior column in tabes as a purely secondary degeneration. The occurrence of changes in the posterior column in cerebrospinal syphilis and tuberculous meningitis has led him to doubt the views of Richter already given. He believes that it is necessary to investigate early cases of tabes in order to be clear about the pathogenesis of the process.

Gagel reports a case of early tabes in order to test the soundness of Richter's ideas. His patient, a woman, aged 36, had suffered for three years from ataxia and stiff pupils, and for six months from a hypotonic paralysis of the lower extremities. Microscopic examination revealed no changes in the Redlich-Obersteiner field. Fatty changes were found in the intramedullary portion of the posterior root, and some loss of myelin sheaths in the same region. These changes were not found in the extramedullary positions.

Gagel believes that his case supports the contention that tabes is due, at least in part, to a primary degeneration of the posterior columns.

ALPERS, Philadelphia.

LA SIGNE DE L'APPROBATIVITÉ DANS LES PSYCHOSES. PAUL COURBON, Ann. méd.-psychol. (pt. 2) **87**: 385 (Dec.) 1929.

To the feeling that many psychotic patients have, a feeling that they are in perfect rapport with their environment, Courbon applies the term "approbativité." This feeling is most characterisically observed in dementia paralytica, but it is not limited to that condition. He conceives of six kinds of "approbativité": imitation, indifference, consolation, conciliation, conviction and convention. The approval of imitation or pretense is observed in the well preserved psychoses where the patient hypocritically puts himself in agreement with his attendants in order to win favors from them. Approval of indifference is the agreeableness of the schizophrenic, the suggestibility, the willingness to obey commands in order to avoid being annoyed. The approval of consolation is that which, for example, the manic patient exacts from his environment. It is the attitude of the attendants who humor the excited patient lest they further excite him. The "approbativité" of conciliation is the attitude of irritable suppliance, observed, for instance, in a senile dement. The approval of convention is the air of patronage exercised by a grandiose patient, a feeling that of course his fellows agree with him—they ought to, they owe it to him. Finally, there is the "approbativité" of conviction—the feeling that everyone around is en rapport with him—experienced, for example, by the patient with dementia paralytica. It is the agreeableness of faith and sincere conviction. Courbon presents his paper as a contribution to the study of psychiatry by examining special symptoms instead of endeavoring to grasp a unified conception of a whole mental disease.

DAVIDSON, Newark, N. J.

TRANSITORY MOTOR OCULI PARALYSIS: PROBABLY OF TUBERCULOUS ORIGIN. M. A. COLRAT, Rev. d'oto-neuro-opht. **7**:339 (May) 1929.

The case reported evidences the difficulty of etiologic diagnosis in transient ocular paralyses but its subsequent course throws light on the etiology. A single woman, aged 24, whose family and past history was unimportant, suddenly became diplopic in March, 1926. A spinal puncture brought a clear fluid containing 0.4 Gm. of albumin and 2 cells per cubic millimeter. Examination of the eye revealed: left eye, normal with 9/10 vision; right eye, vision 6/10, eyeground normal, pupil dilated and an external strabismus. The right eye could not be directed downward and its movement to the left was limited. A diagnosis of paralysis of the motor oculi, affecting especially the iris and the inferior rectus, was made. The nervous system and the visual fields were normal. In November, 1926, the diplopia had disappeared but a feeble dilatation of the pupil and a slight internal strabismus remained. In December, 1927, the patient developed a dorsolumbar Pott's disease with a cold abscess. All ocular symptoms had disappeared.

The interesting points are: Increase of albumin in the spinal fluid with a normal cell count. This was evidently associated with the Pott's disease, which appeared clinically only a year later. There were no evidences of epidemic encephalitis or of specific meningitis, therefore the ocular paralysis was due to a tuberculous lesion, probably a toxic neuritis affecting the trunk of the motor oculi.

DENNIS, Colorado Springs, Colo.

EXPERIMENTAL CATATONIA BY BULBOCAPNINE AND THE CATATONIC SYNDROME IN MAN. H. DE JONG and H. BARUK, Encéphale **25**:97 (Feb.) 1930.

Experiments of a clinical and physiologic nature made on animals with graduated dosages of bulbocapnine yield a series of symptoms which appear to be entirely superimposable on the catatonic syndrome as it presents itself in man. These characteristics of identity include loss of initiative and activity, negativism, occasional hyperkinesis, vegetative symptoms (salivation, respiratory disturbance, vomiting, etc.), sleep crises and epileptiform crises. Even in the psychic sphere, where analogies between human beings and animals are especially dangerous, there is consistently noted the tendency in both men and animals to isolation and loss of contact with the social milieu. Smaller doses produce this characteristic in the animal, so that it may be looked on as a relatively early sign of intoxication, preceding the motor disturbances. The course, which is characterized by the sudden disappearances and reappearances of symptoms in many cases of human catatonia, is likewise similar to that in experimental animals and speaks for the hypothesis of intoxication instead of a destructive, organic type of lesion.

Considerable discussion adduces evidence that the muscular tension of catatonia is not a passive contracture but is a voluntary type of active contraction—a type of physiologic tetanus. The fact of the relative simultaneous alterations in the chronaxia of flexors and extensors supports this view. Experimental catatonia through bulbocapnine is an important point in favor of the organic nature of catatonia.

ANDERSON, Los Angeles.

BASAL METABOLISM AND EMOTIONAL STATES. GEORGE W. HENRY, J. Nerv. & Ment. Dis. **70**:598 (Dec.) 1929.

The author attempts to correlate basal metabolic processes with certain emotional states and their common psychomotor accompaniments and concludes that there is a definite relationship between basal metabolic processes and emotional states regardless of the clinical type of personality disorder. Elated, overtalkative and overactive states are accompanied by acceleration of the basal metabolic processes, while depressed, underactive and undertalkative states are accompanied by retardation of the basal metabolic processes. Apprehensive, tense and agitated states usually imply an acceleration of the basal metabolic processes, but this may be counteracted in some cases in which the feeling of depression is intense. Apathetic

states are accompanied by a retardation in the basal metabolic processes which is not as great, however, as that associated with feelings of depression. It is probable that some of the so-called normal variation in the metabolic rate is due to the different emotional states presented by the person under test. The basal metabolic rate may be altered to a pathologic degree with no other apparent factor than an unusually intense emotional state, and therefore the interpretation of the rate in any given case, regardless of the type of illness, requires the consideration of the prevailing emotional state of the person studied.

HART, Greenwich, Conn.

EXPERIMENTAL INVESTIGATIONS ON THE PENETRATION OF METALLIC SALTS INTO THE CENTRAL NERVOUS SYSTEM BY INHALATION. HANS HOFF, Jahrb. f. Psychiat. u. Neurol. **46**:209, 1929.

1. Mercury can be introduced into the central nervous system by the inhalation of mercurial salts, whereas following intramuscular or intravenous injection of this metal or inunction with it the metal is taken up by the liver and kidneys and does not reach the central nervous system.

2. Arsenic can be introduced into the central nervous system by the inhalation of arsenical salts. The prolonged use of this metal by inhalation is followed by toxic manifestations.

3. Intramuscular and intravenous injections of sodium iodide are followed by the entrance of small amounts of iodide into the central nervous system, and much larger amounts into the liver; the reverse of this occurs following the inhalation of these salts.

4. The inhalation of iodides is frequently followed by toxic manifestations; some of the animals recovered from these thyrotoxic manifestations, following which they showed an unusually high degree of hypersensibility to iodides.

5. No immune bodies were found in the central nervous system or in the cerebrospinal fluid of animals subjected to inhalation of metallic salts.

KESCHNER, New York.

FAMILIAL CONGENITAL SPASTIC DIPLEGIA. FLORENCE POWDERMAKER, Am. J. Dis. Child. **39**:148 (Jan.) 1930.

While it is common to look for prematurity, infection or birth trauma as cause for a case of congenital spastic diplegia, Powdermaker believes that the real origin of the disorder lies in defective germ plasm. She quotes Sigmund Freud, who before he became interested in psychoanalysis was a student of organic neurology and the author of a now classic paper on cerebral diplegias. It was Freud's belief that incidental factors, such as birth injury, had been overestimated, and that the true cause of these conditions lay in primary degenerative processes in the neurons. The author emphasizes her contention by presenting three brothers with diplegia; they were the children of healthy parents, born by normal deliveries and presented no evidence of an infectious factor. That these boys had healthy brothers and sisters is not, Powdermaker thinks, evidence to oppose the theory of prenatal germ defect, because she supposes that such defect might be recessive in the mendelian sense. Her conclusion is that Little's disease, and conditions of congenital spasticity in general, are not due to syphilis, prematurity, birth injury or any other external agent, but rather to defect in the prenatal germ plasm.

DAVIDSON, Newark, N. J.

PHOTORECEPTORS IN MYA ARENARIA, WITH SPECIAL REFERENCE TO THEIR DISTRIBUTION, STRUCTURE, AND FUNCTION. V. E. LIGHT, J. Morphol. & Physiol. **49**:1 (March 5) 1930.

Experiments on the responses to light in *Mya arenaria* indicate that photosensitive tissue is located somewhere near the inner surface of the siphon, and that

the siphon is sensitive throughout its length. Cells of a special type are found throughout the length of the siphon just beneath the inner epithelial layer. They are most abundant where the inner surface of the siphon is most sensitive. They receive nerve elements from branches of sixteen large nerves. Each cell contains a characteristic inner structure, the optic organelle, composed of a rather large hyaline lens, which is surrounded by a network of nerve fibrillae, the retinella. Light rays, reflected from a flat mirror through the lens in these cells, are brought to a focus in the region of the retinella, irrespective of the direction of the rays. The cells are similar in structure and function to visual cells in leeches and photoreceptors in the earthworm. Available data indicate that they function as photoreceptors and that the fibrillae of the retinella are direct receptors of light stimuli. Pigment spots found on the distal third of the siphon, and thought by some to be eyespots, are, owing to simulation of the background, probably protective in nature, rather than functional in photoreception.

WYMAN, Boston.

THE POSTNATAL DEVELOPMENT OF INDIVIDUAL CYTO-ARCHITECTONIC AREAS IN THE DOG. S. SARKISSOW, J. f. Psychol. u. Neurol. **39**:486, 1929.

The area gigantopyramidalis, area frontalis agranularis and area striata differ from each other in the dog at the moment of birth not only by the peculiarities typical of each one of these areas but also by the degree of development of their respective architectonic peculiarities. The representative of the motor zone, the area gigantopyramidalis, is, at birth, definitely better formed than the area striata. In the course of postnatal development, the tempo and character of development of the areas named show great differences, the transformation of the architectonic elements in the area striata being distinctly more rapid than those of the area gigantopyramidalis and area frontalis agranularis. Variations appear in the tempo and character of development not only of the areas themselves but also of the individual layers; these variations occur either in the rapidity of the tempo of development or in an increase or disappearance of the layers. In the process of postnatal development, the area gigantopyramidalis and partly also the area frontalis agranularis remain behind that of the area striata. In contrast with this, the area striata becomes unusually large simultaneously with the rapid development of the architectonic structure and of the cellular elements, as well as regards the size of its surface on the cerebral cortex.

KESCHNER, New York.

BILATERAL CONGENITAL FACIAL PARALYSIS. B. E. BONAR and R. W. OWENS, Am. J. Dis. Child. **38**:1256 (Dec.) 1929.

After reviewing the literature of congenital facial palsy, the authors find only six cases in which both sides were involved, but which were uncomplicated by other involvement of the cranial nerve. They add a seventh case to this list. The condition, the authors believe, represents an agenesis of the facial nuclei. They also consider other aspects of the problem of congenital facial palsy. Because of the difficulty of testing sensory function in babies, they suggest a practical classification into obstetric and nonobstetric types. The former they subdivide into three groups: forceps trauma, antepartum compression and intrapartum compression. Because of the site of the forceps blade, facial palsy due to this manipulation is usually unilateral and peripheral, and usually shows favorable results. Antepartum compression, on the other hand, results in maldevelopment, and the palsy is usually permanent, though ordinarily unilateral. The infants with palsies due to compression during delivery usually recover. Symptomatically, congenital facial palsies present the same clinical picture that the adult forms do.

DAVIDSON, Newark, N. J.

HEADACHE OF TONSILLAR ORIGIN. D. I. VASILIU, Rev. d'oto-neuro-opht. **7**:750 (Dec.) 1929.

This report concerns a man, aged 45, who from childhood had had hypertrophied tonsils and from time to time attacks of peritonsillar abscess. He suffered with persistent and continual occipital headaches which usually became

worse during the attacks of quinsy. Examination revealed enormous tonsils on one of which was a necrotic spot which was exquisitely painful. Three days after removal of the tonsils the headaches disappeared completely, and the patient has remained free from them for a year.

After enumerating the various causes of headache, the author discusses the mechanism of reflex headache, calling attention to the analogy between the innervation of the meninges and that of the tonsils, the latter being supplied by the pharyngeal and tonsillar plexuses with participation of the sympathetic and the former mainly by the trigeminus, sympathetic and parasympathetic systems. In addition to the reflex mechanism, the author thinks that another factor in tonsillar headaches is the continual systemic toxemia due to absorption from the purulent foci in the tonsils. He recommends tonsillectomy for rebellious headaches when their origin cannot be determined.

DENNIS, Colorado Springs, Colo.

CHRONIC SUBDURAL HEMATOMA. FRANKLIN JELSMA, Arch. Surg. **21**:128 (July) 1930.

Trauma, usually slight, is the primary cause of chronic subdural hematoma. The symptoms do not appear until from four to ten weeks after the injury, and by this time the apparently minor accident that precipitated the condition is often forgotten. Headache is the most prominent early symptom, with drowsiness and mental changes occurring later. Organic neurologic signs pointing to a cerebral lesion are usually elicited, choked disks being found in 40 per cent of the cases, while motor disturbances are even more frequent (70 per cent). The clot should be localized by the neurologic picture; if this is inadequate, exploration of the subdural space through burr holes is advisable. Jelsma prefers this method of operative investigation to making an encephalogram or a ventriculogram. Surgical removal of the clot through an osteoplastic flap is advised, a method which yielded an 83 per cent recovery rate in the author's hands. Postoperative cerebral edema may be diminished by the intravenous introduction of a 60 per cent solution of dextrose.

DAVIDSON, Newark, N. J.

THE DEVELOPMENT OF THE SENILE PLAQUE IN ALZHEIMER'S DISEASE AND OTHER SENILE CEREBRAL DISEASES EXAMINED BY APPLYING DEL RIO-HORTEGA'S METHOD OF IMPREGNATION. W. J. C. VERHAART, Acta psychiat. et neurol. **4**:399, 1929.

Verhaart believes that the senile plaque begins as a disturbance in the glia reticulum as shown by the locally increased capacity of the tissue for silver impregnation. There are no cell elements in this incipient plaque, and its nature is not ascertainable by the Hortega method. Some toxic process seems to be initiated by these plaques which attracts Hortega cells. In benign cases the Hortega cells gain the victory over the plaque, and no permanent degeneration in the brain results. If the Hortega cells succumb, a macroglia scar is formed with permanent degeneration of the parenchyma. The plaques in Alzheimer's disease demonstrate their malignancy by the presence of a great number of degenerated Hortega cells. The plaque in itself does not cause disease unless, as a consequence of the number and their toxicity, permanent degenerations in the nervous system result.

PEARSON, Philadelphia.

NON-SPECIFIC PROTEIN THERAPY IN NEUROSYPHILIS. HARRY GOLDSMITH, Am. J. Psychiat. **9**:500 (Nov.) 1929.

Seeking a safer fever therapy than malaria, Goldsmith studied the effects of typhoid inoculation of patients suffering from dementia paralytica and cerebral syphilis. By using twenty doses, injecting a hundred million dead organisms on the first day and increasing to two billion on the last, he was able to produce febrile reactions, the average temperature being 103 F. Among the patients submitted to this treatment there were good remissions in more than half; 36 per

cent of the patients were able to return to work. The therapy is more benign than malaria and is applicable to many patients who are too weak to tolerate plasmodium inoculation. The modification of the serology is particularly striking in the colloidal curve results; over 80 per cent of the patients studied showed a fall from a high zone I reaction to a nonparalytic curve. Goldsmith believes that nonspecific protein treatment is a valuable substitute for malarial therapy, is quite as effective and is of much wider application.

DAVIDSON, Newark, N. J.

THE TOPICAL DIAGNOSIS OF HEADACHE. EDGAR TRAUTMANN, Deutsche Ztschr. f. Nervenh. **110**:67 (Sept.) 1929.

The entire investigation of headache is founded on self-observation and exploration. One can differentiate primary localizations, irradiations and secondary localizations, for the specific etiology of which one is referred to the primary center. The establishment of primary centers is the first task to be undertaken. Delimitation of irradiation is accomplished by the exploration of zones of the slightest degree of painfulness. The pain center itself should be studied for character, degree and duration of the pain itself. The author analyzes the following five types of headaches: (1) migraine; (2) traumatic; (3) frontal lobe syndromes; (4) psychic; (5) headaches occurring in the vegetative neuroses. He regards headaches in which there is absence of cerebral symptoms as relatively harmless. In their presence, however, the importance depends on the localization and functional significance of the part of the brain affected. The establishment of a clinic for headaches is recommended for more accurate topical diagnosis.

HART, Greenwich, Conn.

DERMATOPOLYNEURITIS WITH MOTOR PARALYSIS OF THE LOWER EXTREMITIES. A. B. SCHWARTZ, Am. J. Dis. Child. **39**:359 (Feb.) 1930.

In calling attention to the frequency of neurologic symptoms in acrodynia, Schwartz suggests that this disease be called "erythredemapolyneuritis." In most of these cases striking sensory disturbances have been reported, so that the presence of motor symptoms in the author's case is a matter of especial interest. His patient was a girl, aged 2, who, five months after a severe sore throat, developed hyperhidrosis, irritability, malaise and coldness of the extremities. Then, in order, the following symptoms developed: somnolence, trembling, a rash, urinary incontinence, weakness in the legs, desquamation of the skin of the hands and feet, and, within four months of the onset of the first symptoms, flaccid paralysis of the lower extremities. There was also hypesthesia in the legs. Recovery began about seven months after the onset and was very slow. A complete return to normal health did not occur until a year after the beginning of the symptoms. Faradic stimulation, hygienic care, tonsillectomy and massage were the therapeutic measures employed.

DAVIDSON, Newark, N. J.

THE VEGETATIVE NERVOUS SYSTEM IN SCHIZOPHRENIA. H. H. TIMOFÉEFF Rev. Psychiat., Neurol. & Reflexol. (Liningrad) **4**:263, 1929.

The vegetative nervous system was studied in sixteen cases, most of which were either of the simple or the hebephrenic type. Clinical studies and pharmacodynamic tests were used. The following results were obtained: There is no relationship between the stage of the disease and the vegetative nervous system. The intravegetative ataxia of Ossipov was observed in many of the cases studied. This consists in blotchy discoloration of the skin, inverted oculocardiac reflex and dissociation between the emotional state and the state of the pulse and pupils. In other words, in the presence of an emotional reaction, there was no corresponding dilatation of the pupil or rise of the pulse rate. In the pharmacodynamic tests, experiments with pilocarpine were omitted on account of the unreliability of the test. In experiments with epinephrine, there was no response in cases of dementia simplex, but there was a marked reaction to epinephrine in catatonic and paranoid cases.

KASANIN, Boston.

CHRONIC MENINGOCOCCUS SEPTICEMIA. HARRY VESELL and JOSEPH BARSKY, Am. J. M. Sc. **179**:589 (May) 1930.

Soloman, in 1902, reported the first chronic variety of meningococcus septicemia. The case of a woman, aged 43, running the typical course of the disease, is reported. Usually there is a sudden onset of constant headache, intermittent fever and pains in the joints, followed by chills, and at the end of the first week a characteristic rash of the multiform erythematous type. The average duration of the disease is several months. Meningitis, when it does occur, is usually late and is not necessarily a grave complication. Endocarditis, pericarditis and myocarditis have been described as complications, and the spleen has been found palpable in most cases. Blood cultures taken early in the disease most frequently reveal no growth; later it is usually necessary to use properly enriched culture mediums. Normal spinal fluid was found in the cases in which meningitis was not present. The only treatment found to be of any value was antimeningococcus serum injected intravenously.

MICHAELS, Detroit.

MENINGO-NEPHRITIS (THE INFECTIOUS, LATENT MENINGITIS IN UREMIAS WITH NERVOUS SYMPTOMS, IN NEPHRITIS AND IN ICTERUS GRAVIS). P. SAVY and H. THIERS, Ann. de méd. **27**:39 (Jan.) 1930.

Nervous manifestations in uremia are relatively frequent. They are usually attributed to toxic manifestations following renal insufficiency. The lesions that are found at autopsy are the sequelae of vascular lesions in the form of hemorrhages, softening or meningeal hemorrhages. The authors report a group of cases of uremia in which only mild nervous symptoms were reported, but in which the laboratory examination had demonstrated the presence of pneumococci in the cerebrospinal fluid, though no evidence of meningitis could be demonstrated clinically. Histologic examination, too, did not reveal any meningeal pathology. Similar observations were reported in cases of icterus gravis. It is impossible, however, to decide whether the latent meningitis was produced by the toxins of the pneumococci or by the chemical changes of the body fluids which are always found in uremia.

WEIL, Chicago.

NERVOUS INSTABILITY AND GENIUS. P. A. WITTY and H. C. LEHMAN, J. Abnorm. Psychol. **24**:486 (Jan.-March) 1930.

Judging from the anthology collected by the authors, modern students of the subject seemed to have returned to the old view that genius and nervous instability are inevitably associated. To be sure, Terman does raise a voice in protest when he says that there is no eccentricity in genius and that our intellectual leaders are recruited from gifted children. Except for this one quotation, all of the other comments in the symposium presented by Witty and Lehman are by men who would find neuropathic traits in all geniuses. William James and Francis Galton are given as examples of psychologists who abandoned the theory of the noneccentric genius in favor of a belief in associating intellectual superiority with intellectual abnormality. Nisbet, Lombroso, Nordau, Murchison, Freud and Havelock Ellis are all cited as believers in the close association between genius and madness. The authors' quotations are substantiated by exact references to the authorities cited.

DAVIDSON, Newark, N. J.

ALLEVIATION OF PAIN IN PERITONSILLAR ABSCESS. REPORT OF A METHOD INVOLVING COCAINIZATION OF THE PALATINE NERVES PASSING THROUGH THE SPHENOPALATINE GANGLION. M. REESE GUTTMAN, Arch. Otolaryng. **11**:426 (April) 1930.

After pointing out the distribution of the sphenopalatine ganglion, it is noted that the area involved in quinsy is supplied by the palatine branches of the fifth nerve. Guttman advises blocking the sphenopalatine ganglion in the nose. It may be reached at the posterior palatine foramina, but this is not advisable as it is in the inflammatory area. He quotes cases in which this method was of great

benefit in incising the peritonsillar abscess in a practically painless manner. The author notes that Hoople of Boston has also described this method.

HUNTER, Philadelphia.

CONGENITAL FACIAL DIPLEGIA. R. M. STEWART, J. Neurol. & Psychopath. **10**:317 (April) 1930.

Stewart discusses the literature of congenital facial diplegia and gives a short account of the symptomatology and concurrent defects. The pathology is discussed, but no attempt is made to explain the etiology. This description is transcribed from Heubner's report, and both the author and Heubner believe that this condition results from some slowly progressing disappearance of the nerve cells. A case is presented in which there were several other defects and a mental retardation that would place the case in the imbecile group. The claim is made that this is the only case on record of facial diplegia with mental retardation.

ROBINSON, Kansas City, Mo.

PRESSURE AS A FACTOR IN THE DEVELOPMENT OF NEURITIS OF THE ULNAR AND COMMON PERONEAL NERVES IN BEDRIDDEN PATIENTS. HENRY W. WOLTMAN, Am. J. M. Sc. **179**:528 (April) 1930.

Nine cases of neuritis with pressure as a definite factor are cited; five of these were illustrative of disturbance of the ulnar nerves, four of the peroneal nerves. In about 50 per cent of the cases, infection could not be demonstrated. Sufficient importance has not been given to the compression of these nerves between the bone and the underlying surface and, in the patients with peroneal palsy, additional traction on the nerve and on the muscles supplied by this nerve. Proper attention to bedding, position and support of the limb and massage will aid greatly in the prevention of the neuritis.

MICHAELS, Detroit.

THE RORSCHACH TEST APPLIED TO DIFFERENT GROUPS OF CHILDREN BETWEEN 10 AND 13 YEARS OF AGE. MARGUERITE LOOSLI-USTERI, Arch. de psychol. **22**:51 (Sept.) 1929.

The author applied the Rorschach tests to three groups of children, a group from Zurich, one from Geneva and one from a foundling school. She found that the Zurich children (German extraction) were more infantile in their responses than the Genevese children (French extraction). Pupils in the foundling school were as a rule more infantile in their responses than children living in their own homes. If they showed neuropathic signs, however, they did not show this infantilism, but were more obstinate.

PEARSON, Philadelphia.

AN INSOLUBLE BISMUTH PREPARATION (MESUROL) IN THE TREATMENT OF SYPHILIS. S. GENOVESE and F. VERO, Am. J. Syph. **14**:94 (Jan.) 1930.

The authors used the monomethyl ester of bismuth dioxybenzoic acid; this is commonly called mesurol. It is supposed to be relatively painless and practically free from toxic effect. One cubic centimeter is given intramuscularly twice a week for two months. Neurologists will be interested in their report that very decided clinical improvement was noted in cases of neurosyphilis. Exact figures were not quoted.

THE EFFECT OF HYPNOSIS ON BASAL METABOLISM. JACOB GOLDWYN, Arch. Int. Med. **45**:109 (Jan.) 1930.

Using hypnotism to produce relaxation, Goldwyn observed that an average reduction in the basal metabolism of 4 per cent occurred. There was no case in which the reduction exceeded 9 per cent. All of Goldwyn's subjects (with two exceptions) had a minus basal metabolism to begin with. The author does not describe the method by which hypnosis was induced.

DAVIDSON, Newark, N. J.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

April 17, 1930

E. W. Taylor, M.D., *President,* in the Chair

Operations on the Sympathetic for the Relief of Thoracic Pain. Dr. James C. White.

This report is in the nature of a preliminary communication describing results in six cases of intractable thoracic pain and two cases of atypical neuralgia of the head. The point of attack in each has been the upper dorsal sympathetic ganglia or the communicant rami of these ganglia.

I feel that certain types of painful stimuli from the head, arm and thorax traverse the sympathetic nerves and reach the upper dorsal sympathetic ganglia over two routes: (1) by fibers descending the cervical sympathetic trunk, and (2) by gray rami running from the great vessels in the neck, the pulmonary, cardiac and brachial plexuses directly to the upper five or six dorsal sympathetic ganglia. With a cervical incision, it is impossible to remove the important sensory tracts that joint the sympathetic chain below the stellate ganglion, and as a result, failures from cervical sympathetic trunk resection have been frequent. The anatomic and physiologic proofs of these recently discovered lower connections offered by Jonnesco and Enarchesco, Braeucker, Kuntz, Cannon, Lewis and Britton, and Singer and Spiegel are convincing. I have, therefore, used the posterior approach devised by Henry in all of these operations.

Neuralgias of the Head

Case 1.—G. K., aged 39, single, complained of pain in the face, the side of the head, and the neck for seven years. The pain was burning steady and markedly increased by rubbing the face, by cold or by taking food. She had had an injection of alcohol, a posterior root section of the trigeminal nerve, section of the glossopharyngeal nerve and periarterial sympathectomy of the common carotid without relief. There were no abnormal physical or laboratory observations. Injection of procaine hydrochloride into the stellate ganglion gave temporary relief. The lower portion of the stellate and the second dorsal ganglion were removed with relief of pain in the side and back of the head, but without effect on the pain in the face. A second operation was performed, but on account of scar tissue it was impossible to find and remove the inferior cervical portion of the stellate ganglion.

Case 2.—M. M., a married woman, aged 48, complained of severe burning pain in the left side of the face, the head and neck. The pain was steady but came in attacks lasting several hours; it was brought on by taking food, by cold, or by rubbing the face. The patient had had a posterior root section ten years before, with temporary relief. Facial paralysis occurred after this operation. Injection of procaine hydrochloride into the stellate ganglion gave complete relief for twenty-four hours. The whole stellate ganglion and the second dorsal ganglion were removed with complete relief from pain in the face, head and neck. This relief has persisted up to the present time (two months).

Since operating in the first case, I find that Dr. P. G. Flothow of Seattle has performed this same operation in two similar cases with good results. I have heard recently that Dr. Frazier and Dr. Grant of Philadelphia have also had

two successful cases. I believe that removal of these ganglia may be indicated in severe pain of the face and head when injection of procaine hydrochloride into the cervicodorsal ganglion gives relief and injection of alcohol or posterior root section of the trigeminal nerve is not successful. This operation is as yet in the experimental stage when applied for painful conditions of the head and face.

CARDIO-AORTIC PAIN

CASE 3.—J. B., aged 48, a thick-set man, had an aneurysm of the ascending arch of the aorta, of syphilitic origin, pushing up between the clavicle and first rib. This caused severe pain on the right side from the top of the head down over the posterior part of the scalp and neck and the upper four thoracic segments. Diagnostic injection of procaine hydrochloride showed that the pain could be relieved by blocking the upper two dorsal rami on the right side. Paravertebral injection of alcohol into these two segments gave complete relief until death, three months later, from rupture of the aneurysm.

CASE 4.—G. G., aged 21, an Italian, had severe angina pectoris with a rheumatic destruction of the aortic valve. Severe paroxysms of pain, mostly in the left precordium, came on from four to six times daily. Procaine hydrochloride injected into the region of the stellate and second dorsal ganglia stopped the attacks for twenty-four hours. Resection of the same ganglia gave permanent relief from pain on the left side. Circumstances forced a return to work, and the patient did very well, except for occasional mild attacks of pain on the right side following overexertion. Nine months later, he died of a severe attack of angina on the right side.

CASE 5.—C. A., aged 29, with angina pectoris and syphilitic heart disease, had a resection of the upper three left dorsal ganglia to relieve severe crises of pain. He made an excellent recovery from the operation and was transferred to the medical service for antisyphilitic treatment. On the fourteenth day after the operation, intense pain in the right precordium developed, and he died within a few hours. Autopsy revealed a total occlusion of the orifice of the left coronary artery and 50 per cent occlusion of the right.

CASE 6.—A. C., aged 60, with angina pectoris and arteriosclerotic heart disease, had a resection of the left upper dorsal ganglia. The patient, a woman, appeared to be an unusually good surgical risk. She made an excellent operative recovery and at the end of seven months had had no attacks or the left side. She has mild angina on the right side, as before the operation; this serves as a good danger signal when she is tempted to overexert herself.

CASE 7.—This patient, with arteriosclerotic heart disease, had severe angina on the left side, referred over a wide area, which came on after a mild attack of coronary thrombosis sixteen months before admission. He presented an unusual social problem which made it absolutely necessary for him to work. Under ordinary circumstances I would have treated him by injection of alcohol, but as he stated that he would rather be dead than unable to work, I elected the more radical method. Convalescence was marred by a prolonged attack of pneumonia caused by ether. However, he left the hospital at the end of three weeks apparently in a fair condition. One week later, he reentered, and died of an attack of painless coronary thrombosis. Autopsy revealed a small encapsulated area of empyema and old and new areas of myocardial infarction. The coronary arterial system was nearly entirely obliterated.

Comment.—Two of four of these patients died within a month of the operation. It is hard to form an impartial judgment as to whether death was due to an unfortunate coincidence or directly to the strain of radical operation. Certainly the picture presented at autopsy made it certain that life could not have lasted long in any event. I report these results at this time to bring out the interesting point that all of the patients were relieved from cardiac pain in the operated segments; even in the fatal attacks pain was totally absent.

In deciding how to handle future cases of angina pectoris, I can only recommend operation in preference to injection of alcohol under exceptional circumstances. If medical treatment can keep these patients comfortable, nothing else should be considered. If medical treatment is not satisfactory, the decision must be made whether injection of alcohol or operation should be used. Injection of alcohol carries practically no risk, and good results have followed in 75 per cent of the cases. In every case in which alcohol has been accurately placed, the results have been satisfactory. But as 25 per cent of cases are likely to fail on account of the technical difficulty of accurate injection, I believe that the operation may be recommended for those patients who have a fairly long expectation of life and in whom the ability to work is a vital necessity.

Case 8.—H. L., aged 48, had suffered from severe tabetic crises in the legs, the left side of the abdomen and the thorax for five years. Every recognized form of medical treatment had been tried and had failed. In 1926, section of the sixth to the eleventh posterior roots on the left had failed to relieve the gastric crises. Two successive attempts at cordotomy, in 1927, also failed to control the abdominal attacks, although they relieved the lightning pains in the left leg. This left no known innervation to the painful segments except possible sympathetic pathways traversing anterior roots or running to higher levels before entering the cord. Paravertebral injection of procaine hydrochloride from the seventh to the eleventh dorsal segments relieved the pain for twenty-four hours. A resection of the left splanchnic nerve with the seventh to the ninth thoracic ganglia has given complete relief from the gastric crises for seven months. The deep pain in the left lower part of the chest was likewise controlled by injection of procaine hydrochloride into the third to sixth communicant rami. These were divided four months ago by the technic described by von Gaza.

Von Gaza's operation has been used by Leriche and by Verbrugge and van Bogaert in cases with gastric crises, but I believe that this is the first time that it has been used for the relief of thoracic crises and also the first time that resection of the lower dorsal sympathetic chain has been combined with intrathoracic resection of the great splanchnic nerve for gastric crises.

Conclusions.—It is impossible to draw any definite and striking conclusions from such a small group of cases. Nevertheless, I feel justified in saying that these clinical observations confirm the experimental work of a number of observers, and that certain painful affections of the head and thorax may be relieved by resection of the upper dorsal sympathetic ganglia or by section or alcohol block of their communicant rami.

Herpes Oticus. Dr. C. A. McDonald and Dr. E. W. Taylor.

This article will be published in full in a later issue of the Archives.

Lesions of the Brain in Electric Shock. Dr. Raymond Morrison, Arthur Weeks, Esq., and Dr. Stanley Cobb.

Sublethal electric shocks were administered, by occipital and nasal contact, to laboratory animals, and the brains were subsequently removed and studied histologically with the aim of ascertaining whether electricity produced lesions, and if so, the nature of the lesions in the nervous system. Four types of current were used and were particularly selected because of their wave shapes, so that the factors of direction of flow, polarity, impact, heat value and frequency could be considered. It was found that lesions were produced in the brain, sometimes even after a single shock, and the nature of the lesions depended, at least to some extent, on the nature of the current, similar wave forms producing more or less similar lesions.

There were some overlapping types of lesions, such as hemorrhage, which could be found in all cases, and shrinkage of ganglion cells that could be found in three

classes of cases, but in general, the various combinations of lesions were characteristic of the effects of different types of current.

Nearly all of the structures in the brain were involved in these pathologic processes. The ganglion cells were swollen under some circumstances and shrunken under others. The myelin was affected chiefly in cases in which the glia proliferated and the nerve cells were swollen. In practically all cases there was congestion and hemorrhage. Edema of the brain was a common observation and, in conjunction with the swelling of oligodendroglia and the production of mucocytic or galactolipid degeneration, presented interesting vacuolar lesions. In an attempt to explain the mechanism of the production of these lesions, the heat generated in the brain under most vigorous electric strain was measured, and it was found to be relatively insignificant as long as the blood circulation was maintained, but it increased rapidly when the circulation was stopped.

Furthermore, by means of the special technic devised by Forbes, the vessels on the surface of the brain were observed during electric shock, and it was found that the vessels constricted or dilated during the passage of current, depending on the kind of current used. The condenser discharge and induction coil currents caused the vessels to dilate and the intracranial pressure to rise; histologically, one of the most characteristic lesions from this type of current was swelling, liquefaction and vacuolization of the ganglion cells. The alternating current, on the other hand, brought about a constriction of the pial vessels, a slowing of the blood and, histologically, a pronounced shrinkage of the ganglion cells. It may be, of course, that these lesions were caused by the direct action of electricity on the nerve cells and glia, but in view of the fact that at least some of them could have been caused by these forms of vascular response, attention is directed to the coincidence of vascular response with type of change in the tissue.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, April 17, 1930

LOYAL DAVIS, M.D., *President, Presiding*

PARALYSIS OF THE UPPER EXTREMITIES DUE TO INJURY OF THE ANTERIOR HORNS. DR. VICTOR E. GONDA (by invitation).

History.—This patient, aged 50, was injured on Oct. 12, 1929, when a heavy iron plate fell on the lower part of his neck. He was unconscious for only a short time. A brace was fitted to the neck and a weight applied; in this position the patient remained in bed about six weeks. For the first two weeks he was unable to use his arms. When he left his bed, he could walk with the help of one crutch. At no time were there any bladder or rectal disturbances. Six weeks before presentation, when I first examined the patient, the chief complaints were weakness of the hands, especially the right, and inability to walk a few blocks without becoming exhausted.

Examination.—There was no involvement of the cranial nerves. The Horner syndrome was not present. There was a continual spontaneous nystagmus, but this had been present since childhood and had never disturbed the patient in working. The gait was slightly spastic. The tendon reflexes of the lower extremities were lively, but no Babinski sign nor clonus was elicited. The volume of the intrinsic muscles of both hands, especially of the right hypothenar eminence, was greatly diminished. He was unable to adduct the fingers of the right hand and could not oppose the right thumb. The right triceps muscle was extremely wasted. The left triceps had also lost much of its tone, and its strength was minimal. The flexors of the forearms, the supraspinatus and infraspinatus muscles and the deltoids of both sides were flaccid. All the flabby and seemingly atrophied

muscles, with the exception of the right hypothenar group and the right interossei and lumbricales, reacted to faradic stimuli. There was a definite fibrillary twitching of the right triceps, deltoid and brachioradialis muscles.

The skin over the fingers was glossy. The finger-nails were disfigured and showed some cracking. There were no sensibility disturbances except a questionable diminished sensation to touch and pain over the right little finger, but — and this is important — none of the ulnar side of the ring finger. An old scar on the right palm could have accounted for the sensibility disturbance of the right little finger.

The right lower abdominal reflex was sometimes elicitable. The other abdominal reflexes were definitely absent, and the cremasteric reflexes present. The biceps, triceps, supinator and radial periosteal reflexes were all elicitable.

A roentgen examination showed fractures of the spinous processes of the sixth and seventh cervical vertebrae with downward displacement of these spines. Involvement of the lamina of the first thoracic vertebra was considered, but there was no evidence of fracture of the vertebral bodies proper.

Comment.—The distribution of the atrophy, the absence of sensibility disturbances, the fibrillary wormlike tremors in the wasting muscles, with electrical reactions which were a mixture of the reaction of degeneration and the normal, and the absence of abdominal reflexes, with some spasticity of the lower extremities, led me to the conclusion that pathologic changes in this case were in the anterior horn cells of the lower cervical and upper dorsal segments, with some slight involvement of the pyramidal tracts. The observations indicated a destruction of anterior horn cells, which, however, was not complete, so that some of these cells with their corresponding muscle fibers were normal.

The anterior horn cells had apparently been damaged by concussion and had been pulled and separated from their axons by traction of practically all those anterior roots which unite later in forming the upper, middle and lower brachial plexuses.

DISCUSSION

DR. PETER BASSOE: I showed this patient in my clinic through the courtesy of Dr. Gonda. It is difficult to decide whether the injury was entirely to the cord or to the cervical nerves as they pass through the foramina.

DR. ROY GRINKER: Does Dr. Gonda believe that the fibrillations could be due to initial damage of the anterior horn cell, or that they signify a continuation of a pathologic process?

DR. VICTOR E. GONDA: It is difficult to explain the continuation of the fibrillary twitching. The condition has been getting worse during the last few weeks, and I think that it is progressive. The nystagmus was present at birth, and has no relation to the injury.

CYCLIC OR RHYTHMIC OCULOMOTOR PARALYSIS. ELIAS SELINGER (by invitation).

History.—J. O., a well developed white boy, aged 16, came to the eye department of Rush Medical College on Dec. 12, 1929, complaining of right-sided ptosis, inability to see with the right eye and right divergence, the symptoms having been present since birth. The patient has one younger and two older brothers, all apparently well. The mother has been an inmate of a hospital for mental diseases since the birth of the younger brother, twelve years ago.

Examination.—General physical examination revealed a 4 plus Wassermann reaction of the spinal fluid, negative Wassermann reaction of the blood, and Hutchinson's teeth.

Neurologic examination gave negative results, except for the eye condition to be described. Psychiatric examination showed an intelligence quotient of 71. Antisyphilitic treatment had been given at another institution and was now instituted again. The patient stated he had had two or three operations in childhood for correction of the squint and ptosis.

Ocular Examination.—Vision in the right eye is 3/200, fixation being eccentric. The left eye is normal as far as visual acuity and other conditions are concerned. The changes in the right eye are those of an incomplete paralysis of all branches of the oculomotor nerve and are best described in two stages. First, or flaccid stage: The upper lid droops so that the palpebral fissure is narrowed to 3 mm., the eye deviates outward, the squint angle being from 40 to 45 degrees as measured with a perimeter, and the pupil is dilated to 8 mm. This stage lasts about twenty seconds and is followed by a second or spastic stage, in which the outer one half of the right eyebrow moves up, causing a wrinkling of the skin on that side of the forehead; the upper lid elevates in a jerky manner until the palpebral fissure is from 8 to 9 mm. wide, and a spasm of the internal rectus moves the eyeball nasalward, diminishing the squint angle from 5 to 8 degrees; at the same time the pupil rapidly contracts down to 3 mm. This stage lasts from fifteen to twenty seconds, and is followed again by the flaccid stage. The process of dilatation takes from twenty to twenty-five seconds; contraction, twenty seconds. The entire cycle requires from sixty to eighty seconds, and is constantly repeated. The spastic phenomena are executed more rapidly than the flaccid ones. With the pupillary changes there is an alternating spasm of the ciliary muscle, as evidenced by a 3.5 diopter change in the retinoscopic observations between the contraction and dilatation stage of the pupil.

Light, effort at convergence and accommodation and effort at elevation and depression of the gaze have no effect during the contraction stage, and cause only a momentary twitching of the iris and eyelid during relaxation. A pseudo von Graefe sign is present in the spastic stage — when the patient is asked to look down, the right upper lid remains elevated. Voluntary movements consist of good abduction and poor adduction. There is nasal rotation of the cornea on an effort to look down. The abducens and trochlear nerves are therefore not involved. The eyeball cannot be elevated. The caruncle and plica semilunaris are prominent and displaced forward, possibly owing to a previous operation.

Two per cent pilocarpine caused a contraction of the pupil to 2.5 mm., with no alteration in size, while the lid and adduction phenomena continued as before. When the pupil was contracted the patient complained of pain in the right eye, the pain being rhythmic and most severe during what would correspond to the spastic stage of the ciliary muscle cycle. Homatropine dilated the pupil to 8 mm. and abolished the pain, but had no influence on the lid and rectus medialis changes. The right pupil did not react consensually; the left did. Peripheral visual fields for form and color were normal, and there were no central scotomas. The fundus showed no pathology.

Of the twenty-nine published cases, fifteen were congenital, eight developed during the first year of life and six at a later age. The cyclic phase may not appear until months or years after there has been a definite oculomotor paralysis.

One of Rampoldi's cases was unusual in that both eyes were involved, one eye being in the flaccid stage, while the other was in the spastic stage, thus producing a constant seesaw movement.

Of the twenty-seven cases in which the sex was given, eighteen occurred in females. One oculomotor nerve is affected about as often as the other. In three cases there was also involvement of the fourth nerve, and in Lauber's case the fifth nerve on the same side and seventh and ninth on the opposite side were paretic. The pupil participated in all cases in the cyclic changes, the levator palpebrarum in twenty-one of twenty-nine cases, and the ciliary muscle in eleven cases of the sixteen examined for that symptom. Less frequent and less severe is the involvement of the extrinsic eye muscles. Of these, the internal rectus is the one most commonly affected in all, thirteen times. The spastic features have been observed to continue during sleep. In two cases there was ocular nystagmus, in one of these bilateral; in both the good eye was affected. Amblyopia is a noteworthy symptom. Of eighteen cases in which a functional examination was reported, fourteen showed much poorer vision in the affected eye than in the other. The impairment of vision is probably on the basis of an amblyopia

exanopsia, although vision in the affected eye remained good, even in some of the congenital cases.

As no case has come to autopsy, the nature and location of the lesion is based on theory rather than on pathologic observations. E. Fuchs and others thought that there is a nuclear lesion, while Mosso believed there are not only nutritional disturbances in the nuclear region, but in addition, disturbances in the sympathetic pathways accounting for the rhythmic spastic phenomena. Axenfeld and Schürenberg pointed out that since accommodation innervation is always bilateral and in these cases it is purely unilateral, a congenital anomaly of the connecting fibers of the nuclei of both sides of the brain must be assumed before the site of the lesion can be considered nuclear. Other writers also have called attention to the fact that fibers from the posterior end of each oculomotor nucleus cross over to the other side and that, therefore, a nuclear lesion on one side should, in addition to paralysis of the muscles supplied by the third nerve on one side, show some impairment of the muscles of the other eye. An infranuclear lesion central to the point of emergence of the trunk of the oculomotor nerve from the brain is thought to explain the purely unilateral involvement better than the other theory.

Bielschowsky, in an effort to account for all the phenomena, assumed that there is an anomalous blood supply to the oculomotor nucleus in these cases; he further doubted that fibers from one third nucleus cross over to the opposite side. He believed there is a destructive lesion in the nucleus of the third nerve with sparing of the ganglion cells concerned with the innervation of the muscles taking part in the cyclic changes. The lack of response to volition and physiologic stimuli, he explained on the basis of a supranuclear pathologic process in the vicinity of the nucleus. He thought that in some cases there may be an involvement of the nerve trunk. He accounted for the cyclic spastic features by vasomotor stimulation resulting in increased blood supply to the regions mentioned.

C. Behr stated that the ganglion cells belonging to the automatic moving muscles are grouped closely together in the anterior portion of the oculomotor nucleus, while the cells belonging to the permanently paralyzed muscles are located in the middle posterior portions of the main nucleus. Bernheimer proved that each one of these divisions has its own blood supply — the anterior pole through branches of the posterior communicating artery from the carotid, the posterior pole through branches of the posterior cerebral artery which comes from the basilar artery. Since, with few exceptions, one deals with congenital cases, one may assume, in the presence of the remaining irritability of the anterior nuclei, that there is a congenital aplasia of the corticonuclear paths and reflex contact neurons. By a difference in the blood supply, the cells of the anterior division of the nucleus are spared from complete degeneration while the posterior ones degenerate. The anterior group cannot, because of the abundance of afferent stimuli from aplasia of the contact neurons, be stimulated voluntarily or reflexly. The sparing of part of the nucleus and the centripetal paths offers a possibility that through the normal blood supply there is a cumulative energy storage in the ganglion cells, which discharges itself rhythmically into the muscular periphery; these movements are similar to the athetoid movements in spastic paralysis of the extremities.

DISCUSSION

DR. R. P. MACKAY: I was much interested in the possible relationship between cases similar to this one and the condition known as ophthalmoplegic migraine. There has been reported at least one case of ophthalmoplegic migraine in which the patient exhibited spasmodic contraction of the ocular muscles and also a change in the pupils. This condition was present only during migraine attacks in the patient whose case was reported by Féré (Note sur quelques signes physiques de la migraine et en particulier sur un cas de migraine ophthalmospasmodique, Rev. de méd. **17**:954, 1897). Does this patient have headaches with the paroxysms described, and if so, is there any relationship between the spasmodic condition and the headache?

DR. HARRY L. PARKER: This is a unique presentation. The only analogy that I can see in other diseases of the nervous system is that which occasionally follows lesions of the seventh cranial nerve. I have seen a few cases of paralysis of the facial muscles in which, as the paralysis cleared up, spasmodic twitching movements took its place. The end-result was a spasmodic tic of the facial muscles, far more disagreeable than the original paralysis, which, as far as I remember, in most instances was very slight. Another analogy is represented in an automatic character of these spasmodic movements completely unlike those of voluntary effort.

DR. ELIAS SELINGER: I should have mentioned in this case that the spasms occurred after the patient's first course of antisyphilitic therapy.

So far as the relation to headache is concerned, there was none. Some cases are thought to bear a definite relation to athetoid movements, although it has been pointed out that in this condition voluntary movements are absent, while in athetosis voluntary movements are reestablished before the athetoid movements make their appearance.

OPHTHALMOPLEGIA INTERNUCLEARIS AND OTHER SUPRANUCLEAR PARALYSIS OF THE EYE MOVEMENTS. WILLIAM M. MONCREIFF (by invitation).

This paper will appear in a later issue of the ARCHIVES.

DISCUSSION

DR. JOHN FAVILL: I have been interested in the problem of paralysis of upward gaze, having seen several cases, and I hoped to hear the problem solved tonight, but I see that it is still uncertain. Perhaps I may be pardoned for repeating something I reported before the Society two years ago, but did not have published.

A patient with hemiparesis, in the Cook County Hospital, presented this condition. It occurred to me that it would be a final settlement of whether this was due to a nuclear or supranuclear lesion if one could produce upward movement by that rotation which would normally give vertical nystagmus with the slow movement upward. This was tried, and the upward movement promptly occurred, demonstrating the integrity of the nuclei.

DR. ALBERT KNAPP: Dr. Moncreiff referred to tracts running down from centers upward. This cannot be solved without considering the location of the centers for the conjugate movements in the cortex. Three men, one Frenchman, one Englishman and one Russian, investigated and found the center for the movement. It is in the frontal lobe. The Frenchman investigated it by experimental methods, and there was an interesting result. While clinical investigation pointed to the base of the second lobe just near the anterior central convolution, the experimental investigation located it a little higher, at the base of the first frontal lobe. I am sure that in some years it will be possible to delineate the whole center definitely. But there are not only centers in the frontal lobe; there are places elsewhere in the brain from which the conjugate eye movements are directed. Wernicke believed that he had found such a center in the lower half of the second lobe; others found that there were centers in the occipital lobe; still others, among them myself, found areas in the temporal lobe from which eye movements are influenced. I have the conviction, however, that the latter three places do not represent a center of equal rank with the center in the frontal lobe. Maybe I can explain easily what I mean. You cannot read without moving the eyes, in most instances following the lines from left to right, but if you follow Chinese or Japanese writing, you must move the eyes upward and downward. There must be fibers connecting the centers occupied in reading, and there must be some fibers going down from the oculomotor center in the frontal lobe to the occipital, parietal, temporal and central lobes, and to the nuclei of the ocular nerves. It is necessary to find whether the fibers have been interrupted, and if so, where, making a picture such as that demonstrated.

MENTAL DEPRESSION. JAMES C. HASSALL.

It is difficult, if not impossible, to obtain definite figures concerning the frequency of depression. The present classification of psychoses and neuroses does not always show the mental picture predominating. In a survey of the records in over 1,000 cases, the amount of depression was found to range yearly from 22 to 30 per cent. This is a little higher than is shown by statistics for several state hospitals; these range about 23 per cent. Depression may occur at any age. It varies in duration from a few hours to several years. It is most commonly found under the headings "manic-depressive" and "involution melancholia," but it may occur in dementia praecox, epilepsy, arteriosclerosis, alcoholism, drug addiction, tabes, dementia paralytica or with somatic diseases.

The symptomatology of depression is well known. The onset may be characterized by physical complaints, referable chiefly to the intestinal tract. Fatigue, feelings of weakness, heaviness, dull pain and pressure in the head, cardiac oppression, palpitation and paresthesias may also be present. Sleep is usually defective, with terrifying dreams, and is followed by a feeling of exhaustion in the morning.

The physical distress is usually present until the onset of more definite mental symptoms; then the mental pain usually dominates and physical distress is absent or thrust into the background. It is not uncommon, however, for physical complaints to be present throughout the depression. These disturbances may form the basis for various delusions. As the psychosis progresses, the patient is overcome by profound sadness and unhappiness. He voices many ideas of self-depreciation, reproach and accusation. Initiative and application are lacking. He endeavors to seclude himself from others. There is difficulty of concentration and of recollection, loss of interest and pleasure, a sense of strangeness and of the world's unreality and a hatred of effort and responsibility. At the beginning or end of the attack all of the symptoms may be accompanied with good insight.

In young patients, even when the depression is severe, it is surprising how often no delusions are present. If they are present, they are usually related to a foundation of fact — frail though it may be — and show chiefly a disordered sense of proportion.

Apprehension, hypochondriasis and nihilism are characteristic symptoms of involutional and senile depressions. These symptoms have frequently been observed in manic-depressive patients in whose earlier attacks they were absent. Thoughts of death and nihilism are not to be expected in young patients, because youth does not tend to such ideas. First attacks of depression in later life may be of the manic-depressive type, the patient having been able to weather the storms of life for a longer period than others. Such attacks cannot be expected to manifest themselves as they would in early life, but as they should at the age of the patient.

In the arteriosclerotic period, depression may be accompanied with perplexity and confusion, which is seldom seen in younger patients, and which may be due to some specific interference with intellectual processes. Often it is evident that one is not dealing with a true confusion so much as with a subjective sense of confusion.

In adolescence and early life, it is difficult to differentiate between pure depression and dementia praecox. The depression ushering in an attack of dementia praecox may be mistaken for that of a manic-depressive psychosis. If there are delusions, those of dementia praecox are likely to be grotesque. In catatonia, especially if stupor is present, the diagnosis is difficult; in fact, many patients presenting in the early stage what seems to be a pure depressive syndrome sometimes end with typical schizophrenia. The somatic symptoms, too, of the depressed schizophrenic patients may simulate those of agitated depression.

The neurasthenic as well as the depressed patient finds thinking difficult; his faculty of attention and his memory are defective; efforts to concentrate cause exhaustion and physical exertion causes overfatigue. The neurasthenic is particularly anxious about his physical symptoms, which are extremely disagreeable.

Morbid anxiousness is the symptom most constantly present in anxiety neurosis. In an acute attack, dread may be very intense, and may be accompanied with peculiar sensations in the head and fear of impending mental disease or death. There is usually a sense of embarrassment, a great increase in the heart rate, pain, palpitation, fluttering and irregularity. The pupils are often dilated, and there may be tremor and sweating. In the chronic anxiety state, apprehensive expectation becomes attached to any idea that can justify anxiety. Many physical symptoms are present that are absent in the usual depression. Two cardinal characteristics of anxiety neurosis should be kept in mind: (1) the great tendency for one or another system of organs to dominate the clinical picture, and (2) the subordination of mental manifestations to physical ones.

The prognosis of depression is related to its etiology. Psychopathic heredity increases the probability that the attack will not be an isolated one; if the family disorder is of the phasic type, there is a greater chance that recovery will follow a given attack. Symptoms suggesting schizoid tendencies make the prognosis more grave. Depressions precipitated by the puerperium offer a good outlook. About two thirds of those at the involution period recover, though the attacks are likely to be prolonged, particularly if they are first attacks. Schizoid or paranoid features in the presence of arteriosclerosis make the outlook unfavorable.

DISCUSSION

DR. GEORGE W. HALL: A few points were brought out in Dr. Hassall's paper which I think should be emphasized. One, as Dr. Paskind stated in a recent paper, is the fact that we are overlooking many cases because the attacks are so brief. One week may find the patient in rather a hypomanic condition and the next week, depressed; yet he goes about his work and is not recognized as having a true depression.

Another point I think should be emphasized is the differentiation between psychoneurotic and true depression. Dr. Hassall brought out one or two points in the differential diagnosis. In psychoneurotic depression the important thing is that the patient has definite fears of losing his mind, which are not so definitely expressed in true depression. Another point is that the attacks may occur at any age. Today I saw a boy, aged 15, who seemingly has a true depression; yet it is difficult to make a definite diagnosis from one observation. One may be misled into making a diagnosis of true depression when one is dealing with a case of dementia praecox.

DR. PETER BASSOE: When I look over my records I find in many cases that I put down a diagnosis of anxiety neurosis, and within a few months when I again see the same patient, I have to scratch that diagnosis and write "depressive stage of manic-depressive psychosis." I should like to ask Dr. Hassall whether there is an essential difference, or whether it is a difference in degree rather than in kind.

DR. HUGH T. PATRICK: I think the disorders are absolutely different. Psychoneurosis is one thing and primary depression is another, something *sui generis*. The cause is not known. I know that sometimes it is difficult to tell, especially in the beginning, whether the trouble is primary depression, a psychoneurosis with secondary depression or dementia praecox; but I believe that there is an essential, fundamental difference, not simply one of degree.

DR. JAMES C. HASSALL: In making a diagnosis of an anxiety state, I usually depend more on the persistence of physical symptoms than on depression, such as the fear that something will happen — the heart will stop, or something of that sort — which is not seen in the ordinary simple depression.

METASTATIC ABSCESS OF THE BRAIN: A CLINICAL STUDY. HARRY L. PARKER, Rochester, Minn.

Twenty cases of metastatic abscess of the brain were studied clinically. The diagnosis had been established either by necropsy or by surgical exploration. For convenience the cases were classified in three groups: (1) three cases in which the

abscesses were secondary to an overwhelming or progressive general septic process; (2) fourteen cases secondary to pulmonary or pleural suppuration, and (3) three cases secondary to an original septic process that had apparently healed, but the signs of cerebral suppuration had appeared later and progressed to menace the life of the patient.

The prognosis depends entirely on the severity and progress of the original infection. In group 1, the condition was hopeless. In group 2, it was grave; but in some of the cases it might have been modified by operation. In group 3, surgical treatment at a favorable time was possible and the outcome favorable.

The course of these abscesses was much shorter than that of abscesses due to other causes. This no doubt depended largely on the debility of the patient. The clinical signs and symptoms, however, differed little. The severity of the original infection added a complicating feature, often making the diagnosis extremely difficult.

In ten of the twenty cases a single abscess was situated in one or the other lobe of the cerebrum. In many of these a certain degree of encapsulation was present, but rupture into the ventricle, nevertheless, occurred readily. In the other ten cases the brain was the seat of multiple foci of suppuration.

DISCUSSION

Dr. Peter Bassoe: In connection with the case in which the abscess was removed on the supposition that it was a tumor, I am reminded of a case reported by Dr. Diamond and myself. Left hemiplegia and aphasia gradually developed in a woman, and there was a distinct thickening in the bone over the parietal region. This bone was removed, and immediately under the dura was a round, firm, globular tumor mass, which was removed, and the wound was closed. The specimen was taken to the laboratory, where a day or two later a technician accidentally dropped it, and pus ran out. Six weeks later the patient came to necropsy, and at that time it was found that a new abscess had formed inside the old cavity. Dr. Diamond then had an opportunity to examine the wall of an abscess, the exact age of which we knew.

Dr. Percival Bailey: I wish to report a rather amusing experience. Dr. Bassoe sent a patient to me, and I removed what I thought to be a meningeal tumor from the temporal region. It was attached to the petrous bone. The specimen was taken to the laboratory, where I cut it, and was surprised to see pus roll out. I had remarked at the time of operation that I was surprised to see so little blood, but when I found what the tumor actually was, the explanation was simple. The wound was immediately opened and a drain inserted. It has been about a year since the operation, and the patient remains well.

Dr. Harry L. Parker: One thing I forgot to mention in the last group is that the original infection may be hard to place. In some cases there was difficulty in finding where the abscess originated, and that adds considerably to the whole diagnostic problem.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, April 25, 1930

Williams B. Cadwalader, M.D., *President, in the Chair*

Cerebrospinal Fluid Pressure During Alterations in Posture. Dr. William Duane, Dr. R. M. Lewis and Dr. I. S. Ravdin.

In a study of the variations of pressure of the cerebrospinal fluid during alterations in posture of the animal, it was found necessary to build a suitable tilting table and to devise a closed apparatus for measuring pressures in closed

systems that was accurate and did away with the common errors inherent in open manometers such as inertia and displacement. Such a closed apparatus was made, and the cerebrospinal pressures were recorded from a cisternal puncture and the venous pressures of the torcular herophili in dogs under sodium amytal anesthesia.

Starting with a dog lying horizontally on its abdomen, rotating it to its side and later to its back, with the cerebrospinal system always horizontal, it was found that the cerebrospinal fluid pressure and the torcular venous pressures increased as the animal was turned. The actual values found for the cerebrospinal fluid pressure in animals in each position corresponded fairly closely. Their values in each position also corresponded to the figures reported by other observers when the posture of the animals was known.

Some of Weed's experiments on the relation of head-up and head-down position to cerebrospinal fluid pressure were repeated. Greater shifts in pressure were observed than those reported by Weed. This may be partly explained by the fact that Weed placed his animals first on their sides, whereas, our animals were at first on their abdomens, so that the initial pressure readings in our series were lower than Weed's. Subatmospheric pressures were found to exist in the subarachnoid space of the dog as measured at the cisterna magna in the head-up position and the lumbar area in the head-down position.

DISCUSSION

Dr. I. S. Ravdin: There are a number of interesting points in connection with the measuring of cerebrospinal fluid pressure. In a space as small as the subarachnoid space, and with a fluid pressure as small as that normally present, the pressure should be measured, if possible, without the loss of any fluid. At high pressures, the loss of very small amounts, of the magnitude of 0.5 cc., has at times caused a drop of pressure of nearly 100 mm. as estimated with a straight manometer of 1 mm. bore. The method devised by Dr. Lewis has permitted the estimation of cerebrospinal fluid pressure in the cisterna magna without any loss of fluid, and with the maintenance of a closed space. The pressure can be read directly, or photographs of the shift in pressure can be made. The apparatus is based on a simple physical principle and should provide a method with an accuracy not heretofore available. The inertia of the column of water in the ordinary straight manometer is eliminated, so that the lower pressure, which occurs during inspiration, can be satisfactorily recorded.

Dr. W. J. Gardner: The dynamics of the cerebrospinal fluid is interesting. Several years ago I made an observation, which I then thought was original, namely, that the intracranial pressure in the erect position is negative. This conclusion was drawn from the fact that if a lumbar puncture is performed with the subject in the sitting posture, the meniscus in the water manometer does not attain a level even with the top of the patient's head. I subsequently found, however, that this observation was made by a German author shortly after the introduction of spinal puncture by Quincke. The respiratory oscillations of the spinal fluid are apparently of abdominal origin, while the pulse waves originate in the cranium. The proof of this statement is furnished by the fact that by spinal puncture in cases of complete spinal subarachnoid block, one can demonstrate pure respiratory waves below the level and pure pulse waves above.

The authors are to be congratulated on the perfection of a piece of apparatus that will without doubt throw light on many obscure points in cerebrospinal fluid dynamics.

Intracranial Dermoid Cyst. Dr. S. T. Kwan.

True intracranial dermoid is rare, judging from the fact that only thirty-two cases have been recorded in the literature up to the present time. Horrax gave the frequency as 0.4 per cent of all intracranial growths in 750 verified brain tumors from Cushing's clinic.

A case of dermoid of the posterior fossa can be added to the literature. This occurred in a young woman, aged 19, who entered the neurosurgical service of the University Hospital on Nov. 26, 1929, complaining of headache, blindness and difficulty in walking for eight months. The positive observations were: The patient was dull and apathetic; intelligence was fair, but memory was poor. Station was unsteady, and the Romberg sign was positive. Dysmetria was definitely present, and she would consistently past-point to the right in the vertical, and go under in the horizontal planes. Nystagmus was present on lateral gaze. Ophthalmoscopic examination showed tortuous arteries, overfilled veins and secondary optic atrophy with a choked disk of 7 diopters in the left eye and 6 in the right. The cerebrospinal fluid pressure measured 640 mm. of water in the horizontal posture. Routine laboratory examinations all gave negative results. Roentgen examination of the head showed a perforation of the occipital bone just below the external occipital protuberance to the left of the midline, in addition to evidence of the presence of a possible tumor of the brain in the posterior fossa obstructing the ventricular system.

The diagnosis was tumor of the cerebellum, probably in the midline. On the day before operation the patient developed a cerebellar crisis to which she succumbed.

At autopsy a large cystic tumor, 5 cm. in diameter, was found between the cerebellar hemispheres. The tumor had a pedicle which extended through the perforation in the occipital bone and came to fuse with the under surface of the scalp. The content of the tumor was largely a grayish, soft, putty-like mass in the midst of which a few hairs of auburn shade were embedded. Chemical analysis of the content of the cyst showed abundant cholesterol. Microscopically, the tumor was a dermoid. The pedicle was lined by stratified epithelium, and in the dermal layer many hair follicles and groups of sebaceous glands were found.

This case is of interest, not only because the lesion is rare, but because of the significance of a shadow of a perforation in the roentgenograms which might lead one to suspect a dermoid.

CHRONIC SUBDURAL HEMORRHAGE. DR. FRANCIS C. GRANT.

To establish direct connection between a head injury and its immediate consequences is easy in most instances. Occasionally, however, the accident may have been so trivial, occurring weeks or even months previously, that to consider it as the underlying cause of the serious symptoms presented may not seem logical. Yet, it must be remembered, if proper diagnosis is to be made, that a chronic subdural hemorrhage can be produced by a relatively mild degree of trauma, and that it may lie dormant over the brain for a long time without any definite neurologic evidence of its presence. That chronic subdural hemorrhage is not an uncommon result of cranial trauma is evidenced by the fact that ten cases have passed through the neurosurgical clinic of the University Hospital in the past four years.

Time does not permit a detailed discussion of the pathologic processes involved in the production of these slowly forming subdural clots. According to Virchow, the first step is spontaneous chronic inflammation of the dura, evidenced by the exudation of a thin layer of film, often blood-stained, over its inner surface. With the scattered dural vessels as their source, numerous thin-walled capillaries burrow out into this film and vascularize it. Under the stress of increased intracranial pressure these newly formed vessels may rupture, with hemorrhage and clot formation, resulting in the condition Virchow described as pachymeningitis haemorrhagica. However, cranial trauma preceded the formation of the clot in all the cases here reported. In brief, I agree with Spesling, Huegenin and Wiglesworth, and with the more recent Putnam and Cushing, that in the post-traumatic type of subdural hemorrhage at least, the clot appears first, and the resulting pathologic processes are merely the result of an effort by the dura to organize the clot. Since there are blood vessels in the dura, but none in the arachnoid, the greater part of the organizing process occurs from the under surface

of the dura. Consequent on the reaction, a definite cyst wall forms on the under surface of the dura and is extended over that part of the clot lying against the arachnoid as a very thin web of fibrous tissue covered by a layer of mesothelial cells. Definite fine adhesions are formed between the clot and the underlying dura; but there is no attachment to the arachnoid unless it has been torn. The dura in the region involved is thickened and its under surface is covered with a reddish, velvety tissue, resembling granulations, on which delicate bleeding points appear when it is stripped back from the clot. The blood contained within the membrane in all the cases here reported was fluid, with clots adherent to the walls of the cyst. The physical characteristics of the confined blood may be greatly changed from the normal, depending on the length of time it has been present. In all my cases of over ten days' duration, the fluid was greenish brown and of a somewhat oily consistency. In one instance, the cyst contents gave a faintly positive test for bile salts.

With regard to the way the clot formed in the cases here reported, I agree with Trotter that following a mild trauma, a small vein running unprotected from the cortex to the dura is ruptured and hemorrhage into the subdural space follows. While the falx prohibits lateral change in the position of the cerebral hemispheres, a small amount of anteroposterior dislocation can follow a sharp trauma. Movements of a hemisphere in this direction carry it at right angles to the course of veins passing from the longitudinal sinus to the cortex or from the dura to the surface of the cerebrum in the frontal or occipital regions. In this connection, it is interesting to note in four of my cases that there were small frontal or occipital scars showing the point of application of the injury to the skull and its probable radiation in an anteroposterior direction. In addition, in this series one patient gave a definite history of a fall on the occiput; another had been struck on the forehead, and in a third case, in which calcification of the clot occurred, the hemorrhage followed a birth trauma, a condition in which it is generally accepted that the hemorrhage is due to rupture of a subdural vein. Consequently, in six of the ten cases it seems not improbable that tearing of a vein may have initiated the clot. The bleeding may then continue from the numerous large, thin-walled capillaries just described, which form from the dural vessels as organization of the clot goes forward. Hemorrhage from this source would be slow, accounting for the gradually progressive nature of the clinical symptoms with their long, latent period. I do not believe, in posttraumatic cases at least, that the dural reaction is primary and that the clot results in the beginning from these newly formed vessels beneath the dura. I have two definite reasons for this opinion. If the clot is due to bleeding from newly formed tissue beneath the dura, because of a chronic dural inflammation resulting from trauma, alcoholism, infection or other cause, why is the whole dura not involved instead of a single area over one hemisphere? Second, if the process in its inception is due to bleeding from vessels in a chronic inflammation of the dura, it is curious that the dura overlying the clot may be left in place following an operation without another hemorrhage resulting. In none of the six cases in which operation was performed in this series of ten was the dura over the clot excised, and in none of them did the condition recur. I lean strongly, therefore, to the theory that trauma is always the underlying cause of the hemorrhage. As a result of the injury a vein is ruptured and bleeds into the subdural space. The reaction seen on the under surface of the dura is caused by a natural effort to encapsulate and organize the clot. It is possible, however, that once this organization occurs, further hemorrhage into the clot may take place from the new blood vessels formed on the under surface of the dura. It is only in this way that the laminated clots, reported in several cases described in the literature, can be explained.

The clinical picture resulting from a chronic subdural hemorrhage is curiously uniform in development. If it is remembered that this condition can follow a mild injury of the head, and if the history in the case is obtained in a sufficiently painstaking manner to unearth this fact, a shrewd guess as to the true condition of affairs may readily be hazarded. It is the long interval between the injury

and the onset of serious symptoms that may cloud the issue. Disregarding for the moment three of my cases, in two of which the symptoms had been present for only seven and nine days, and in the third for fifteen years, the average duration of the condition from injury to operation was eight weeks. All the patients seemingly recovered completely from the apparently trivial head injury and were able to return to work. The first symptom suggesting the presence of an intracranial lesion in all my cases was headache, at first intermittent and then persistent, followed by a slight obtunding of the mentality. This change in personality may be vague at first, but later is apparent even to the patient himself. Another striking feature is the rapidity with which signs of severe intracranial pressure may develop. In spite of headache and slight mental lethargy, the patient may be able to be about his business, when suddenly he may have a convulsion, sink into stupor or give other evidence of a serious cerebral condition. It seems as though the brain can accommodate itself to a slowly developing lesion up to a certain point and no farther. When this point is reached, any slight increase in pressure due to exertion, excitement or other cause may usher in a startlingly abrupt collapse. The following case from my series is a good illustration.

Clinical History.—F. B. F., a lawyer, aged 43, was referred by Dr. Karl J. Kurz, of Mount Airy, Philadelphia. While bathing in the surf, four weeks before admission, he was knocked down and roughly handled by a heavy sea. On leaving the water he noticed a buzzing in both ears and was slightly dizzy. About an hour later while dressing, a severe bitemporal headache developed. During the next hour, vision became blurred, and finally complete blindness developed in the left eye, with marked visual loss in the right. He consulted a local physician, who put him to bed and purged him vigorously. The next morning he had recovered sight completely, and the headache had somewhat abated. He stayed in bed that day, but twenty-four hours later felt well enough to return home by train. A week after the onset he was back at work, although his head felt heavy, and he was less alert mentally than usual.

Four days before admission and three weeks after the injury, he left home on an important business trip involving much mental strain. Forty-eight hours later, while examining a witness, he suddenly was at a loss for the proper word, and within six hours became completely aphasic, answering only "yes" and "no." He was brought home at once, arriving in a semistuporous condition. Convulsions, beginning in the right side of the face and involving the right arm, next developed. Later he became deeply stuporous.

Examination.—The patient was in deep stupor, with frequent convulsive twitchings of the entire right side of the face and right arm and occasional involvement of the left side of the forehead. Breathing was noisy and stertorous; the face was somewhat livid. The pupils were normal in size, shape and reactions. The disk margins were blurred, the vessels, overfull and tortuous, but there was no measurable choking. The lower jaw and tongue tended to deviate to the right and the right seventh nerve showed definite weakness. All the extremities were spastic, especially the right arm and hand. All the reflexes were exaggerated, especially in the right arm. There was a bilateral Babinski sign. No sensory loss could be demonstrated. All extremities were moved on pin prick equally well. The pulse and respiration rates were slightly retarded. The temperature was normal.

Diagnosis.—An abscess or a hemorrhage into a left temporoparietal brain tumor was diagnosed. The trauma involved seemed of minor importance. At best, the condition seemed desperate.

Operation.—Since an abscess was suspected, a trephine opening in the left temporoparietal region was made under procaine hydrochloride. On inspection, the dura was deeply bluish green. The presence of a hemorrhage was then realized for the first time. With infiltration anesthesia, a left temporoparietal bone flap was turned back. The appearance of the dura was characteristic: tense, pulseless and greenish blue. On incision, a bloody, grumous fluid escaped. At this point, the patient had a violent right-sided convulsion, and respiration ceased. Under artificial respiration, breathing commenced again, and the dura was opened widely.

This membrane was much thickened, especially over the underlying clot. This clot covered the entire hemisphere from the frontal to the occipital pole, and extended over into the median fissure and beneath the brain in the frontal, middle and posterior fossae. It was rather firmly adherent to the dura, from which it could be peeled. As it came away, fine fibrous trabeculations attaching it to the dura were noted. It was not at all attached to the arachnoid.

In its thickest part, directly over the temporoparietal region, the clot was 4 cm. thick, tapering off to 0.5 cm. in the frontal and occipital regions. All the accessible clot was removed by suction, but parts of it could not be reached at the base and in the median fissure. The brain was so compressed that a large clotted mass was easily removed, with gentle retraction of the frontal lobe, from about the left optic nerve and chiasm. The cortex was flattened, anemic and pale yellow. A section of the thickened dura was removed for examination. Sufficient clot was withdrawn piecemeal to fill a 6 ounce (178 cc.) glass, besides that which disappeared into the suction tube. No definite bleeding point could be discovered, but the inner surface of the thickened dura was definitely roughened and oozed freely. The brain was rapidly regaining its normal contour when, after careful hemostasis, the dura was closed. The bone flap was replaced and the galea and skin closed as usual in layers without drainage.

Course.—After a rather stormy convalescence, the man recovered, and within a year was able to return to work. (This is case 3 of a previous report: Chronic Subdural Hematoma, Ann. Surg. **86**:485, 1927.)

Comment.—The minor nature of the cranial trauma, the long latent period and the abrupt onset of symptoms may easily be appreciated. As was stated, the discovery of a subdural hemorrhage was entirely unexpected. However, as a result of such an experience, in view of a head injury, a correct diagnosis was made preoperatively in another case.

History.—R. M. P., a man, aged 45, was admitted to the University Hospital on Feb. 3, 1930, with headache and mental confusion. On Nov. 11, 1929, he had been unconscious for about an hour following an automobile accident. He remained in another hospital overnight and was discharged to the care of his physician. A wound in the left occipital region required dressing for a week. He then returned to work as an automobile salesman. Two months after the injury, he noted dull headaches on the left side, centering over the eye and radiating to the occiput. Two weeks before admission, he became mentally confused and partially aphasic for an hour or two. He had a severe spell of vomiting at this time. A week later, he became much confused in filling out an order blank, a routine procedure which he had carried out many times before. During the week before admission, the headache became more severe.

Neurologic Examination.—The man appeared placid and comfortable. He was definitely paraphasic, although at times this was difficult to demonstrate with certainty. There were fine lateral nystagmus and blurring of both optic disks. The right side of the face was doubtfully weaker than the left. The spinal pressure was slightly increased, although the fluid itself was clear. The reflexes were all prompt and active. No Babinski sign nor clonus were present. The headache lessened in the hospital under the administration of hypertonic dextrose, and the speech defect became less marked.

Diagnosis.—It seemed almost unjustifiable to subject a man with so few symptoms to an operation. But an encephalogram seemed contra-indicated because of the intracranial pressure. Furthermore, to some of us, the diagnosis seemed sufficiently clear to warrant surgical intervention without the added risk of injection.

Operation.—It was decided to make an exploratory trephine opening under local anesthesia to inspect the dura over the left temporal lobe, and to throw back a flap if a clot were found. This was done; the dura was greenish blue, and when a cut through it had been carefully made, a clot was seen. An osteoplastic flap was at once reflected, and a large encysted clot, slightly adherent to the dura,

was revealed. The cyst contained about 2½ ounces (74 cc.) of a brownish-green degenerated blood clot. The cyst wall peeled readily off the cortex, which was stained a deep yellow. Following the removal of the clot and the surrounding cyst, the dura was replaced over the brain and the wound closed.

Course.—Convalescence was uneventful. The headache disappeared at once, and speech was normal within a week. Three weeks after discharge from the hospital, the patient was at work again.

Comment.—The differential diagnosis between traumatic subdural hemorrhage and other expanding intracranial lesions, as tumor or abscess, depends on the discovery of the antecedent trauma. With regard to the localization of the hemorrhage, the fact that it occurs over the cerebral hemisphere in by far the larger number of cases, is of importance. The neurologic signs, considering the extent of the hemorrhage, are as a rule, surprisingly few. A careful study with a keen search for localizing evidence may be essential in these patients. The intracranial pressure may not be increased; in fact, in over half the cases here reported, neither lumbar puncture nor retinoscopic study suggested tension. It is interesting to note in the hemorrhages over the left cerebral hemisphere that apparently involved the whole surface of the brain in this region, that difficulty in speech was the first symptom. This suggests that if the whole left hemisphere is uniformly compressed, a complicated mechanism, such as speech, is interfered with before the less elaborate functions of motor movement or sensation are involved.

The treatment of traumatic subdural hemorrhage is essentially surgical. Once the diagnosis is made from clinical evidence, air injection or exploratory trephining, an osteoplastic flap should be reflected. An osteoplastic flap is much more satisfactory than a decompression, because these hemorrhages are always considerably larger than the symptoms suggest, and only by the use of a wide exposure can the whole clot be removed with certainty. On exposure, the dura will have the curious blue-green tinge to which attention has already been called. After opening the dura, the whole clot and its confining cyst membrane should be extirpated. It is not necessary to excise the overlying dura to prevent recurrence of the clot. A most favorable sign, once the clot has been evacuated, is to have the brain commence to pulsate and to swell out and fill the empty space. Reports from the literature suggest that in many fatal cases the brain remained compressed, and no benefit resulted from relief of pressure. Frequently, these hemorrhages are bilateral. It is important to realize this, for if evacuation of the clot on one side fails to improve conditions, an exploratory opening should be made on the other side.

If the diagnosis is made sufficiently early and if operation promptly follows, the prognosis is good. All the patients in my cases who survived were able to return to their usual occupations in a relatively short time. None of them have complained of annoying after-effects.

DISCUSSION

Dr. N. W. Winkelman: I do not believe that the clinical picture is difficult to explain. One must keep in mind, however, that this is venous bleeding in contrast to arterial bleeding, such as is seen in epidural hemorrhage. The picture Dr. Gardner drew of the subdural hemorrhage is not that which I have seen in many cases. There is in contrast a comparatively thin layer of blood, not over a few centimeters in thickness. My conception of the process has been somewhat as follows: As the result of a mild trauma, many times in the lateral portion of the skull, there has been a tearing of one or more of the veins and bleeding into the longitudinal sinus. Clotting soon occurs, but under emotional or physical stress, bleeding begins again, and so on, until a sizable clot is present. In my experience, bilateral subdural hemorrhage has been rare, although I have seen it.

Dr. Williams B. Cadwalader: In cases of chronic subdural hemorrhage, similar to those Dr. Grant has just discussed, in which the cerebral cortex is severely compressed, I have noticed the comparative infrequency of jacksonian seizures.

DR. CHARLES H. FRAZIER: May I emphasize one point in Dr. Grant's interesting presentation? In a considerable number of cases of chronic subdural hemorrhage the lesion is bilateral. It is important to remember this from the standpoint not only of treatment, but of prognosis. Only recently, one of my patients succumbed because of this. This was a patient with a history of intolerable headache for a month. There were reasons why one should have suspected a chronic subdural hemorrhage. The diagnosis was confirmed by exploration on one side. The patient did not respond well to this operation, and died before a second operation could be performed to uncover the lesion on the opposite side.

DR. W. J. GARDNER: No one as yet has satisfactorily explained the latent period between the inception of the trauma and the appearance of pressure signs in patients with subdural hematoma. A possible explanation is that, following encystment and liquefaction of the hematoma, the latter absorbs cerebrospinal fluid by osmosis through the semipermeable arachnoid and cyst membranes, with progressive enlargement of the hemorrhagic cyst, until pressure signs ensue.

DR. FRANCIS C. GRANT: I wish to refer to the probable venous origin of the blood in these cases. As I stated, it is my belief that the bleeding occurs from the veins running from the cortex to the dura. In a recent article, Trotter stated his belief that these lesions commonly follow a blow on the front or back of the head, so that the movement of the brain is in an anteroposterior direction. In this connection it is interesting to note in the majority of these cases that the injury was in either the frontal or the occipital region, which makes it probable that the movement of the brain within the skull was along an anteroposterior plane. While the hemorrhage is practically always on the surface, only five patients had jacksonian convulsions as a prominent symptom.

THE REACTION OF THE CENTRAL NERVOUS SYSTEM TO EXPERIMENTAL UREA POISONING. DR. BERNARD J. ALPERS.

This article appeared in the September issue of the ARCHIVES (**24**:492, 1930).

TUMOR OF THE SPINAL CORD ASSOCIATED WITH BILATERAL ACOUSTIC TUMORS: REPORT OF A CASE. DR. W. J. GARDNER.

This article appeared in the November issue of the ARCHIVES (**24**:1014, 1930).

PROPRIOCEPTIVE BODY REACTIONS IN TOPICAL BRAIN DIAGNOSIS, WITH SPECIAL REFERENCE TO CEREBELLAR LESIONS. DR. C. W. IRISH.

This article appeared in the November issue of the ARCHIVES (**24**:978, 1930).

CEREBRAL PSEUDOTUMORS. DR. C. H. FRAZIER.

This article appears in this issue of the ARCHIVES, p. 1117.

Book Reviews

PSYCHOTHERAPIE: VORAUSSETZUNGEN—WESEN-GRENZEN. By HANS PRINZHORN. Price, 14 marks. Pp. 334. Leipzig: Georg Thieme, 1929.

When a rhetorician with a facile pen surveys a broad subject like psychotherapy and its philosophic background, the reviewer has no easy task. Prinzhorn calls his book "an attempt to clear the foundations" of psychotherapy. If one uses the standard of what positive contributions a book contains, if not in facts then at least in clear thinking and new perspectives, it would not be necessary to give this book very much attention. But there is a serious aspect to it. Not only psychiatrists, but also neurologists and internists are beginning to realize more and more the tremendous importance of psychotherapy for a very wide group of heterogeneous disorders. Psychotherapy itself, however, is as yet in a stage where different schools disagree with one another with no little vehemence, and where it is difficult to outline the most essential points for those who would know something about it. If, therefore, a medical author writes a book which is subjective to a degree most unusual in scientific literature, it is necessary, for those who regard the development of psychotherapy and instruction in that branch for the general physician as among the important tasks of modern medicine, to take a definite stand and assert themselves about the "clarification of foundations" which Prinzhorn attempts.

A large part of the book is taken up by a discussion of the personality of the psychotherapeutist. In fact, the author says: "The chief problem of psychotherapy, that is the therapeutist." This sentence really gives the keynote of the whole book. The naive medical reader may think that the chief problem of any therapy is the patient. But the troubles of the patient play a very small part in this book on psychotherapy. Scientific knowledge, according to the author, counts for very little in psychotherapeutic success. The psychotherapeutist is born, not made. It is not possible to give a scientific basis to psychotherapy. It is all a question of the personality of the therapeutist, his knowledge of people, his "easy self-objectivation," his inborn leadership. Psychotherapy is really leadership. Ideals of objectivity and equality can never be its foundation."

The philosophy and psychology of Nietzsche and Klages are for the author the only real foundation of any rational psychotherapy. He regards Klages especially as the real leader in the development of a philosophic and psychologic background of psychotherapy. Unfortunately he does not explain why he assigns to Klages such an important rôle, and the reason is not at all evident from the text. Does it really help psychotherapy to subscribe to Klages' view that the psychopath—whatever may be meant by such a categoric formulation—is incurable?

The fundamental defect of the book, and unfortunately at the same time its fundamental note, is the endeavor to arrive at absolutes and to see all problems in the form of either—or. The physician who does not possess an absolutely correct "Weltbild" cannot be a psychotherapeutist to be taken seriously. If he attempts to combine several points of view in order to do justice to the manifoldness of life, the author calls him just a "mediator," a "liberal." Attempts to help patients adjust themselves to their environment by methods of social service are called by him "socialistic." In this way Prinzhorn does not really take issue with the formulations of other points of view. Instead, he attempts to interpret the psychologic or social background of other authors. The criticisms, as indeed the whole book, are written in a rather unusual style of egotistic self-assurance. For example, he complains that Freud did not read more of Nietzsche, for then "we all . . . would have been spared an infinite amount of further thought." Or he interprets the lack of response to one of his previous books as "embarrassed silence," and concludes from this that he has in this book hit the nail on the head.

One might be tempted to use the author's method on the author himself, and to ask some questions about the contents of his book. Why does he write with so much resentment about other authors? Why is he attracted by psychoanalysis as a moth is by light, though at the same time he attacks this mode of psychotherapy so vehemently and with obviously overrationalized reasons? Why does he stress so much the existence of a universal crisis of psychotherapy outside of his own personality? Why does he seek the explanation of other psychologists' points of view in their personalities rather than in their facts and conclusions? And, finally, why does he feel constrained to look to the writings of Klages, a not very lucid but aggressively critical writer, as a philosophic panacea for the supposed general problems of psychotherapy? It is not difficult from a perusal of the book to find the answer to such questions. But from a scientific book one expects anything but a self-revelation of this type.

PROGRESSIVE RELAXATION. A PHYSIOLOGICAL AND CLINICAL INVESTIGATION OF MUSCULAR STATES AND THEIR SIGNIFICANCE IN PSYCHOLOGY AND MEDICAL PRACTICE. By EDMUND JACOBSON. Price, $5. Pp. 428. Chicago: University of Chicago Press, 1929.

It is hardly possible to do justice to this brilliant and original work in the scope of a review. The book deals with a method of treating the neuromuscular system so as "to quiet the nervous system, including the mind," the history of this method and the principles on which it is based, and the possible range of its usefulness. It covers studies by the author which began twenty years ago and which are still being continued. The practical method of "progressive relaxation" which the author advocates is based on a long series of careful physiologic experiments.

In 1908, the author investigated the excessive responses that take place after sudden unexpected stimuli. This "involuntary start" reaction is familiar to every one. He came to explain variations in the results in the following way: "The muscles contracted with a jerk when the strong stimulus came because they were previously contracting, although in less degree, during attention. If there was no previous contraction, as during inattention, no start took place." The effects of the involuntary start are widespread throughout the skeletal and visceral systems of the body. The hypothesis was reached that "all subjective irritation or distress might be reduced if the individual were to become sufficiently relaxed." When the subjects reported a state of effortlessness it was found that there was an increase of relaxation as externally manifested. "What is commonly called effort in our subjective experience consists in part of readily observable contractions of skeletal muscles." The keystone of the method is therefore the observation that "cerebral activity apparently diminishes in the presence of advancing relaxation." Progressive relaxation is "an extreme degree of relaxation" in which all "residual tension" is abolished. "Doing away with residual tension is the essential feature of the present method." It consists of "voluntary continued reduction of contraction or tonus or activity of muscle groups and of motor and associated portions of the nervous system. When the relaxation is limited to a particular muscle group or to a part, it is called local; when it includes practically the entire body, lying down, it is called general."

The author has found that the neurotic person does not know when he is tense. He has to learn to cultivate muscle sense. He is made aware of sensations of muscular contraction, and then when his attention is drawn to it he sees what parts he should relax.

The manifold relationships between "progressive relaxation" and the field of general medicine and physiology are discussed in great detail. There are, of course, many connections between this method and its physiologic background and theoretical and practical problems of psychopathology. Jacobson suggests that in almost all cases the term "neuromuscular hypertension" should be substituted for "neurasthenia."

It is not possible to describe here the practice of "progressive relaxation," nor can the many topics touched on by the author be outlined. The book deserves the highest recommendation as an excellent presentation of a much neglected topic which is of great practical and theoretic importance. Especial attention should be drawn to the carefully compiled bibliography, which is indispensable to any one who is interested in neuromuscular physiology.

CATATONIA AND ITS CONNECTION WITH THE OPTOSTRIATE NUCLEI. By EDGARD PINTO CESAR. Memorias do Hospital de Juquery, 1928-1929, vols. 5 and 6, nos. 5 and 6, p. 39.

The author seeks to establish a connection between the catatonic syndrome and lesions of the basal ganglions. The pathologic changes in these regions have not been thoroughly investigated in dementia praecox. The similarity in clinical pictures has already been commented on by many authors, notably Klippel, Widal, Bernadou, Claude, Mey, Koenig and Westphal, and in certain cases a differential diagnosis between parkinsonism and catatonia has been considered difficult, particularly when an accurate history is lacking. Bernadou has raised the question whether both conditions may not be due to the same neurotropic virus. Kleist has claimed that the hyperkinetic syndrome, which is not rare in catatonic dementia praecox, is due to an alteration in the extrapyramidal system, but the lesions he has distributed diffusely over frontal lobes, pons, corpus striatum, cerebellum, etc. The author's claim that almost all the extrapyramidal symptoms of postencephalitic parkinsonism have been observed in catatonia, for example, the sunken head, hanging arms, expressionless physiognomy and loss of mimic function, the increased muscle tonus and posture tonus, catalepsy, bradykinesia, hyperkinesia, disturbance of sleep rhythm, ocular phenomena, tremor and even choreo-athetoid movements has been described in catatonic dementia praecox. The author associates catatonic disturbances in affectivity with lesions in the thalamus, which has been regarded by Head as the center of consciousness for affectivity, and to explain the frequent transitory nature of these affective changes in catatonics he introduces the theory of toxic action on the synapses. The loss of apperceptivity, memory, stereotypy, loss of initiative and vague fears described by Bianchi, Bechterew and others in cases with lesions in the frontal lobes lead the author to suspect this lesion of the brain also in catatonia. He even terms dementia praecox a diffuse toxic neuraxitis. The material studied by the author consists of ten cases diagnosed as dementia praecox in patients under 30 years of age, which he describes in detail both clinically and pathologically. Sections were studied by cresyl-violet, hematoxylin-eosin and the Bielschowsky, Cajal and Weigert technics; in all cases degenerative lesions were found in the optostriate nuclei, with diminution of the cellular elements and proliferation of neuroglia. These lesions, though not specific, were diffuse rather than focal. The association zones in the frontal lobe were those chiefly involved.

The author concludes with the hypothesis that an infection creating degenerative lesions, similar to those of encephalitis, lies at the base of catatonia, and he feels convinced that the similarity of the clinical picture must exist because of an identity in localization of lesions rather than in infectious agents.

GRUNDZÜGE EINER GENETISCHEN PSYCHOLOGIE. By OTTO RANK. Teil I. Price, 8 marks. Pp. 166. TEIL II. GESTALTUNG UND AUSDRUCK DER PERSÖNLICHKEIT. Price, 5 marks. Pp. 104. Leipzig: Franz Deuticke, 1927-1928.

The first volume of this work consists of a lecture course given at the New York School of Social Work, the second of a course given at the Pennsylvania School for Social and Health Work in Philadelphia. Rank aspires to a new orientation of psychoanalysis. He feels that his previous attempts in this direction have not been sufficiently taken into account by other psychoanalysts. For Rank,

anxiety is the central problem of the neuroses. The revision which Freud has made with regard to the problem of anxiety in his "Hemmung, Symptom und Angst," Rank refers to his own contributions. But he regrets that Freud has not gone the whole way of his own solution of the problem of anxiety, and credits this—as also the negative attitude of other psychoanalysts—to personal "resistances." According to Rank, the mechanism of the transformation of libido into anxiety has never been cleared up. Even if anxiety can disappear after sexual gratification, it is not proved that it originated in repressed sexual libido. The study of the normal elaboration of anxiety experiences in the ego, i. e., in the character, shows that anxiety has a relation to the object, just as the libido has. The development of the relation to the object parallels the development of the ego. What Rank arrives at is a general critique of psychoanalysis in which the therapeutic value of pure analysis is denied; the necessity of constructive, educative measures is postulated. The ethical roots of the guilt experience are especially pointed out. In the course of the presentation of these conceptions the author introduces some interesting discussions on details of psychoanalytic theory and practice.

The psychiatric reviewer of this work feels the difficulty of outlining it against a background of general psychopathology. Not only in terminology but also in the consideration of facts described by other authors—or rather the lack of such consideration—the author goes entirely his own way. One gets the impression, which I do not think is correct, that the problems dealt with by the author, namely, the critique of psychoanalysis and the building up of his "genetic psychology," lie in a narrow, sharply delimited field which one can hardly understand without being a "specialist" oneself. The author spins and spins the thread of a cocoon, in which the threads are fine enough, and certainly get longer and longer. Yet one misses a clear relationship, not only to the writings of other authors who have gone over similar and partly identical ground, but also to the many data belonging to the author's own problem, although they lie outside of his cocoon. The two volumes contain some interesting contributions in their details. But bearing in mind the great influence of the author in the psychologic orientation of the mental hygiene movement, one might rightly wish that this movement would seek guidance a little nearer to scientific normality.

DIE STÖRUNGEN DES BEWUSSTSEINS. By M. ROSENFELD. Price, 16 marks. Pp. 247. Leipzig: Georg Thieme, 1929.

The limitation of general classifications in psychiatry is well shown by the fact that it would be rather difficult to delimit clearly the scope of the disorders included in this book. Rosenfeld is concerned less with describing a special syndromatic entity or a reaction type than with the attempt to present a series of clinical facts that have a great deal in common and can be grouped together conveniently as "disorders of consciousness." He believes that disorders of consciousness are always accompanied by "changes in the bodily-nervous sphere." From the point of view of systematic psychopathology his attempt is rather problematic. Disorders of consciousness occur in almost any psychopathologic reaction type, and it is misleading to infer from their existence a physical, or as one says nowadays, "biologic," foundation. The author assumes, for example, that hysteria has a biologic foundation. Owing to the ubiquity of prolonged or episodic disorders of consciousness, one would have to survey the whole field of psychiatry if one wished to do justice to the subject indicated by the title of this book. And this indeed the author does. His book is a condensed survey of psychiatry from the point of view of the study of disorders of consciousness. In the general part he discusses the psychic symptomatology, the neurologic accompanying symptoms and the somatic accompanying symptoms. In the special part he takes up nosologic entities—from hysteria to schizophrenia—in which disorders of consciousness occur.

. Consciousness is disturbed when the normal preparedness for single mental acts is changed, restricted or lost and when normal mental contact with the immediate environment cannot be maintained. Or it may happen that the patient has the inner experience that a change, a disharmony, in his most central processes is approaching, that it only threatens to approach, or that it has already occurred. In the first case one might speak of "subjective disorders of consciousness," in the latter of "objective" ones.

The book is remarkably free from theoretic discussions, restricting itself mainly to clinical observations and their interpretation. Its main emphasis is on the association of mental and physical disorders. In going over the author's level-headed presentation, one becomes again aware of how unsatisfactory is the present status of the somatopathology of mental disease. In its sphere this book has considerable usefulness.

LA PRATIQUE PSYCHIATRIQUE. By M. LAIGNEL-LAVASTINE, ANDRÉ BARBÉ and DELMAS. Second edition, revised and corrected. Pp. 891. Paris: J. B. Baillière et fils, 1929.

In the development of American psychiatry it has come about that the conceptions of French authors have had little influence. Today it is difficult for those not well versed in the historical development of French psychiatry to find their way in the terminology of French authors. The aim of the textbook edited by Laignel-Lavastine is to make psychiatry easily understandable to students of general medicine and to practitioners, to show that at least an elementary knowledge of psychiatry is necessary to every physician, and to point out that "the psychiatric anarchy is much more in words than in facts." The unity of the volume is assured by the fact that the three authors are all pupils of the eminent Gilbert Ballet. The first part, by Barbé, takes up the general symptomatology of mental diseases; the second part, by Delmas, takes up the nosology. Laignel-Lavastine discusses legal medicine in the third part, which is based on lectures given in Paris. The first edition of this book was successful. This edition has been revised but not enlarged.

One might make the generalization that French psychiatry views phenomena more with a sharp lens, emphasizing the details, while German psychiatry views them more broadly and discovers larger entities. (This explains why Wernicke has been more read in France than other German authors.) Neither of these two aspects should be neglected. The presentation of psychiatry in this volume will strike the reader as rather formal; but it is useful as an introduction to the French point of view. How different this point of view is from current American psychopathology can be seen for example in the classification; one finds manic-depressive psychoses under the constitutional psychoses, neurasthenia under the toxic-infectious psychoses and dementia praecox under the organic psychoses. While the first two parts of the book are more elementary, the section on forensic psychiatry contains a great deal of interesting material. The lack of an index is conspicuous in a book that is evidently not intended to be read in one stretch from cover to cover.

DIE GEISTIGE ENTWICKLUNG DES KINDES. By KARL BÜHLER. Fifth edition. Price, 15 marks. Pp. 484. Jena: Gustav Fischer, 1929.

This is the fifth edition of Bühler's monographic treatise on the mental development of the child. It contains in compact form a large amount of material, so that it is not possible to cover it adequately in a review, especially since the subject itself makes it necessary that heterogeneous groups of facts and theories be included.

The material is grouped in large sections. The first is devoted to general problems, such as comparative psychology, the inheritance of mental qualities, the scope of psychology of the child and the child's bodily development. Other

chapters deal with the first year of life, perceptions, the development of representative activity, the development of language, the development of drawing, the development of thinking and a general theory of mental development. In the author's opinion, the development of drawing has especial interest, and he discusses it fully. Perhaps the most original part of the book is the author's outline of the problem of language and language development. Koffka's book on the foundations of psychic development is criticized; Bühler regards it as too simple and as not doing justice to the variety of facts. It seems a regrettable omission that Bühler has not availed himself of the opportunity to enlarge and broaden his outline and his data by the more recent observations concerning the psychopathology of childhood. Inclusion of a discussion of psychoanalytic observations, for example, seems to be not a question of theory or point of view, but rather a question of scientific completeness in any book which aims to cover the mental development of children. Of course, how much one may include, or from what standpoint one may consider such data, is a different question. Bühler ignores psychoanalysis in toto; neither the name of Freud nor that of any one of his followers appears in the index. In the chapters on heredity and on anthropology, likewise, newer contributions are insufficiently considered. Despite these qualifications, this book may be considered at present one of the standard works on the subject. The book is carefully organized. Each chapter is followed by a brief bibliography, and the book as a whole is adequately indexed. Such a book deserves the careful attention of the neuropsychiatrist.

UEBER DEN INSTINKT. By L. R. MÜLLER. Price, 1.20 marks. Pp. 27. Munich: J. F. Lehmanns, 1929.

By way of his studies on the vegetative nervous system the author has come to a consideration of instinctive behavior. In this booklet he gives a survey of his views on instincts in general. Instinctive acts are executed as if the individual had foreknowledge of future events. Müller opposes the idea that instincts have something to do with the cerebral nervous system. They are not "inherited habitual acts" or "mechanical volitional acts," nor are they due to the "inherited modification of the brain" of which Darwin spoke. Insects, like ants, bees or spiders, have very few ganglion cells. Their minute cerebral nervous system, therefore, cannot be the seat of their manifold instinctive reactions. Instincts develop with the general bodily development; they are closely intertwined with reflexes, from which they sometimes cannot be separated. The basis of instinct is the need to use a given organ. Instinctive acts on the one hand and the growth of bodily structures on the other are nature's way of guarding the organism against harmful influences from the environment. It is important that the accomplishment of procedures incited by instinct causes feelings of pleasure, whereas their hindrance causes feelings of displeasure. Instincts have a phylogenetic development like that of the morphologic structure of the body. "While we have to distinguish sharply between instinctive acts, which occur without foreknowledge of their purpose, and intelligent acts dictated by experience, it must nevertheless be conceded that instinctive acts in animals that have a well developed central nervous system may be influenced by memory impressions and their combinations, that is to say, by psychic processes of the brain." Processes inside the body, although they may appear to be "purposeful," as for example in antitoxin formation, are not "instinctive." The latter term is reserved for actions outwardly recognizable. There is for the brain as for all other bodily organs an instinctive impulse toward activity. It is this "organ instinct of the brain" which is at the basis of all creative work and not intelligent reflection or education or environmental influences.

This discussion is enlightened by a large number of examples of instinctive behavior in animals. It is written in an easy style and avoids consideration of the more complicated problems of instinctive life.

LE RÉVE. By DR. MARTIN GOMES. Pp. 179. Rio de Janeiro: Rodrigues & Cia, 1928.

This book contains material which the author describes as preliminary to a larger work on the "Direction of Thought." In it he has endeavored to apply to the study and analysis of dreams some general psychologic principles concerning the factors that lead to the particular form of action that is chosen in any set of circumstances. He illustrates this by a diagram similar to the parallelogram of forces and points out that the reaction which occurs is the resultant of forces that constitute the real aim and those that are represented by sense perceptions, both visceral and external, with their concomitant emotional and sentimental tendencies. The various tendencies and the modifications which they cause in the state of receptivity to stimuli are discussed in some detail.

Chapter 2 is devoted to an analysis of the love sentiments, and chapter 3 deals with the sequence of representations. Chapter 4 discusses hypnagogic states and Chapter 5 the dream states. These are admirably illustrated by an analysis of dreams and hypnagogic states reported in the literature by various authors as well as by references to literary characters. In chapter 6 is given a brief but excellent criticism of freudian interpretations which pays tribute to the genius and accomplishments of Freud while recognizing that his work is that of a physician and pragmatist rather than of a psychologist.

The book contains an excellent psychologic review of the facts of dreams and their significance in which no interpretation is forced, but in which it is shown that they are subject to rules that govern all psychic activity with modifications due to the particular plane of consciousness in which they occur. Throughout the activity that results is "comme une recherche du moins désagréable." It is unfortunate that the proofs were not read, as noted by the author, before publication as the text contains numerous errata, some of which make the reading somewhat difficult. Any student or physician who is interested in an understanding of the meaning of dreams should certainly study this book.

DIE PSYCHIATRISCH-NEUROLOGISCHE BEGUTACHTUNG IN DER LEBENSVERSICHERUNGSMEDIZIN. By A. H. HÜBNER. Price, 13 marks. Pp. 170. Leipzig, Georg Thieme, 1928.

This book is an exhaustive, systematic treatise on psychiatric-neurologic observations in relation to life insurance. The first part deals with the legal aspects; the second part discusses the different neurologic and psychiatric diseases that are significant for the question of life insurance. The whole subject is thoroughly considered. Instructive examples are given abundantly, with full use of the literature on the subject. For all persons dealing with practical problems connected with life insurance this book should be a great help. The impression is conveyed that life insurance companies would save a great deal of money if they made more use of competent psychiatrists.

But the book is also of considerable interest apart from the special subject of life insurance medicine. Most instructive examples are given of wrong diagnoses. For instance, in one case the physician described the knee reflexes as present on both sides, the gait as normal. On a second examination the man was found to have one false leg. In another case, three physicians certified that the pupils reacted well to light and to accommodation. That man had a false eye. The variety of methods of simulation used by insurance candidates can throw light on the problem of simulation under other circumstances. It is interesting that, according to the author's large material (526 cases), mental disease and the existence of psychopathic traits play a large part in suicide, contrary to the statement of Leoncini. In 44.48 per cent of suicides there was a definite mental disease. The author's investigation on the causes of death in cases of mental disease and psychopathic deviations are also of considerable interest.

DIE UNFALLNEUROSE ALS PROBLEM DER GEGENWARTSMEDIZINE. By WALTHER RIESE. Price, 8.50 marks. Pp. 261. Stuttgart: Hippokrates, 1929.

The problem of "traumatic neuroses" in its various aspects is discussed in a series of brief papers. The contributors are Wittgenstein, Walther Riese, Honigmann, Fränkel, Karl Landauer, Sperling, Meng, Levy-Suhl, Hertha Riese, Rosenstein (Moskau), Meyer (Köppern), Monakow and Eliasberg. All the contributors agree that "expert opinions" in cases of traumatic neurosis should not be based on any dogmatic schemes or questions of precedence. Traumatic neuroses are recognized as real pathologic conditions. Careful examination in each individual case and attempts at psychotherapy are recommended. The need for the application of social service methods to the not cured and not compensated patient with traumatic neurosis is stressed. (Why only for these?) The book is intended as a counter move to the "reigning doctrine of medical science" concerning traumatic neuroses. Although the book contains a great deal of chaff, the fundamental tendency of the authors; namely, their stand against too rigid decisions based on the assumption that compensation desires are at the root of practically all traumatic neuroses, is undoubtedly correct.

GEISTESKRANKHEIT IN ALTER UND NEUER ZEIT. By H. A. ADAM. Price, 10 marks. Pp. 160. Regensburg: Ludwig Rath, 1928.

The author, chief physician of the state hospital at Regensburg, gives a survey of the treatment of psychiatric patients from old times to the present day. The book is intended for nonmedical as well as medical readers. In clear language and with well chosen concrete examples is sketched the historical development of hospital care of patients with mental disease. Although the material is not the product of any new researches, but is based on standard works on the subject, it is presented in an original and most satisfactory manner. In the last part of the book a description of some modern state hospitals is given, and the methods of treatment are outlined. Occupational therapy takes a prominent place. This book fulfils excellently the task of explaining to the layman what a state hospital stands for. The psychiatrist, too, will find here a historical survey which makes interesting reading. The numerous illustrations are well selected. Of especial interest are the photographs of a wooden tankard dating from the eighteenth century. It is covered with figures in low relief carved by a patient to represent scenes from his hospital experience.

AESCULAPIUS. A One-Act Play. By BARBARA RING. Pp. 40. Boston: Walter H. Baker Company, 1930.

This little play, suitable for sanitariums or hospitals, can be given either indoors or in the open air. Full directions are given as to staging, costumes, lighting, etc. The play is written around the mythical story that Hades, the god of the lower regions, became incensed at Aesculapius for healing the sick and withholding from him their shades. For this offense Hades killed Aesculapius with a thunderbolt. Whereupon Apollo, the god of the healing art, deified Aesculapius and dedicated the caduceus to his followers.

PATHOLOGIE DES UNBEWUSSTEN. By ARMIN STEYERTHAL. Price, 3.80 marks. Pp. 47. Stuttgart: Ferdinand Enke, 1929.

It is difficult to understand why so eminent a scientist as Moll should include this pamphlet in the series "Psychotherapy and Medical Psychology" which he edits. The author remains entirely on the uppermost surface of the problem which he intends to treat. The pamphlet contains a series of platitudes, a few historical remarks and the expression of an emotional outbrust of the author against psychoanalysis.

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