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Archives of Neurology and Psychiatry

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SUPERIOR OLIVARY NUCLEUS

ITS FIBER CONNECTIONS *

JAMES W. PAPEZ, M.D.

ITHACA, N. Y.

The groups of experiments herein reported were carried out in order to determine what connection the superior olive makes with other centers through the olivary peduncle. An abstract of this work appeared in the *Anatomical Record*.[1] The experiments were undertaken with the supposition that the superior olive probably does send fibers into the reticular formation which make connections with various motor centers in addition to the usual connections believed to exist with the nucleus of the abducens nerve.

In the cat (fig. 3), the superior olive consists of a large lateral S-shaped segment and a smaller medial or accessory segment which is a straight piece. Ventral to these are the preolivary nuclei of Cajal,[2] and medial to them in the trapezoid body is the nucleus of the trapezoid body. I have adopted these terms because there is evidence that the (medial) nucleus of the trapezoid body differs essentially in its fiber connections from the olivary and preolivary nuclei. Many collaterals pass from the trapezoid body into the superior olive and the preolivary nuclei.

Reconstructions in wax of the superior olivary nuclei are shown in figures 1 and 2. The medial segment or accessory olive (*aso*), as will be seen from the figures, is the longer of the two segments, extending the entire length of the olive. It is narrowest at its caudal end and gradually enlarges toward its cephalic end. Its ventral border is connected by cell strands with the medial preolivary nucleus. Its dorsal border has a lateral curve which overhangs its lateral concave surface. Its medial surface is convex.

The lateral segment or superior olive proper (*so*) is a folded S-shaped lamina. Its medial limb is connected by cell strands with the lateral preolivary nucleus on which its ventral surface as a whole

* Submitted for publication, Dec. 5, 1929.

* From the Department of Anatomy, Cornell University Medical College.

* Read at a meeting of the American Association of Anatomists, Nashville, Tenn., April 15, 1927.

1. Papez, J. W.: Anat. Rec. **35**:20, 1927.

2. Cajal, S. R.: Histologie du système nerveux, French translation, Paris, A. Maloine, 1909, vol. 1, p. 796.

lies. The medial fold is the larger and longer, extending about three fourths of the length of the medial segment (fig. 2). The lateral fold is relatively short, being developed strongly only in the lower half of the olive. Its cephalic end is reduced in size and joins the preolivary nucleus. Winkler [3] showed that this segment was greatly hypertrophied in a micro-ophthalmic animal in which vision was much reduced. The lateral segment is rudimentary in man.

The preolivary nuclei (*pro*) are looser masses of cells ventral to the compact olivary segments with whose extremities and ventral margins they are in continuity. Their lack of a definite form is due

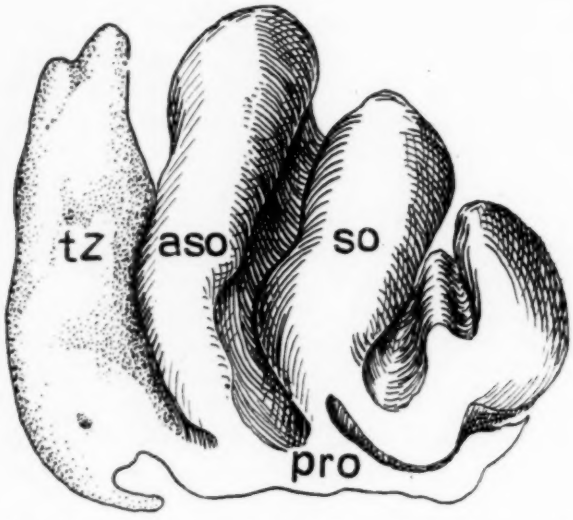

Fig. 1.—Reconstruction in wax of the right superior olivary nuclei in the cat, caudal end viewed from below; × 13. *Tz* indicates the trapezoid nucleus; *aso*, the medial segment of the superior olive; *so*, the superior olive, lateral segment; *pro*, preolivary nucleus.

to the fact that they are pierced by the fiber bundles of the trapezoid body and by the numerous fascicles of collaterals that stream dorsally into the olivary segments. At the caudal end two portions are distinguishable, the medial and lateral preolivary nuclei, each underlying and joining the corresponding segment of the olive. Proceeding upward, they are united into a single mass which at the cephalic end becomes continuous with the lower extremity of the nucleus of the lateral lemniscus (*nll*).

The nucleus of the trapezoid body (*tz*) lies medial to the preolivary nuclei and to the medial segment of the olive. It is pierced by the

3. Winkler, C.: Anatomie du système nerveux, Haarlem, de Erven F. Bohn, 1920, vol. 2, p. 242.

root filaments of the abducens nerve but lies chiefly lateral to them. Its dorsal margin extends almost as far dorsally as does the medial segment. Its medial surface lies obliquely across the outgoing root filaments of the abducens nerve. Its ventral margin cannot be sharply separated from the medial preolivary nucleus. It has no direct continuity with the nucleus of the lateral lemniscus.

The earlier researches of Bechterew,[4] Monakow,[5] Held,[6] Cajal [7] and others on the connections of the acoustic nerves and their central connections are indicated in Cajal's "Histologie" [2] and will not be dis-

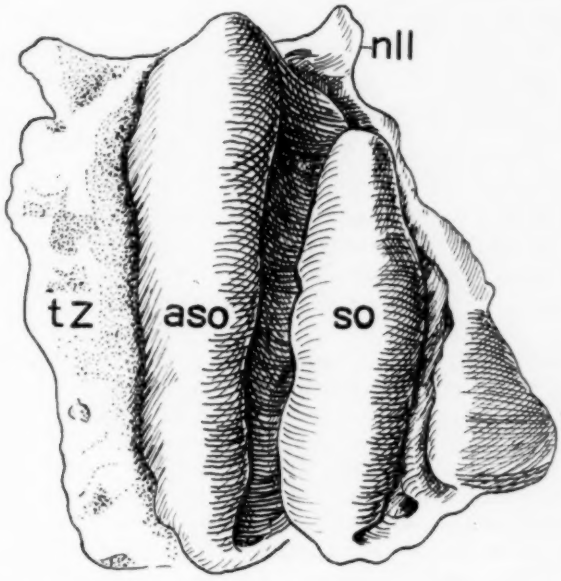

Fig. 2.—Reconstruction in wax of the right superior olivary nuclei in the cat, viewed from the dorsal side; × 13. *Tz* indicates the trapezoid nucleus; *aso*, the medial segment of superior olive; *so*, the superior olive, lateral segment, and *nll*, the nucleus of the lateral lemniscus.

cussed here. This author has also given a complete study of the cell structure of the superior olive.

4. Bechterew, W.: Ueber die Verbindungen der oberen Oliven und ihre wahrscheinliche physiologische Bedeutung, Neurol. Centralbl. **4**:489, 1885.

5. Monakow, C.: Ueber Striae acousticae und unterer Schleife, Arch. f. Psychiat. **22**:1, 1886.

6. Held, H.: Die centralen Bahnen des Nervens acousticus bei der Katze, Arch. f. Anat. u. Psysiol. Anat. Abtheil. **15**:271, 1891; Die centrale Gehörleitung, ibid. **17**:201, 1893.

7. Cajal, S. R.: Beiträge zum Studium der Medulla oblongata, des Kleinhirns und Ursprungs der Hirnnerven, Leipzig, Johann Barth, 1896, p. 77.

The fibers that reach the superior olive from the reticular formation have long been known as the olivary peduncle. They have been traced by Bechterew [4] as far dorsally as the abducens nucleus where he and most authors believe they terminate. Other authors, as Winkler,[3] have them join the medial longitudinal fasciculus and end also on the oculomotor nuclei. Bechterew [8] thought that the fastigial nucleus of the cerebellum was connected by means of the uncinate tract with the superior olive. Monakow [5] and Koelliker [9] stated that the acoustic stria connected with the superior olives of both sides. This fact has been shown by degenerations of the stria by Held,[6] van Gehuchten,[10] Winkler [3] and other authors. The fibers of the stria pass lateral and parallel to the olivary peduncle but are independent of it.

Cajal [2] (page 763) has described and figured the crossed bundle of the vestibular nerve which I believe is nothing other than the outgoing limb of the olivary peduncle which my experiments have shown crossed the midline and passed out with the vestibular nerve. Some fibers of the vestibular nerves probably do cross, but the main bundle which he described is undoubtedly identical with the outgoing limb of the olivary peduncle in these experiments.

Cajal's description is as follows:

Faisceau vestibulaire croisé.—Dans le bulbe des fœtus de chat et de souris, nous avons vu, de façon très nette, partir du nerf vestibulaire, un peu avant la bifurcation de ses tubes, un faisceau de fibres serrées, qui semblent être des fibres radiculaires. Ces fibres se portent en dedans, en passant derrière la branche descendante du trijumeau et en avant du coude du facial; elles cotôient ensuite la face postérieure du noyau du moteur oculaire externe, franchissent la ligne médiane et se perdent enfin dans la moitié opposée de la moelle allongée. Origine et parcours de ce faisceau sont représentés dans la figure 319.

Dans la première partie de son trajet, ce faisceau est compact; mais, à son passage en arrière de la substance grise, il se désagrège en fascicules entrecroisés, d'où un aspect plexiforme qui s'accentue encore dans les portions plus internes. Il est malheureusement impossible de suivre ses fibres jusqu' à leur terminaison, car, au moment où elles traversent la ligne médiane, elles perdent leur disposition fasciculée et se mêlent à des tubes venus de divers côtés, mais surtout à ceux qu'envoie le ganglion de Deiters de la moitié bulbaire opposée.

Ces fibres ne se bifurquent pas durant leur parcours; elles n'émettent pas davantage de collatérales. Quoique nous n'avons pas observé leur traject entier, nous ne serions pas surpris si elles se terminaient dans les foyers vestibulaires du côté opposé. Les fibres de faisceaux homologues, que nous avons trouvés récemment

8. Bechterew, W.: Les voies de conduction du cerveau et de la moelle, 1900, p. 411.

9. Koelliker, A.: Handbuch der Gewebelehre des Menschen, Leipzig, W. Engelmann, 1896, vol. 2, p. 379.

10. Van Gehuchten, A.: La voie acoustique centrale, Névraxe **4**:253, 1902-1903. 1903.

chez les poissons, ont cependant une orientation qui ne semble pas corroborer cette opinion; elles se portent, en effet, à la substance réticulée où elles prennent une direction descendante.

(Crossed vestibular bundle: In the fetal bulb of the cat and the mouse, we have seen clearly a bundle of compact fibers which seem to be radicular fibers leaving the vestibular nerve a little in front of the bifurcation of its tubes. These fibers travel inward, in passing behind the descending branch of the trigeminal and in front of the bend of the facial; thereupon, they travel along the posterior side of the nucleus of the abducens nerve, cross the median line and disappear finally in the opposite half of the medulla oblongata. The origin and course of this bundle are shown in figure 319.

In the first part of its course, this bundle is compact, but on its passage behind the gray substance, it separates into interwoven fasciculi, giving it a plexiform aspect which is accentuated still more in the more internal portions. Unfortunately, it is impossible to follow its fibers to their termination, because at the point where they traverse the median line they lose their fascicular tendency and mingle with the tubes coming from various sides, especially with those which are sent by Deiters' ganglion from the opposite bulbar half.

These fibers are not bifurcated during their course; they do not emit further collaterals. Although we have not observed their entire course, we would not be surprised if they ended in the vestibular centers of the opposite side. The fibers of homologous bundles which we have recently found in fish have, however, a course which does not seem to corroborate this opinion; they travel, in fact, to the reticular substance, from which they take a descending direction.)

Cajal believed that the superior olives are acoustic reflex centers and send their fibers into the reticular formation to make connections with motor nuclei. In his "Histologie" [2] (page 829) he has figured these fibers as making connections with the nucleus of the spinal accessory nerve and other cephalogyric nerves. Malone [11] recently described the cells of the olive as being cytologically of the motor type.

To test out these hypotheses, I have placed lesions in the region of the superior olive from the ventral side. All of the experiments were performed on cats.

OPERATIVE PROCEDURE

The operative approach was from the ventral side of the neck, carried out under anesthesia and with aseptic precautions. The hair was removed with a solution of sodium sulphide. The skin was cleansed and incised lengthwise near the midline. The commissure of the external jugular veins in this region was doubly ligated and cut. The larynx and pharynx were separated from the infrahyoid muscles on one side for some distance on a level with the external auditory meatus and the tympanic bulla. The vessels and nerves in the carotid sheath were displaced to the side with their surrounding fascias. The prevertebral fascia was opened to expose the longus capitis muscle. This muscle was cut across opposite the middle of the tympanic bulla. Then the cut ends of the muscle were pushed cephalad and caudad to expose the basioccipital bone and the ventral atlanto-

11. Malone, E. F.: The Cell Structure of the Superior Olive in Man, J. Comp. Neurol. **35**:205, 1923.

occipital ligament. This ligament and the bulla were used as landmarks for placing the opening in the skull. In the average medium-sized cat, the trapezoid body and superior olive were located about 1 cm. cephalad to the middle of the margin of the foramen magnum opposite the middle or most prominent portion of the tympanic bulla. When this point was located, a drill hole was made through one side of the base close to the bulla with a roundheaded dental drill 2 mm. in diameter. These drills had the form of the large Cushing trephine, and would not tear through the dura as would the pointed dental drills. In placing the drill hole it was found important to have deep, properly fitting retractors in the wound so that no connective tissue would be twined around the drill. This accident would injure the nerves and vessels in the carotid sheath where they entered the skull through foramina around the bulla. The drill hole exposed the dura. When the hole was properly placed no veins were apparent in the dura. A probe with a narrow sharply pointed and curved end about 1 mm. in diameter was used to make the lesion. A drop of solder, about 3 mm. in diameter, was attached about 6 mm. above the end of the probe to fix the depth to which the probe entered the skull. In large animals a slightly longer end was used. The probe was introduced through the drill hole in such a way as to puncture the dura and the surface of the brain. Then the handle was raised to a vertical position, and the probe was rotated several times and withdrawn. With some practice in avoiding the large veins in the base of the skull, the operation was practically bloodless except in the cutaneous region. The skin was sewed up and sealed with collodion. The animals were killed on the tenth or eleventh day. The brain was removed and fixed in formaldehyde over night. The dorsal part of the cerebellum was cut away. The medulla oblongata and pons were cut into suitable blocks, hardened in 3 per cent dichromate solution for ten days, and thereafter treated with an addition of 1 per cent solution of osmic acid. The blocks were embedded in celloidin, sectioned and examined under the microscope for the total extent of the lesions and degeneration of fiber tracts.

Several days after the operation, the animals were tested for the presence or absence of the corneal, nictitating membrane, labial, ear flicking and nasal head retraction reflexes, and other motor or sensory disturbances on the two sides of the head region.

LESIONS AND DEGENERATED FIBER TRACTS IN INDIVIDUAL CATS

From a larger number of experiments the following twenty specimens have been selected as representing a variety of lesions in and around the superior olive. Since the main demonstration concerned the origin of the peduncle of the superior olive, lesions of the surrounding region are also included in the records. The lesions that injured the medial nuclei of the olive or the trapezoid nucleus produced the degeneration of the olivary peduncle, and because they would repeat the main results, only typical ones have been included. A brief synopsis of the records is given.

CAT 458.—The lesion lay just lateral to the lateral S-shaped segment of the left superior olive, extending its entire length. It was close to the olive, but nowhere did it directly damage it. The lesion was a mechanical injury which contained no blood clot, and it may be inferred that no pressure was exerted on the olive as is the case when a blood clot is present. The lesion extended to the depth of the dorsal surface of the olive but not beyond. The probe entered ventral to

the cochlear nuclei through the trapezoid body and spinocerebellar tract. At a more caudal level the probe injured the lateral part of the facial nucleus, especially its dorsal and lateral cell groups. The lesion was maximal between the olive and the facial nucleus. Here it extended also to the ventral side of the facial nucleus into the region of the ventral reticular nucleus, but nowhere did it touch the nucleus of the trapezoid body.

Six degenerated fiber systems were readily identified and followed out in the sections:

1. Fibers of the caudal portion of the trapezoid body including the small dorsal division were degenerated in a medial direction. Great quantities of degenerated terminals entered the superior olive of the same side. A smaller number of fibers crossed to the other side.

2. Reticulocerebellar fibers extending to the floccular lobes of both sides were degenerated. Those that entered the lower surface of the left restiform body and thence into the fiber stratum of the floccular lobes were most abundant. Those that crossed to the other side were less abundant, being about 10 per cent of those to the left. These fibers did not pass dorsally over the restiform body to enter the peduncle of the flocculus, but they passed on the medial ventral side of the restiform body close to the entering vestibular nerve. As the restiform body turned dorsally to enter the cerebellum, the reticulocerebellar fibers turned laterally under its ventral border and entered the floccular lobe.

3. The left ventral spinocerebellar tract was degenerated above the lesion. It curved over the surface of the brachium conjunctivum, and entered the vermis. About one half of the fibers passed around the lingula to the opposite side of the vermis. Both halves of the bundle were distributed to the cerebellar folia that line the caudal wall of the fissura prima.

4. The lateral and anterior fibers of the pars prima of the facial nerve leaving the lateral and superior cell groups of the facial nucleus were completely degenerated. They accumulated in the lateral segment of the genu of the facial nerve. In this animal the bulbar retraction reflex was absent on the left side, and the corneal reflex was sluggish. The ear reflexes, labial reflexes and nasal head retraction reflexes were normal. There were no definite motor symptoms.

5. The peduncle of the superior olive that came from the left side had a few degenerated fibers in it. The fibers accumulated on the medial side of the genu of the left facial nerve and crossed the midline to pass in the outgoing limb of the peduncle on the right side. The outgoing limb of the olivary peduncle on the left side showed no degeneration. The degenerations in the olivary peduncle coming from the left side can be attributed to the proximity of the ventral part of the lesion to the nucleus of the trapezoid body, since these fibers did not appear to arise from the lateral segment of the superior olive.

6. Some degenerated fibers occurred widely scattered in the dorsal part of the reticular formation lateral to the medial longitudinal bundle of the same side. A few degenerated fibers occurred in the ventral part of the opposite medial longitudinal bundle.

CAT 454.—The lesion was a small puncture wound just caudal to the left superior olive, and injured chiefly the cephalic end of the facial nucleus and the reticular formation in front of it. It injured also the posterior part of the trapezoid body as far cephalad as the lower end of the superior olive. There was, however, no direct injury of the superior olive itself. The lesion extended

dorsalward close to the ventral surface of the descending trigeminal nucleus which was slightly involved. The following degenerations were followed out in the sections:

1. Fibers of the pars prima of the left facial nerve involving about 10 per cent of the nerve were degenerated.

2. The caudal and ventral fibers of the trapezoid body were degenerated from left to right, the degenerated fibers ending in the superior olives.

3. Above the lesion, some ascending fibers of the ventral spinocerebellar tract extended over the surface of the brachium conjunctivum into the vermis of the cerebellum.

4. There were a few degenerated fibers in the ventral and caudal parts of the left abducens nucleus.

5. There was a well defined group of degenerated fibers in the left medial longitudinal fasciculus. There was no degeneration of the peduncle of the superior olive.

In this cat, the bulbar retraction or nictitating membrane reflex was absent on the left side. The corneal reflex was present. The degenerated fibers in the abducens nucleus appeared to arise from the injured upper part of the trigeminal nucleus. They passed dorsomedially along the lateral side of the pars prima of the facial nerve, and at the genu of the facial nerve they entered the abducens nucleus. They can be explained as a reflex arc connection between this portion of the trigeminal nucleus and the abducens nucleus, and in all probability they mediate the bulbar retraction reflex. The ear-flicking reflex was absent on the left side, which can be explained as due to the injury of the cephalic fibers of the facial nerve.

Cat 453.—The lesion in this animal was a small wound which penetrated the trapezoid body, the left ventral spinocerebellar tract medial to the outgoing limb of the facial nerve close to the lateral surface of the superior olive, which was not itself definitely injured. The following degenerations were traced through the sections:

1. Some fibers of the rubrospinal tract were degenerated downward.

2. Ascending, degenerated fibers in the ventral spinocerebellar tract extended into the fastigial region of the cerebellum.

3. Fibers of the trapezoid body were degenerated medially. There was no degeneration of the peduncle of the superior olive.

In this animal, there was a loss of the corneal reflex and the bulbar retraction reflex on the left side. At rest, the eye was depressed, and the nictitating membrane was partly drawn over the eye.

Cat 456.—The lesion penetrated the trapezoid body and destroyed nearly all of the caudal part of the lateral, S-shaped segment of the left superior olive. The medial segment of the olive and the trapezoid nucleus were not injured. The following degenerations were traced in the sections:

1. Fibers of the trapezoid body were degenerated from left to right.

2. The acoustic stria passed across the dorsal surface and through the vestibular nucleus, and divided to pass ventrally to the injured olive and also across the midline to the superior olive of the other side. The peduncle of the left superior olive showed no definite degenerations.

3. The ear-flicking reflexes and the labial reflexes were less active on the left side.

CAT 460.—The lesion was a small wound which penetrated the caudal border of the trapezoid body medial to the ventral cochlear nucleus, and which ended in the ventral surface of the descending trigeminal nucleus. The lesion was lateral to the caudal end of the superior olive, but there was no definite injury of the olive or nucleus of the trapezoid body. The following degenerations were traced in the sections:

1. The deep or internal division of the trapezoid body was degenerated from the left side. It passed over the restiform body, and entering the reticular formation divided into two streams of degenerated fibers, one which passed to the superior olive of the same side, and the other which crossed the midline to the olive of the other side.

2. Ventral fibers in the trapezoid body were degenerated from the site of the lesion to the superior olives of both sides.

3. There was an extensive degeneration of fibers passing from the lesion under the restiform body into the left floccular lobe. The position of the lesion justifies the inference that these are the reticulocerebellar fibers and not those coming from the superior olive.

4. There was some degeneration of fibers of the dorsal spinocerebellar tract and of the laterocerebellar fibers. There was no degeneration of the peduncle of the superior olive.

5. The medial rootlets of the left abducens nerve were totally degenerated. There was a loss of the bulbar retraction reflex, and the nictitating membrane was partly drawn over the eyeball.

CAT 328.—The lesion was a large vascular wound of the right tegmentum of the pons which destroyed a small part of the cephalic end of the lateral S-shaped segment of the right superior olive.[12] The medial segment of the olive and the trapezoid nucleus were not directly involved in the lesion, but so large a blood clot as was present in the lesion must have exerted considerable pressure on these surrounding structures. The following degenerations have previously been described: (1) the right rubrospinal tract passing down into the spinal cord, (2) a small part of the descending root of the right trigeminal nerve, (3) the dorsal and upper portion of the trapezoid body, (4) the dorsal spinocerebellar tract into the cerebellar vermis and (5) some degenerated fibers crossing under the genu of the right facial nerve and then over the midline.

CAT 329.—The lesion destroyed most of the lateral S-shaped segment of the right superior olive, but the medial segment of the olive and of the trapezoid nucleus were not injured. At a lower level the lesion destroyed the restiform body, the descending root and nucleus of the trigeminus, the descending root of the vestibular nerve and the adjacent reticular formation. The upward extent of the lesion ended on a level of the outgoing limb of the facial nerve.[13]

There was no evidence in this specimen that the peduncle of the right superior olive was degenerated. One can infer that this peduncle does not come from the lateral S-shaped segment which was destroyed in this case. The reticulocerebellar fibers were grossly degenerated and passed to the floccular lobe.

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12. Gray, Lloyd P.: Some Experimental Evidence on the Connections of the Vestibular Mechanisms in the Cat, J. Comp. Neurol. **41**:334, 1926. Papez, James W.: Reticulo-Spinal Tracts in the Cat: Marchi Method, J. Comp. Neurol. **41**:378, 1926.

13. Gray: J. Comp. Neurol. **41**:337, 1926.

CAT 362.—The lesion destroyed the lateral half of the S-shaped segment on the left side. There was, in addition, a small injury to the ventral end of the medial segment of the superior olive and to the preolivary nucleus in its anterior extent. The following degenerations were traced in the sections:

1. The left acoustic stria was degenerated. This specimen showed clearly how the fibers of the acoustic stria pass into the reticular formation and divide into two streams; one passes to the upper border of the lateral S-shaped segment of the same side, and the other crosses the midline and reaches the upper border of the S-shaped segment of the other side.

2. The dorsal spinocerebellar tract was degenerated above the lesion into the vermis of the cerebellum.

3. The reticulocerebellar fibers appeared degenerated under the left restiform body.

4. The left rubrospinal tract was degenerated below the lesion.

5. Fibers of the trapezoid body were degenerated from left to right.

6. A few fibers in the peduncle of the left superior olive were degenerated. Most of the peduncle, however, appeared normal. This specimen showed that the peduncle does not arise from the lateral S-shaped segment, which was severely injured by the lesion.

CAT 503.—The entering wound was a straight thrust through the lateral third of the right pyramidal tract just medial to the nucleus of the trapezoid body which was largely damaged. The main part of the lesion was a vascular wound situated medial and then dorsal to the medial segment of the right superior olive (fig. 3). From this dorsal extension of the wound, degenerated fibers streamed dorsally to the genu of the facial nerve. The medial segment of the olive was partly damaged, but the lateral S-shaped segment was not involved. There was much fine black precipitate in the surface of the sections. The following degenerations were traced in the sections:

1. The ventral and dorsal fibers of the trapezoid body were degenerated from the right across the midline to the left.

2. The acoustic stria from the frontal surface of the lesion across the midline to the left superior olive was degenerated.

3. The reticulocerebellar fibers were grossly degenerated from the lesion to the right floccular lobe along the ventral surface of the restiform body.

4. The peduncle of the right superior olive was completely degenerated (fig. 3). The fibers appeared to come from the region of the medial segment of the superior olive. They passed dorsally and medially as a scattered strand through the lateral border of the abducens nucleus, and accumulated into a compact bundle ventral to the genu of the right facial nerve. The fibers could be traced through the abducens nucleus into the compact bundle. The bundle passed between the extreme cephalic parts of the two facial genua decussating with its fellow across the dorsal surface of the medial longitudinal fasciculi. On the left side, it passed beneath the vestibular nucleus and then over the descending root of the trigeminal nerve. Over the lateral surface of the trigeminal root, it became associated with the ventral side of the incoming root of the vestibular nerve. It disappeared from view ventral to the cochlear nucleus, where it appeared to pass out with the vestibular nerve. There was no definite evidence that the bundle turned in a medial and cephalic direction to join the lateral leminscus. The degenerated fibers in the trapezoid body entering the left lateral lemniscus did not permit a positive settlement of this important point.

CAT 500.—The lesion was a penetrating wound which pierced the trapezoid body between the pyramidal tract and nucleus of the trapezoid body. It destroyed the outgoing root of the left abducens nerve. The nucleus of the trapezoid body was partially damaged, but the two segments of the left olive were intact. The following degenerated fibers were traced in the series of broken and obliquely cut sections:

1. Fibers of the trapezoid body were degenerated from left to right.

2. Some reticulocerebellar fibers appeared degenerated on the left side.

3. An abundance of degenerated fibers occurred in the commissural region between the genua of the facial nerves. These fibers passed out in the typical position lateral to the genu of the right facial nerve. The sections through this region were badly cut and broken up, but the course of the fibers was evidently the same as in other specimens.

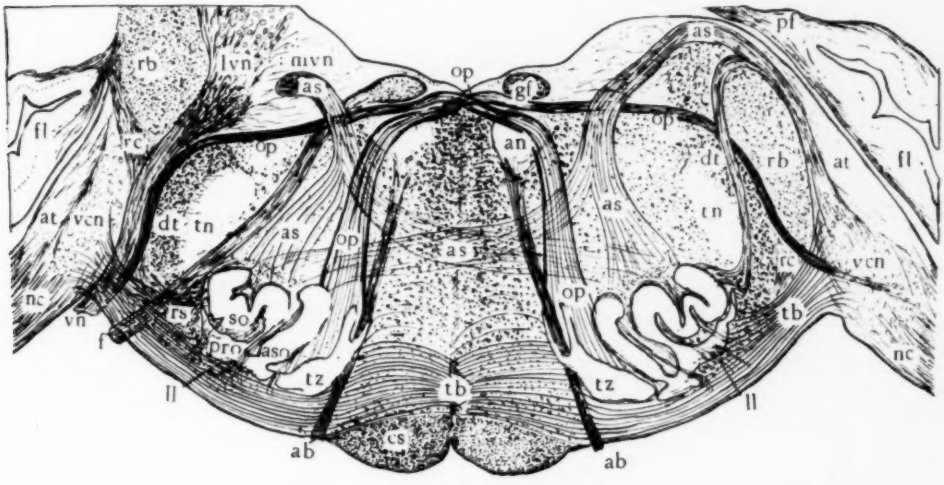

Fig. 3.—A diagrammatic section through the region of the superior olive of the cat representing the arrangement of fiber tracts in this region and showing the course of the olivary peduncles. *Fl* indicates the flocculus; *at*, acoustic tubercle; *nc*, cochlear nerve; *vcn*, ventral cochlear nucleus; *vn*, vestibular root; *f*, facial nerve; *rb*, restiform body; *rc*, reticulocerebellar fibers; *dt*, trigeminal root; *rs*, rubrospinal tract; *ll*, lateral lemniscus; *lvn*, lateral vestibular nucleus; *tn*, trigeminal nucleus; *op*, olivary peduncle; *as*, acoustic stria; *mvn*, medial vestibular nucleus; *tz*, trapezoid nucleus; *ab*, abducens nerve; *aso*, medial segment of superior olive; *pro*, preolivary nucleus; *so*, superior olive, lateral segment; *cs*, cerebrospinal tract; *tb*, trapezoid body; *gf*, genu of the facial nerve; *an*, abducens nucleus; *pf*, peduncle of the flocculus.

CAT 502.—The lesion was a large vascular wound medial to the right superior olive and extended somewhat over its medial segment so that it cut the fibers above the medial segment of the olive and the trapezoid nucleus. The lesion was much like the one in cat 503. The following degenerations were traced in the sections:

1. Fibers of the trapezoid body were degenerated from right to left.

2. The fibers of the peduncle of the right superior olive were abundantly degenerated. They passed dorsally through the right abducens nucleus and formed a compact bundle under the genu of the facial nerve. They crossed the midline over the medial longitudinal fasciculi. Then they passed through the upper end of the outgoing limb of the left facial nerve, ventral to the vestibular nucleus and over the dorsal border and lateral surface of the descending root of the trigeminal nerve to join the entering root of the vestibular nerve.

Cat 504.—The lesion penetrated the trapezoid body on the left side and injured the nucleus of the trapezoid body. The lesion extended dorsally into the reticular formation along the course of the left abducens nerve medial to the superior olive. The following degenerations were traced in the sections:

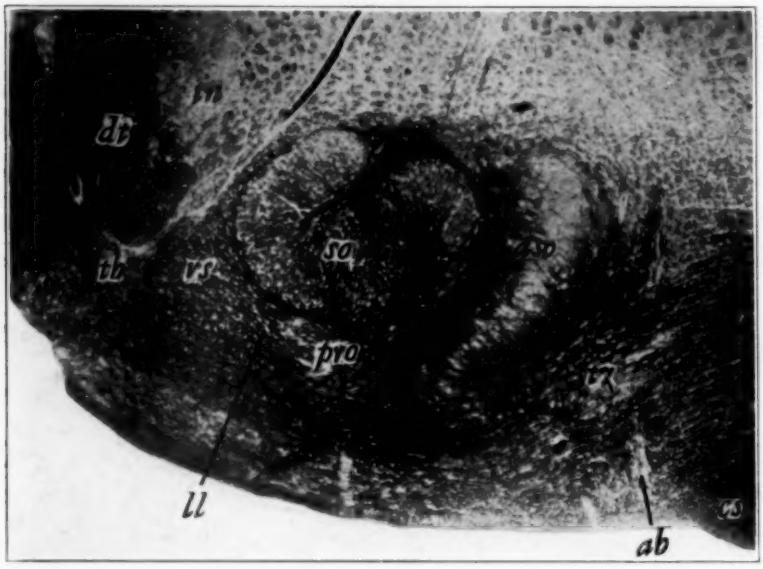

Fig. 4.—A transverse section through the left superior olivary nucleus of the cat; × 17. *Dt* indicates the trigeminal root; *tb*, the trapezoid body; *tn*, the trigeminal nucleus; *rs*, the rubrospinal tract; *so*, the superior olive, lateral segment; *ll*, lateral lemnicus; *pro*, preolivary nucleus; *aso*, medial segment of superior olive; *tz*, trapezoid nucleus; *ab*, abducens nucleus; *cs*, cerebrospinal tract.

1. Fibers of the trapezoid body were degenerated from right to left.

2. Fibers of the right lateral reticulospinal tract were degenerated downward.

3. The peduncle of the left superior olive was degenerated. The fibers passed dorsally and under the genu of the left facial nerve. Then they crossed the midline, passed under the genu of the right facial nerve and out over the descending root of the trigeminal nerve.

Cat 450.—The lesion completely destroyed the left nucleus of the trapezoid body and a small part of the medial segment of the superior olive, and possibly the lesion grazed the cephalic end of the olive. The ventral reticular nucleus of the oblongata was also injured. The following degenerations were traced through the sections:

1. The trapezoid fibers were completely degenerated from left to right.

2. The reticulocerebellar fibers were grossly degenerated on the left side. They passed along the ventral surface of the restiform body to enter the floccular lobes of both sides of the cerebellum. They appeared to come from the ventral reticular nucleus and not from the olivary segments.

3. The medial reticulospinal fibers in both medial longitudinal fasciculi were degenerated in a considerable number, passing in a downward direction.

4. The fibers of the left olivary peduncle were degenerated. They appeared to come from the injured trapezoid nucleus. They streamed dorsally and medially through the abducens nucleus and accumulated into a compact bundle under the genu of the left facial nerve. They crossed the midline over the medial longitudinal fasciculi and passed laterally through the outgoing limb of the facial nerve, then beneath the vestibular nucleus on the right side and out over the descending root of the trigeminus to enter the root of the vestibular nerve.

5. There was a considerable degeneration of the fibers of the left abducens nerve.

CAT 360.—The lesion was a wound of the left side that cut through the brachium conjunctivum and left reticular formation and injured the ventral and medial portions of the superior olive and ended cephalad to the olive in the lateral lemniscus and vestibular region.[14] The following fiber degenerations were traced through the sections: (1) the direct vestibulospinal tract on the left side downward, (2) the crossed vestibulospinal tract downward, (3) the rubrospinal tract down on the left side, (4) the medial reticulospinal tract on the left side, (5) Probst's tract down on the left side to the salivary nuclei, (6) the crossed reticulospinal tracts on both sides, (7) the uncinate or cerebellovestibular fasciculus on the left side, (8) fibers in the trapezoid body from left to right, (9) the acoustic stria from left to right, (10) some fibers in the left facial nerve, (11) the reticulocerebellar fibers to the floccular lobe and (12) the superior olivary peduncles of both sides. The dorsally situated lesion on the left side cut the outgoing limb of the right peduncle, while the lesion of the ventral portion of the left olive caused a degeneration of the entire extent of the left peduncle to its exit on the right side. The peduncles passed ventral to the genua of the facial nerves, intercrossed in the midline, passed ventral to the vestibular nuclei and out over the descending roots of the trigeminal nerves where they joined the roots of the vestibular nerves.

CAT 373.—The lesion in this animal was a V-shaped cut in the floor of the ventricle to the left side of the midline between the genua of the facial nerves.[15] In addition to the degeneration already described in this specimen, the outgoing limbs of both olivary peduncles were degenerated. The median lesion cut the decussation of these peduncles between the genua of the facial nerves. The peduncles passed laterally beneath the vestibular nuclei and out over the dorsal borders and lateral surfaces of the descending trigeminal roots to join the vestibular roots.

CAT 363.—The lesion and degenerations in this animal have been described by Gray.[16] The lesion was located mainly in the upper left vestibular nuclei where

14. Gray: J. Comp. Neurol. **41**:325, 1926. Papez: J. Comp. Neurol. **41**:390, 1926.

15. Gray (footnote 14, p. 335).

16. Gray (footnote 14, p. 328).

it severed the outgoing limb of the right superior olivary peduncle just to the lateral side of the left facial genu. The peduncle passed laterally beneath the vestibular nuclei and out over the descending root of the trigeminal nerve.

Cat 344.—The lesion and degenerations in this animal have been described by Gray and by myself.[17] The upper part of the lesion cut through the pars prima of the right facial nerve under its genu, and here it also severed the right peduncle of the superior olive. The degenerated peduncle passed across to the left side ventral to the vestibular nucleus and out over the trigeminal root. The acoustic striae passing across to the left side were also degenerated.

Cat 457.—This was a large lesion which destroyed the entire left superior olive, the facial nucleus and two thirds of the ventral reticular formation on the left side. It also injured the left abducens nucleus and completely severed the trapezoid body. The following degenerations were traced in the sections: (1)

Fig. 5.—Photomicrograph of a section through the olivary region of cat 503, showing the location of the lesion and the degenerated peduncle of the superior olive; × 6. *Op* indicates the olivary peduncle; *tz*, the trapezoid nucleus and *so*, the superior olive, lateral segment.

complete degeneration of the left direct vestibulospinal tract, (2) degeneration of the left rubrospinal tract from the lesion downward, (3) the left reticulospinal tract in the medial longitudinal fasciculus, (4) the right crossed vestibulospinal tract in the left medial longitudinal fasciculus, (5) the ventral spinocerebellar tract above the lesion on the left side, (6) the reticulocerebellar fibers to the floccular lobe of both sides, most abundant on the left side, (7) the trapezoid body from left to right, (8) the acoustic stria across to the right, (9) scattered degenerations in the left descending trigeminal root and (10) the superior olivary peduncles of both sides in their outward course. The one from the right side could not be clearly isolated on account of the extensive degeneration of other fibers dorsal to the lesion on the left side. The one from the left side was followed under the facial genua, across the midline, beneath the right vestibular nucleus and over the descending root of the right trigeminal nerve where it

17. Gray: J. Comp. Neurol. **41**:338, 1926. Papez: J. Comp. Neurol. **41**:389, 1926.

joined the entering root of the right vestibular nerve. In sections through the right vestibular nerve there was a definite bundle of degenerated nerve fibers beyond the border of the medulla oblongata. There was no evidence that the peduncle curved medially to joint the lateral lemniscus, but the degenerated fibers clearly passed out into the vestibular nerve.

CAT 501.—The lesion was a large, oval hemorrhage just cephalad to the left superior olive placed in the nucleus reticularis pontis of Bechterew which lies dorsal to the medial part of the medial lemniscus. There was much peripheral precipitate stained darkly with osmic acid, probably due to a meningitis. Though the sections were unsatisfactory on this account for an accurate study of fiber degenerations, the olivary peduncles showed no specific degenerations. It can be said that a lesion in this region in front of the superior olive does not cause a degeneration of the olivary peduncle.

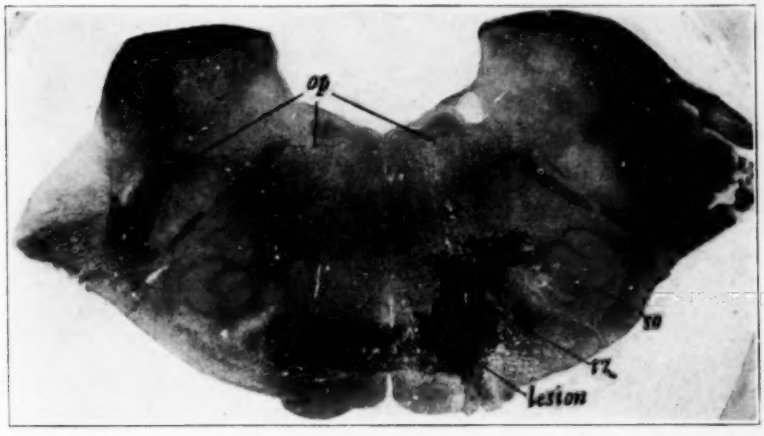

Fig. 6.—Photomicrograph of a section through the olivary region of cat 503, showing the location of the lesion and the degenerated peduncle of the superior olive; × 6. *Op* indicates the olivary peduncle; *tz*, the trapezoid nucleus; *so*, the superior olive, lateral segment.

CAT 327.—The lesion was a large vascular degeneration cephalad to the superior olive in the right half of the pons as already described by Gray [18] and by myself.[18] The superior olive was not injured, and there was no degeneration of the olivary peduncle.

RESULTS

The foregoing specimens were selected to show the results of various olivary and circumolivary lesions. Only the direct olivary lesions test the various hypotheses that concern the connections of the olivary peduncle. The circumolivary lesions have been used for the purpose of showing that the olivary peduncle does not arise outside of the olive. The relations of centers and fiber tracts in this region are shown diagrammatically in figure 3. The main degenerations in the twenty specimens are set down in the accompanying table.

18. Gray: J. Comp. Neurol. **41**:332, 1926. Papez: J. Comp. Neurol. **41**:379, 1926.

The table summarizes the results obtained from lesions in the twenty animals reported herein. In every case in which the lesion was in the lateral segment of the superior olive there was no degeneration of the olivary peduncle. Lesions in the medial segment in every case produced a degeneration of the peduncle. Lesions between the genua of the facial nerves produced a bilateral degeneration of the peduncles. Lesions above and below the olives did not produce the degeneration.

Most of the test animals showed degeneration of the trapezoid body, which was injured by the entering probe. A number of lesions placed on the lateral side of the olive and medial to the cochlear nuclei, as

Summary of the Main Degenerations Observed in the Twenty Specimens Described

Number of Cat	Location of Lesion	Degenerations							
		Olivary Peduncle	Reticulocerebellar	Trapezoid Body	Acoustic Stria	Gowers' Tract	Rubrospinal	Reticulospinal	Vestibulospinal
458	Lateral to superior olive	—	+	+	..	+	+	—	+?
454	Lateral to superior olive	—	..	+	..	+	..	..	+
453	Lateral to superior olive	—	..	+	..	+	+	..	..
456	Caudal end of superior olive	—	..	+	+	—	—	—	—
460	Ventral cochlear nucleus	—	+	+	—	—	..	..	..
328	Lateral segment of superior olive	—	+	+	—	..	+	—	+
329	Lateral segment of superior olive	—	+	+	+	+	+	—	—
362	Lateral segment of superior olive	—	+	+	+	+	+	—	..
503	Medial and dorsal to superior olive	+	+	+	+	—	—	—	—
500	Medial to superior olive	+	—	+	—	—	—	—	—
502	Medial side of superior olive	+	..	+	—	—	—	—	—
504	Ventral and medial to superior olive	+	..	+	—	—	—	+	—
450	Trapezoid nucleus	+	+	+	—	—	—	+	—
360	Between genua of 7th	+	+	+	+	—	+	+	+
373	Between genua of 7th	+	—	—	+	—	—	+	—
363	Lateral to facial genu	—	+	—	+	+	—	+	+
344	Under facial genu	+	..	—	+	—	—	+	+
457	Entire superior olive	+	+	+	+	+	+	+	+
501	Cephalad to superior olive	—	..	+	—	—	—	—	—
327	Cephalad to superior olive	—	—	—	—	+	+	+	—

in cat 362, or in the dorsal part of the reticular formation produced a degeneration of the acoustic stria. The fibers of the stria sprayed out in the reticular formation dorsal to the superior olive and separated into two nearly equal groups. One group turned ventrally and joined the superior surface of the lateral S-shaped segment of the olive on the same side. The other group passed obliquely across the midline to the dorsal surface of the S-shaped segment of the opposite olive. The fibers appeared to send collaterals into the lateral segments of the superior olives. However, they did not stop there but passed upward just dorsal to the olive, and formed the medial fibers of the lateral lemniscus which passed without interruption to the medial side of the inferior colliculus.

Some of the lesions of the olive were very large, thus totally destroying the superior olive and the reticular formation dorsal to it or around it, as in cat 457. In such cases, the trapezoid body and the acoustic stria were completely degenerated. In addition, a small flat band of degenerated fibers was seen passing under the facial genua and out on the other side. This was found to be an extension of the peduncle of the superior olive.

In a number of animals the lesions were located on the side so as to destroy a part or the whole of the lateral S-shaped segment of the superior olive. Only the trapezoid body and the acoustic stria were degenerated. In none of these preparations could a definite degeneration of the olivary peduncle be demonstrated. In other words, a lesion of the large lateral segment did not give rise to dorsally passing degenerated fibers as did the lesions on the medial side. Likewise, the lesions which totally destroyed the facial nucleus or reticular formation cephalad to the superior olive did not produce a degeneration of the superior olivary peduncle. This was true also when the lesion was exceedingly large, as in cat 329, in which there was no injury to the medial half of the reticular formation, to the medial segment of the olive or to the nucleus of the trapezoid body.

When the lesion was placed on the medial side, as in cat 503, so as to damage the medial segment of the olive or the medial nucleus and nucleus of the trapezoid body, degenerated fibers were seen to stream dorsally. They passed through the abducens nuclei and outward on the opposite side. Even when the lesion in this location was rather small, as in cat 504, the fibers of the superior olivary peduncle appeared degenerated. Likewise, a lesion restricted to the nucleus of the trapezoid body produced a total degeneration of this band of fibers, as in cat 450.

The lesions placed to the medial or dorsal side of the superior olive, as in cat 360, produced a degeneration of the outgoing fibers of the olivary peduncle.

A lesion placed between the facial genua from the dorsal side, as in cat 373, produced a double degeneration of these bundles, seen crossing between the facial genua and passing out on both sides.

The structure herein described as the outgoing limb of the olivary peduncle is undoubtedly identical with the structure described by Cajal as the crossed bundle of the vestibular nerve.

The lesions that involved the outgoing root of the abducens nerve, as in cat 503, always caused a loss of the bulbar retraction reflex. When the lesion was placed lateral to the superior olive, so as to compromise the nucleus of the trigeminus, an irritative condition of this reflex occurred, as in cat 360. In such cases the nictitating mem-

brane was drawn over the partly retracted eyeball, and the reflex was sluggish or absent. In some of the specimens degenerated fibers passed from the trigeminal nucleus dorsally and medially parallel and lateral to the pars prima of the facial nerve into the reticular formation lateral to the abducens nucleus. There were indications that these represent a reflex connection between the upper end of the descending trigeminal nucleus and the abducens nucleus. The degeneration of these fibers probably in some cases was responsible for the loss of the bulbar retraction reflex. In other cases it was doubtless attributable to the direct injury of the root filaments of the abducens nerve. Cajal [19] has figured and described fibers arising from the trigeminal nucleus passing dorsally and medially, lateral to the pars prima of the facial nerve and entering the reticular formation in its dorsal portion. He called these fibers the short central trigeminal tracts. I believe that these fibers represent, at least in part, reflex connections between this upper region of the trigeminal nucleus and the nucleus of the abducens nerve, and that they mediate the bulbar retraction or nictitating membrane reflex.

When the ventral reticular nuclei in the reticular formation cephalad to the facial nucleus were injured the reticulocerebellar fibers of van Gehuchten [20] were degenerated, usually most heavily on the side of the lesion. These fibers were traced into the ventral side of the restiform body beneath which they turned laterally to enter the medullary center of the floccular lobe independent of the peduncle of the flocculus. Owing to the obliquity of this lobe in the cat, it was not determined whether the fibers ended in the flocculus or the paraflocculus.

Bechterew thought that the superior olives were connected with the vermis of the cerebellum, and this view is expressed by other workers. It is true that any lesion in the superior olive or lateral to it gave a degenerated strand of fibers which passed into the floccular lobe. But lesions to the medial side and caudal to it which damaged the ventral reticular nuclei gave origin to the same degenerations. The course of these fibers in relation to the caudal, ventral and lateral surfaces of the olive accounts for their degeneration in other cases. From Marchi preparations alone it was difficult to assign a positive origin to these fibers, but the evidence was against the view that they arose in the superior olive.

In all cases in which the olivary peduncle was degenerated by a ventral lesion, the fibers appeared to arise from one of three nuclei; from the medial segment of the olive, from the medial preolivary nucleus or from the nucleus of the trapezoid body. The fibers reached

19. Cajal (footnote 2, p. 869).

20. Van Gehuchten, A.: Le corps restiforme: Fibres reticulo-cérébelleuses ventrales, Névraxe **5**:135, 1904; **4**:40, 1902.

dorsally, passed through the abducens nucleus of the same side. Beneath the facial genua they accumulated into a compact bundle which crossed the midline between the facial genua thus decussating with its fellow of the other side. On the other side, it passed laterally under the vestibular nucleus and curved out over the descending root of the trigeminus and joined the medial surface of the incoming root of the vestibular nerve. It disappeared in the anterior side of the cochlear nucleus between the nucleus and the vestibular nerve.

The precise origin of the olivary peduncle cannot be determined from Marchi preparations. However, in some of the specimens, as in cat 373, in which the outgoing limb of the peduncle was cut there was direct evidence of acute degeneration of the cells of the nucleus of the trapezoid body. After removal of the cochlear nuclei on one side in the rabbit, Winkler [3] observed that the contralateral trapezoid nucleus had undergone atrophy to almost complete extinction. Such a lesion destroyed the outgoing limb of the olivary peduncle, and if the peduncle arises from the contralateral trapezoid nucleus the degeneration of this nucleus in Winkler's rabbit can be explained.

All other fibers from the superior olives appeared to be contributed to the lateral lemniscus and not to the reticular formation or cerebellum.

In not a single case was there any evidence of olivary fibers being contributed to the reticular formation as supposed by Cajal. With this evidence against such a view, the superior olive cannot be looked on as an acoustic reflex center.

SUMMARY

Lesions were made through the base of the skull in and around the superior olives in a number of cats to determine the fiber connections of these organs. After eleven days, the brains were treated by the Marchi method for fiber degenerations and were sectioned. From a study of these sections, the following conclusions are made:

1. The acoustic stria cross through the vestibular nuclei, enter the reticular formation and divide into two fiber streams, one that enters the lateral side of the olivary peduncle of the same side and joins the lateral S-shaped segment, and the other that crosses the raphe forming the decussation of the stria and joins the S-shaped segment of the other side. Both streams continue upward to the inferior colliculi.

2. A lesion of the medial segment of the superior olive or of the adjoining medial preolivary nucleus or the nucleus of the trapezoid body caused a degeneration of the medial fibers of the olivary peduncle that passed dorsally through the abducens nerve across the midline and passed out on the other side by crossing over the descending root of the trigeminus. They appeared to join the vestibular nerve.

3. From these experiments it also appears that no longitudinal fibers are contributed by the superior olive into the reticular formation dorsal to the superior olive such as have been postulated by Cajal. In other words, the olive does not appear to play the rôle of a reflex center.

4. All fibers arising from the superior olive, except the peduncle, appeared to be contributed to the lateral lemniscus.

5. The superior olives did not appear to be connected with the cerebellum as was thought by Bechterew and others.

6. An injury of the ventral reticular nucleus of the medulla oblongata and pons produced a constant degeneration of the reticulocerebellar fibers of van Gehuchten which entered the floccular lobes by passing ventral to the dorsally turning limb of the restiform bodies.

7. An injury to the roots of the abducens nerve produced a loss of the nictitating membrane reflex or bulbar retraction. Likewise, a lesion in the trigeminus nucleus lateral to the outgoing limb of the facial caused a loss of the same reflex.

OPTIC NYSTAGMUS

III. CHARACTERISTICS OF THE SLOW PHASE *

RAYMOND DODGE, PH.D., SC.D.
Professor of Psychology

ROLAND C. TRAVIS, PH.D.
Research Associate

AND

JAMES CHARLES FOX, JR., M.D.
Assistant Clinical Professor of Neurology

NEW HAVEN, CONN.

It is commonly supposed that the speed of the slow phases of normal optic nystagmus varies with the speed of visual objects. The accuracy and limits of that correlation are, however, unknown. As we pointed out in the first paper of this series, the slow phase represents a kind of optic pursuit of moving objects and might be called with propriety the pursuit phase.

For the purposes of this discussion the pursuit may be called adequate when it is sufficiently accurate to permit clear vision. Whenever the line of regard lags behind or overshoots the moving object, vision became more or less blurred and the pursuit may be called inadequate. Determination of the degree of adequacy with which normal eyes pursue at various velocities of the visual object seems important, not only to provide a base line for the study of abnormalities, but also for an understanding of the mechanism of nystagmus. We regard it as one of the fundamental problems in optic nystagmus.

UPPER LIMIT OF ADEQUATE PURSUIT

While a scientific answer to this question makes exacting technical demands, a rough indication of the differences between adequate and inadequate pursuit may be found in the appearance of the environment as one looks from the windows of a train which is traversing a flat terrain. Under such circumstances, doubtless, many of the readers of this report have noticed a band in the middle distance beyond which objects in the landscape are clear and distinct, whereas nearer objects are more or less blurred, while objects in immediate proximity to the train are so completely blurred that they fuse into horizontal, parallel

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* From the Institute of Psychology and the Department of Internal Medicine, Yale University School of Medicine.

lines. The upper limits of adequate ocular pursuit could be computed under these circumstances if one knew the speed of the train and the distance from the observer's eyes to the line of demarcation between the clear and the blurred images.

A clinical instrument to measure the upper limits of adequate pursuit could be designed on the principles involved in this common experience, but neither the crude observations nor the clinical instrument would give a picture of what actually happens to the eye movements when the subject is attempting to follow the closer zones. Instead of subjective judgments of clearness in an accidental field of view, scientific exploration of the upper limits of ocular adjustment requires nystagmographic records of the behavior of the eyes to a predetermined series of known objects, moving at various known velocities.

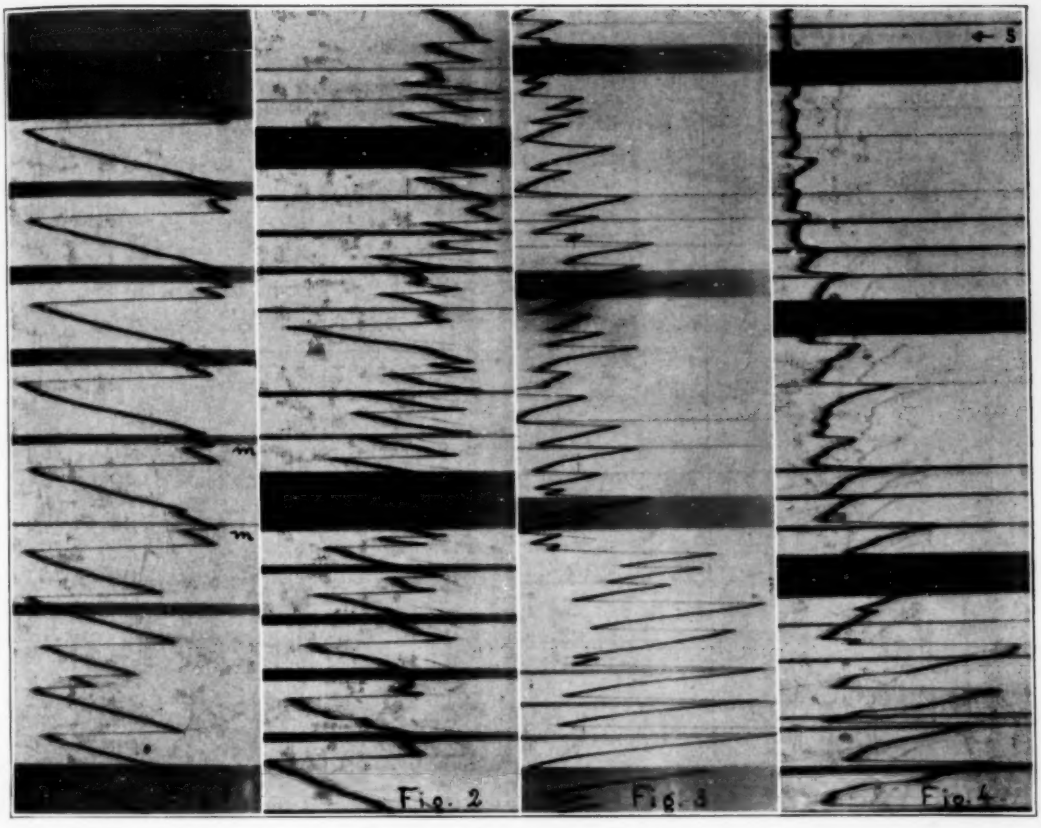
TECHNIC

For the sake of brevity the reader is referred to the description of the nystagmograph given in the first and second papers in this series.[1] The chief modification of the instrument in the present experiments was to substitute for the pictures and letters which were used as visual objects in the previous work twelve vertical black bars approximately 2 degrees in width. Six of these bars were placed at intervals of 20 degrees on the revolving screen, and six at 40 degrees. Furthermore, the screen was rotated by a separate motor permitting gradual variations in its velocity up to 120 degrees a second, while a flashlight signal, operated by a commutator, indicated on the record each 40 degrees of rotation of the screen.

ADEQUATE PURSUIT

Figure 1 is a record of normal optic nystagmus in response to movement of the screen at an approximately constant angular velocity of 35 degrees a second. The first six bars, at 20 degree intervals, are commonly pursued for a distance of approximately 20 degrees instead of for the full 30 degree distance permitted by the aperture in the intervening screen. The pursuit is interrupted by the alternating quick phase which serves to fixate the next oncoming bar. During pursuit of these bars there is no evidence of pursuit of the mesh of the moving screen, indicating the relatively greater stimulus value of the bars whenever they are in the field of view. In response to instructions to watch

1. Dodge, R., and Fox, J. C.: Optic Nystagmus: I. Technical Introduction with Observations in a Case with Central Scotoma in the Right Eye and External Rectus Palsy in the Left Eye, Arch. Neurol. & Psychiat. **20**:812 (Oct.) 1928; Fox, J. C., and Dodge, R.: Optic Nystagmus: II. Variations in Nystagmographic Records of Eye Movement, ibid. **22**:55 (July) 1929.



Figs. 1-4.—Sections from nystagmographic records of four normal subjects, each record representing 10.5 seconds. The records should be read from below upward, the heavier oblique lines corresponding to the slow pursuit phases, the fainter almost horizontal lines corresponding to the quick phases. The coarse horizontal lines occur at each 40 degrees of rotation of the screen. Poor contact makes some of them faint, with occasional misses. In figures 1 and 2 the bars were moving in a direction from the subject's left to right, whereas in figures 3 and 4 they were moving from his right to left. The actual photographic record of each eye movement is reversed in direction, because of the fact that the beam of light from the mirror recorder moves opposite to the direction of eye movement. The left eye is recording in each case; the subject actually watches the bars with his right eye only. In figure 1 (subject T), the angular velocity of the screen was constant—35 degrees a second. Adequate pursuit of each of the twelve bars, the first six at 20 degree intervals, the second six at 40 degree intervals, is readily distinguished. Optic nystagmus of small amplitude, such as occurred at places marked *m*, was a response to the mesh of the moving screen. In figure 2 (subject H), there was gradual acceleration of the angular velocity of the screen to 80 degrees a second during two revolutions of the screen, after previous gradual acceleration to 40 degrees per second at the beginning of the record. The pursuit pattern is irregular. In figure 3 (subject F), there was acceleration of angular velocity to 120 degrees a second during three revolutions of the screen, after previous gradual acceleration to 90 degrees a second at the beginning of the record. The pursuit pattern becomes increasingly irregular, bordering on complete inadequacy. In figure 4 (subject G) there was fairly constant angular velocity of 110 degrees a second during three revolutions of the screen, after previous gradual acceleration to 90 degrees a second at the beginning of the record. The pursuit pattern is entirely inadequate, with complete breakdown at the point marked *s*.

each bar, the eye pursues the other six bars at 40 degree intervals through the entire screen aperture, the maximal pursuit permitted by the experimental conditions. Since the field of view is limited by an aperture of 30 degrees, there is a brief interval between each of the six more widely separated bars when no bars are visible. During this 10 degree interval, the record shows optic nystagmus of small amplitude, presumptively in response to the mesh of the moving screen background. This mesh pursuit regularly appears in that section of the records in which oncoming but unseen bars are expected (m). The emergence of each bar from behind the edge of the screen is shown in the record by the abrupt transition from the short mesh pursuit to a long pursuit of a bar.

For eight normal subjects angular velocities of from 30 to 40 degrees a second regularly permitted adequate pursuit. This closely corresponds with the optimal speed of rotation, as found by Fox and Holmes [2] in their clinical study of optic nystagmus, although they used a drum whose surface moved 34 cm. per second at a distance of 60 cm. from the patient's eyes (33 degrees a second). The nystagmographic pattern under these conditions is fairly uniform and characteristic as already described. Two subjects showed consistent differences between the adequacy of nasalward and temporalward pursuit, whether as a result of the influence of the blind spot or of neuromuscular or other factors has not yet been determined.

No angular velocity used in our experiments was too slow to evoke adequate pursuit. This observation is confirmed by the oscillation experiments, which are described later in this paper. With a gradual acceleration of the angular velocity of the visual objects, the nystagmographic pattern changes its character in the following particulars, as shown in figure 2: (*a*) The contrast between the longer pursuits of the bars at 40 degrees and the shorter pursuits of the bars at 20 degrees becomes less marked; (*b*) the nystagmus evoked by the mesh becomes less apparent; (*c*) the pursuit movements do not begin at the point where the oncoming object appears through the aperture but at some later point in its course. These changes were found to vary considerably in different subjects at similar angular velocities. They represent the transition from adequate to inadequate pursuit.

INADEQUATE PURSUIT

With still further increase in angular velocity, i. e., beyond 90 degrees per second, the situation becomes increasingly more difficult for the normal subject and definite features of inadequacy of adjustment appear (figs. 3 and 4). These may be enumerated as follows:

2. Fox, J. C., and Holmes, G.: Optic Nystagmus and Its Value in the Localization of Cerebral Lesions, Brain **49**:333 (Sept.) 1926.

1. *Shortening of Pursuit.*—Instead of the previous 20 to 30 degree pursuit evoked at the slower speeds there is a progressive shortening of the slow phases. Within our experience this decrease in amplitude is a universal phenomenon of inadequate pursuit at high velocities of the visual object.

2. *Slowing of Pursuit.*—Whereas the accelerating velocity of the visual objects might be expected to evoke more and more rapid ocular pursuit, the angular velocity of the pursuit not only fails to correspond to that of the objects but actually decreases. This is shown by greater obliquity of the pursuit lines in figure 4. Optically this must mean that the visual images of the bars are blurred throughout their course in the field of view. Introspection tends to support this interpretation, although the subjective impressions of blurred and clear images are not reliable.

3. *Irregularity of Pursuit.*—This phenomenon is obvious if one compares figure 1 with the others. At an angular velocity of 35 degrees per second the slopes are approximately parallel; at velocities from 80 to 120 degrees per second, successive slopes are often far from parallel. It will be noted that the irregularity applies to amplitude as well as velocity, though an occasional adequate pursuit is discoverable.

Hypothesis.—One conjectures that this irregularity of ocular pursuit at high angular velocities is a prototype of those vagaries of reaction that occur mixed with adequate response when any objective situation becomes too difficult for adequate adjustment.

4. *Elision of Major Pursuits.*—During a time interval represented by one complete revolution of the screen at the higher velocities, one or more bars may pass without pursuit fixation. Consequently there may be eleven and in some records as few as five pursuit phases instead of the twelve that appear during adequate adjustment. These elisions commonly occur first in the series of bars at 20 degree intervals, indicating the direct influence of stimulus frequency on the inadequacy of response.

5. *Failure of Pursuit.*—Complete disappearance of the slow phase occurs occasionally at angular velocities above 100 degrees a second; during these periods, the eyes are approximately still, instead of attempting pursuit (fig. 4,s). This is characteristic of the majority of subjects but does not appear universally at any given speed. Most records show a tendency toward the repetition of a partial though imperfect pursuit response rather than the complete abandonment of the kind of reaction that is persistently inadequate.

Hypothesis.—One conjectures that this type of defective pursuit adjustment, characterized by repetition of inadequate response with occasional abandonment, is a prototype of the reaction shown by the normal individual to a too rapidly changing environment. At present one can

only guess what kind of pursuit adjustment might be shown by a patient whose history revealed an inability to respond to rapid environmental changes, but the presumption is in favor of the early abandonment of pursuit.

Comparative study of the records indicates that there is considerable variation between the subjects in the incidence and completeness of the described inadequacies of pursuit at the higher angular velocities of the visual objects (compare figs. 3 and 4). There are also certain individual peculiarities of response to the higher speeds, such as excessive winking and transient fixation on the sides of the aperture.

Emphasis, however, should be placed on the fact that the higher angular velocities do not evoke ocular disorders which are supposedly of neuromuscular origin, such as "overshooting" of the quick phase. Moreover, as the velocity increases beyond a certain limit, the inadequate pursuit phases actually show a smaller number of corrective refixations in the direction of pursuit than at medium speeds.

Thus far our description of the modification of pursuit by increasing the angular velocity of the visual objects has been largely qualitative. The chief barrier to a quantitative statement of these changes in optic nystagmus is the difficulty of finding a satisfactory common measure of their magnitude. For example, the amplitude of any given slow phase depends not only on adequacy of pursuit, but also on factors of relative interest. The pursuit phase may be terminated at any instant by a quick phase in the opposite direction, evoked by an oncoming object competing for fixation. The quick phase in such instances is not a corrective refixation but rather the fixation of a new object of interest.

In view of this fact it seemed desirable to isolate the pursuit phase from the quick phase of optic nystagmus and to study it in its simplest form. Such an experimental situation seemed to be provided by the pursuit fixation of a simple object oscillating in harmonic motion on either side of the primary position of the eye. To this end we constructed a new instrument by the aid of which experiments were undertaken to determine quantitatively the upper limits of pursuit of an accelerated object and to describe in quantitative terms the phenomena of inadequate pursuit.

ESSENTIALS OF AN INSTRUMENT FOR RECORDING THE VISUAL PURSUIT MOVEMENTS OF THE EYES FOLLOWING A SINGLE OBJECT

The object of protracted visual pursuit was an illuminated upright slit in a vertical screen on the moving end of a long inverted pendulum. It was oscillated right and left in a plane perpendicular to the primary line of regard, across the distal end of a dark tube which was 75 cm. long and about 15 cm. square in cross section. A horizontal offset at the midpoint of the vertical line determined the plane of pursuit movements. The pendulum was driven in harmonic motion by a

rod attached eccentrically to a motor driven pully. The observer looked through the dark tube with the open right eye, while the conjugate movements of the mechanically closed left eye were recorded by a mirror recorder. A beam of light was reflected from the concave mirror of the recorder which rested on the closed lid tangential to the cornea, and fell on vertically moving sensitive paper through a horizontal slit in a stationary recording camera.

A lever to record the movements of the object was attached to the inverted pendulum and cast a double shadow across the camera slit. This double shadow appears in the records as double white lines, whereas the curves representing the eye movements are black. Clockwise movements of both the eyes and the pendulum produced counterclockwise excursions in both recording lines. The records of both eye and object are consequently directly comparable as to time and direction. Their respective amplitudes, however, are not directly comparable, since they are mechanically conditioned by unrelated physical constants of the recording lever and the recording eye. Time in tenths of a second was recorded by an electric marker in series with a calibrated vibrating reed. The observer was comfortably seated and was stabilized by supports for the back and head. The head was further held by an adjustable mouth rest.

The inverted pendulum carrying the object of regard was oscillated at various speeds, including those which proved to be too rapid for adequate pursuit. The amplitude of oscillation in all cases was 6 degrees of the visual field at a distance of approximately 75 cm. from the eye. This angular excursion was determined by two practical considerations. On the one hand, it was important to exclude such obliquity of the object line as would occur at greater amplitudes of pendular oscillation. On the other hand, the arc of movement must produce eye movement records of sufficient amplitude to be accurately measured. Magnification of the record depended on the distance of the camera from the eye. In all these experiments, this distance was arbitrarily fixed at 60 cm.

Ocular pendular pursuit records are reproduced as figures 5 to 7. The first two of these were taken from the same subject at different frequencies of oscillation, as the respective records show. The third, figure 7, was selected from the records of that subject who showed the most pronounced after-effects.

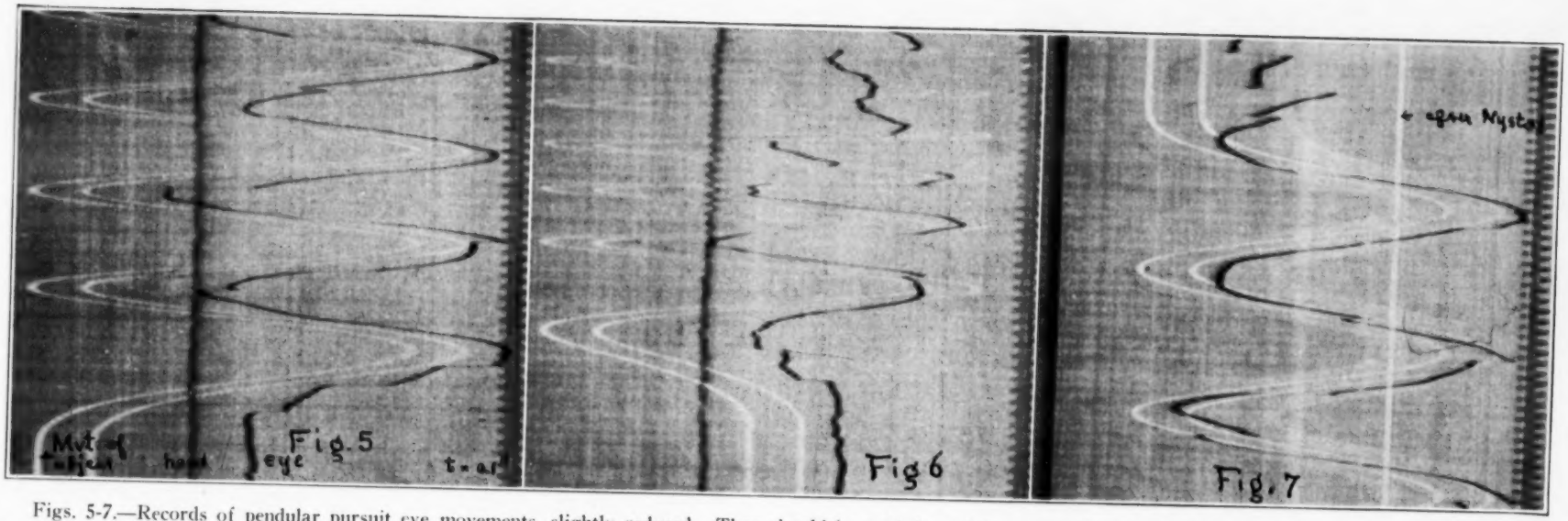
COMMENT ON RECORDS 5, 6 AND 7

The records of visual pursuit of a suddenly moving object regularly begin with a more or less protracted latency, a kind of neuromuscular inertia before the eye gets started after the object begins to move. This latency is apparent in the records as an approximately straight horizontal fixation line continuing upward after the object moves (lower end of fig. 5). It averaged about 0.19 second in duration for all eight subjects and all speeds, and is of the same order as was previously found by Dodge,[3] Diefendorf and Dodge [4] and Dodge and Benedict.[5] It tends to decrease with practice.

3. Dodge, R.: The Reaction Time of the Eye. Psychol. Rev. **6**:477, 1899.

4. Diefendorf, A. R., and Dodge, R.: An Experimental Study of the Ocular Reactions of the Insane from Photographic Records, Brain **31**:451, 1908.

5. Dodge and Benedict: Psychological Effects of Alcohol, Carnegie Institution of Washington, number 232, 1915, p. 75.



Figs. 5-7.—Records of pendular pursuit eye movements, slightly reduced. They should be read from below upward. The double white lines show the phases of harmonic motion of the stimulus object; the three dark lines are records respectively of time, head movement and visual pursuit. Time is shown at the right of each record in tenths of a second. Head movements are shown in figures 5 and 6 by the approximately straight vertical lines near the center of the figures and by the dark line at the extreme left in figure 7. Movements of the eyes to right and left are shown by oblique excursions of the eye line record to the left and right, respectively. Still fixation of the eyes appears in the records as approximately straight vertical lines. Both pursuit and still fixation records are commonly complicated by pulse waves, tremors and rapid corrective movements. Quick refixations and corrective eye movements appear as faint approximately horizontal lines. Adequate pursuit appears as oblique lines approximately in phase with the white record lines of the object.

The first eye movement after movement of the object may be either a quick refixation movement or a drift (fig. 5). The former is evoked by a lapse of central fixation which occurs when the fixated object starts oscillation and is shown by a fine almost horizontal excursion of the record line. This is commonly followed in normal subjects by an approximate pursuit. Preliminary drifts, when they occur, appear as short oblique lines. They are often clearly related to irregularities of still fixation but are not yet clearly understood. The angular velocity of the first real pursuit movement commonly corresponds in velocity to the beginning of objective movement rather than to the contemporaneous speed of the object. The beginning of pursuit is shown in the records by a darker more or less oblique line which emerges out of the first quick movement. Approximately adequate pursuit commonly appears first on the second or return phase of the oscillation of the object and is shown by the approximately smooth sine-wave curves in phase with the record of the movement of the object. It is notable that patterns of even practiced pursuit do not arise immediately when objective motion begins, but may and commonly do show a period of development such as we have just described. The conditions and course of that development are significant both for a theory of optic nystagmus and for the interpretation of abnormalities. Approximately adequate pursuit, however, regularly shows more or less numerous rapid refixation or quick corrective eye movements. They appear in the records as breaks in the otherwise smooth pursuit curves. Usually, but not universally, these quick eye movements occur in the direction of pursuit and are more numerous during oscillation at medium frequencies. Corrective eye movements also occur sometimes during the slower angular movements of the object at the turn of the direction of its oscillation. They may correct overshooting and in that case be in a direction opposite to that of the preceding pursuit movement, or they may correct an anticipatory reversal and in that case be in the same direction as the preceding movement of pursuit.

As the frequency of oscillation increases with a corresponding increase in the maximal angular velocity of the object, pursuit becomes less and less adequate in the following respect: The amplitude of pure pursuit movement becomes smaller until it finally approximates still fixation. This is shown on the records by the small amplitude of those pursuit movements which are in phase with the movements of the object either with or without increase of the fine horizontal lines which show rapid corrective eye movements. Total breakdown of pursuit (upper end of fig. 6) is recorded as an approximately straight vertical line characteristic of still fixation, in place of the oblique lines in phase with the oscillation of the object. These changes in pursuit of an oscillating object, as its angular velocity increases, are essentially the same as the

changes in the slow phase of nystagmus which were found under analogous circumstances when a screen was rotated in the visual field, except that the pursuit of the single oscillating object is not complicated by the fixation of new objects of interest All quick movements in this case may consequently be interpreted as indicating corrections of pursuit fixation.

In figure 5, at the frequency of the one complete oscillation of the object in 0.8 second, the eye followed the moving object quite well after the first half oscillation. Figure 6 shows the pursuit movements of the eye when each oscillation gradually accelerated to one oscillation in 0.38 second. In this record the quick corrective movements become longer at the midspeeds and practically disappear at higher speeds. At the final highest speed, pursuit eye movements broke down into approximately still fixation. This breakdown also duplicates phenomena found in the pursuit phases of optic nystagmus at the higher angular velocities.

There was no consistent indication in the records of difference in ability to pursue from left to right and from right to left. Rapid corrective movements seemed to occur in pursuit in one direction as often as in the other and the amplitude of eye movement remained approximately the same for pursuit in the two directions for six of the eight subjects. The two exceptions also showed differences in the two directions of optic nystagmus.

Figure 7 reproduces the end of a record of approximately adequate pursuit at a frequency of one oscillation in 1.4 seconds. The important feature in this record is the continuation of optic nystagmus after the object stopped oscillating. This phenomenon has importance as an indicator of the neuromuscular elaboration of ocular pursuit.

THE COURSE OF PURSUIT ADJUSTMENT

The pattern of the course of adjustment, indicated in our records, is first a phase of rough approximations in which response follows the adequate stimulus after a considerable delay corresponding in duration to cortical reaction times or latencies. In this first phase, the corrective movements are gross and pursuit is quite inadequate. This was followed by a return phase which showed much closer approximation to adequate pursuit with finer and relatively infrequent corrections. Developed adjustment at this stage seemed to indicate a preelaboration of response which often anticipated its normal stimulus and persisted after oscillation of the object stopped. As the frequency of oscillation became greater and the optical situation more difficult, corrective eye movements constituted the major part of the response, only to disappear entirely at still greater speeds. In protracted stimulation at this level of difficulty, the amplitude of pursuit became very small and pursuit broke down into approximate still fixation of the eyes.

EXPERIMENTAL DATA

Quantitative expression of these changes in the adequacy of pursuit in reaction to various frequencies of oscillation of a single object is given for several subjects in the accompanying table. Graphic expres-

Characteristics of Eye Movements in Pursuit of an Oscillating Object with a Displacement of Six Degrees.

Time of One Complete Oscillation, Seconds	Average Total Amplitude of Eye Movement, Mm.	Average Amplitude of Pure Pursuit, Mm.	Average Amplitude of Quick Movements, Mm.	Average Number of Quick Movements	Average Number of Half Oscillations	Average Pursuit Latency, Seconds
Subject C.						
3.7	36	35	1	5	15	0.16
1.5	36	32	4	21	20	0.16
0.9	35	29	6	20	19	0.16
0.5	23	19	4	11	21	0.17
0.38	10	7	3	6	20	0.16
Subject T.						
3.1	29	28	1	1	15	0.16
1.5	29	28	1	6	18	0.16
0.9	28	25	3	10	19	0.19
0.5	28	21	7	10	19	0.14
0.38	14	12	2	5	19	0.16
Subject Hil.						
3.7	39	38	1	2	12	0.24
1.4	37	34	3	10	19	0.18
0.9	31	27	4	6	15	0.16
0.5	25	20	5	7	20	0.15
0.38	8	6	2	4	20	0.12
Subject F.						
3.8	34	33	1	4	10	0.10
1.5	34	31	3	20	19	0.25
0.9	30	23	7	25	20	0.16
0.5	15	10	5	12	20	0.20
0.4	6	4	2	5	16	0.14
Subject Han.						
4.0	45	43	2	16	15	0.19
1.5	44	39	5	19	18	0.18
0.9	44	34	10	15	14	0.17
0.6	37	29	8	10	17	0.16
0.4	14	12	2	4	20	0.23
Subject RD.						
3.6	36	33	3	14	11	0.26
1.5	36	33	3	12	17	0.27
0.9	32	20	12	18	17	0.26
0.6	20	8	12	23	28	0.23
0.4	9	2	7	8	20	0.17
Subject M.						
3.4	27	26	1	5	12	0.14
1.4	26	24	2	15	20	0.22
0.9	27	22	5	25	20	0.28
0.5	11	8	3	7	20	0.20
0.36	5	3	2	4	20	0.12
Subject G.						
5.2	37	36	1	2	8	0.26
1.4	29	25	4	18	18	0.32
0.7	16	13	3	7	20	0.15
0.6	9	7	2	7	20	0.29

sion of the extent of pure pursuit for all subjects is given as distributions of amplitude of eye movement in millimeters at various frequencies of oscillation in figure 8.

COMMENT ON TABLE

The amplitude of oscillation of the object was kept constant at 6 degrees for all subjects. The time for one complete oscillation for each subject was varied systematically from 3 or 4 seconds to 0.36

second, as shown in column 1, although these speeds varied slightly from subject to subject. During each complete oscillation, each phase of 6 degree amplitude was composed of 3 degrees positive acceleration and 3 degrees negative acceleration. Thus the object actually moved 12 degrees for each double oscillation with four phases of acceleration. The maximum angular velocity of the object occurred twice, once in each phase of oscillation. It varied from 4.7 degrees a second during one double oscillation in 4 seconds to 52.4 degrees per second during one complete oscillation in 0.36 second.

The average total amplitude of eye movement decreased as the speed of oscillation of the object increased, as shown in column 2. For all subjects there was a sudden decrease in amplitude as the time of oscillation reached a value less than 0.5 second. While there is a considerable difference in the amount of this decrease, all subjects showed the same general tendency. The variation in the maximum extent of eye movements at the slow speeds is due, in part at least, to differences in the physical characteristics of the eyeball from subject to subject.

Column 3 gives the average amplitude of pure pursuit, exclusive of quick corrective movements. This is probably the best single measure of pursuit adequacy. It shows a more consistent decrease with increasing speeds than the total amplitudes in column 2. The amplitudes of pure pursuits, whose averages are given in column 3, are also shown as surfaces of distribution for all subjects and for all speeds in figure 8.

The average amplitude and the number of quick corrective movements are given in columns 4 and 5. The number of half oscillations of the object for each speed is given in column 6. Columns 5 and 6 should be compared with each other to get the relative frequency of the rapid corrective movements. In general, one may say that as the frequency of oscillation increased from that which permitted adequate pursuit to that which evoked inadequate pursuit, the number and amplitude of the quick corrective movements first increased, then decreased, as the speed approached the point of more or less complete breakdown. A further inspection of columns 4, 5 and 6 will show a positive correlation between the number and the amplitude of the rapid corrective movements. On the whole, the greatest relative number of rapid corrective movements occurred at the same speed at which they also showed the greatest amplitude.

The average pursuit latencies for the various frequencies are given in column 7. For most of the subjects there is considerable variation from frequency to frequency, but this variation is not consistent from subject to subject. On the whole, there seems to be very little correlation between pursuit latency and speed of oscillation.

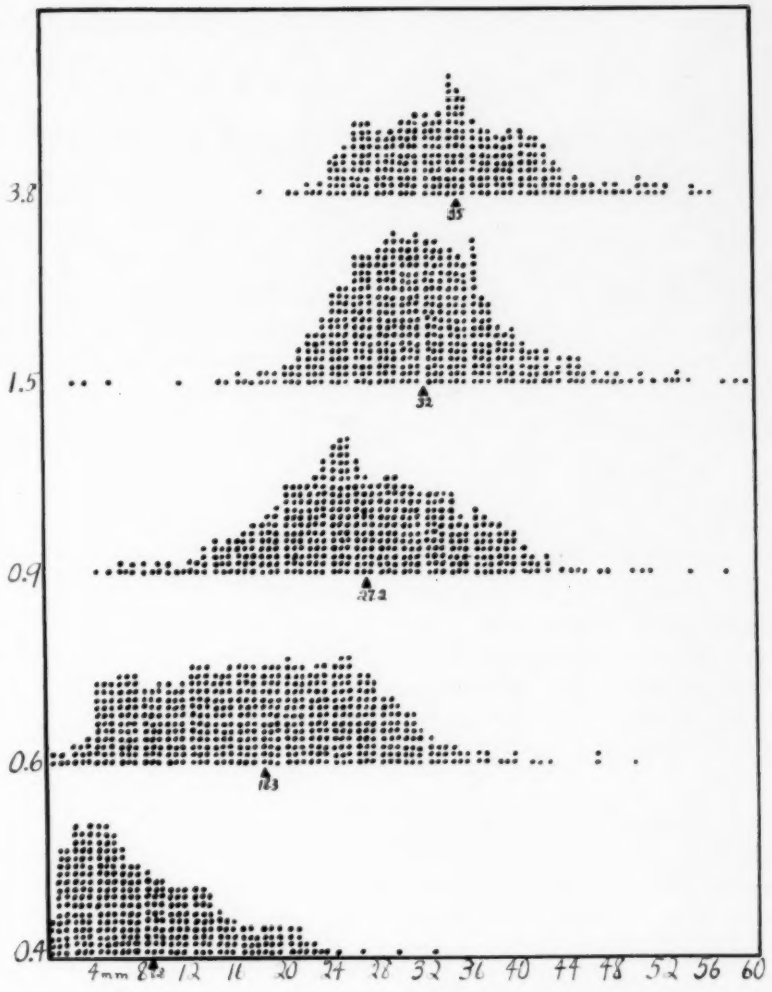


Fig. 8.—The extent of pure pursuit phases at various speeds of oscillation of a single object in the form of distribution areas. Speed is shown at the left in seconds per oscillation. The extent of pure pursuit is represented on the abscissae in millimeters, as indicated by the numerals at the bottom. The number of pure pursuit phases of each indicated extent is shown for each indicated speed of oscillation by the number of dots in areas above the corresponding numerals. For example, at the highest speed used, one oscillation in 0.4 second, shown in the lowest level, the greatest number of records showed short pursuit excursions about 4 mm. in amplitude. There were fewer excursions around 8 mm., and progressively fewer around 12, 16, 20 and 24 mm. There was only one pure pursuit with an amplitude between 28 and 32 and one between 32 and 36. This last value approximated adequate pursuit.

The spread of all distributions is somewhat exaggerated by the fact that the data from all settings of the records and all subjects are combined in one chart. While this gives a distorted picture at each speed level, comparison between the speed levels should not be adversely affected.

The general rule is clear: Reading from above downward, as the speed of oscillation increases, the extent of pure pursuit decreases and the pursuit phase becomes less adequate. The averages at each level, indicated by arrow heads below the several distribution areas, reveal that this relationship follows a regular curve, which is obviously approaching its limit at the upper end and zero at the lower.

To conserve space, the distributions of pure pursuit amplitudes for all subjects were pooled. They are given in figure 8. The variability in each distribution is due in some measure to individual differences from subject to subject. Most of the subjects, however, showed variability to about one-half the extent manifested in the figure. The variability was less for the slower and faster speeds than for intermediate speeds. A curve may be drawn through the arithmetic means which are indicated below the base line of each distribution. It represents a fairly regular curve showing the fundamental tendency of pursuit to decrease with increasing speed of objects.

SUMMARY

1. The slow phases of optic nystagmus are essentially eye movements of pursuit. They are termed adequate when the point of regard consistently falls on one of the moving objects of interest; inadequate when the point of regard overshoots or lags behind the visual object.

2. Angular velocities below 40 degrees per second evoke approximately adequate pursuit movements as shown in the fairly uniform photographic records of the eye movements obtained from eight normal subjects. With gradual increase in the angular velocity the nystagmographic pattern gradually becomes less regular.

3. At angular velocities above 90 degrees per second, records from the average normal subject show the following features of inadequacy in the pursuit movements: shortening, slowing, irregularity, elision and final complete failure of pursuit.

4. Quantitative data on the relation of speed to inadequate pursuit were sought with a new instrument that recorded the eye movements in pursuit of an oscillating single object.

5. With increasing speed of oscillation the rapid corrective eye movements first increase and then decrease in both number and amplitude, whereas the pursuit movements steadily decrease in amplitude, occasionally reaching still fixation.

6. The most consistent measure of adequacy is the amplitude of pure pursuit, which is approximately inversely proportional to the speed of the object.

THE PRINCIPAL SINISTRAL TYPES

AN EXPERIMENTAL STUDY PARTICULARLY AS REGARDS THEIR RELATION TO THE SO-CALLED CONSTITUTIONAL PSYCHOPATHIC STATES *

CLARENCE QUINAN, M.D.
SAN FRANCISCO

Although the term "sinistrality" usually suggests merely the idea of left-handedness, it is collective in its significance and embraces various manifestations obviously due to functional predominance on the part of the right cerebral hemisphere. Here it is used, in a somewhat restricted sense, in connection with certain paired activities of the upper extremities and the eyes, sometimes spoken of as handedness and eyedness. Remarkable though they are, these coordinated activities of the hands and eyes seem to have attracted comparatively little attention. Clinically, they are easy to recognize. There are four principal forms, two of them being symmetric and two asymmetric. The two symmetric or homonymous forms are best known. They were defined first, I believe, nearly fifty years ago, by Prof. Joseph Le Conte.[1] Since his description of these peculiarities will serve well for the present purpose, I shall quote his words. Under the caption "Double Images," he said:

> I have found that nearly all persons neglect the right-hand image—i. e., the image belonging to the left eye. In other words, they are right-eyed as well as right-handed. I have also tried the same experiment on several left-handed persons, and have found that these neglected the left image—i. e., the image belonging to the right eye. In other words, they were left-eyed as well as left-handed.

If, for the sake of brevity, the two kinds of persons thus described are designated, respectively, as RR's and LL's, the first letter being that of the preferred hand, the second, that of the dominant eye, then, in like manner, by means of the respective formulas RL and LR, one can indicate the corresponding peculiarities characteristic of those persons who constitute the two remaining asymmetric groups.

Little seems to be known as to the relative importance of these four groups in point of size, though it is generally conceded that from 4 to 6 per cent of all people are left-handed.

Having employed sinistrality tests in private practice routinely for over six years, I have been struck by the fact that sinistrals differ from dextrals in certain qualitative psychologic respects; that is, they seem in

* Submitted for publication, July 6, 1929.

1. Le Conte, Joseph: Sight: An Exposition of the Principles of Monocular and Binocular Vision, New York, D. Appleton & Company, 1881, p. 94.

many ways to be more sensitive and also, temperamentally, more idealistic. Often, along with these characteristics, one notes various evidences of constitutional instability; these take the form of bizarre disturbances of function of one sort or another, together with signs that point to a low threshold value as regards painful impressions.

Recently, in meditating on such facts as I had been able to gather with respect to the clinical significance of visuomanual symmetry and asymmetry, it occurred to me that further light might be thrown on this rather obscure problem by means of a comparative study of selected groups of persons, the central idea being that sinistrals, supposing them to be more delicately constituted in a psychologic sense than dextrals, would gravitate naturally into certain vocations. I had some reason to suspect that they might show such a trend because, in a previous study [2] of musicians and mechanics I had found a much larger percentage of sinistrals in the former class. It seemed also as though something might be learned by collecting sinistrality data among persons suffering from various forms of mental disease.

The work reported here had therefore three objectives: To note the relative numbers of dextrals and sinistrals to be found (1) in control groups of normal people, (2) among persons engaged in certain vocations and (3) among persons with mental diseases.

Before discussing the general results obtained in this research, I shall first describe the experimental technic employed and give some additional details as to the various types of handedness and eyedness noted in the course of the work.

METHOD OF TESTING

In order that dependable results may be obtained, it is important that the persons about to be tested be kept in a state of complete ignorance as to the real purpose of the experiments. It should be understood, however, that this precautionary measure has no object other than that of minimizing the tendency on the part of any subject to exercise conscious control over the reactions he exhibits in response to the tests. The examiner soon learns by experience that it is easy to satisfy the curiosity of the people with whom he plans to work. For example, he can readily obtain the cooperation of a group of persons merely by asking them to take part in a few simple "coordination experiments"; or he can say to them, by way of explanation, that the object he has in view is to obtain statistical data as to certain normal peculiarities of vision. No reference whatever need be made to the subject of right-handedness and left-handedness; it has been my experience that one rarely has to answer questions on this score.

Since observations as to handedness and eyedness, particularly when large groups of people are concerned, often have to be made under conditions of more or less hurry and bustle, it is a matter of some importance to conserve time. For this reason, it is a great advantage to employ a working method that combines

2. Quinan, C.: A Study of Sinistrality and Muscle Coordination in Musicians, Iron-Workers and Others, Arch. Neurol. & Psychiat. **7**:352 (March) 1922.

simplicity with a fair degree of accuracy. Many experiments in this direction led finally to the adoption of certain pantomimic tests. These tests, which are accurate enough for ordinary clinical purposes, are based on the principle of functional dominance. For example, the question as to the handedness of any given person is decided by noting with which arm he executes the primitive act of throwing.

It should be understood, therefore, that in the tests about to be described each person examined automatically registers his own handedness and eyedness simply by the use he makes of two test objects—a soft rubber ball and a wooden pistol with a barrel 9 inches long, provided with sights about ¾ inch high.

The method of procedure is as follows: A table having been placed before the examiner at a distance of 10 feet or more with the test objects laid on it, each person in the group under observation is requested: (1) to seize the ball and go through the motion of throwing it with great force; (2) to take up the pistol, hold it at arm's length, and then, while standing squarely face to face with him and with both eyes open, to point it at the examiner's nose, and to do this first with one hand and then with the other.

All of the data reported in this paper were obtained by means of these simple tests, though in some instances the experiments had to be modified slightly to meet special situations. These pantomimic tests may be used to advantage among normal white persons, and they are ideal for the purpose of securing sinistrality data among Chinese school children; on the other hand, I found, when working among persons with mental diseases, that it was sometimes necessary to question them individually with respect to handedness.

It may be as well to state here, as regards selection, that in collecting the materials on which this report is based, every care was exercised to note down only such results as were obtained from persons who believed themselves to possess fairly normal vision in both eyes. However, for obvious reasons there may be small plus or minus errors in the results obtained among the Chinese school children and also in those obtained among persons with mental diseases.

TYPES OF HANDEDNESS AND EYEDNESS

Although most persons are either wholly right-handed or wholly left-handed, as the case may be, it is also true that in any large group of people one usually finds a few strongly right-handed persons—seemingly, less than 5 per cent of the whole number examined—who use the left hand habitually for some particular purpose; for instance, they perhaps deal cards with this hand or wield a knife or some other implement with it. In classifying such cases, I accept as decisive the evidence yielded by the throwing test. These mixed varieties of handedness are very puzzling. Possibly in some instances the cause of a variant type of this sort may be traced to a change of handedness effected in early life. My own observations on this subject seem to indicate, however, that a peculiarity of this sort is usually congenital.

In similar fashion, some persons exhibit ambilaterality as regards the use of the eyes in sighting; that is to say, they appear to have no

master eye. That this is so is shown by the fact that they invariably sight with the right eye when the pistol is held in the right hand or, if the pistol is held in the left hand they sight with the left eye. Most of these ambilaterals are strongly right-handed. There is, however, a left-handed type, but it seems to be rare. At any rate, I found only a single person of that description among 1,000 university students.

There remains to be spoken of another class of persons who cannot be classified definitely as to eyedness. Now and again one meets with a person who responds to the pointing test in such an uncertain manner that the examiner may find himself unable to decide which eye is being used for sighting purposes. Commonly, when such a person points with the pistol it will be seen that he holds it in such a way that its

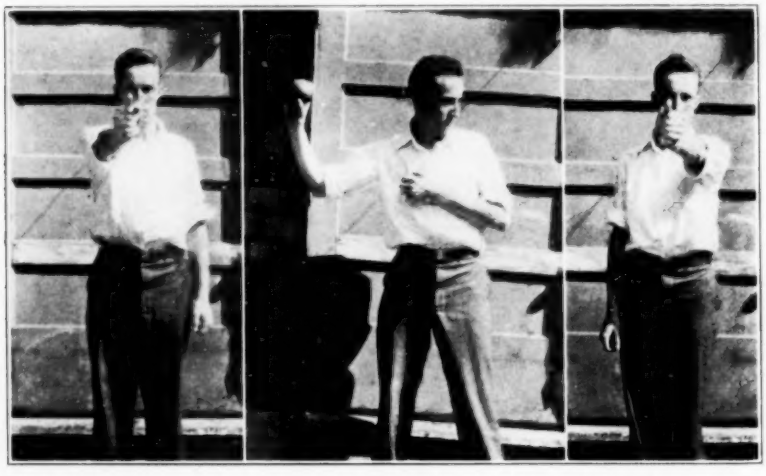


Fig. 1.—Symmetric dextral—right-handed and right-eyed (RR). Note that he throws with the right hand and sights only with the right eye.

barrel is nearly in the median line between the eyes, and that the front sight then wavers uncertainly, swinging now to one eye, now to the other.

Collectively, the persons who make up the two preceding classes are few. In the tables that accompany this article they are recorded together under the heading "ambilaterals and other types." In calculating percentages, however, they were counted as dextrals. Just what these aberrant forms of eyedness signify is problematic; perhaps, however, for want of a better explanation, each one of them may be regarded simply as standing in the relation of a transitional phase to some one or other of the four principal types of handedness and eyedness. Typical examples of the latter strongly-marked clinical varieties are given in the photographic illustrations, figures 1, 2, 3 and 4, the two

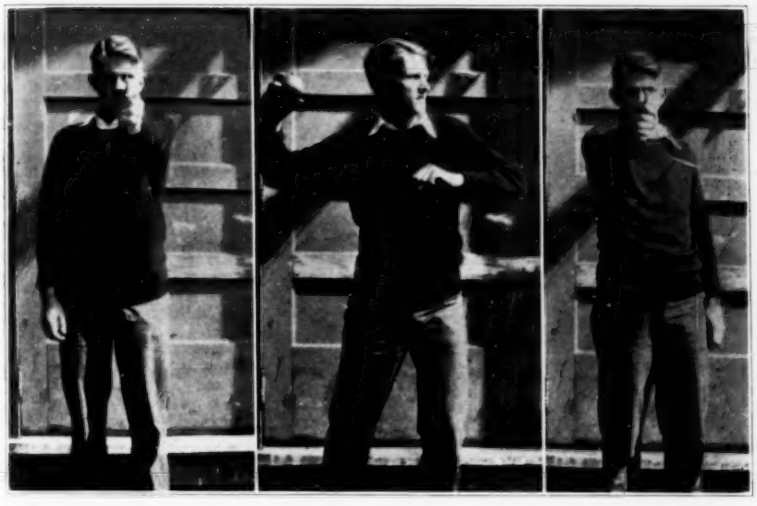


Fig. 2.—Asymmetric or crosswired dextral—right-handed and left-eyed (RL). Note that he throws with the right hand and sights only with the left eye.

Fig. 3.—Symmetric sinistral—left-handed and left-eyed (LL). Note that he throws with left hand and sights only with the left eye.

symmetric types being shown in figures 1 and 3, the two asymmetric in figures 2 and 4.

EXPERIMENTAL RESULTS

Relative Numbers of Dextrals and Sinistrals in Control Groups of Normal People.—Two fairly large, though widely dissimilar, groups of persons were examined. One consisted exclusively of undergraduate students in the University of California—mostly freshmen and sophomores; the other was composed of young Chinese pupils in attendance at five of the mission schools of San Francisco. The university students, all men, of an average age of about 18.5 years, were tested at the University gymnasium, the experiments being conducted

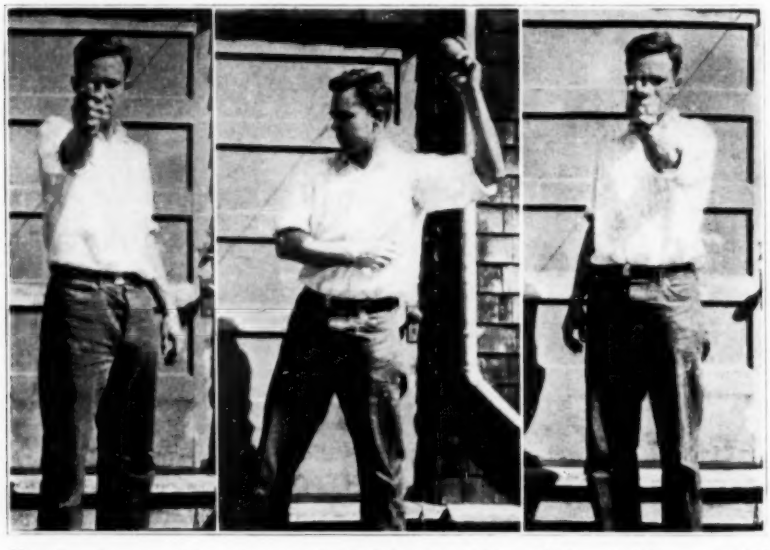


Fig. 4.—Asymmetric or cross-wired sinistral—left-handed and right-eyed (LR). Note that he throws with the left hand and sights only with the right eye.

twice weekly for a period of a little over three weeks. The oriental pupils, most of whom were boys, averaged about 12 years of age; they were examined in the class-rooms of the schools, each child rising in his place when called on to perform the tests. The simple pantomimic game with the ball and the pistol seemed to appeal to them; for they entered into it with the utmost intelligence and enthusiasm.

The sets of data obtained from these two racial groups are brought together in table 1.

It will be seen that the two series of figures agree closely throughout. The parallelism seems especially remarkable because of the tremendous cultural gap that separates the subjects from which these figures were obtained. However, it is also important in another sense. It appears

to show that the values recorded in table 1 are basic values. At any rate, it would seem that about 3.5 per cent of people in the general population may be classified in LL's and a like number as LR's; while about 18 per cent are of the asymmetric type RL. This leaves only 75 per cent of people in the purely dextral group RR. From these facts it follows that approximately one person in four exhibits some form of sinistrality; and that the right-handed sinistrals outnumber all other sinistrals in the ratio of three to one.

The figures require little comment. However, with respect to the sinistral groups, it may be pointed out: 1. That apparently, among both Americans and Chinese, the proportional number of left-handed persons to be found in any large group stabilizes itself at about 7 per cent. This appears to be particularly noteworthy because of the circumstance that to most parents of the Chinese coolie and small shop-keeper classes it probably matters little whether a child prefers to manipulate his chop-sticks with the right hand or with the left. 2. That of 1,000 uni-

TABLE 1.—*Percentage Incidence of the Principal H-E Types Among Normal People*

Subjects Examined	Dextrals, RR*	Sinistrals			Ambilaterals and Other Types	Total Sinistrality
		RL	LL	LR		
1,000 U. C. undergraduates.....	70.4	18.5	4.0	3.6	3.5	26.1
317 Chinese children.....	69.7	20.8	4.7	2.2	2.5	27.7

* The first letter indicates handedness, the second eyedness.

versity students, 185, or 18.5 per cent, were found to belong in the cross-wired group RL. Hence there must be at least twenty million people in this country who exhibit this visuomanual peculiarity.

Relative Numbers of Dextrals and Sinistrals in Certain Vocational Groups.—As already stated, a series of H-E tests[3] carried out several years ago among musicians and mechanics seemed to show that while few sinistrals are to be found in the ranks of iron-workers, many of their number make a living as players of stringed instruments. This surprising and unexpected result having made it seem worth while to carry out similar experiments among persons engaged in other vocations, two groups of persons were examined—one at the California School of Fine Arts, in San Francisco, the other at the Pacific School of Religion in Berkeley.

The average age of the art students, a majority of whom were women, appeared to be about 20 years; that of the divinity students, who seemed to be evenly divided between men and women, I judged to be nearly 30.

3. This term is employed hereafter in place of the words handedness and eyedness.

The results obtained in these tests, as well as those noted in the earlier experiments, are shown in table 2.

Unfortunately, I was able to test only a small number of art students and a still smaller number of divinity students, so that the observations made in these groups have little statistical value; nevertheless, when considered in connection with some data of another kind to be introduced presently, they have, I believe, rather unusual suggestive interest.

It will be noted that nearly 13 per cent of the art students are left-handed. This observation, which I am inclined to believe was not a purely casual one, would make it seem reasonable to infer that left-handed persons are endowed with some innate quality or leaning which causes them, instinctively, to seek a career in art. Still more striking, however, are the data with respect to the sinistrals of the dominant type RL; for the evidence goes to show, not only that many persons

TABLE 2.—*Percentage Incidence of the Principal H-E Types in Vocational Groups*

Subjects Examined	Dextrals, RR*	Sinistrals			Ambilaterals and Other Types	Total Sinistrality
		RL	LL	LR		
100 journeymen mechanics	92.0	4.0	4.0	0.0	0.0	8.0
100 professional musicians	68.0	24.0	6.0	2.0	0.0	32.0
70 pupils in California School of Fine Arts	54.2	28.5	4.2	8.5	4.2	41.2
41 pupils in Pacific School of Religion	46.3	48.7	0.0	0.0	4.8	48.7

* The first letter indicates handedness, the second eyedness.

of this type take up the muscian's calling, but that others of their kind, in much greater numbers, strive to become artists or to become ministers of the gospel.

Since there seems to be no doubt that this phenomenon of selective vocational concentration actually takes place and that it is characteristic of sinistrals, some experimental evidence may be introduced that affords at least a clue as to its cause. A few words will serve to explain why this evidence was sought.

Earlier experiments with musicians had shown (table 2) that 32 per cent of their number were sinistrals. As this work was carried out among professional musicians and was limited almost exclusively to players of stringed instruments, it is probable that the figure cited gives a fair idea as to the percentage of sinistrality characteristic of that vocation. On the other hand, these experiments leave one entirely in the dark (1) as to the number of persons in the population at large who possess musical talent and (2) as to the H-E peculiarities of such persons. It seemed desirable, therefore, to elucidate both of these points. Accordingly, while the sinistrality tests were in progress, 815 of the university students were asked this question: Do you play any kind

of a musical instrument? Table 3 gives a detailed analysis of the data thus obtained.

As regards the first question, it will be noted that 234, or 28.7 per cent, of these young men produce instrumental music of one sort or another. This is probably an average value as far as university students are concerned. In the absence of more extensive data on the subject, it may be accepted, provisionally, as applicable in a wider sense to the general public. Here, then, is at least a tentative answer to the query.

Turning now to the second question—that as to a possible correlation between H-E peculiarities and musical talent—the results of the H-E tests show that the 815 students fall into two unequal groups—a large one of 590 dextrals and a smaller one of 225 sinistrals. Singularly enough, the members of these two groups seem to be quite unlike as regards the extent of their musical accomplishments. It will be noted that 141, or 23.8 per cent, of the dextrals are able to play on a piano

TABLE 3.—*Classification of 234 Amateur Musicians Found Among 815 University Students*

Students Examined	Musicians Found		Preferences as to Instruments (Percentages)					
	Number	Per Cent	Piano	Violin	Clarinet	Trombone	Trumpet	All Other
590 dextrals.....	141	23.8	53.6	17.3	10.1	4.3	5.0	9.4
225 sinistrals.....	93	41.3	41.9	26.7	12.9	6.4	3.2	8.6

or other instrument, while 93, or 41.3 per cent, of the sinistrals are gifted in this way. These data, I believe, justify the conclusion that individuals of the types RL, LL and LR probably have a quicker sense of harmony than those of the type RR and therefore, more commonly than dextrals, try to express themselves through the medium of a musical instrument. Significant, too, in this connection is the fact that sinistrals, in the choice of an instrument, seem to show a decided preference for the violin.

Another circumstance that may be noted here, for what it is worth, is that of a total of forty-one divinity students examined, twenty, or 48.7 per cent, are amateur musicians; and that, as regards type, all of these instrumentalists are RL's. I did not secure data of this sort from the art students.

On the whole then, if entitled to any credence, the foregoing observations would seem to warrant the assumption that sinistrals, as compared with dextrals, are more delicately constituted; that their perceptive faculties are perhaps more sensitive and impressionable; I am disposed to believe, moreover, that in some way this factor determines the vocational trend to which attention has been drawn.

Relative Numbers of Dextrals and Sinistrals among Persons with Mental Diseases.—For the sake of comparison, two groups of neuropsychiatric patients were examined. One of these groups was made up of 483 ex-soldiers of an average age around 35 years. The other consisted of 210 patients in the Mendocino State Hospital, Talmadge, California; the average age appeared to be about 50 years. This latter was a mixed group, as it contained 129 men and 81 women.

The experiments were carried out in the wards. The procedure followed in each ward was to check off the patients from a nominal list and at the same time, opposite the names of those persons whose responses had been clear and unmistakable, to note the reactions to the H-E tests. With the data thus secured in any given ward, a revised nominal list was made out in which the patients were grouped according to their H-E peculiarities. This list then was taken to the admin-

TABLE 4.—*Percentage Incidence of the Principal H-E Types Among Persons with Mental Disease*

Patients Examined	Condition	Dextrals, RR*	Sinistrals			Ambilaterals and Other Types	Total Sinistrality
			RL	LL	LR		
327 ex-soldier	Dementia praecox	66.8	16.2	3.3	5.2	8.2	24.7
136 ex-soldier	Miscellaneous	62.0	20.4	2.2	5.1	10.2	27.7
20 ex-soldier	Constitutional psychiatric state	25.0	45.0	5.0	10.0	15.0	60.0
166 civilian	Dementia praecox	53.0	28.3	4.2	3.6	10.8	36.1
44 civilian	Miscellaneous	47.7	31.8	2.2	9.0	9.0	43.0
70 patients (consolidated group)	Constitutional psychiatric state	37.1	38.5	7.1	8.5	8.5	54.1

* The first letter indicates handedness, the second, eyedness.

istration building where the diagnoses were written in, for, while the work was in progress, I had no means of knowing with what clinical types of mental disease I was dealing.

With respect to the varieties of mental disease encountered in the soldier group, it will be observed (table 4) that 327, or 67.7 per cent, of the men had dementia praecox of one form or another, while 136, or 28.1 per cent, suffered from other, "miscellaneous" disorders, and 20, or a little over 4 per cent, are listed under the heading "constitutional psychopathic state."

It may be as well to state here that the miscellaneous group included 56 cases of dementia paralytica; 22 of manic-depressive psychosis; 18 of epileptic psychosis; 10 of cerebrospinal syphilis; 9 of a psychosis with mental deficiency; 9 of undiagnosed psychoses; 4 of hysteria; 2 of traumatic psychoses; 2 of encephalitis; 2 of chronic alcoholism; 1 of spastic paraplegia and 1 labeled presenile psychosis.

If now the question as to a possible correlation between sinistrality and mental disease is taken up, the figures in table 4 show that the percentages of sinistrality noted in the dementia praecox group and

also in that marked "miscellaneous" are about the same as those noted previously among normal people—that is, around 25 per cent; while in the remaining group—that made up of persons with constitutional psychopathic states—no less than 60 per cent are sinistrals. However, it should be mentioned that very high values were noted also in two other small groups; i. e., among the patients listed as having a psychosis combined with mental deficiency and among those suffering from chronic alcoholism.

It will be noted that slightly different values were obtained among the patients at the state hospital.

Of these patients there were two principal groups; one consisted of 166 cases of dementia praecox, the other of 44 miscellaneous cases. This latter group included 1 case of epileptic psychosis, 1 of senile psychosis, 2 of manic-depressive psychosis and 1 of dementia paralytica. It also included 39 cases carried on the hospital records, collectively, as "psychopathic personalities" (made available by Dr. D. R. Smith, the medical superintendent). These, in detail, consisted of 15 cases of chronic alcoholism; 6 of drug addiction without psychosis; 11 of psychosis with mental deficiency and 7 listed as constitutional psychopathic states.

Briefly stated, the H-E tests carried out at this institution gave the following results:

Taking the larger group first, of a total of 166 patients with dementia praecox examined, 60, or 36.1 per cent, were sinistrals of one type or another (table 4). As regards the sex factor, a slight difference was observed; of the women, who numbered 64, 21, or 32.8 per cent, were sinistrals, while in the male group of 102 patients the number of sinistrals was 39, or 38.2 per cent.

In the smaller, miscellaneous group of 44 patients there were altogether 19 sinistrals, or 43.1 per cent. Of the 39 members of the group listed as psychopathic personalities 18, or 46.1 per cent, were sinistrals.

On comparing the data obtained respectively from the two groups of patients with dementia praecox, it will be seen that the total sinistrality value noted in the case of the civilian group is about 11 per cent higher than the corresponding figure obtained from the group of military patients. This discrepancy, I believe, may be attributed to the process of selection carried out by the draft boards, since there can be no doubt that many persons obviously unfit for military service because of mental trouble were thereby weeded out.

The general results obtained among persons with mental disease by means of the H-E tests can be summed up briefly as follows:

In two fairly large groups of patients with dementia praecox, much lower percentages of sinistrals were found than were found in groups

made up of persons with constitutional psychopathic states. The contrast in this respect is indeed striking if one consolidates the groups and then compares the average values thus obtained. In the case of the 493 patients with dementia praecox the value is 30.4 per cent, while in the case of the 70 persons with constitutional psychopathic states—a consolidated group in which are included the drug addicts, the chronic alcoholic patients and the patients said to be mentally deficient—the average sinistrality figure is 54.1. As regards the relative numbers of the three types of sinistrals among the 70 psychopathic persons, there were 27 of the type RL, 5 of the type LL and 6 of the type LR.

On the whole, then, the experimental evidence accumulated in this research seems to show that: (1) about one quarter of the people who make up the general public are sinistrals; (2) under normal conditions, sinistrals tend to concentrate in certain vocations, and (3) among persons with mental disease, sinistrals are most numerous in the classes of patients known as psychopathic personalities.

COMMENT

Sinistrality data may be of service to a psychiatrist in several ways. For example, the information derived from H-E tests seems to throw light from a new angle on the much debated problem as to the propriety of effecting a change of handedness in the case of any given person. This question would only be raised in the case of a child born left-handed. Since established left-handedness is divided nearly equally between persons of the respective types LL and LR, it would appear logical to recommend a compulsory change of handedness only in the case of a child of the type LR because, apparently, such a change would convert it forthwith into a symmetric dextral. However, the matter does not end here, as in every instance of this sort there is a mental as well as a physical factor to be considered. The truth is that a sinistral child is apt to be hypersensitive in many ways and it does not seem at all likely that such a child's inborn traits of character would be modified to any noticeable extent, if at all, merely by virtue of a change in handedness. Moreover, the vast majority of sinistrals are strongly right-handed. On the whole, therefore, perhaps the best course to pursue is to let the child follow its own natural bias in this matter, allowing it to use whichever hand it prefers.

Again, to consider a more practical use that may be made of visuomanual data of the kind under discussion, there is reason to believe that a classification of persons according to their H-E peculiarities would prove helpful to persons who give vocational advice. The figures shown in table 2 suggest that in many instances a sinistral may possess natural aptitudes that would render it difficult for him to adjust himself to the conditions imposed by any shut-in humdrum form of employment.

Finally, from the clinical standpoint, whether a patient is sound or unsound of mind, the discovery that he is a sinistral of one type or another sometimes may afford a clue, not to be had in any other way, on the one hand, as to some personality problem, and, on the other, as to the nature of some baffling "nervous disorder." And since sinistrals, especially those of the RL type, are apt to show signs of constitutional instability, in any obscure case it would seem advisable to canvass this possibility before treatment is instituted.

CONCLUSIONS

1. Of 1,000 university students examined with reference to handedness and eyedness, 704 were right-handed and right-eyed; 185 were right-handed and left-eyed; 40 were left-handed and left-eyed; 36 were left-handed and right-eyed; 35 are classified as ambilaterals and other types. Therefore, 26.1 per cent of these young men are sinistrals.

2. Nearly the same percentage of sinistrals was found in a study of 317 Chinese school children.

3. There seems to be a marked tendency for sinistrals to concentrate in certain of the esthetic vocations.

4. On the basis of data collected from 815 university students, it can be stated that sinistrals are definitely more musical in their tastes than dextrals.

5. In a survey of 693 neuropsychiatric patients, it was found that in dementia praecox and in most other forms of mental disease the percentage of sinistrals was 30.4, while in a mixed group of persons with constitutional psychopathic states the average value was 54.1 per cent.

6. It is suggested that sinistrals, especially those of the RL type, are apt to show signs of constitutional instability.

2512 Washington Street.

EFFECT OF ALCOHOL ON THE PATELLAR TENDON REFLEX TIME IN DOGS *

JOHN M. DORSEY, M.D.
ANN ARBOR, MICH.
AND
LEE EDWARD TRAVIS, PH.D.
IOWA CITY

A previous study demonstrated that alcohol markedly shortened the patellar tendon reflex time in human subjects.[1] Both it and another study [2] indicated that the central nervous system of man enjoys a constant relationship existing between its higher and lower levels. This relationship appears to be in the direction of dominance of the superstructures so that the higher levels of irradiation play a functional part in the lower levels of irradiation—alterations in the former determining a depth effect on the latter. The present comparative study is reported to indicate a similar relationship existing in a central nervous system of a lower developmental order.

METHOD

Again we determined the reflex time with the action current technic. In general, each of five dogs, ranging in weight from 31 to 37 pounds (14.1 to 16.8 Kg.), was given intravenously rapidly 20 cc. of 97 per cent alcohol in 80 cc. of physiologic solution of sodium chloride. Each dog quickly became drowsy, one of them after resuscitation, the others spontaneously, recovering a good degree of vigilance within an hour and a half.

EXPERIMENTAL DATA

In each of the accompanying five graphs the ordinate scale denotes reflex time in sigma. Each circle represents a knee jerk. These graphs picture the consistent but irregular shortening of the reflex time after the injections of alcohol. This result is identical with our observation of the effect of alcohol on the patellar tendon reflex time in man.

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* Submitted for publication, Oct. 20, 1929.

* From the Psychopathic Hospital at Ann Arbor and at Iowa City.

1. Travis, L. E., and Dorsey, J. M.: Effect of Alcohol on Patellar Tendon Reflex Time, Arch. Neurol. & Psychiat. **21**:613 (March) 1929.

2. Travis, L. E., and Dorsey, J. M.: Patellar Tendon Reflex Time in Psychiatric and in Neurologic Cases, Arch. Neurol. & Psychiat. **22**:99 (July) 1929.

The increase in action current duration and the production of other noteworthy reported phenomena of the latter study were not detected in this work.

The accompanying table reveals the following information: After injections of alcohol, in all dogs the mean reflex times were less, in all but one dog (dog 2) the ranges of reflex time were greater and in all dogs the lower limits of the ranges were less and their upper limits never more than those of the normal ranges.

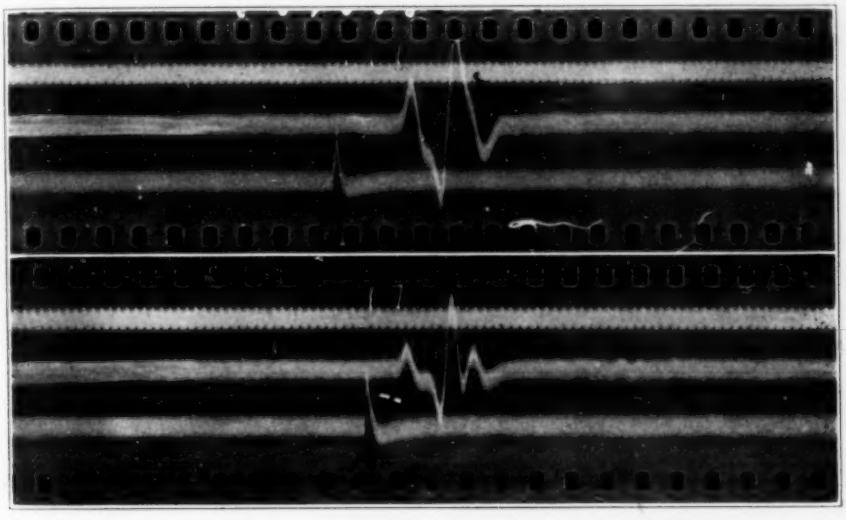

Fig. 1.—The upper figure is the patellar tendon reflex time record of dog 1 before the injection of alcohol. Reading from above down here and in the lower figure the first is the time line in sigma, the second is the action current line and the third is the signal line. *A* indicates the instant of stimulation of the tendon, and *B* indicates the initiation of the action currents. The reflex time is the number of sigma (read as 7.5) between *A* and *B*. The lower figure is the patellar tendon reflex time record of dog 1 after the injection of alcohol. Here the reflex time was read as 4 sigma.

Means and Ranges of Reflex Times Before and After Injections of Alcohol

	Before Injection		Following Injection	
Dog	Mean Reflex Time	Range Reflex Time	Mean Reflex Time	Range Reflex Time
1	8.4	8.0 - 9.0	7.6	6.0 - 9.0
2	10.8	10.0 -11.0	7.9	7.5 - 8.5
3	8.7	8.0 - 9.0	7.4	7.0 - 8.5
4	9.0	9.0 - 9.5	7.1	5.0 - 7.5
5	7.0	6.5 - 7.0	6.4	4.0 - 7.0

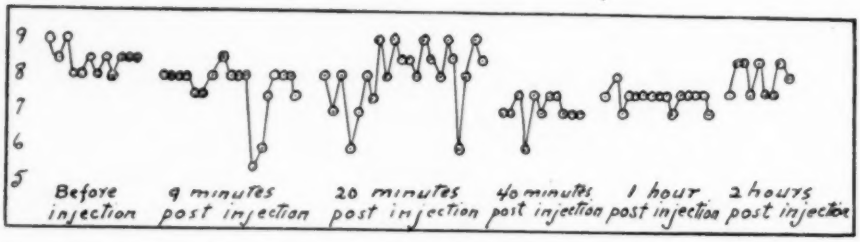

Fig. 2.—Graph of the reflex times of dog 1.

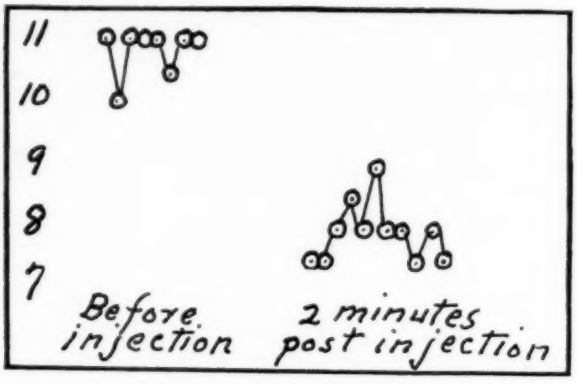

Fig. 3.—Graph of the reflex times of dog 2.

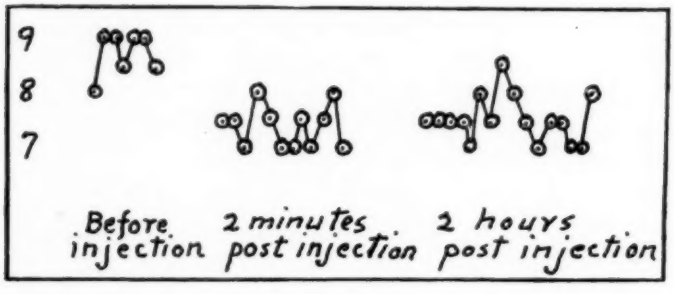

Fig. 4.—Graph of the reflex times of dog 3.

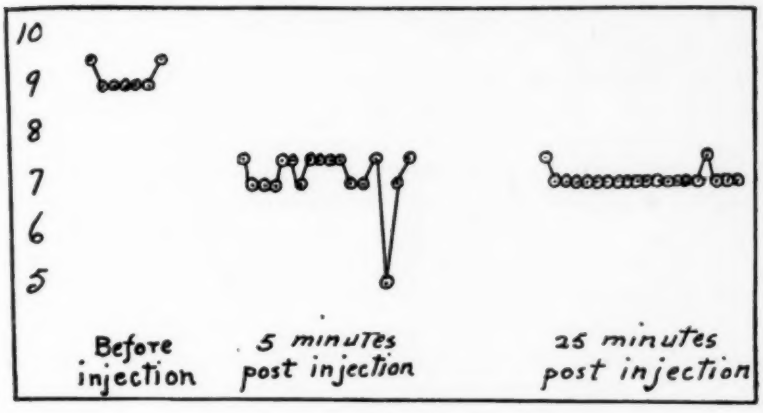

Fig. 5.—Graph of the reflex times of dog 4.

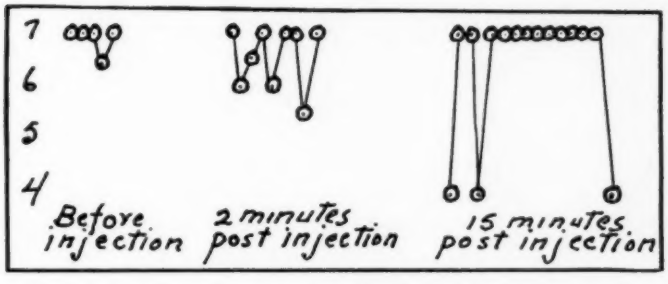

Fig. 6.—Graph of the reflex times of dog 5.

SUMMARY

1. Alcohol consistently but irregularly reduces the patellar tendon reflex time in dogs.

2. In our studies this observation is comparable to the effect of alcohol on patellar tendon reflex time in man.

3. As was the case with our human subjects, the alcohol affected the animals to make them stuporous.

4. This study lends further support to the proposition that the central nervous system of higher animals is a pyramid of dependencies subserving superstructures which maintain their dominance by virtue of their inhibitory effect on the subjacent and possibly adjacent structures they environ.

5. Increase in action current duration following injection of alcohol was not detected in the case of dogs.

MYATONIA CONGENITA

WITH PARTICULAR REFERENCE TO PATHOLOGY AND FAMILIAL TENDENCY *

E. S. GURDJIAN, M.D.
ANN ARBOR, MICH.

Among the myopathies of childhood and infancy, myatonia congenita has an important place. This disease was described by Oppenheim [1] in 1900. Later contributions of note are those of Collier and Wilson,[2] Rothman,[3] Griffith and Spiller,[4] Grinker,[5] Greenfield and Stern [6] and others. The condition usually appears at birth. It is exceptional for it to appear in a postnatal form, although Collier and Wilson stressed the possibility of such types. According to the earlier investigators, the disease has no hereditary or familial tendencies. Some of the more recent contributions, however, have shown definitely that more than one ease. Particularly the postnatal type follows an acute infection, such in association with other myopathies in the same family (Greenfield and Stern, 1927). One group to be described by me is also interesting from the familial standpoint. Sorgente [7] described a familial type of the disease. Particularly the postnatal type follows an acute infection, such as tonsillitis and infection of the middle ear. In most of these instances the condition has appeared within the first fifteen or twenty months of the patient's life.

The condition is characterized by extreme flaccidity of muscles, particularly in the lower extremities, although the upper extremities are also frequently involved. There are associated lengthening and flaccidity of the ligaments of joints in such a way that the extremities may be placed in any bizarre position. The reflexes are at first lost, although

* Submitted for publication, Oct. 14, 1929.

* From the Department of Neurology, University of Michigan Medical School.

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2. Collier, J., and Wilson, S. A. K.: Amyotonia Congenita, Brain **31**:1, 1908.

3. Rothman, M.: Ueber die anatomische Grundlage der Myatonia congenita, Monatschr. f. Psychiat. u. Neurol. **25**:161, 1909.

4. Griffith, J. P. C., and Spiller, W. G.: Amyotonia Congenita: A Clinical and Pathological Study, Am. J. M. Sc. **142**:165, 1911.

5. Grinker, R. R.: The Pathology of Amyotonia Congenita, Arch. Neurol. & Psychiat. **18**:982 (Dec.) 1927.

6. Greenfield, J. G., and Stern, R. O.: The Anatomical Identity of the Werdnig-Hoffmann and Oppenheim Forms of Infantile Muscular Atrophy, Brain **50**:652, 1927.

7. Sorgente, quoted by Collier and Wilson (footnote 2).

with amelioration of the condition they may return. There are no atrophies, a condition unlike the primary myopathies of infancy. The muscles are soft and velvety, and one cannot usually distinguish between subcutaneous fat and muscular tissue. Although the extremities involved appear paralyzed, the condition is one of weakness, so that practically all movements are possible in any given extremity. The involvement is bilateral. There are no fibrillary tremors. The face usually is exempt, but it also may be involved. The electrical reaction of muscles shows a weakness in contraction rather than a reaction of degeneration. As already stated, the disease is capable of amelioration. Rothman, Greenfield and Stern noted that there is a definite anatomic similarity in the pathology of myatonia congenita and the disease described by Werdnig and Hoffmann. Greenfield and Stern collected all the reports of cases of myatonia congenita in which autopsy was performed in the literature and they prove their point. Anatomic identity of pathologic changes, of course, does not necessarily mean that the conditions are alike clinically. I shall next report certain cases of this disease which add a few new data on the pathology and clinical observations.

REPORT OF CASES

CASE 1.—*History.*—N. E. E., a girl, aged 2½, was brought to the University Hospital because of inability to walk. The family history was essentially unimportant. The patient began to walk at 11 months. The first tooth erupted at 14 months. Three weeks before admission, the child fell on her back; three days later, it was noticed that she would stumble easily and fall.

Examination.—On entrance the child was able to walk with help. She appeared to be fairly well developed for her age. She was not acutely ill and cooperated well. She seemed to be mentally alert. There were no atrophies or deformities in the extremities. The tendon reflexes could not be elicited. The abdominal reflexes were present. The grip was weak in both hands. There were no areas of tenderness to palpation anywhere in the body. The patient was able to sit up and support her head, but she was unable to change from a lying to a sitting position. She was also unable to change from a sitting or creeping position to one of standing. She could take a few steps with help. Cutaneous sensation was apparently normal throughout the body. There was extreme myatonia, particularly in the lower extremities. During the entire examination no fibrillary tremors were noted.

The laboratory studies gave essentially negative results. The Wassermann test was negative with both the blood and the spinal fluid. The urine and blood were normal on her entrance to the hospital. The spinal fluid was clear; it contained from 2 to 3 cells per cubic millimeter; Pandy's reaction was slightly positive.

Course.—During her stay in the hospital, the child developed bronchopneumonia. Routine nasal cultures were positive for diphtheria, and she was given 40,000 units of antitoxin. Four days after the onset of pneumonia, she died.

Postmortem Examination.—An autopsy was performed nine hours after death. The cause of death was subacute lobular pneumonia with abscess formation and bronchiectasis, and fibrinopurulent pleuritis with right-sided empyema.

The spinal cord was secured through a dorsal approach. The vertebrae and the spinal canal looked normal. The cord appeared normal macroscopically. The muscles of the calf and the pelvic region looked normal except for slight emaciation.

Microscopic Examination.—The cord at various levels showed fewer cells in the anterior horns than normally, with definite intracellular changes. The normal contour of the cells was often distorted. There was swelling of cells. The Nissl substance was wanting in many. In some instances there was a migration of the nucleus to the periphery. These changes were found particularly in the lumbar

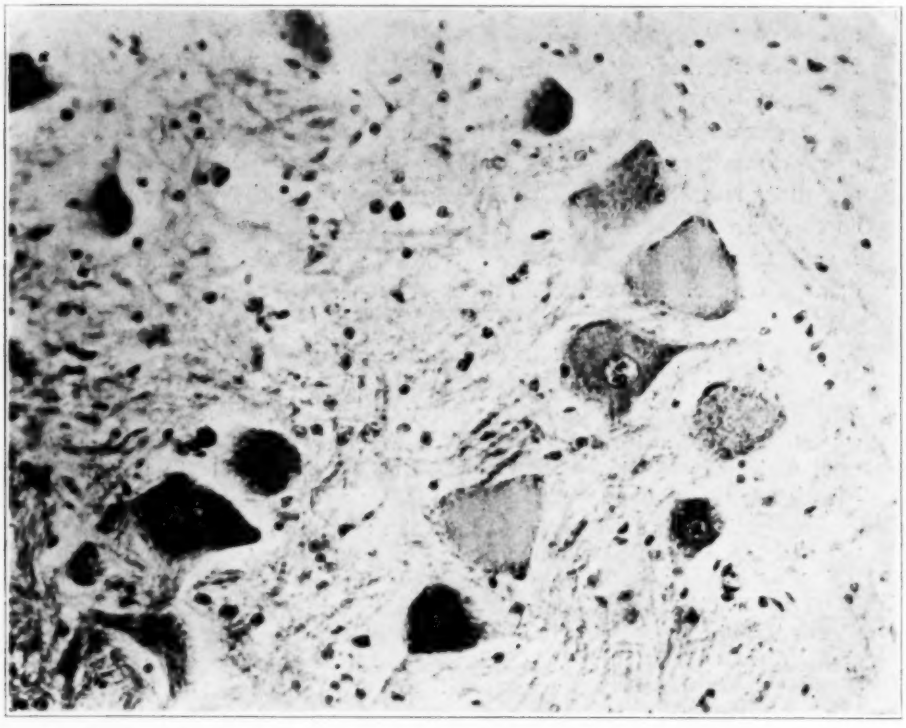

Fig. 1.—The anterior horn cells in the sacral cord. Note the number of cells with marked degenerative changes; chromatolysis is far advanced in several; migration of nuclei to the periphery may also be seen in one cell; there is a marked absence of Nissl bodies. Hematoxylin-eosin preparation.

and lower dorsal regions (fig. 1). The Weigert-Pal method revealed a definite discrepancy in myelination in the anterior and posterior roots. The anterior roots were poor in myelin substance, as shown in figure 2. The discrepancy was noted particularly in the cauda equina and the lumbar region. The anterior roots were smaller than normal. The cells in Clarke's column also showed marked changes, characterized by chromatolysis, migration of nuclei to the periphery, disappearance of Nissl substance and swelling of the cells.

Microscopic examination of the gastrocnemius and the iliopsoas muscles showed atrophy of individual fibers with occasional hypertrophic fibers. Some fibers appeared much paler than their fellows. There was an increase in the interstitial

nuclei between the muscle fibers, these being made up of oval or elongated nuclear material. There was also perivascular infiltration with small round cells (lymphocytes and plasma cells). The longitudinal and cross-striations were not so well preserved as normally (fig. 4).

The heart showed subendocardial fatty degenerative infiltration. There was marked serous atrophy of the subepicardial fat. There was an increased number

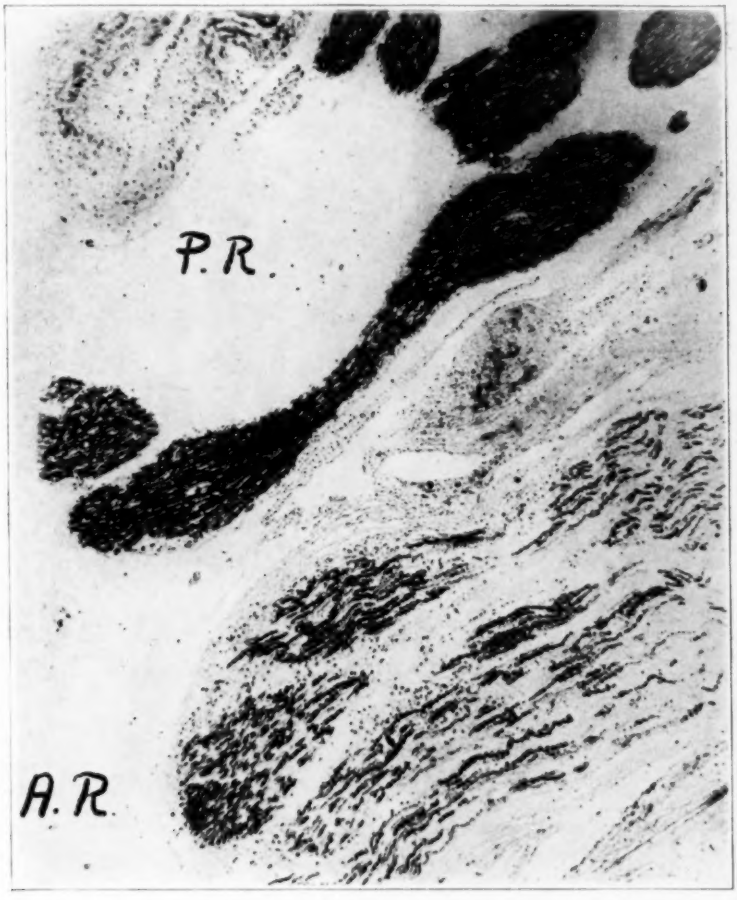

Fig. 2.—A section through the junction of the anterior (*A.R.*) and posterior (*P.R.*) roots. Note the difference in the staining power of these two components. There is a marked change in the myelin of the anterior root fibers; many are broken and poorly stained and form a marked contrast to the posterior root fibers. Weigert preparation.

of wandering cells in the subserous material, chiefly plasma cells and lymphocytes. There was atrophy of muscle fibers with increased interstitial nuclei and areas of small perivascular infiltration.

Microscopic observations on the remainder of the body are irrelevant and are therefore omitted.

Comment.—In this case the condition apparently began acutely with a fall. It may be that the patient fell because of an insidious pathologic change in the spinal cord and muscles. It apparently progressed rapidly until the patient was incapacitated. There was no familial history of the disease. The marked myatonia was apparently due to both a pathologic process in the peripheral muscles and a central neural involvement. It is interesting to note that the region of Clarke's column was so acutely inflamed and showed such extreme cellular change. It is true that curi-

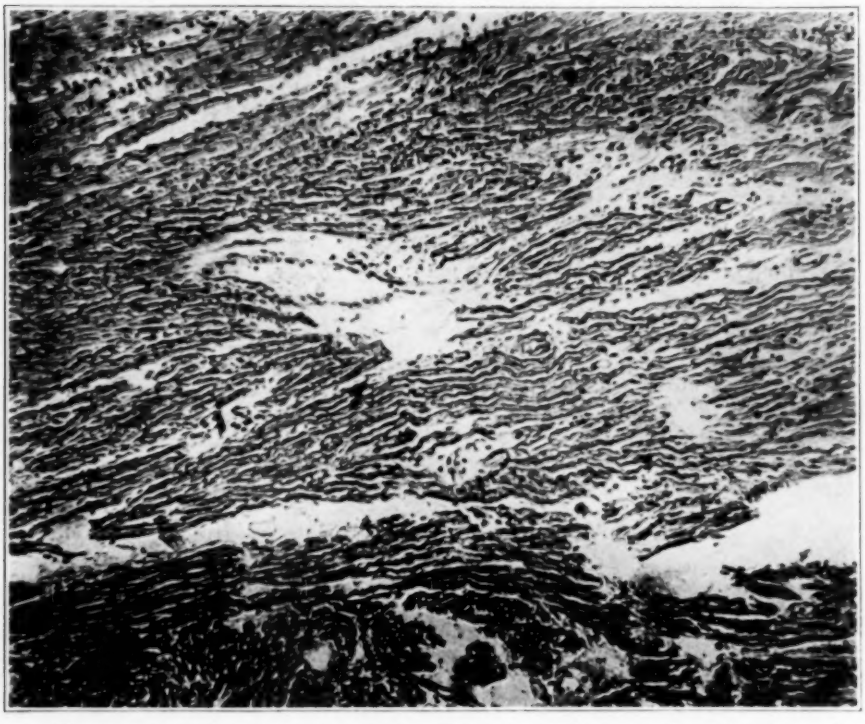

Fig. 3.—A section of heart muscle. There is marked cellular infiltration of lymphocytes and plasma cells. Zenker's necrosis is present in places, and also marked atrophy of the muscle fibers. The longitudinal and cross striations are destroyed in several fields. Hematoxylin-eosin preparation.

ous changes may be observed in Clarke's column even in the normal cord, but in this instance I think that the alterations were definitely pathologic. Possibly the lack of cerebellar coordinating mechanisms is a contributing factor in the production of atonia. The case is of interest in that it appeared in a child who was of apparently normal development, and that it showed such extensive microscopic changes without more clinical manifestations than have been related.

Earlier investigators considered the disease to be nonhereditary and nonfamilial. Several textbooks of the present day still cling to this belief. There are several cases on record in which familial tendencies have been noted (Beevor, Bibegeil and others quoted by Greenfield and Stern). Even different types of myopathy may afflict the progeny in the same family. I shall next report the history of the M family, in which myotonia congenita affected two of six children and in which

Fig. 4.—A section of striated muscle (gastrocnemius). The marked perivascular infiltration of lymphocytes and plasma cells should be noted, also the increase in the number of interstitial nuclei in the entire structure. The cross-striations are absent in this section. The individual fibers are from 15 to 40 microns in diameter. Hematoxylin-eosin preparation.

one child died at the age of 3 from either Oppenheim's or the Werdnig-Hoffmann disease.

Cases 2, 3 and 4.—*The M family.*—There were six pregnancies. The oldest child, aged 11, was apparently well. The next child, Phyllis, aged 8, showed typical weakness, particularly of the lower extremities. There were no hypertrophies or atrophies. The tendon reflexes were absent throughout. There were

no sensory changes. At the age of 16 months the patient had a severe sore throat with an infection of the middle ear, after which she was unable to use the limbs. There was no urinary or fecal incontinence. The patient apparently showed a definite amelioration in that on entrance she was able to walk with help. Although movements were possible in the lower extremities, the latter seemed almost completely paralyzed. The mentality was apparently normal.

The third child, Robert, a boy, aged 6, was apparently well.

The fourth child, a boy, died at the age of 3. He had been abnormal from birth. He did not have the use of the arms or legs. His mentality seemed normal to the parents. They stated that the condition remained stationary until death from pneumonia.

The fifth pregnancy terminated in a miscarriage.

The sixth child, a girl, aged 15 months, sat up alone at the age of 8 months but had never been able to stand. The tendon reflexes were not elicited. There was marked weakness of the lower extremities. There was marked myatonia, particularly of the lower but also of the upper limbs. These parts could be placed in practically any position with no difficulty. There were no fibrillary tremors. There was no definite paralysis of the extremities. There were no localized atrophies. Electrical examination showed diminution in reaction, particularly in the lower limbs.

Comment.—In this group of cases there was a definite familial tendency. One boy, who died at 3, had never been normal, and, according to the parents, had showed weakness of both the upper and the lower extremities. From this description alone it is difficult to make a diagnosis, but the condition undoubtedly belongs in either the myatonia or the infantile progressive muscular atrophy group. The fact that the patient died from intercurrent disease rather than from a progression of the paralytic condition favors myatonia congenita. In another case there was marked myatonia, particularly in the lower extremities. During the examination of the patient no fibrillary tremors were noted over the entire body, although the parents stated that the patient would "frequently twitch at night," particularly in the lower limbs. In this case the condition began after a severe sore throat at the age of 16 months, following which the extremities were practically completely paralyzed, but in the course of several years there was definite amelioration. It is rather difficult to assume that the diagnosis in this case was an acute anterior poliomyelitis. The symmetrical involvement and the lack of local muscular atrophies rule out acute poliomyelitis. In the sixth child, examination showed marked weakness and extreme myatonia of the lower extremities. The electrical reactions were indicative of slight diminution in contraction of muscles.

In this family three of the six children were afflicted with a similar condition (I am sure that at least two had myatonia congenita). The onset in one case was typical of the postnatal myatonia congenita type; i.e., it began after an acute infection.

CONCLUSIONS

I have described several cases with characteristic atonia of the muscles. In one case an autopsy was performed. Microscopic examination of the central nervous system and striated muscle indicated involvement of both. There was marked change in the anterior horns, and the peripheral mechanism (muscle) also showed perivascular infiltration, increase in interstitial cells and cloudy swelling and atrophy of muscle fibers. The longitudinal and cross striations were not so well defined as in a normal specimen. These observations are in accord with those of several other investigators. It is possible that the peripheral muscular changes were due to disease of the anterior horn cells, but one is impressed by the fact that the entire neuromuscular system was involved, with the possible exception of the sensory fibers. I think that the muscular changes represented more than a reaction to pathologic changes in the anterior horns. The generalized perivascular infiltrations, the marked increase in interstitial cells in the muscles, with generalized degenerative changes in the anterior horns (and other motor cranial nuclei), within a period of three or four weeks after the onset of the illness, speaks in favor of their reaction to some toxic process. A better theory, therefore, would be that a common factor (a toxic one) involving the neuromuscular system may be responsible for amyotonia congenita (at least in a large group of cases). The postnatal cases of the disease, with an onset usually after an acute infection, speaks in favor of this assumption. The congenital types could also be explained on a toxic theory in that, under the influence of such a factor, the anterior horn neurons and the muscles (the neuromuscular system) either degenerate or do not develop normally. The lower motor neuron is not fully developed for several months after birth. On the toxic theory one may also be able to explain the possible amelioration in some cases of this disease; with the cessation of toxic causes the anterior horn cells and the muscles recover function sufficiently to operate. The familial tendency in some cases would predispose the neuromuscular system to the deleterious effects of the toxic causes.

Concerning the pathologic identity of the diseases of Oppenheim and Werdnig-Hoffmann, there is undoubtedly a definite clinical difference between the two syndromes. Is the Werdnig-Hoffmann type a more advanced stage of involvement of the central and the peripheral systems? Rothman, Greenfield and Stern answer this question in the affirmative. My studies point to the same conclusion. Patients with the Werdnig-Hoffmann type are seriously ill and die in from four to six years after the onset of irritation of the anterior horn. Muscles may show a reaction of degeneration. In myatonia congenita tremors do not form a part of the picture. The amelioration observed in cases

of Oppenheim's disease may also be explained on the basis of corrective measures and gymnastics. According to various authors, however, practically complete recovery may take place, which would not be compatible with this assumption. It may be that in some cases of myatonia the changes in the neuromuscular system are so slight that recovery of function is a possibility. The assumption that Oppenheim's disease constitutes a form of chronic infantile anterior poliomyelitis (Marburg [8]) does not seem justifiable. In anterior poliomyelitis there are localized inflammatory foci in the anterior horns, whereas in myatonia congenita the entire neuromuscular system is involved (lower motor neurons and muscle tissue). The extreme symmetry of the condition and the absence of localized atrophies also speak against anterior poliomyelitis.

SUMMARY

Cases of myatonia congenita are reported, one with observations at autopsy.

The most important pathologic change was involvement of the neuromuscular system (anterior horn cells, anterior nerve roots and striated muscle). These changes were particularly marked in the lower part of the spinal cord and the lower extremities.

Such universal involvement of both peripheral and central factors suggests the possibility of the operation of a toxic agent.

A family of six children is described in which three cases of myatonia congenita occurred; this establishes the familial tendency of the disease, at least in some cases.

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NIEMANN-PICK'S DISEASE

PATHOLOGIC STUDIES OF A CASE *

GEORGE B. HASSIN, M.D.
Professor of Neurology, College of Medicine, University of Illinois;
Histologist to Illinois State Psychopathic Institute
CHICAGO

Some clinical manifestations of Niemann-Pick's disease, particularly those pertaining to the central nervous system, so much resemble the infantile form (type Tay-Sachs) of amaurotic family idiocy that the question naturally arises whether these two morbid conditions are not one disease. In a very few instances [1] it was possible to show their identity by pathologic studies, while in rare instances it was possible to prove the presence in Niemann-Pick's disease of a cherry-red spot which is pathognomonic of the infantile form of amaurotic family idiocy. Though in the case recorded here such a spot was absent, the histologic as well as the gross changes were decidedly those of amaurotic family idiocy. There were some differences, which will be pointed out.

The clinical and general pathologic features of this case were typical of Niemann-Pick's disease. They will be recorded by Dr. Poncher and Dr. Boikan, while Mr. MacFate will report his extensive studies on the chemical problems, such as the nature of the lipoids present in the ganglion cells. Here only the changes in the brain will be described.

The brain was divided at the necropsy by a sagittal section into two halves. One was sent to Mr. MacFate for chemical studies, the other to me.

REPORT OF EXAMINATION OF BRAIN

Macroscopic Examination of the Brain.—The brain was of normal configuration, but rather large and hard and was leathery to the touch; the sylvian and interparietal fissures were gaping; the frontal convolutions were markedly atrophied. The pia was congested, transparent and not adherent to the brain; the ventricles were of normal size, except the fourth ventricle, which was

* Submitted for publication, Nov. 29, 1929.

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

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dilated. The cerebellum was of normal size, and the corpus callosum appeared thin. In general, the brain exhibited characteristics some of which, such as unusual hardness, have been emphasized by Sachs [2] in his first contribution, while other features (gaping of some fissures) have been pointed out by Schaffer [3] and described by me.[4] The foregoing features, unnatural leathery hardness and gaping of the sylvian and interparietal fissures, were so typical that a diagnosis of amaurotic family idiocy had been made even before the microscopic studies were begun.

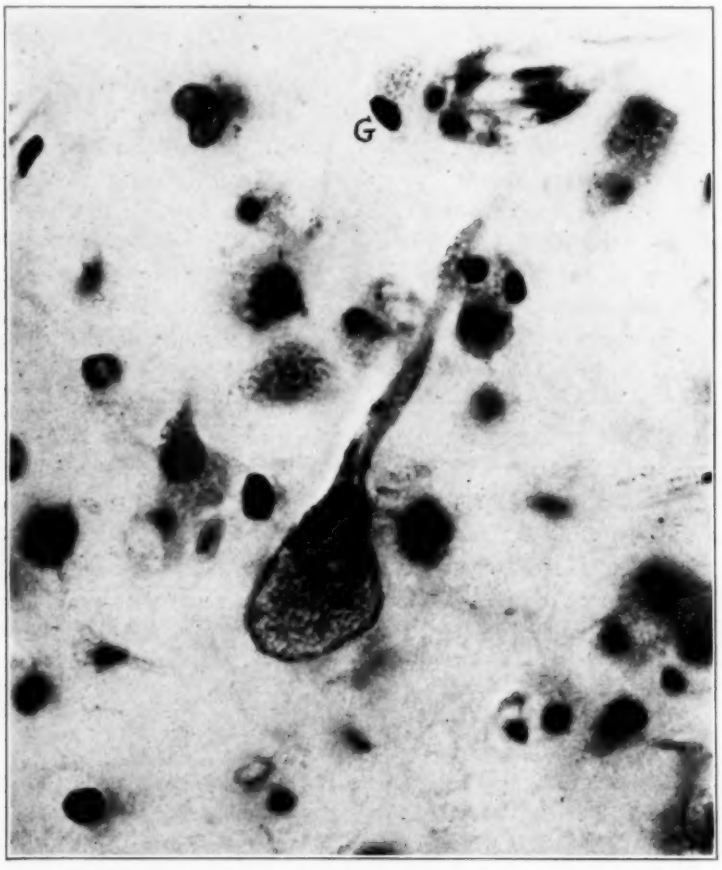

Fig. 1.—The majority of the cell bodies are ganglion cells one of which is well shown in the center; at *G* is a gitter cell. Toluidine blue; × 750.

2. Sachs, B.: An Arrested Cerebral Development, with Special Reference to Its Cortical Pathology, J. Nerv. & Ment. Dis. **14**:541, 1887.

3. Schaffer, K.: Thatsächliches und hypothetisches aus der Histopathologie der infantile-amaurotischen Idiotie, Arch. f. Psychiat. **64**:570, 1922.

4. Hassin, G. B.: A Study of the Histopathology of Amaurotic Family Idiocy (Infantile Type of Tay-Sachs), Arch. Neurol. & Psychiat. **12**:640 (Dec.) 1924; A Case of Amaurotic Family Idiocy, Late Infantile Type (Bielschowsky) with Clinical Picture of Decerebrate Rigidity, Arch. Neurol. & Psychiat. **16**:708 (Dec.) 1926.

Microscopic Examination.—The cortex exhibited a mass of cells which little resembled ganglion cells. They appeared as pale, expanded bodies of large size, honeycombed in structure and containing a darkly stained nucleus. This was peripherally located, sometimes protruding from the swollen cell body the processes of which were also swollen, winding and rather well stained (fig. 1). Such cells were present in every cortical layer and were intermingled with a mass of other cells of similar structure, the so-called gitter cells (fig. 1). These so much resembled the former in size and shape that on superficial examination

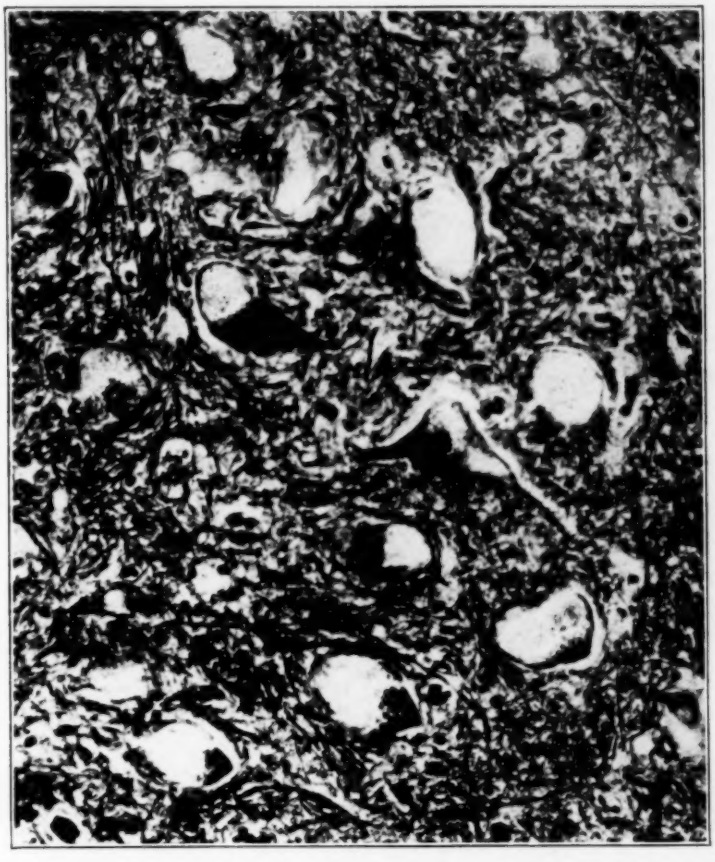

Fig. 2.—Ganglion cells of the hypoglossal nerve. The dark portion around or near the nucleus contains well preserved Nissl bodies. The light portion appears reticular in some cells; in some, it is broken up in a dustlike mass which is poorly stainable. Bielschowsky stain; × 285.

they appeared alike. Their nucleus, also located at the periphery, was, however, smaller and darker and did not protrude from the cell body, which showed no processes whatever. Yet in some instances the distinction was exceedingly difficult, especially when the processes of the ganglion cells did not stain distinctly and therefore did not show. In such cases the differentiation could be made only by microchemical stains. For instance, when stained with aniline dyes, such

as toluidine blue, the cell body showed a reticulum (fig. 1) the meshes of which appeared empty. In other areas, such as the nucleus of the hypoglossal nerve (fig. 2), only a part of the cell body was reticular while the portion near the nucleus contained an abundance of Nissl bodies. In the cortex and the large ganglia, especially in the optic thalamus, the ganglion cells, except some Betz cells, showed no Nissl granules whatever, even near the nucleus, where they are usually present. In such instances the pale portions of the cells stained well with the hematoxylin methods of Weigert-Pal, Heidenhain and Spielmeyer. The dark granules, some small, some larger, were scattered over the cell bodies (fig. 3) and were generally more abundant than in amaurotic family idiocy. The other type of honeycombed cells, the gitter cells, did not harbor such

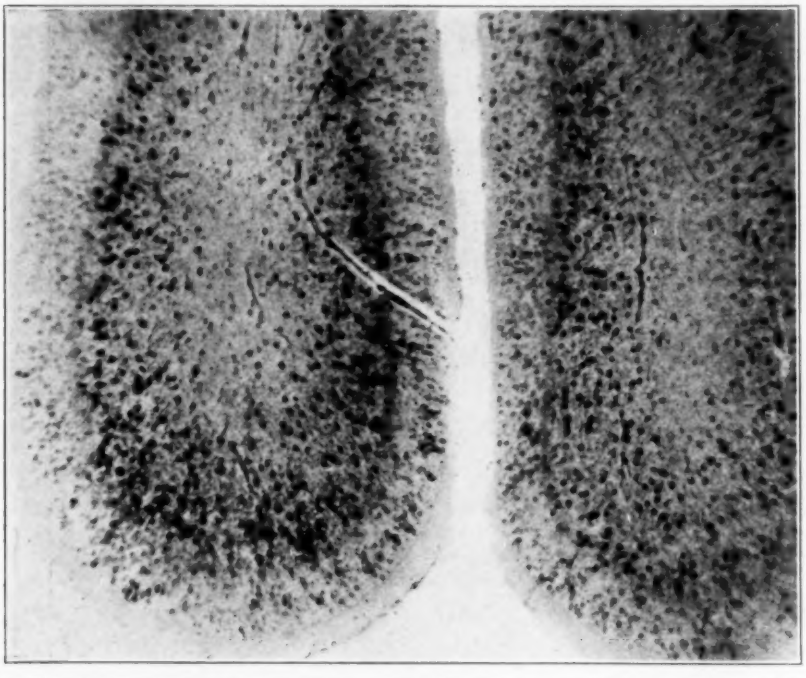

Fig. 3.—Cerebellum. The layers, especially the one assigned to the Purkinje cells, are filled with hematoxylinophilic granules. Spielmeyer stain; × 45.

granules. Their contents, instead, stained bright red with scarlet red, which stained the former only pale orange. Such lipoid-laden cells were scattered over the field regardless of the region of the brain—in the cortex, basal ganglia, corpus callosum or medulla oblongata. They were especially numerous around the blood vessels, as well as in the cerebral subarachnoid space. In some areas, such as the corpus callosum, these cells predominated, and the field (fig. 4) accordingly appeared densely covered by them.

In sections stained with the methods of Bielschowsky or Schultze-Stöhr (fig. 2), the ganglion cells also appeared vacuolated and honeycombed. Some vacuoles were especially large, and the ganglion cell in such cases much resembled the so-called α-gitter cell of Jakob. Neurofibrils were few and were always pushed to the periphery; they were noticeable in the dendrons, while the cytoplasm was represented by a dustlike mass. Of especial interest were the changes

in the cerebellum. As figure 5 shows, the molecular layer was well brought out, though it was smaller than usual. It was bordered by a band of dense glial feltwork, and covered by numerous vacuolated bodies. Some of these stained bright red with scarlet red; the majority, however, stained with the method of Spielmeyer or Weigert (fig. 3); that is, some cells contained lipoids in the form of neutral fats, some contained so-called prelipoids. As in the brain, the former (neutral fats) were especially abundant around the blood vessels. The Purkinje

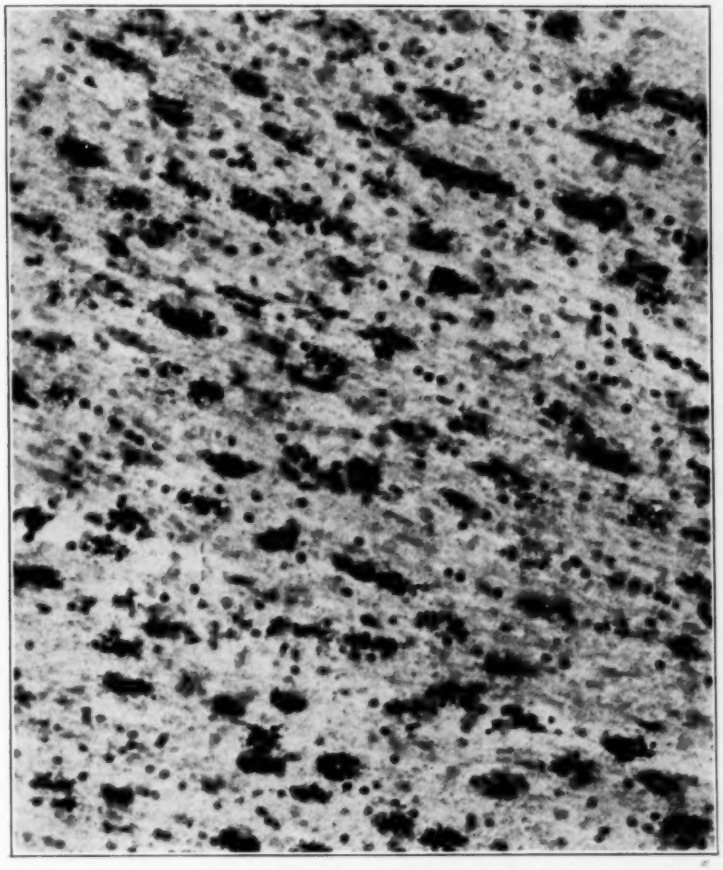

Fig. 4.—Corpus callosum. The dark masses are lipoids (neutral fats); the small round dots are oligodendroglia cells. Scarlet red and hematoxylin; × 240.

layer showed no typical Purkinje cells, but instead, exhibited under a lower power lens a mass of nuclei, "foam" cells and dense glia fibers. Only exceptionally and with great difficulty was anything seen that resembled a Purkinje cell (fig. 6). The cell pictured in figure 6 is greatly expanded and is partly filled with a dustlike substance; the apical dendron appearing antler-like is greatly expanded and pale, while the axon emanating from the base is deviating from its course and is winding its way upward. It appeared smooth, devoid of thickenings, expansions and similar formations. However, the whole row normally

taken up by Purkinje cells appeared dark in specimens stained with the methods of Spielmeyer, Heidenhain or Weigert (fig. 3). Most likely the nuclei described were partly those of gitter cells, partly of Purkinje cells that were profoundly changed. As in the rest of the brain, numerous round cells were scattered—filled with lipoids and prelipoids—as is well seen in figure 5. The granular layer was preserved, but in specimens stained with the methods of Bielschowsky and Schultze-Stöhr it often appeared less rich in cells than it does in amaurotic

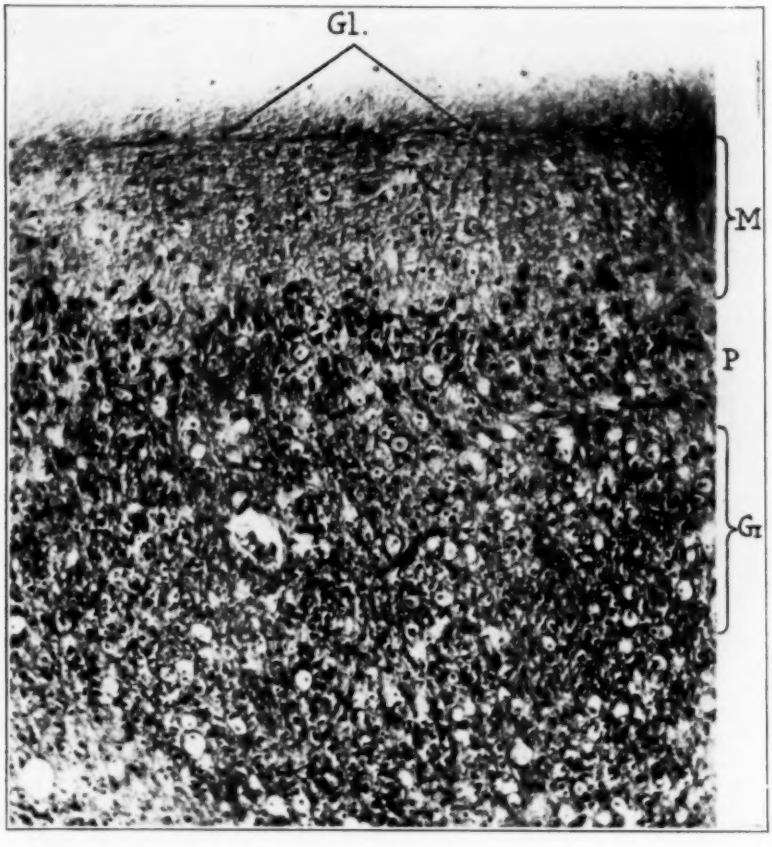

Fig. 5.—Cerebellum. The three layers can be made out distinctly. They are all covered with round ("foam") cells which are especially abundant in the granular layer. The molecular layer (*M*) is bordered by a stripe of dense glia fibers (*Gl*), while the Purkinje cell layer (*P*) shows an abundance of nuclei which belong mainly to the expanded and changed Purkinje cell; below is the granular layer (*Gr*). Bielschowsky stain; × 140.

family idiocy. In the infantile type of this disease the cerebellum possesses a wealth of Purkinje cells, which in this case were lacking and were replaced by cells greatly changed in size and form and by neuroglia tissue. As a matter of fact, when stained with the method of Holzer (fig. 7), the dense glial network was much in evidence, not only in this particular layer, but throughout the other layers of the cerebellar cortex. It is rather noteworthy that the gold

corrosive sublimate stain of Cajal showed here only a scanty amount of cytoplasmic glia cells (macroglia), while in the cortex they were present in immense masses and could be well demonstrated also by the silver stains of Bielschowsky or Schultze-Stöhr (fig. 8). Figure 8, for instance, shows the monster cytoplasmic glia cells with their huge processes inserted in the blood vessel walls, while in other places the processes envelop also the honeycombed bodies, both the ganglion and gitter cells. As to the optic thalamus, this, like the cerebellum,

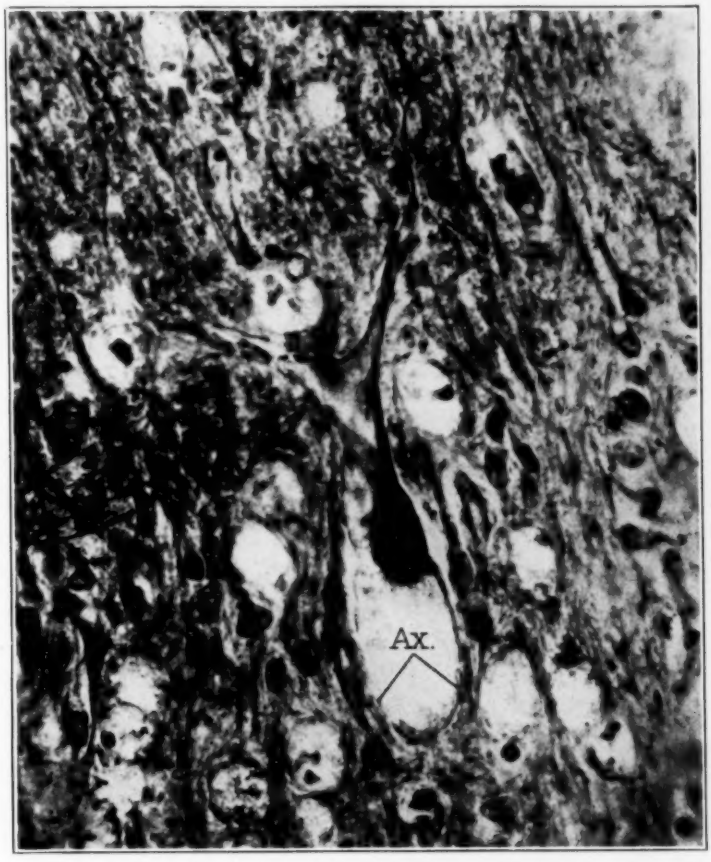

Fig. 6.—In the center is a greatly expanded Purkinje cell. The apical dendron is antler-like; the axon (*Ax*) emanating from the dark portion of the cell is winding upward around its pale portion. The dark portion is the perinuclear mass of the neurofibrils; the scattered round pale bodies are partly gitter cells, partly changed Purkinje cells. Bielschowsky stain; × 500.

was transformed into a glial scar, with a relative scarcity of cytoplasmic glia cells and a wealth of fibrous glia tissue.

Notwithstanding such huge masses of glia cells and glia fibers, many cortical areas occasionally appeared rarefied and reticular. Even in the cerebellum, which resembled a solid tissue scar, there were areas devoid of parenchyma and containing only faintly stained glia fibers and a shrunken blood vessel in the center.

The parenchymatous and glial changes described were associated with changes in the blood vessels and the nerve fibers. The former, as mentioned, were infiltrated with gitter cells which were packed with lipoids; their endothelium was lightly stained; it was bulging; the blood vessels were numerous, and many were newly formed. Nerve fibers in the cortical regions were very sparse and thin; in the cerebellar cortex they were exceptionally sparse, often exhibiting varicosities and thickenings. In the subcortical matter, however, the nerve fibers were, in contrast, exceptionally rich; they were so also in the medulla, notwithstanding the marked changes of the ganglion cells. The macroglia cells in the

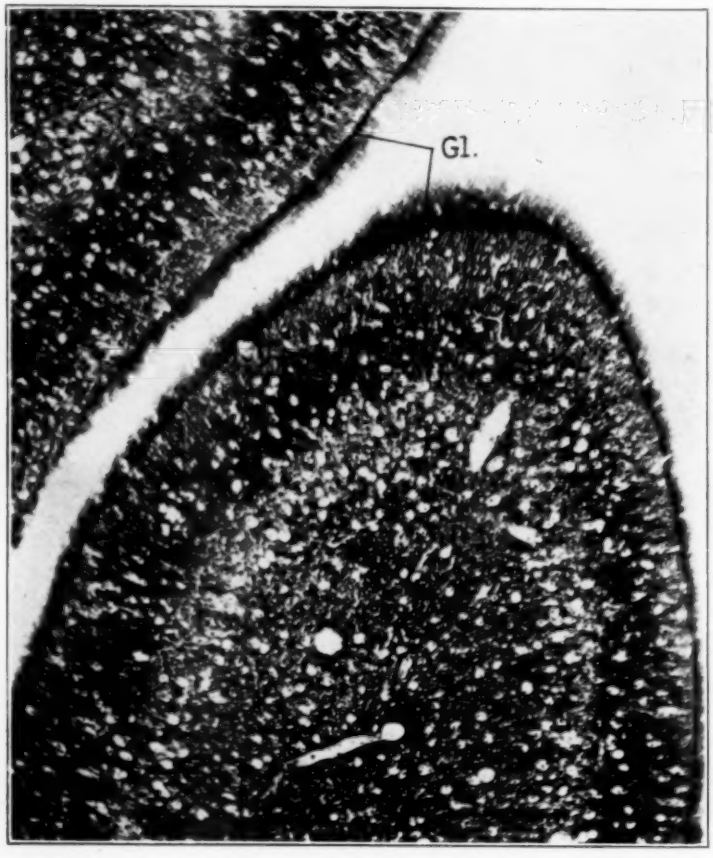

Fig. 7.—Cerebellar cortex transformed into a mass of glia tissue interspersed with "foam" cells. The marginal glia bands (*Gl*) of the molecular layer are especially well shown. Holzer stain; × 85.

subcortex were less abundant than in the cortex, and were often represented by so-called "gemästete Zellen," large cytoplasmic cells with short and scarce processes. These were also present in the upper strata of the cortex and even in the optic thalamus. On the other hand, the oligodendroglia was as abundant as normally; it exhibited no appreciable changes, and only in the pons was some cytoplasm visible around the nucleus. The subarachnoid space contained a wealth

of cellular elements, lymphocytes, polyblasts, fibroblasts and an enormous number of gitter cells packed with fat. The arachnoid stripped from the pia was studied in toto, and showed numerous honeycombed foam cells filled with hematoxylinophilic granules. The ependyma of the sylvian aqueduct was crowded by foam cells, which were also present in the pineal body. In the choroid plexus the tuft cells were expanded, pale in appearance and often resembled the foam cells of other organs (the viscera, bones and lymph glands). These were carefully studied by Dr. Boikan, who found the changes to be similar to those described repeatedly in Niemann-Pick's disease.

Fig. 8.—The cortex of the brain. The spider cells are macroglia; their processes are attached to the blood vessel walls; the round reticular cells scattered among them are partly changed ganglion cells, partly gitter cells. Schultze-Stöhr silver stain; × 240.

COMMENT

The histologic changes recorded exhibited great similarity to those seen in the various types of amaurotic family idiocy. There were differences, of which the most striking were the changes in the cerebellum. Marked as the latter are in some late infantile types of

amaurotic family idiocy, they were much more marked in the case recorded. The prevalence of glia tissue and blood vessels in the various cortical layers, the extreme scarcity of Purkinje cells, the density of the fibrous glia and the abundance of neutral fat substances in the perivascular spaces of the blood vessels suggest a far advanced parenchymatous lesion with transformation of the cerebellar cortex and optic thalamus into a so-called glial scar. One gains the impression that in this disease the pathologic phenomena are much farther advanced than in ordinary amaurotic family idiocy, though the fundamental type of changes is generally the same. Here one has the same expansion of the ganglion cell bodies with practical obliteration of the dendrites, the identical accumulation in them of prelipoids, and the same pronounced cerebellar and thalamic changes. The microchemical changes also are similar and denote that the conditions under discussion are members of one pathologic group. As Bloom [5] well put it, Niemann-Pick's disease appears to be "a profound disturbance of metabolism in which the infants do not thrive; there is a piling up of lipoid material, especially phosphatids, in phagocytic cells throughout the body while neutral fats are more or less gone or destroyed. In this they differ from Gaucher disease where no deep metabolic disorder is present, for such patients may live up to 59 years."

In other words, the two morbid conditions are due to a metabolic disturbance. However, one is so far hardly justified in considering Niemann-Pick's disease and amaurotic family idiocy one disease process. As Sachs [6] justly pointed out, it is rather singular that no striking visceral changes such as are seen in Niemann-Pick's disease have been described in amaurotic family idiocy. This would be the case if they were one and the same condition. Nor do all cases of Niemann-Pick's disease, as shown elsewhere,[7] exhibit cerebral changes typical of amaurotic family idiocy. However, the pathologic material has not been sufficiently abundant, nor were the visceral organs in amaurotic family idiocy thoroughly studied. Until such studies are forthcoming, it may be considered that the two conditions are caused by a metabolic disorder which in some instances affects preferably the central nervous system (amaurotic idiocy), in others preferably the visceral organs (Niemann-Pick's disease) and in the minority of cases, the two.

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6. Sachs, B.: Amaurotic Family Idiocy and General Lipoid Degeneration, Arch. Neurol. & Psychiat. **21**:247 (Feb.) 1929.

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HYALINE DEGENERATION IN DEMENTIA PARALYTICA *

ABNER WOLF, M.D.
JERSEY CITY, N. J.

In rare cases of dementia paralytica an interesting phenomenon has been observed. Here and there, scattered through the brain stem but chiefly in the gray matter of the cortex, a homogeneous, translucent, tough and cartilaginous substance has been found. It varies from microscopic deposits to clumps many centimeters in diameter and gives the staining reactions of hyaline. It is found exclusively in cases of dementia paralytica, the nonparetic early cases referred to by Alzheimer having had ill founded pathologic diagnoses.

This striking condition was first systematically described by Alzheimer [1] in 1898. In his admirable presentation, he also reviewed the preexisting literature, which was scanty and unequal in quality. Since that time there have been approximately a dozen publications on the subject, dealing invariably with cases of neurosyphilis. Without exception these have been carefully and excellently worked out. The problems involved were as to the nature of the hyaline substance and the reason for its deposition. I have no intention of reviewing here this earnestly developed material. It has been done repeatedly and thoroughly by Sträussler and Koskinas,[2] Löwenberg,[3] Dürck [4] and others. Much of what I shall present is in agreement with and substantiation of what has previously been written on the subject. A line of thought and a few facts are interesting and new. In the hope that these will add something to the elucidation of the problem, I present the following case.

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* Submitted for publication, Nov. 17, 1929.

* From the Pathological Laboratory (Prof. Dr. A. Jakob) of the University Psychiatric Clinic and State Hospital, Hamburg — Friedrichsberg (Prof. Dr. Weygandt).

1. Alzheimer: Die Kolloidentartung des Gehirns, Arch. f. Psychiat., 1898, p. 30.

2. Sträussler and Koskinas: Ueber "kolloide" "hyaline" Degeneration und ueber "Koagulationsnekrose" im Gehirn, Ztschr. f. d. ges. Neurol. u. Psychiat., 1926, nos. 1 to 3, p. 100.

3. Löwenberg, K.: Ueber hyaline Degeneration der Grosshirnrinde bei progressiver Paralyse, Ztschr. f. d. ges. Neurol. u. Psychiat., 1924, vol. 93; Hyaline Degeneration of the Blood Vessels in Neurosyphilis, Arch. Neurol. & Psychiat. **20:** 731 (Oct.) 1928.

4. Dürck, H.: Ueber die sogennante Kolloidedegeneration in der Grosshirnrinde, Ztschr. f. d. ges. Neurol. u. Psychiat., 1924, p. 88.

REPORT OF CASE

Clinical History.—P. B., aged 48, a moving picture technician, was admitted to the State Hospital at Friedrichsberg, near Hamburg, in August, 1925. He was found to have a typical case of paresis without unusual symptoms, signs or laboratory observations. He received malarial treatment. The result was only moderately good. He remained under observation until December, when he was discharged on his own repeated requests.

His second admission occurred in September, 1927, when he presented the symptoms of dementia paralytica in its terminal stages. After a slow but progressively downhill course, he died in June.

Autopsy.—The entire frontal pole of the brain was somewhat softened. There was a depressed, hardened area, approximately 2 cm. in diameter, in the upper

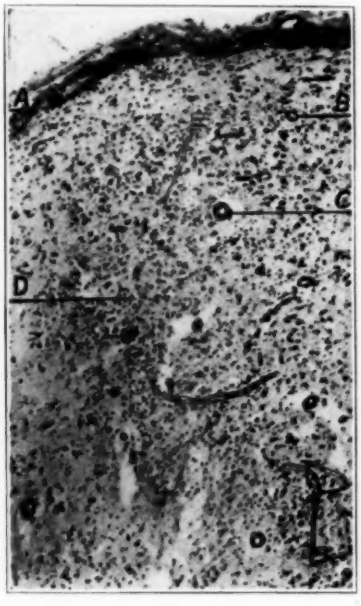

Figure 1

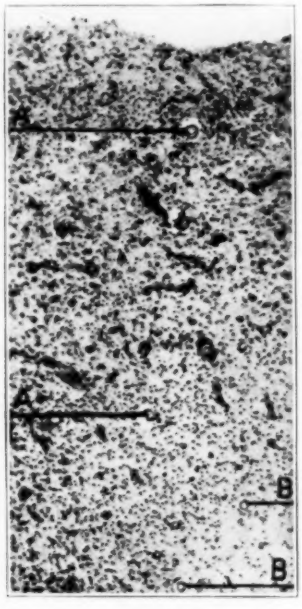

Figure 2

Fig. 1.—Photomicrograph of dementia paralytica changes in the pia and cortex. First appearance of hyaline in the vessel walls. *A* indicates the thickened, infiltrated pia; *B*, unaffected vessel; *C*, a hyalinzed vessel; *D*, stratified structure of the cortex no longer visible. Nissl stain.

Fig. 2.—Photomicrograph showing first appearance of hyaline in the vessel walls in the cortex. *A* indicates a hyalinized vessel; *B*, unaffected vessels. Nissl stain.

portion of the left precentral gyrus. Incision showed that it extended to a depth of about 3 cm. The normal cortical markings were gone. The material was glassy, translucent and of almost cartilaginous toughness. Similar smaller areas were found in various regions of the brain stem, both occipital lobes, second left temporal gyrus, both gyri recti and the thalamus. The rest of the macroscopic observations were those of ordinary dementia paralytica.

The microscopic observations were typical of dementia paralytica. There were a moderate reaction in and of the pia and cortical vessels, architectural disturbances in the gray matter, proliferation of the Hortega glia, deposits of iron, etc. The hyaline substance proved to be the same as that in cases already described in the literature. It gave none of the reactions of amyloid. With toluidine blue (Nissl) it stained a light greenish blue and appeared granular; with methyl violet a grayish blue; with hematoxylin-eosin pink; with iodine sulphate straw colored; with bright green fuchsin an intense homogeneous green, and with van Gieson a homogeneous reddish yellow. In silver stains, it showed itself to be argentophile. As in previous investigations, these various stains proved the substance to be hyaline and carminophile but gave no information as to its nature.

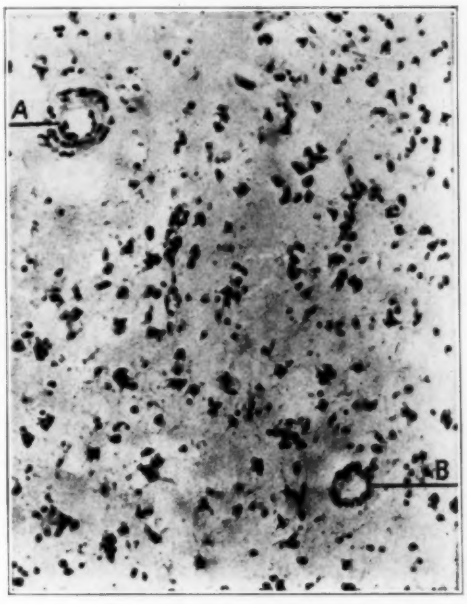

Fig. 3.—Higher power photomicrograph of early hyalinization. *A* indicates hyaline in the wall of a cortical arteriole; *B*, an unaffected arteriole. Nissl stain.

What was interesting in this case was the possibility presented of tracing some sequence in the series of histologic events. Repeatedly, areas were found in which a certain number of cortical vessels showed a beginning hyaline deposit in their walls while adjacent vessels remained free (figs. 1, 2 and 3). A gradual progress could be traced from involvement of a few vessels to the hyalinization of almost all the vessels of a given area of the cortex. Next in order seemed to be regions in which a definite reaction to the process was going on. On the margins of these foci were the hyalinized arterioles and capillaries. As one approached the center of such an area, the usual paretic vascular infiltration became many times intensified. Large homogeneous macroglia cells appeared and became more numerous the nearer one approached to the heart of the given focus. They infiltrated the entire width of the cortex but were most developed in the first two layers (fig. 4). In the densest collections they were quite large and shiny, and had eccentric nuclei

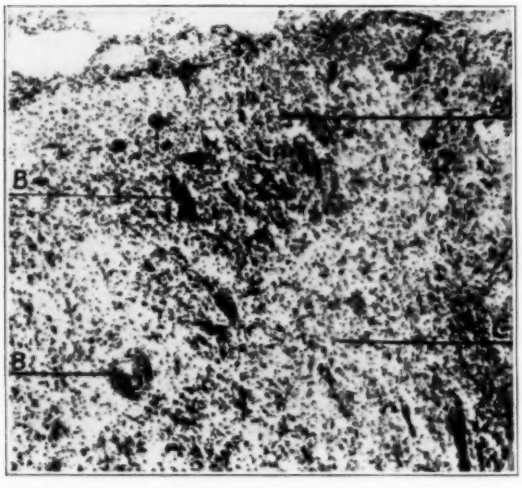

Fig. 4.—Photomicrograph of intense perivascular and glial recation to the invasion of hyaline in the cortex. *A* indicates masses of homogeneous macroglia cells; *B*, intense perivascular infiltration; *C*, proliferation and hypertrophy of

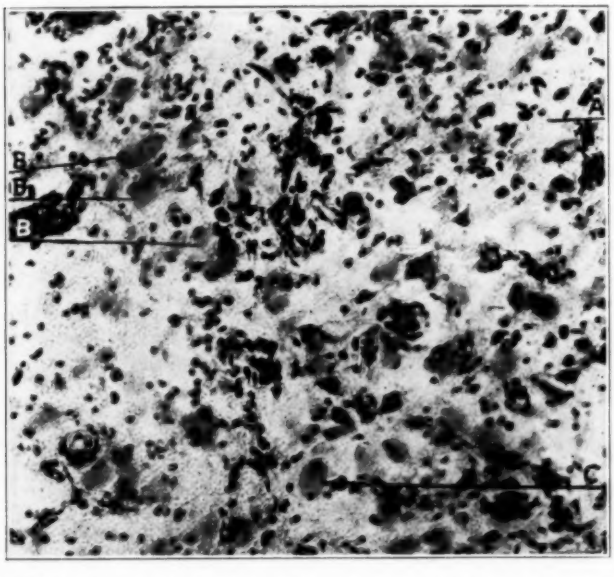

Fig. 5.—High power photomicrograph of macroglial reaction in the cortex. *A* indicates coarsely granular ground substance; *B*, large, homogeneous, glassy, macroglia cells; *C*, large homogeneous macroglia cell and eccentric nucleus. Nissl stain.

(fig. 5). The ground substance seemed to have become more coarsely granular (Nissl preparations which form the basis of this description) in these places. This seemed to be followed by a stage in which the massive inflammatory reaction in the vascular walls disappeared and the ground substance became still coarser and

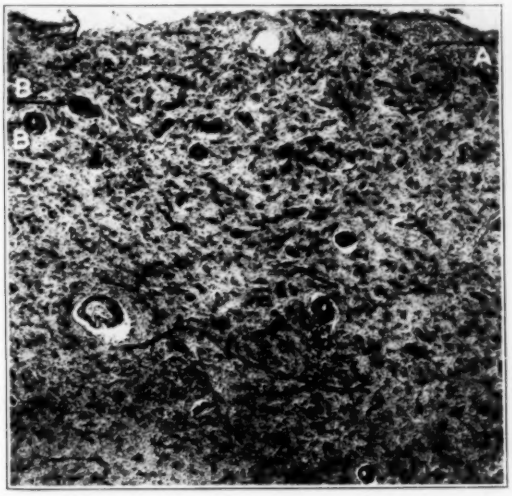

Fig. 6.—Photomicrograph of first deposition of hyaline on the outer margin of the cortex; reaction disappearing. *A* indicates hyaline; *B*, two hyalinized arterioles.

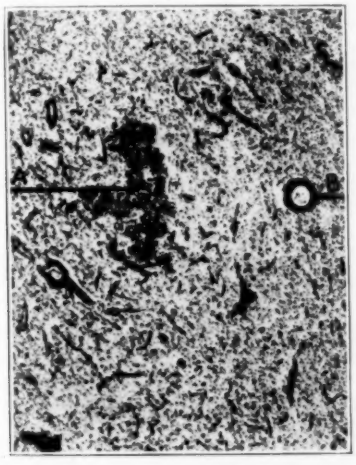

Fig. 7.—Photomicrograph of early disposition of hyaline in the deeper layers of the cortex. *A* indicates hyaline; *B*, a hyalinized vessel. Van Gieson stain.

more granular, while little isolated deposits of hyaline began to appear in it (figs. 6 and 7). At the same time ganglion cells, homogeneous macroglia and other cell elements progressively disappeared, apparently due to the pressure of the invading hyaline. The latter gradually increased in amount, chiefly in the marginal layers

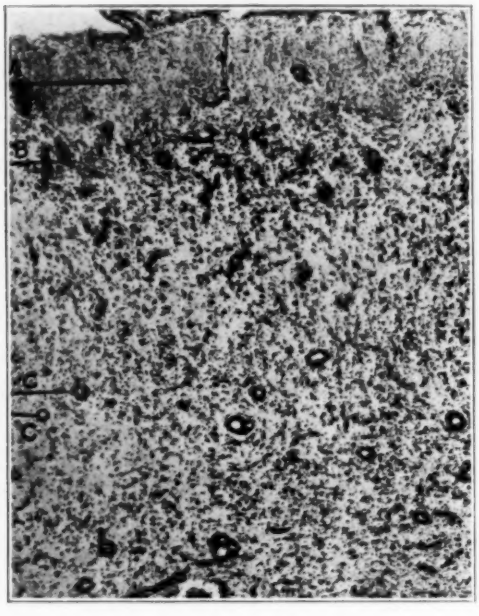

Fig. 8.—Photomicrograph of further advance of deposition of hyaline in the first two layers of the cortex. *A* indicates hyaline; *B*, intense perivascular reaction; *C*, hyalinized vessels. Nissl stain.

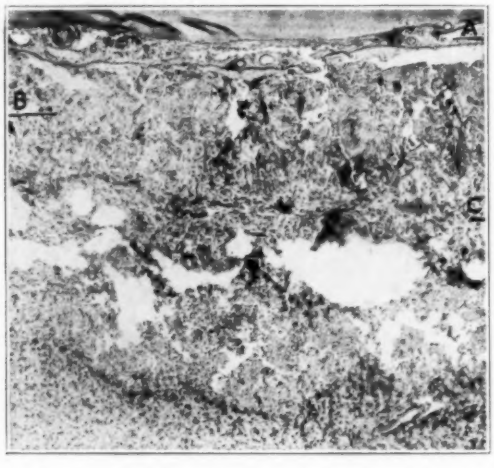

Fig. 9.—Photomicrograph of advanced hyalinization of the cortex; invasion of the pia. *A* indicates the invaded pia; *B*, masses of hyaline; *C*, cortical structure gone. Nissl stain.

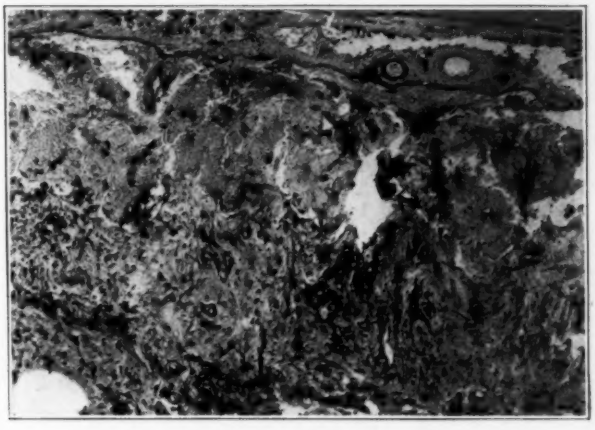

Fig. 10.—Photomicrograph showing large homogeneous masses of hyaline in an advanced stage. Nissl stain.

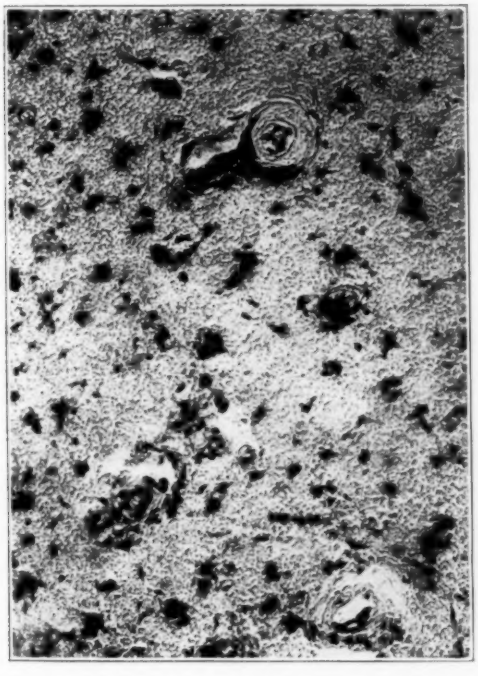

Fig. 11.—High power photomicrograph showing intensely hyalinized area. Most of the cellular elements were destroyed by compression. Nissl stain.

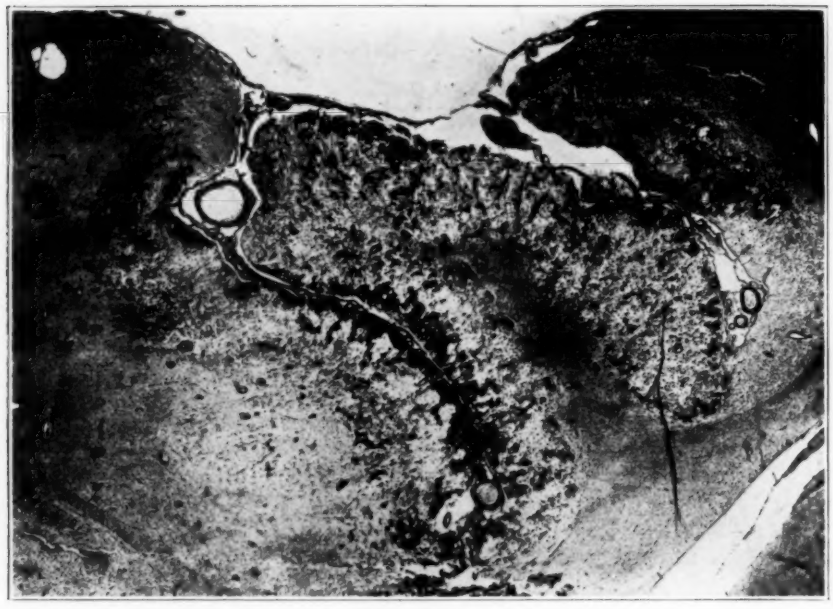

Fig. 12.—Photomicrograph showing status spongiosus after absorption of some of the hyaline. Nissl stain.

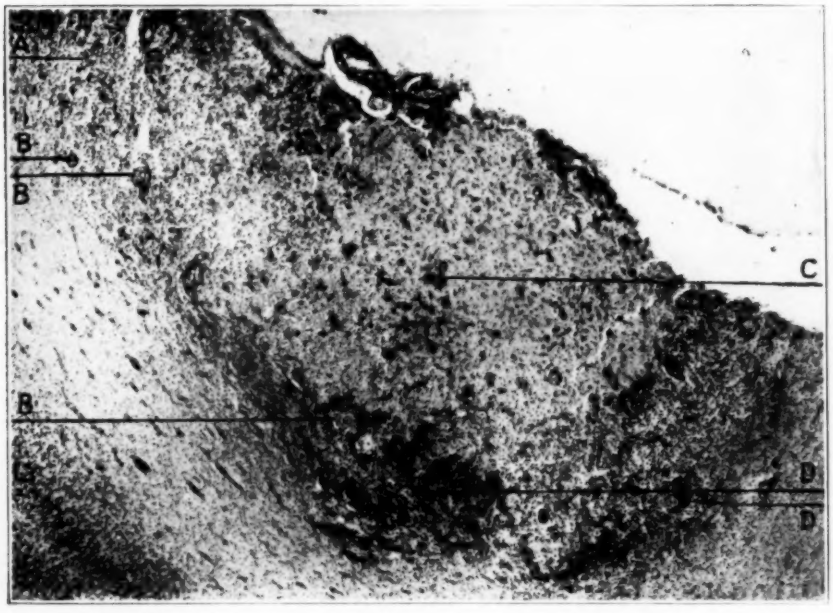

Fig. 13.—Photomicrograph, showing an area illustrative of the various stages of deposition of the hyaline. *A* indicates an unaffected area of the cortex; *B*, hyalinized vessels; *C*, masses of hyaline, with disappearance of cellular elements; *D*, glial and perivascular reactions tending to form a reactive zone against hyaline. Nissl stain.

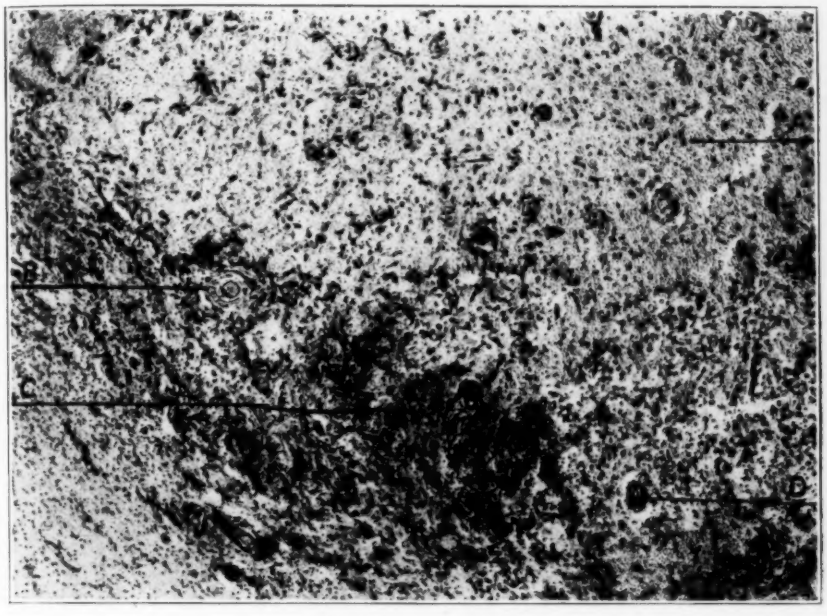

Fig. 14.—Higher power photomicrograph of reaction zone in figure 13. *A* indicates hyaline masses; *B*, a hyalinized vessel; *C*, reaction zone, with large homogeneous glia; *D*, perivascular reaction. Nissl stain.

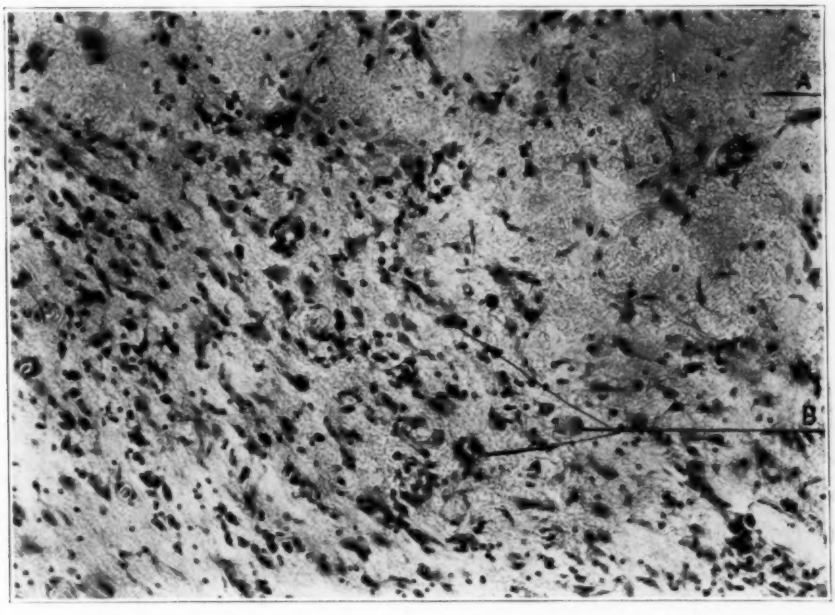

Fig. 15.—Photomicrograph showing large homogeneous macroglia in reaction zone. *A* indicates hyaline; *B*, large homogeneous macroglia. Nissl stain.

of the cortex (fig. 8), but also at the junction of the cortex and medulla. Soon the hyaline took up the whole width of the gray matter in which only the degenerated remains of cells were evident, and also invaded the pia (figs. 9, 10 and 11). The vessel walls became steadily more thickly infiltrated with hyaline so that they seemed merely thick-walled hyaline tubes (fig. 16). Finally there were areas, spongy and vacuolated, in which some of the hyaline seemed to have disappeared and left a so-called status spongiosus as the terminal stage (fig. 12).

There was one region which excellently illustrated all the stages of the process described) (fig. 13). Toward the surface of the cortex lay a large mass of hyaline in which the structural elements were nearly all gone (figs. 14, 15 and 16). About it there was a reactive layer in which an intense vascular reaction, homogeneous macroglia and hyalinized vessels could be seen. Beyond this lay an area

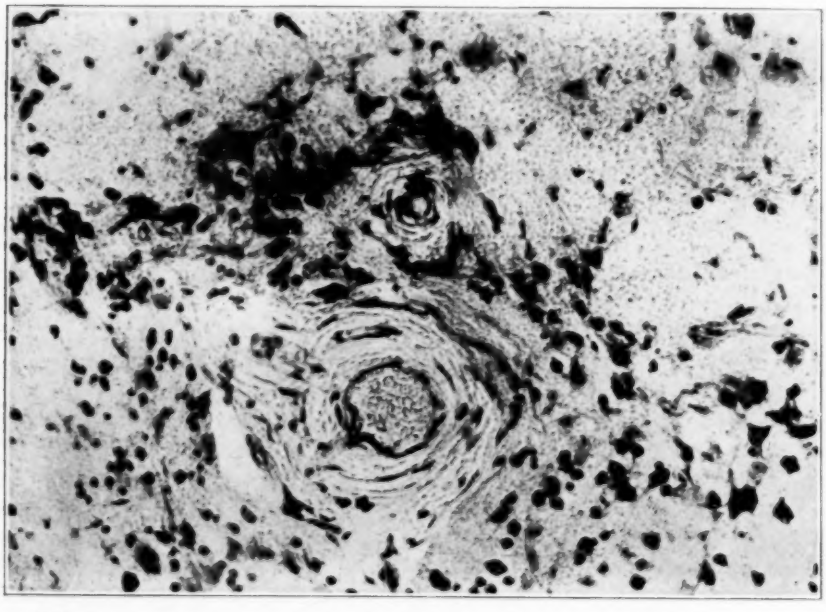

Fig. 16.—Photomicrograph showing heavily hyalinized, much thickened vessel in the reaction zone. Nissl stain.

in which only the vessels were hyalinized and beyond that only the usual paretic changes.

COMMENT

The attempt on the part of neuropathologists to arrive at the nature of this hyaline substance was of course hampered by the scarcity of material. The general pathologist, however, has had little better fortune in investigating the hyaline substances in his domain. As the excellent summary of Leupold [5] shows, little more is known of hyaline today than

5. Leupold, E.: Amyloid und Hyalin, Ergebn. d. allg. Path. u. path. Anat. **21**:1, 1925.

was known twenty-five years ago. Beyond the fact that it is protein in nature and related to amyloid, practically nothing is known. The latter fact, however, permits a little insight into the subject. According to Lubarsch,[6] Fahr,[7] Herxheimer, Leupold and others, there is a close relationship between amyloid and hyaline, and knowledge of the former has increased a good deal in the last ten years. Kuczynski,[8] Domagk,[9] Leupold and others have succeeded in producing amyloid artificially. Their work has shown that when the blood is oversaturated with partially broken-down proteins, artificially introduced, produced by a pathologic process or due to a change in protein metabolism, this circulating protein is precipitated as amyloid in areas in which it

Fig. 17.—Photomicrograph showing connective tissue framework in hyalinized area of the cortex. *A* indicates the outer margin of the cortex; *B*, proliferated connective tissue arising from blood vessels; *C*, regions in which hyaline lies. Perdrau stain.

6. Lubarsch: Milz, in Henke and Lubarsch: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926.

7. Fahr, T.: Niere, in Henke and Lubarsch: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926.

8. Kuczynski, M. H.: Neue Beitrage zur Lehre vom Amyloid, Klin. Wchnschr. **2**:727, 1923.

9. Domagk, G.: Untersuchungen über die Bedeutung des retikuloendothelialen Systems für die Enstehung des Amyloids, Virchows Arch. f. path. Anat. **253**:594, 1924.

encounters a proteolytic ferment (diseased areas, for instance). Although one cannot draw any conclusions as to the formation of hyaline from these investigations of amyloid, the close relationship of the two substances makes a similar mode of origin likely. The work of Karczog, Paunz and Nemeth shows that both are of protein origin, although amyloid originates from more complex proteins.

In the light of these considerations, the deposition of hyaline, as traced in the case cited, takes on a certain logical sequence. It has been agreed by most previous writers on the subject that the process

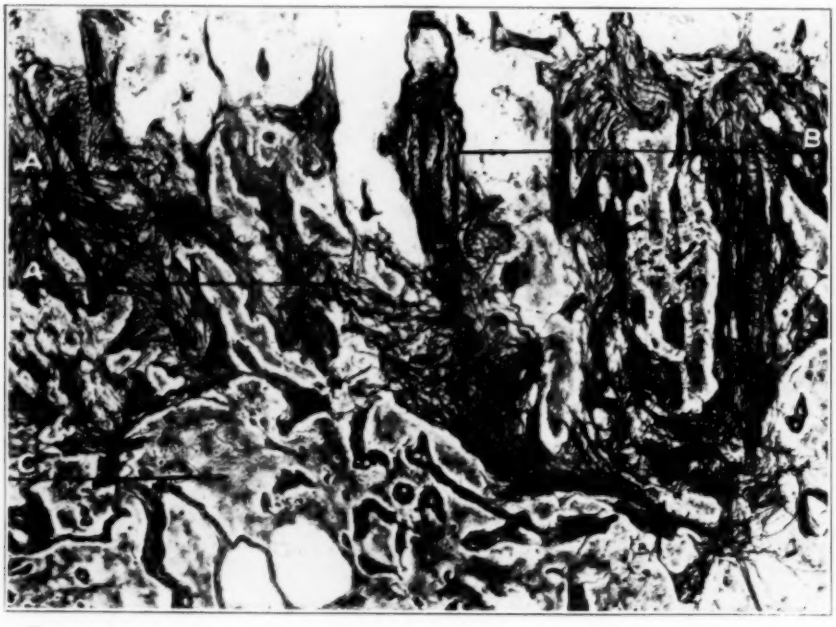

Fig. 18.—Higher power photomicrograph of figure 17, showing vascular origin of connective tissue framework. The area between *A* and *A* indicates an intervascular, continuous, lacey net of connective tissue; *B*, a vessel tangentially cut, showing the proliferation of connective tissue from its wall; *C*, position of hyaline in the connective tissue net. Perdrau stain.

is vascular linked and probably coupled with a disturbance in protein metabolism. The latter provides the partly broken-down proteins circulating in the blood. The nerve tissues, especially the cortex, showing the severe degenerative and lesser inflammatory changes of paresis, undoubtedly contain proteolytic ferments. In some areas a sufficient barrier is apparently formed by the vessels which are perhaps less diseased than elsewhere, and so none of the foreign protein gets beyond the vascular walls, where it is deposited here and there. When the

diseased vessels can no longer form a barrier, more and more of them become thickly infiltrated with hyaline, and there arises about them a defense wall of intense inflammation and reaction of macroglia. The large, homogeneous, glassy appearance of these macroglia cells, casually mentioned by Löwenberg, and more exactly described by Sträussler and Koskinas, inevitably suggests that they contain the foreign protein, if not as hyaline, then as a related substance. There seems to be an attempt on the part of these cells to take up and dispose of the invading foreign substance, yet they seem to lack the proper ferments and disintegrate without having accomplished their purpose. This secondary defense,

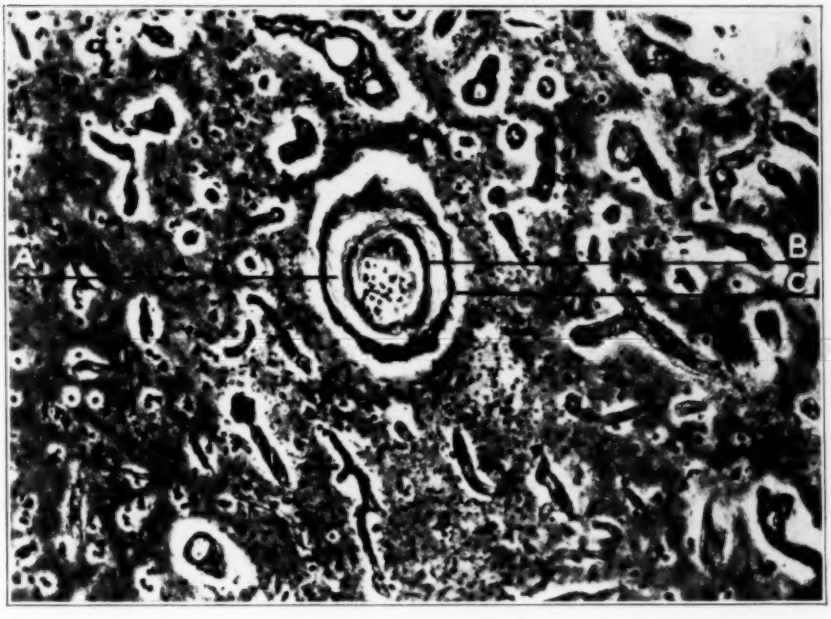

Fig. 19.—Photomicrograph of hyalinized vessel, showing double layer of connective tissue. *A* indicates the position of the hyaline; *B*, the inner and *C*, the outer layers of connective tissue surrounding the hyaline. Perdrau stain.

mesodermal and ectodermal, breaks down as well, and the glial and perivascular reactions disappear while the hyaline begins to be precipitated in the tissues. It seems to be finely granular at first, giving the ground substance its roughened appearance, but becomes increasingly more homogeneous. The vessel walls become more solidly hyaline and thicker. The normal nervous and supporting structures are destroyed by the pressure of the foreign material. Finally some of the hyaline seems to be absorbed, leaving a status spongiosus. This seems to me to be the likely course of events in such cases of deposition of hyaline in dementia paralytica.

Hyaline in plasma cells, as described by Alzheimer, Dürck and Kufs,[10] foreign body giant cells, as described by Löwenberg, Dürck and Sträussler and Koskinas, the localization of the hyaline in the vessels of definite layers of the cortex seen by Löwenberg, Sträussler and Koskinas, as well as calcification of the hyaline, were not observed in this case.

A final interesting point in my case was a remarkable proliferation of the connective tissue of the blood vessels in the affected areas. This hypertrophied fibrous tissue extended from vessel to vessel, interlacing through the entire hyalinized area and forming a protective framework for it (figs. 17 and 18). Most of the vessels showed a distinct division into outer and inner connective tissue layers (fig. 19). It was evident that the hyaline was enclosed between the two. It seemed, therefore, that within and outside the vascular walls there was an increase in connective tissue to surround and bind the hyaline.

SUMMARY

1. A case of paresis showed many areas of hyaline degeneration in the brain.

2. An attempt was made to analyze this process:

(*a*) There is a disturbance of protein metabolism in dementia paralytica.

(*b*) Diseased nervous tissues contain proteolytic ferments.

(*c*) The vessel walls form the first barrier, and are the first site of deposition.

(*d*) After breaking down of the vascular defense, the second defense mechanism is a ring of inflammation and reaction of macroglia.

(*e*) This also becomes insufficient. Hyaline is gradually precipitated. At first granular, it becomes progressively more homogeneous.

(*f*) There is a disappearance of nervous and supporting elements, which are destroyed by pressure of the hyaline.

(*g*) The hyaline is partly absorbed, giving rise to a status spongiosus.

(*h*) A connective tissue framework arising from the blood vessels forms a supporting structure for the degenerated areas.

10. Kufs, H.: Ueber ausgedehnte Kolloiddegeneration des Gehirns bei einem 74 Jahrig alten Paralytiker und andere Fälle dieser Hirnentartung, Ztschr. f. d. ges. Neurol. u. Psychiat. **95**:151, 1925.

SUBARACHNOID HEMORRHAGE AS A CLINICAL COMPLICATION OF NEUROSYPHILIS*

IRVING J. SANDS, M.D.
Associate in Neurology, Columbia University
BROOKLYN

The recognition of the various forms of neurosyphilis has, during recent years, become relatively easy. This is due to a better understanding of the underlying pathologic process as well as to the standardization of the Wassermann test and the addition of the Kahn test. A thorough neurologic examination as well as a serologic study usually leads to the correct diagnosis. Failure to diagnose neurosyphilis correctly is due to error of omission rather than of commission. The therapy of these disorders has also become more or less standardized. The different arsphenamine preparations, mercury, bismuth, tryparsamide, and malaria inoculation, selected to suit each particular case, comprise a fairly effectual group of remedies in the treatment for these diseases. The following two cases are reported because of their unusual clinical picture, their striking diagnostic difficulties and therapeutic problems, and the interesting pathologic observations in one of them.

REPORT OF CASES

CASE 1.—*History.*—A. B., a white woman, aged 40, was seen by me on Dec. 9, 1927, at the request of her family physician. The family history was unimportant. In 1910, she had an attack of so-called ptomaine poisoning; she suffered from extreme headache, protracted vomiting and diplopia. She also had rigidity of the neck and a bilateral Kernig sign. She was treated in a hospital for three weeks. She married in 1909, and had five living, healthy children. The menstrual periods were regular in every respect. She had never had a miscarriage.

The present illness dated back to the early part of November, 1927, when she complained of headache and drowsiness, and occasionally saw double on looking to the right. She was seen by an ophthalmologist, who could not detect any diplopia at first, but one week later found a vertical diplopia. She also experienced a peculiar sensation when ascending stairs, claiming that the stairs were approaching her. She complained of pain in the lumbar region. The family physician made a Wassermann test of the blood which was reported as one plus. For that reason he sent her to me to exclude any possible neurologic complication. On examination there was found irregularity of both pupils, the right being practically fixed to light and in accommodation, while the left reacted very sluggishly to light and in accommodation and consensually. There was a little resistance of the head to flexion, and some suggestion of a Kernig sign. On spinal puncture the fluid

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* Read at a Meeting of the Section of Neurology and Psychiatry of the New York Academy of Medicine, Feb. 12, 1929.

came out under moderately increased pressure, but was bloody, the impression being that the blood was of old rather than of recent origin. The specimen gave a four plus Wassermann reaction.

Course.—The suspicion of neurosyphilis was conveyed to the husband, and the patient was taken to another neurologist, who obtained a negative Wassermann reaction of the blood but who did not have a spinal fluid examination made. He assured the husband that syphilis was entirely out of consideration, and advised an ocean trip as a therapeutic measure. On Jan. 2, 1928, the couple sailed for Bermuda. Six hours after reaching the hotel she was found unconscious in the bath room. She had a hematoma over the occipital region and was unconscious for approximately twelve hours. She then complained of excruciating headache and of pains in the lower extremities. The physician who treated her informed the husband that she was suffering from neuritis. She returned to this country on Jan. 11, 1928, and was admitted to the Jewish Hospital. She then complained of extreme dizziness on turning the head, and of pain in the small of the back and in the calves.

Examination.—The pupils were unequal, the left being the larger. The left pupil reacted fairly well to light, in accommodation and consensually. The right pupil was almost fixed to light but reacted sluggishly in accommodation. No extra-ocular palsies were found. There was a moderate right supranuclear facial weakness. There was marked diminution of all deep reflexes; the ankle jerks were absent. All sensations, including muscle, joint and vibratory sensibilities, were intact. There was a moderate Kernig sign and this maneuver caused excruciating pains in the thighs. The blood pressure was 120 systolic and 80 diastolic. A blood count showed: red cells, 4,800,000; hemoglobin, 80 per cent; white cells, 8,000, with 64 per cent polymorphonuclears. The urine was normal. The temperature was 100.4 F.; the pulse rate was 96, and the respirations were 22. Spinal puncture again produced a bloody fluid, which again showed a four plus Wassermann reaction. The Wassermann reaction of the blood was two plus. Roentgen examination of the skull and spinal column disclosed no evidence of any pathologic changes. The stools were examined for ova and parasites but none were detected.

Course.—The patient continued to complain of pain in the muscles of both calves and in the small of the back, as well as dizziness on turning the head from side to side. On January 17, there was moderate blurring of the left disk margin. The vessels of both disks were moderately engorged. The patient remained in the hospital for two weeks, receiving an occasional hypertonic solution of dextrose intravenously. No further puncture was done as she strongly objected to that procedure. The patient then insisted on going home, and was discharged from the hospital.

At this time she was able to walk about and to take meals with the family, complaining only of headache and of pain in the small of the back and in the calves. The question of antisyphilitic therapy was discussed, and it seemed advisable to start it. On Jan. 27, 1928, she received 0.1 Gm. of sulpharsphenamine intravenously. This was followed on the next day by a rash over the body, and by an elevation of temperature to 102 F. The fever subsided at the end of three days. The patient, however, complained of excruciating frontal and suboccipital headaches, and of pain in the small of the back radiating down the thighs. She also had considerable vomiting. She was then readmitted to the hospital on Feb. 2, 1928.

Examination at Second Admission.—At this time she again presented unequal pupils, the left being somewhat the larger; the left reacted very sluggishly and

the right practically none to light or in accommodation. There was moderate rigidity of the neck and a moderate Kernig sign. The deep reflexes were diminished. There was tenderness in the calf and gluteal muscles. The pulse rate was 92, the temperature 100 F. and the respirations 18. There was a rash over the entire body, and the patient complained of itching of the skin. Spinal puncture again revealed blood-tinged fluid.

Course.—On February 6, the patient complained of intense headache and vomited considerably. The headache commenced in the suboccipital region, radiating upward to the vertex. She then had a convulsion lasting for a few minutes, in which the facial muscles were mostly involved. She was given hypertonic solutions of dextrose intravenously and spinal drainage every second or third day. The condition gradually improved, and on February 11, it was decided again to try the effect of antisyphilitic remedies. She was given bismuth and mercury intramuscularly, three injections of each, on alternate days. On February 17, she received 0.1 Gm. of sulpharsphenamine intravenously. This was followed immediately by the reappearance of the rash, elevation of temperature to 102 F., and by complaints of intense suboccipital headache and pains in the muscles of the back and thighs. A blood count at this time showed 8,000 white cells with 70 per cent polymorphonuclears. The urine was normal except for an occasional leukocyte. The fever gradually subsided. The patient showed considerable improvement and on February 17, was discharged at her request.

Since then the patient has not received any antisyphilitic therapy. She has resumed her activities as a busy housewife and active participant in social activities. She still has the pupillary signs but is free from symptoms otherwise. The blood of the children was examined and showed a negative Wassermann reaction. The husband asserted that the Wassermann reaction of his blood is negative.

Case 2.—*History.*—P. M., a white man, married, an operator on cloaks, was admitted to the Brownsville Hospital on Jan. 30, 1928, and died on Feb. 22, 1928. The family history was without significance. The personal history was that he had had fairly normal habits, smoked twenty cigarets a day, was married in 1913, and had three living children. He was operated on for hemorrhoids and hernia in 1918. For the past ten years he had been complaining of drawing pains in both lower extremities during inclement weather. He said that he had never had a venereal disease. The present illness dated back to the latter part of December, 1927, when he began to have headaches, localized in the parietal region. He showed a tendency to drowsiness. On Jan. 23, 1928, he began to vomit. On Jan. 26, 1928, while sitting at the dinner table, he suddenly collapsed and had to be carried to bed. He was unconscious for a few hours.

Examination.—On admission to the hospital he was conscious and well oriented, and complained of intense headache and of drowsiness. Physical examination disclosed unequal pupils, the left larger than the right. Both were irregular; neither reacted to light, but both reacted in accommodation. The eyegrounds showed moderate blurring of the disks. No extra-ocular palsies were elicited. There was no involvement of the fifth nerve. There was a slight right facial paresis. Hearing was intact. The tongue was protruded in the midline. The biceps and triceps jerks were active; the knee and ankle jerks were absent. No pathologic reflexes were elicited. There were moderate cervical rigidity, a bilateral Kernig sign and a positive Brudzinski sign. There was definite dermographia. Both calves were extremely tender. The temperature was 101 F.; the pulse rate was 60 and respirations 28. A blood count showed: red cells, 4,200,000; hemoglobin, 81 per cent; white cells, 11,000 with 82 per cent polymorphonuclears. The urine was normal.

In view of the close similarity of the clinical pictures between this case and case 1, I concluded and noted that the neurologic signs, with the history of unconsciousness and headache, and the lack of the mental signs observed in epidemic encephalitis, point to an unusual type of syphilitic meningo-encephalitis hemorrhagica.

A spinal tap resulted in a bloody spinal fluid under four plus pressure. The spinal fluid was centrifugated, and the supernatant clear fluid gave a four plus Wassermann reaction. The Wassermann reaction of the blood was also reported as four plus. The blood chemistry was normal. A Widal test was negative. On

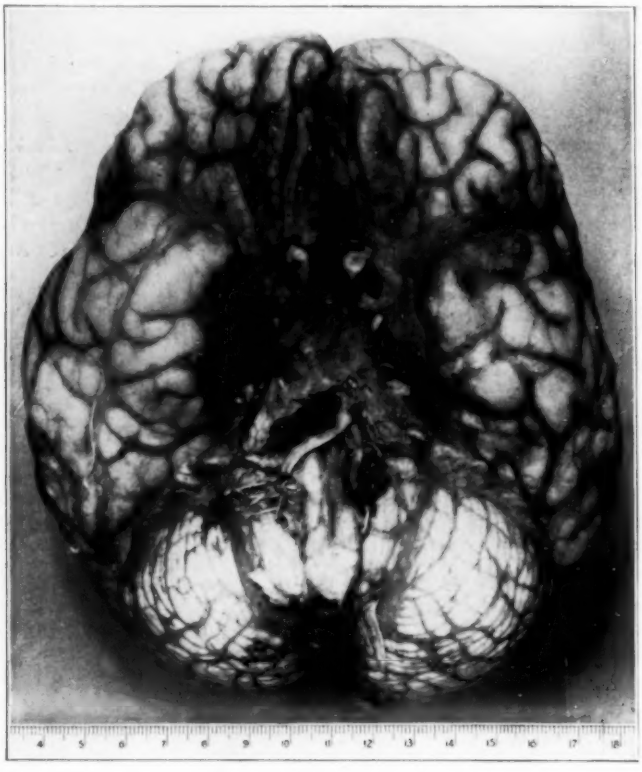

Fig. 1.—Extensive basal hemorrhage.

admission to the hospital the patient said that he had never had syphilis. When confronted with the serologic reports, he admitted having had a chancre in 1910.

Course.—On Feb. 10, 1928, it was noted that the patient showed a persistence of all neurologic signs with increasing rigidity of the neck and an increasing Kernig sign. A spinal puncture again resulted in bloody fluid. On Feb. 16, 1928, 25 cc. of blood-tinged fluid was removed under three plus pressure.

On Feb. 19, 1928, the patient still complained of headache and pain in the calves. The pupils were unequal, the left larger than the right, and both were of the Argyll Robertson type. The left eyeground showed blurring of the margins and a general hyperemia of the disk. There was marked cervical rigidity and a

bilateral Kernig sign. The deep reflexes were not elicited. Fifteen cubic centimeters of xanthochromic fluid, not bloody, was obtained under normal pressure. He was given three injections of bismuth and three of mercury on alternate days. Medication was discontinued on February 5, in view of our experience with the first patient. The temperature ranged between 100 and 101 F., the pulse rate between 76 and 96, and the respirations from 28 to 18 from the day of admission until February 6. The temperature then became normal. The patient showed considerable improvement. On February 18, he was permitted to sit up. On February 22, at 5 a. m., he asked the nurse for some water. Half an hour later he had a convulsion, which lasted for a few minutes, and died.

Postmortem Examination.—The dura appeared clear. When it was incised a large amount of bloody fluid escaped. The brain weighed 1,350 Gm. At the

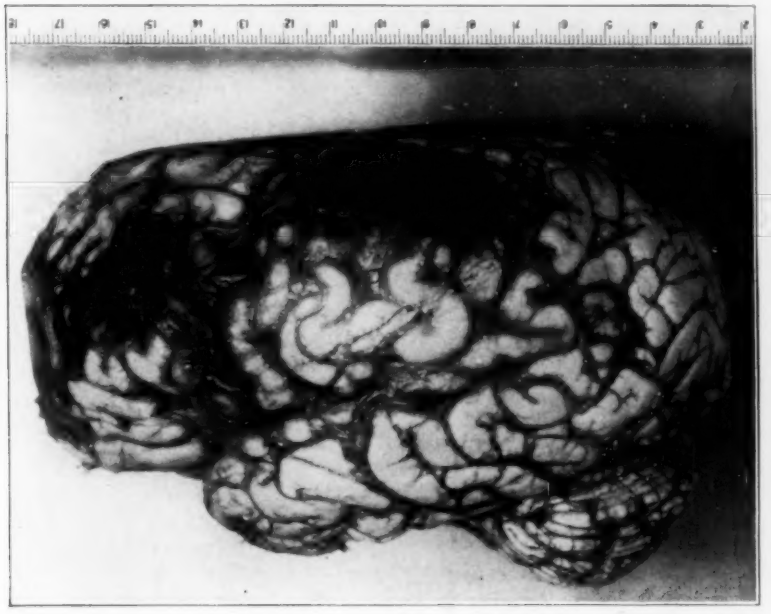

Fig. 2.—Hemorrhage over the frontal and parietal regions.

base of the brain (fig. 1) there was an extensive hemorrhage which filled the middle and posterior fossae, and surrounded the midbrain, pons, medulla and cerebellum. On the vertex of the brain (fig. 2), the pia-arachnoid showed extensive bloody infiltration over the frontal and parietal areas. The fourth ventricle was filled with blood.

Microscopic examination showed an apparently normal dura; no hyperplasia nor inflammatory areas were found. The pia-arachnoid showed marked hyperplasia and thickening. A considerable amount of hemorrhage was found in many areas which separated the pia from the arachnoid (fig. 3). There was marked infiltration of the pia-arachnoid with lymphocytes and plasma cells. In several areas this infiltration was so extensive and dense as to suggest the appearance of a miliary gumma. The pial vessels showed an endarteritis typical of syphilis. The vessels were surrounded by a collar of lymphocytes and plasma cells (fig. 3).

In a few the lumens were obliterated. The cortical vessels showed infiltration of the Virchow-Robin lymph spaces with lymphocytes and plasma cells. The ganglion cells showed a normal layer arrangement but presented considerable cloudy swelling, chromatolysis, satellitosis and neuronophagia. There was marked neuroglial reaction in the cortex. Numerous protoplasmic and fibrous glia cells, as well as oligodendroglia and microglia, were seen. No hemorrhagic areas were noted in

Fig. 3.—Note the hemorrhage and the characteristic vascular changes in the medulla.

the brain tissue proper. Sections from the medulla revealed considerable hemorrhage, especially in the lateral recess (fig. 3) and considerable endarteritis of the vessels. The superficial medullary capillaries showed considerable infiltration of the walls with lymphocytes and plasma cells.

The aorta showed linear scars and puckering in the arch and thoracic portions. Microscopically, there was extensive proliferation of the media with infiltration by lymphocytes.

COMMENT

The outstanding features in the cases were the onset of headache, the tendency to drowsiness, pain in the occipital region and in the small of the back, followed in a few weeks by unconsciousness with excruciating headache and pain in the lower extremities. There was evidence of some blurring of the disks. There was moderate cervical rigidity and Kernig phenomena. The deep reflexes were diminished. The spinal fluid was bloody; it showed uniform coloring of the different specimens collected in separate test tubes. There was no coagulation of this bloody spinal fluid, and, when centrifugated, the supernatant fluid was a golden yellow. These signs and symptoms pointed to the presence of a subarachnoid hemorrhage. Obviously there was leakage in the subarachnoid space for several weeks, the blood slowly invading the sheaths of the optic nerves and especially those of the lumbosacral plexus. Finally, there must have been an excessive hemorrhage into the subarachnoid space resulting in increased intracranial pressure causing headache, vomiting and convulsions.

The syndrome of subarachnoid hemorrhage has been ably discussed by Bramwell,[1] Neal,[2] and especially by Symonds,[3] who analyzed 127 cases and presented his observations in a classical paper. He noted that syphilis was the primary cause in ten cases, in which five patients died and five recovered. Herman [4] reported twenty-four cases of subarachnoid hemorrhage, of which two were of syphilitic origin. Neylahn [5] reported two cases of spontaneous diffuse meningeal hemorrhage, but could not demonstrate any ruptured vessel in the pathologic material. One of these cases was caused by syphilitic disease of the meningeal blood vessels. He concluded that this condition was due to diapedesis of red blood cells, claiming that the cause of the production of the hemorrhage was a disturbance in the function of the vasomotor mechanisms secondary to disease of the vessel walls.

One must remember that, as a general rule, hemorrhage is not commonly encountered in cases of neurosyphilis, except in dementia paralytica in which repeated bleeding produces chronic internal hemorrhagic pachymeningitis. In an exhaustive study of fifty-five cases of syphilitic

1. Bramwell, B.: Clinical and Pathological Memoranda, Edinburgh M. J. **32**:1 (July) 1886.

2. Neal, Josephine B.: Spontaneous Meningeal Hemorrhage, J. A. M. A. **86**:6 (Jan. 2) 1926.

3. Symonds, C. P.: Spontaneous Subarachnoid Hemorrhage, Quart. J. Med. **18**:93 (Oct.) 1924.

4. Herman, E.: Zur Frage der subarachnoidal Blutungen, Ztschr. f. d. ges. Neurol. u. Psychiat. **105**:667 (Nov.) 1926.

5. Neylahn, K.: Ueber spontane diffuse Meningealblutungen, Deutsche Ztschr. f. Nervenh. **78**:78 (April) 1923.

leptomeningitis, Le Count and Dewey [6] recorded only three in which pial hemorrhage was found. One must therefore agree with Turner [7] that cerebral hemorrhage arising directly from syphilitic causes is rare. Ordinarily there is in neurosyphilis a characteristic endarteritis resulting in thrombosis. This obliterating endarteritis is mentioned by both Malamud [8] and Robustow [9] as playing the dominant rôle in the pathology of neurosyphilis. Warthin,[10] in his classic monograph on the pathology of syphilis, describing syphilis of the nervous system stated that thickening of the wall and more or less obliteration of the meningeal vessels are usually associated with local fibrosis and focal thickenings of the leptomeninges, the endarteritis playing a prominent part in the disease process. However, in some cases there is intense inflammation with necrosis in the adventitia and media before the latter becomes greatly thickened, resulting in rupture of the degenerated vessel wall and in the production of hemorrhage. In a critical analysis of two cases of hypertrophic pachymeningitis, Hassin [11] concluded that vascular changes, especially in the pia-arachnoid, are the real primary factors instrumental in the formation and organization of the pseudomembranes found in that disease process. He said that the ruptured vessels lead to hemorrhages which fill up the subarachnoid space.

The serologic observations in my cases gave the clew to the underlying etiology and pathology. Case 1 illustrates the fallacy of depending on one negative Wassermann reaction of the blood as the sole criterion on which to exclude neurosyphilis. Such a step led a neurologist to make a wrong diagnosis and to give advice which jeopardized the life of the patient.

Considerable difficulty was encountered in treating these patients. The question of spinal drainage is indeed a delicate one to decide. When the diseased capillary or vessel wall ruptures, a coagulum is formed at the site of the rupture. This coagulum is partly supported by the surrounding cerebrospinal fluid. Spinal drainage, therefore, may cause the removal of this protecting spinal fluid water jacket, and result in the

6. Le Count, E. R., and Dewey, K.: Syphilitic Leptomeningitis, J. Infect. Dis. **16**:142 (March) 1915.

7. Turner, W. A., in Albutt and Rolleston: System of Medicine, New York, The Macmillan Company, 1911, vol. 8, p. 339.

8. Malamud: Zur Klinik und Histopathologie der chronischen Gefässlues im Zentralnervensystem, Ztschr. f. d. ges. Neurol. & Psychiat. **102**:778 (June) 1926.

9. Robustow: Klinische und histologische Beiträge aus dem Gebiete der chronischen Syphilis des Zentralnervensystems mit besonderer Berücksichtigung der Gefässlues, Ztschr. f. d. ges. Neurol. u. Psychiat. **102**:757 (June) 1926.

10. Warthin, A. S.: The New Pathology of Syphilis, Am. J. Syph. **2**:425 (July) 1918.

11. Hassin, G. B.: Histogenesis of Cerebral Hypertrophic Pachymeningitis and Its Relation to Syphilis, Am. J. Syph. **2**:714 (Oct.) 1918.

giving way of the coagulum because of the blood pressure, and produce a new hemorrhage. In the later stages, when the coagulum is well organized and especially when there are signs of increasing intracranial pressure, spinal drainage may be judiciously employed.

The institution of specific syphilitic therapy is also a debatable question. The administration of arsphenamine, even though in minute doses, resulted in a Herxheimer reaction in the first patient on two occasions. It would therefore seem that the different arsphenamine preparations should be delayed until recovery from the subarachnoid hemorrhage is fully established. Possibly the administration of bismuth and mercury might suffice for some time.

SUMMARY

1. Subarachnoid hemorrhage complicating neurosyphilis is a clinical entity probably caused by rupture of diseased pial capillaries or vessels.

2. The underlying pathologic process is syphilitic meningitis demonstrated by thickened leptomeninges, infiltration with lymphocytes and plasma cells, marked endarteritis of the fine capillaries of the pia and hemorrhage in the subarachnoid space. The brain tissue proper shows marked glial reaction and syphilitic endarteritic processes in the capillaries.

3. The clinical picture presented is that of subarachnoid hemorrhage plus the presence of positive serologic tests.

4. Two cases are presented illustrating these points.

202 New York Avenue.

MEASUREMENTS OF CEREBRAL AND CEREBELLAR SURFACES

IX. MEASUREMENT OF CORTICAL AREAS IN CAT, DOG AND MONKEY.*

JOSEPH J. MICHAELS, M.D.
DETROIT
AND
WALTER M. KRAUS, M.D.
NEW YORK

In a previous article [1] the motor surface areas of the cat, dog, monkey and man were measured, and certain relationships were shown. This area was chosen first because of its grosser characteristics and its easier delimitation.

German writers contributed extensively in the field of mensuration and cyto-architecture in different animals. Brodmann [2] studied the cortical fields in the animals that are most commonly employed in laboratory experiments. Gurewitsch, Chatschaturian and Chatschaturow,[3] in 1928, devised a new method for measuring cortical fields in their studies on the cat which is somewhat analogous to the method described by Kraus, Davison and Weil.[4] They made use of parallel sections, and the side surface of each single section was equal to the surface area of a trapezoid. The length of the parallel sides of the trapezoid corresponds to the length of the contours which border the section. The height of the trapezoid is known, being equal to the thickness of the section; the side surfaces of the highest and lowest sections are taken as triangles. In a chart they showed the sections, which, after being drawn, were unrolled from the same given point, thereby showing the visible and invisible sur-

* Submitted for publication, Dec. 2, 1929.
* From the Neuropathological Laboratory, Montefiore Hospital, New York.
* This work was carried on under the auspices of the Hilda Stich Stroock Scholarship.

1. Michaels, J. J., and Davison, C.: Measurement of Cerebral and Cerebellar Surfaces: VIII. The Measurement of Motor Area in Some Vertebrates and Man, Arch. Neurol. & Psychiat. **23**:1212 (June) 1930.

2. Brodmann, K.: Beiträge zur histologischen Lokalisation der Grosshirnrinde. Mitteilung I-VII, J. f. Psychol. u. Neurol. **2**:79 and 133, 1903; **4**:177, 1905; **6**:108 and 275, 1905; **10**:231, 1907; 287, 1908.

3. Gurewitsch, M.; Chatschaturian, A., and Chatschaturow, A.: Methodik der Zeichnung cytoarchitektonischer Karten und der Messung der Felder, Ztschr. f. d. ges. Neurol. u. Psychiat. **114**:50, 1928.

4. Kraus, W. M.; Davison, C., and Weil, A.: Measurement of Cerebral and Cerebellar Surfaces: III. Problems Encountered in Measuring the Cerebral Cortical Surface in Man, Arch. Neurol. & Psychiat. **19**:454 (March) 1928.

faces. They found that the area striata was 15.9 per cent, the motor area 5.4 per cent and the total surface of one hemisphere 16.40 sq. cm. in the cat, of which 29 per cent was in the depths of the fissures.

S. Rose,[5] in 1927, measured the particular cortical regions in the allocortex of man and of various animals. Some of his conclusions were that the receptive surface for olfactory impressions is smallest in primates; the olfactory bulb as compared with the entire cortex is

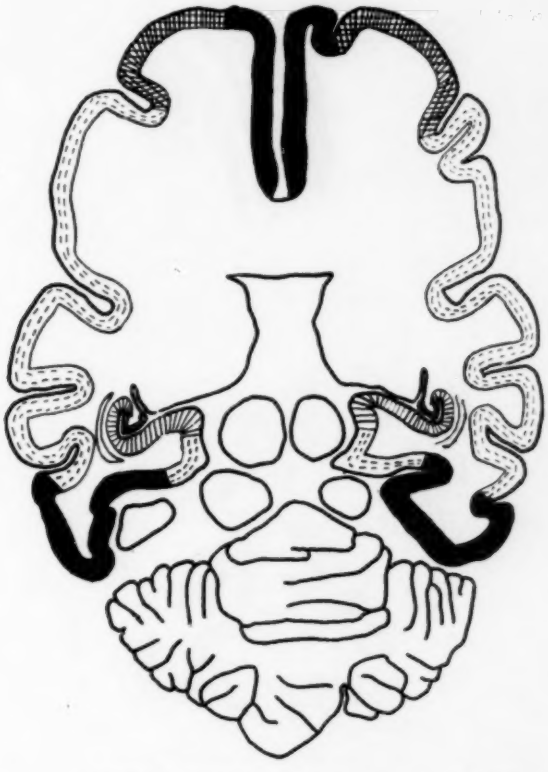

Fig. 1.—Horizontal section of the brain of a cat 11 mm. from the top; shaded area, anteriorly, frontal area; shaded area, posteriorly, visual area; crosshatched area, motor area; dotted lines, temporoparietal area; straight lines, olfactory area; × 6.

considerably larger in lower mammals than in primates. In the dog, he found that the bulbus olfactorius, tuberculum olfactorium, cortex semiparietinus and cornu ammonis were 11.80 sq. cm. All of the aforementioned writers have not considered shrinkage as a factor.

5. Rose, S.: Comparative Measurements of the Allocortex in Animals and in Man, abstr. by M. Keschner, J. f. Psychol. u. Neurol. **34**:250, 1927.

METHOD OF PROCEDURE

The present work deals with the functional areas of the brain, and accordingly more difficulty was encountered in dividing the brain into measurable areas. Five large divisions were used because it was thought that further differentiation would lead to too many errors in deciding between the boundaries of the functional areas. The areas used were the olfactory, the visual, the motor, the temporoparietal and the frontal areas in the cat, dog and monkey. The technical procedure and calculations were the same as the method used in the determination of the motor surface area.[1]

Fig. 2.—Horizontal section of the brain of a dog 16 mm. from the top: shaded area, anteriorly, frontal area; shaded area, posteriorly, visual area; dotted lines, temporoparietal area; straight lines, olfactory area; × 6.

Ammon's horn was considered as the olfactory area. Part of the olfactory bulb in which definite cortical layers could be seen was also included in this area.

As the basis for the limits of the visual area, the line of Gennari was used. In the cat and dog, the line of Gennari was much more difficult to visualize because of its reduction in comparison to that in the monkey.

The temporoparietal region was considered as the area between the motor area and the occipital area. In the cat and dog, the temporoparietal region included the postsensory, the ectosylvian, the extrarhinic and the limbic regions

TABLE 1.—*Measurement of the Olfactory Area*

Animal	Total Perimeter of the Total Surface	Olfactory Perimeter	Ratio of $\frac{t\ p}{o\ p}$	Surface Area	Olfactory Surface Without Shrinkage	Olfactory Surface With Shrinkage
Cat	213.929	51.78	4.13	23.571	5.70	7.60
Dog	645.305	133.23	4.85	75.828	15.60	20.17
Monkey	484.227	40.817	11.88	67.54	5.68	6.76

TABLE 2.—*Measurement of the Visual Area*

Animal	Total Perimeter	Visual Perimeter	Ratio	Surface Area	Visual Surface Area Without Shrinkage	Visual Surface Area With Shrinkage
Cat	235.148	32.19	7.3	33.21	4.56	6.08
Dog	1024.49	129.24	7.93	126.188	15.92	20.59
Monkey	636,885	122.621	5.19	91.256	17.6	20.94

TABLE 3.—*Measurement of the Temporoparietal Area*

Animal	Total Perimeter	Temporo-parietal Perimeter	Ratio	Surface Area	Temporo-parietal Surface Without Shrinkage	Temporo-parietal Surface With Shrinkage
Cat	361.092	295.38	1.22	47.04	38.50	51.36
Dog	1212.755	743.77	1.63	155.288	95.30	123.22
Monkey	846.762	564.53	1.5	127.096	84.70	100.80

TABLE 4.—*Measurement of the Frontal Area*

Animal	Total Perimeter	Frontal Perimeter	Ratio	Surface Area	Frontal Surface Area Without Shrinkage	Frontal Surface Area With Shrinkage
Cat	174.379	41.36	4.22	18.516	4.38	5.84
Dog	1094.68	197.88	5.54	137.288	24.8	32.07
Monkey	788.395	203.47	3.88	117.406	30.2	35.94

TABLE 5.—*Total Measurement of Cortical Areas*

Animal	Total Surface Area	Percentage of Shrinkage	Olfactory Surface Area	Percentage to Total	Visual Surface Area	Percentage to Total	Motor Surface Area	Percentage to Total	Temporoparietal Surface Area	Percentage to Total	Frontal Surface Area	Percentage to Total
Cat	74.54	33.4	7.60	10.19	6.08	8.15	3.66	4.9	51.36	68.7	5.84	7.8
Dog	211.33	29.3	20.17	9.55	20.59	9.75	14.28	6.75	123.22	58.4	32.07	15.2
Monkey ...	169.24	19.0	6.76	4.00	20.94	12.50	4.79	2.83	100.80	59.5	35.94	21.20

(Campbell [6]). Temporoparietal is not an exact term, but for our purposes it was used to include the general sensory fields grouped into one region (the homolog of the second, third and fourth temporal gyri is undefinable in lower forms). In our measurements, our total surface areas of the three animals was larger than that given by Davison and Kraus,[7] because every twentieth section was measured, and the olfactory regions were included.

It may be emphasized that the visual area in the cat and dog was found more dorsally and laterally, and in the monkey migrated to a more medialward posi-

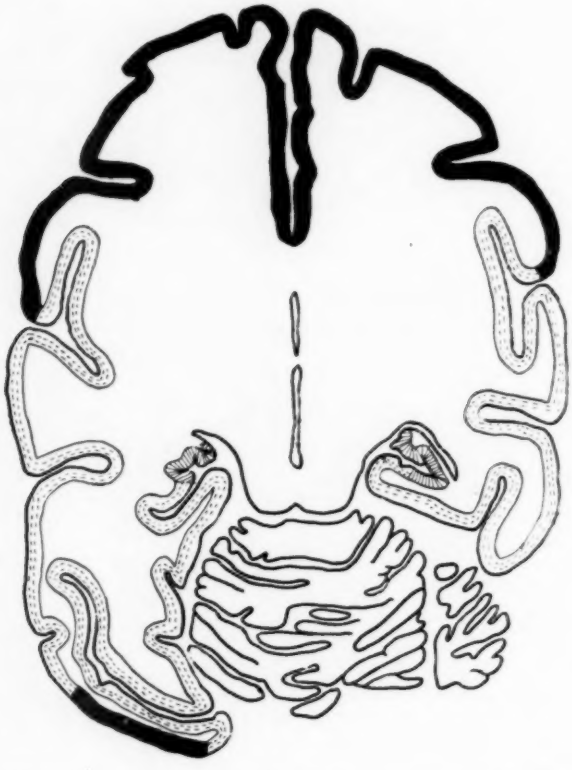

Fig. 3.—Horizontal section of the brain of a monkey 17 mm. from the top: shaded area, anteriorly, frontal area; shaded area, posteriorly, visual area; dotted lines, temporoparietal area; straight lines, olfactory area; × 6.

tion. As the animal scale is ascended, the distance between the postcentral and occipital regions increases steadily, and the motor area from a most frontal loca-

6. Campbell, A. W.: Localisation of Cerebral Function, London, Cambridge Univ. Press, 1905.

7. Davison, C., and Kraus, W. M.: The Measurement of Cerebral and Cerebellar Surfaces: VII. The Measurement of Visible and Total Cerebral Surfaces of Some Vertebrates and of Man, Arch. Neurol. & Psychiat. **22**:105 (July) 1929.

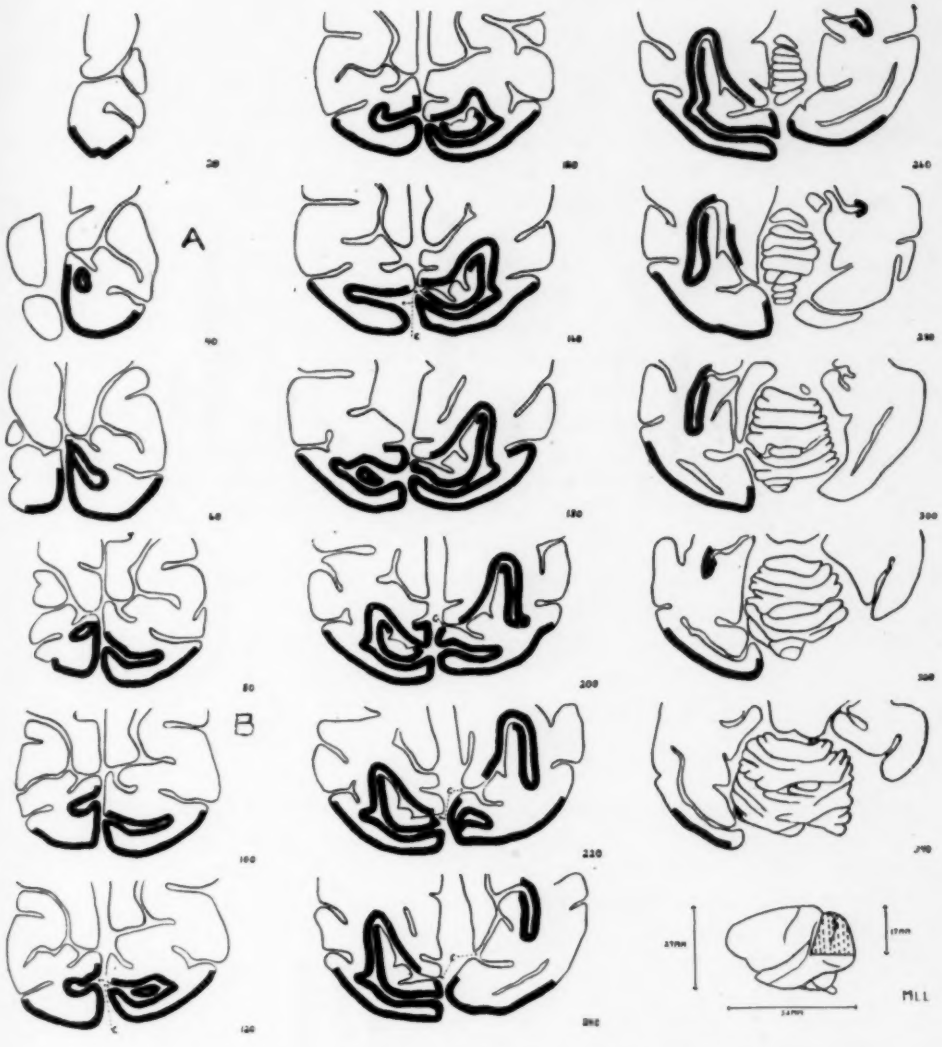

Fig. 4.—Visual area in monkey (area striata), the cortical area containing the area striata. The first section 20 is the uppermost. Every twentieth section (50 microns thick) is represented. Section 340 is the lowermost. This shows the great extent of the cortical area which is concealed (invisible) as well as that which is on the surface (visible). The diagram in the right hand lower corner shows the upper and lower limits of the area striata. The horizontal line beneath the shaded area represents the lower limit.

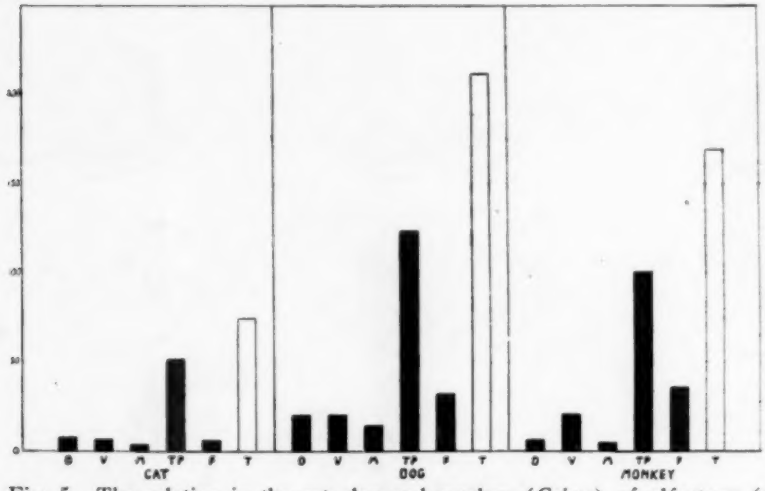

Fig. 5.—The relation in the cat, dog and monkey (*Cebus*) of olfactory (*o*), visual (area striata) (*v*), Betz cell area (*m*), temporoparietal (*tp*), frontal (*f*) areas and total (*t*) expressed in square centimeters.

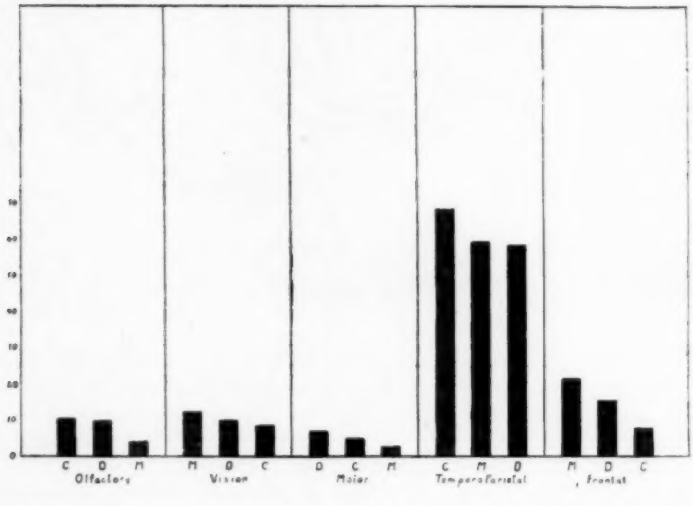

Fig. 6.—The relation in the cat (*c*), dog (*d*) and monkey (*Cebus*) (*m*) of olfactory, visual (area striata), Betz cell area, temporoparietal and frontal areas expressed in percentages.

tion moves in a caudal direction. Figure 4 shows that the distribution of the area striata in the monkey is present to a large extent in the invisible portion of the cortex as well as in the visible surface.

COMMENT

The various areas in the cat, dog and monkeys were measured and calculated as shown in the accompanying tables. Figures 5 and 6 show that in the monkey the visual and frontal areas are actually and relatively larger than in the cat and dog; the olfactory and visual areas are about equal in the cat and dog, whereas in the monkey the visual area is more than twice the olfactory area. In all the animals, the temporoparietal area, which includes a number of areas of different functions, was relatively the highest. It is generally accepted that in the lower forms the regions of the cortex primarily essential for survival are the largest, namely, olfactory, visual, hearing, general sensation and locomotion. In comparing the cat and the monkey, the percentage of olfactory area to total surface was 10.19 and 4, whereas the percentage of frontal area to total surface in the cat and the monkey was 7.8 and 21.2, respectively.

In 1912, Brodmann [8] gave the percentage that the frontal area represented of the total surface in the cat as 3.4; in the dog, 6.9; in the monkey, from 8.3 to 16.9, and in man, 29 per cent. It is interesting that our figures are relatively higher, although a certain approximation occurs in the ratio of 1:2:3. Brodmann gave the area striata in apes as about 10 per cent and in lemurs as 15 per cent. Our observations of 12.5 per cent compare favorably. Von Economo and Koskinas [9] mentioned that the area striata in man is from 2 to 2.5 per cent of the total surface.

CONCLUSIONS

The cortical areas of the cat, dog and monkey were measured. The temporoparietal area in each animal formed the greatest amount of cortical surface area. The olfactory cortex was relatively high in the cat and dog and low in the monkey; the frontal and visual (area striata) cortex was highest in the monkey, and lowest in the cat. The Betz cell area was relatively larger in the dog and cat, and smaller in the monkey.

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8. Brodmann, K.: Neue Ergebnisse über die vergleichende histologische Localisation der Grosshirnrinde mit besonderer Berücksichtigung des Stirnhirns, Anatomischer Anzeiger, Verhandlungen der anatomischen Gesellschaft, April, 1912, p. 211.

9. Von Economo, C., and Koskinas, G. N.: Die Cytoarchitektonik der Hirnrinde des erwachsenen Menschen, Berlin, Julius Springer, 1925.

RADIOGENIC MICROCEPHALY

A SURVEY OF NINETEEN RECORDED CASES, WITH SPECIAL REFERENCE TO OPHTHALMIC DEFECTS *

LEOPOLD GOLDSTEIN, M.D.
PHILADELPHIA

During an investigation of the effect of maternal pelvic irradiation on the health and development of subsequent children, several interesting and vitally important observations have been made, as reported in earlier papers.[1] The most significant conclusion reached is that pelvic radiotherapy during pregnancy is extremely injurious to the fetus.

Severe disturbances of the central nervous system were observed to be of common occurrence among children irradiated in utero. Thus, of seventy-five children so exposed, twenty (26.6 per cent) exhibited marked defects, eighteen (24 per cent) being microcephalic idiots.

Since the completion of these studies, attention has been directed to a report of another microcephalic child who also had been irradiated in utero.[2] The present paper deals with these nineteen microcephalic children, and particularly stresses the ocular defects.

In a previous paper, a survey was made of the factors possibly responsible for the ill health of thirty-eight children irradiated in utero.[3] Heredity, syphilis, the general health of the mother prior to and during pregnancy and the circumstances incident to labor were among the conditions studied. Sixteen cases of microcephaly, however, could not be ascribed to any of these factors and were therefore attributed to the fetal irradiation. In two of these cases there seemed to be some evi-

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* Submitted for publication, Aug. 5, 1929.

* Microcephaly caused by the deleterious action of radium, the roentgen ray or mesothorium on the fetus in utero.

* Read at a meeting of the Philadelphia Neurological Society, Oct. 25, 1929.

* From the Gynecean Hospital Institute of Gynecologic Research, University of Pennsylvania.

1. Goldstein, L., and Murphy, D. P.: Etiology of the Ill-Health in Children Born After Postconception Pelvic Irradiation, Am. J. Roentgenol. **22**:322, 1929; Amenorrhea Following Serial Roentgen Pelvic Exposures Due to Intervening Pregnancy, Am. J. Obst. & Gynec. **18**:696, 1929. Murphy, D. P.: Ovarian Irradiation and the Health of the Subsequent Child: A Review of More than 200 Previously Unreported Pregnancies in Women Subjected to Pelvic Irradiation, Surg. Gynec. Obst. **48**:766, 1929. Murphy, D. P., and Goldstein, L.: Etiology of the Ill Health in Children Born After Preconception Pelvic Irradiation, Am. J. Roentgenol. **22**: 207, 1929.

2. Zimmermann, R.: Cervix Carcinoma und Schwangerschaft, unter Berucksichtigung der Frage einer Strahlenschaedigung der Frucht, Strahlentherapie **29**: 108, 1928.

3. Goldstein and Murphy (footnote 1, first reference).

dence that parental influences had played a rôle: in one, the mother was tuberculous and the father was a victim of chronic alcoholism; in the other,[4] the mother had (prior to irradiation) borne a child who exhibited a nervous condition associated with "spasms." In both cases, the mothers had received intensive pelvic irradiation in the early months of pregnancy, and this treatment may have been the sole etiologic factor in the production of the microcephaly. For this reason, these two children have been included in the nineteen discussed here. Although several other mothers suffered from "delicate constitution," tuberculous peritonitis or other abnormal conditions, and although almost all of them manifested some type of pelvic disease or disturbance of the menstrual function, these pathologic conditions were not thought to be etiologically related to the arrested mental development of the children.

For the purpose of this study, I have accepted the conclusion reached in previous papers from this Institute, viz., that pelvic radium or roentgen irradiation during pregnancy frequently inhibits the development of the central nervous system, as manifested by microcephaly at or shortly after birth.

Since the microcephalic children were not described in detail in previous papers, and because they had certain pathologic conditions in common, they are discussed in this paper.

Disturbances of the eye, ranging from congenital abnormalities to inflammatory conditions, were frequently observed in these children. This suggested that there might be a genetic relationship between the fetal irradiation and the congenital ophthalmic defects. If such a relationship could be demonstrated, certain defects of the eyes might then be considered pathognomonic of "radiogenic microcephaly."

Data concerning the details of the maternal irradiation and the health of the subsequent microcephalic children were secured through a review of the literature, supplemented by personal communications from the original observers.

The first case of microcephaly resulting from fetal irradiation was reported by Aschenheim.[4] Since then, a number of foreign observers and one American author[5] have placed additional cases on record. Murphy and I[6] recently reported a case of microcephaly following pelvic irradiation for a carcinoma of the cervix complicating pregnancy.

4. Aschenheim, E.: Personal communication to the author; Schaedigung einer menschlichen Frucht durch Roentgenstrahlen, Arch. f. Kinderh. **68**:131, 1920.

5. Little, H.: Personal communication to the author; in discussion on Matthews, H. B.: The Effect of Radium Rays upon the Ovary: An Experimental, Pathological and Clinical Study, Am. J. Obst. & Gynec. **6**:615, 1923.

6. Goldstein, L., and Murphy, D. P.: Microcephalic Idiocy Following Radium Therapy for Cancer of the Cervix During Pregnancy, Am. J. Obst. & Gynec. **18**:189, 1929.

MATERNAL TREATMENT

The information relative to the amount of maternal irradiation is presented in table 1. Sixteen women were treated with the roentgen ray, two with radium and one with mesothorium. From an analysis of the details of treatment it can be stated that relatively large doses

TABLE 1.—*Details of Maternal Treatment*

Author and Date of First Report	Irradiation	Number of Treatments	Total Dosage of Exposure	Time of Treatment	Indication
Abels: Wien. klin. Wchnschr. **37**: 869, 1924	X-ray	3	8 S. E. D.*	2nd month	Myoma uteri
Apert and Kermorgant: Presse méd. **21**: 1020, 1923	X-ray	17	Deep therapy	1st to 3rd month	Myoma uteri
Aschenheim,[4] 1920	X-ray	2	80 minutes	1st to 3rd month	Myoma (probably only pregnancy)
Deutsch: Monatschr. f. Kinderh. **31**: 284, 1926	X-ray	2	2 S. E. D.	3rd month	Myoma uteri
Falkenheim: Discussion on Deutsch, 1926	X-ray	2	Unknown	1st to 3rd month	Sterilization because of frequent pregnancies
Flatau: Zentralbl. f. Gynäk. **45**: 1545, 1921	X-ray	2	"Ovarial dose"	2nd month	Myoma (probably only pregnancy)
Ganzoni and Widmer: Strahlentherapie **19**: 485, 1925	X-ray	1	36 minutes over 4 fields	3rd month	Induction of abortion and sterilization (pulmonary tuberculosis)
Goldstein and Murphy,[6] 1929	Radium	1	4,440 mg.	6th month	Carcinoma of cervix
Koehmann: Discussion on Deutsch, 1926	X-ray	Unknown	Unknown	1st month	Myoma uteri
Kupferberg: Zentralbl. f. Gynäk. **48**: 2031, 1924	X-ray	1	⅓ "castration dose"	5th month	Tuberculous peritonitis
Little,[5] 1923	Radium	1	Unknown	2nd month	Myoma (probably only pregnancy)
Naujoks: Monatschr. f. Geburtsh. u. Gynäk. **68**: 40, 1924	X-ray	3	"Sarcoma dose"	1st to 4th month	Postoperative sarcoma of one ovary
Petenyi: Klin. Wchnschr. **2**: 566, 1923	X-ray	Unknown	Deep therapy	5th to 7th month	Carcinoma of cervix
Schiffer: Cited by Petenyi, 1923	X-ray	2	Deep therapy	3rd to 4th month	Myoma uteri
Schwaab: Presse méd. **32**: 566, 1924	X-ray	16	Unknown	1st to 5th month	Myoma uteri
Stettner: Jahrb. f. Kinderh. **95**: 43, 1921	X-ray	4	60-70% S. E. D.	3rd month	Myoma uteri
Werner: Arch. f. Gynäk. **129**: 157, 1926	X-ray	3	Unknown	1st to 3rd month	Metrorrhagia
Zappert: Wien. klin. Wchnschr. **28**: 609, 1925	X-ray	2	6 H †	2nd month	Amenorrhea
Zimmermann,[3] 1928	Mesothorium	3	8,400 mg.	5th to 7th month	Carcinoma of cervix

* Skin erythema dose.
† Holzknecht unit.

were given to all of the women. Many of them received more than one roentgen treatment (table 1).

Fifteen women received radiotherapy during the first four months of gestation and several of them were given one or more treatments in the first month. The relationship between the time of treatment and the characteristic defects manifested by the microcephalic children

is discussed later. In seven instances the embryo was irradiated in the first few weeks of its existence, at a period when the central nervous system is in its most active stage of differentiation and development. It is of interest to note that several women had received one or more roentgen exposures prior to the onset of pregnancy. That conception may take place between any two of a series of roentgen treatments has already been pointed out.[7]

The various indications for radiotherapy are recorded in table 1. It is observed that myoma uteri was the most common indication for the treatment, being present in seven of the nineteen women. As pointed out in a previous publication,[8] pelvic disease probably exerted no influence in the production of abnormalities or malformations in the children.

In three cases, a pregnant uterus was mistaken for a myomatous one. Preliminary curettage of the uterus would eliminate the danger of unwittingly irradiating an embryo. Norris [8] has emphasized the value of diagnostic curettage in determining the exact pathologic condition of the endometrium before employing either radium or the roentgen ray in the treatment for pelvic disease. This procedure would disclose the presence of a previously unsuspected fundal carcinoma, not infrequently associated with myoma uteri.

Two observers attempted to abort and sterilize a patient by the roentgen ray, in one case because of a rapid succession of pregnancies and in the other because of pulmonary tuberculosis. In both instances the irradiation failed to induce abortion and, on the refusal of the patients to allow further efforts to empty the uterus, the gestation continued to term.

Pregnancy was complicated by carcinoma of the cervix in three of the women, while tuberculous peritonitis, functional metrorrhagia, amenorrhea and sarcoma of the ovary (oophorectomy prior to irradiation) were the conditions for which the patients were treated in the remaining four cases.

DESCRIPTION OF MICROCEPHALIC CHILDREN

The characteristics of the nineteen microcephalic children are listed in tables 2 and 3. The weights at birth, the various defects of the eyes and other abnormalities, as well as the condition of the children at the time of last observation, are recorded in these tables.

Eleven of the fourteen children, whose birth weights are known, weighed below 2,500 Gm. at birth, while only three weighed over 3,000

7. Goldstein and Murphy (footnote 1, second reference).

8. Norris, C. C.: Personal communication to the author.

Gm. The average weight was 2,130 Gm. According to DeLee, the average weight of the new-born infant is 3,400 Gm. It is seen, therefore, that thirteen children were distinctly underweight at birth.

Fifteen children were born at term; the four reported by Falkenheim, Flatau, Stettner and Werner were born prematurely. Of these children, three weighed 2,500 Gm. or less (the weight of the child reported by Werner is unknown). The small size of the full-term children at birth may probably be correctly attributed to the general inhibiting influence of irradiation on the entire embryo.

TABLE 2.—*Contour of Head in Microcephaly*

Author	Shape of Head	Mongolism	Fronto-Occipital Circumference
Abels	Occiput flattened	Absent	27.5 cm. at birth
Apert and Kermorgant	Small round	Present	Not stated
Aschenheim	Prominent parietal bosses	Absent	Normal (49.5 cm.) 42 cm. at 3½ years
Deutsch	Oxycephalus, "birdlike"	Absent	20 cm. at birth
Falkenheim	Occiput flattened	Absent	39.5 cm. at 9 months
Flatau	Small round	Not stated	Not stated
Ganzoni and Widmer	Oxycephalus "turmschaedel"	Absent	Not stated
Goldstein and Murphy	Occiput flattened	Absent	Not stated
Kochmann	Not stated	Not stated	Not stated
Kupferberg	Small round	Present	Not stated
Little	Small round	Absent	Not stated
Naujoks	Oxycephalus, "birdlike"	Absent	39 cm. at 1 year (normal, 46 cm.)
Petenyi	Right occiput and parietal region flattened	Absent	Not stated
Schifler	Not stated	Not stated	Not stated
Schwaab	Small round	Present	27 cm. at birth
Stettner	Small round	Absent	27.5 cm. at birth
Werner	Small round	Present	Not stated
Zappert	Small round	Present	35 cm. at 8 months (normal, 44 cm.)
Zimmermann	Small round	Absent	Not stated

General Features.—As is shown in table 2, the configuration of the heads of seventeen children is known. The heads of nine children were described as being "small and round." Three had the oxycephalic type of head, also known as "tower head," "domelike" or "turmschaedel." In four children the occiput was noticeably flattened, while in one case the parietal bosses were prominent.

Five children, with small round heads, exhibited the facial features of the mongoloid idiot. Mongolism was pronounced absent in eleven, and was not mentioned in three cases.

The circumference of the head of the microcephalic children is recorded in table 2. Three children had a head girth of from 27 to 27.5 cm. at birth, and one had a girth of only 20 cm.

OCULAR DEFECTS IN MICROCEPHALIC CHILDREN IRRADIATED IN UTERO

Reports were available concerning ocular defects in fifteen of the nineteen children. The various disturbances have been described in table 3. Twelve children exhibited various more or less serious path-

TABLE 3.—*Observations on Nineteen Microcephalic Idiots Irradiated in Utero*

Author and Date of Last Report	Birth-Weight in Grams	Microphthalmia	Other Eye Disturbances	Other Defects	Condition at Last Report
Abels, 1924	1,950	Present	Microcornea, ankyloblepharon	Periostitis, hypoplastic genitalia	3 weeks old
Apert and Kermorgant, 1924	Not stated	Absent	None	Multiple nevi	Mentally backward at 8 years
Aschenheim, 1920	4,370	Present	Bilateral chorioretinitis; bilateral optic atrophy; right cataract	Muscular spasms	Imbecile at 3½ years
Deutsch, 1926	1,050	Present	Nystagmus, blepharon	Small genitalia	Feebleminded at 6 months
Falkenheim, 1928	2,500	Present	Defect in retina, strabismus	Arrested physical development	Feebleminded at 4 years
Flatau, 1921	2,500	Not stated	Not stated	Not stated	No report
Ganzoni and Widmer, 1925	2,000	Absent	Convergent strabismus, nystagmus, normal eyegrounds	None	Idiotic at 2 years
Goldstein and Murphy, 1929	1,310	Absent	None *	Muscular rigidity	Imbecilic at 12 years
Kochmann, 1926	Not stated	Not stated	Amaurosis	Not stated	No report
Kupferberg, 1928	1,060	Absent	Not stated *	Cleft palate	Died at 4 weeks
Little, 1928	1,680	Not stated	Amaurosis	Underdeveloped	Died at 2 years
Naujoks, 1928	3,200	Absent	Abnormal retinal pigmentations	Underdeveloped	Idiotic at 6 years
Petenyi, 1923	Not stated	Not stated	Albinic eyegrounds *	Hydrocephalus	Imbecilic at 3 years
Schifler, 1923	Not stated	Not stated	Not stated	Not stated	6 months old
Schwaab, 1928	1,620	Present	Convergent strabismus	Underdeveloped	Feebleminded, died at 2 years of tumor of liver
Stettner, 1921	1,550	Present	Chorioretinitis, optic atrophy, strabismus	Malformed ears, hypospadia	Feebleminded at 2½ years
Werner, 1926	Not stated	Not stated	Not stated	None	Idiotic at 5 years
Zappert, 1925	2,000	Present	Right optic atrophy, ankyloblepharon, convergent strabismus, bilateral epicanthus	Hypophalangia of big toes	Idiotic at 8 months
Zimmermann, 1928	3,125	Absent	None *	None	Feebleminded at 5 years

* Children irradiated late in pregnancy.

ologic conditions of the eyes. Microphthalmia, although a rare congenital anomaly, was common among these children, being known as present in seven. Other optic defects were associated with the microphthalmia. Four children were almost completely amaurotic, the blindness being due to optic atrophy, to chorioretinitis or to both. Strabismus, nystagmus and abnormal retinal pigmentations were also observed.

It is known that of the fifteen children exposed to irradiation in the first four months of gestation, eleven (73.3 per cent) manifested disturbances of the eyes. The remaining four children were irradiated for the first time during or after the fifth month of gestation. Only one of these, reported by Petenyi,[9] suffered from any ophthalmic defect, exhibiting albinic eyegrounds. Two had normal eyes—one reported by Zimmermann [2] and the other by Murphy and me [6]—whereas the eyes of the fourth child, reported by Kupferberg,[10] were not described.

The relation of the malformation of the head to the presence of ocular defects is shown in table 4. This relationship has been studied, since it has been shown that types of deformities of the head—especially oxycephaly or turritum caput—are very likely to be associated with serious ocular anomalies; optic neuritis and atrophy with resulting impairment of vision, exophthalmos, nystagmus and strabismus being frequently observed. The optic atrophy associated with oxycephaly has

TABLE 4.—*Eye Defects According to Contour of Head*

Head Contour	Number of Microcephalies	Ocular Disturbances		
		Present	Absent	Not Stated
Small round head, including 3 with occipital flattening	14	8	3*	3
Oxycephalus (tower skull)	3	3	0	0
Not stated	2	1	0	1
Total	19	12	3	4

* Two children irradiated after the fifth month of pregnancy included here.

been attributed to increased intracranial pressure due to the malformation of the head.

It is observed that of the fourteen microcephalic children who had the round small type of head, eight (57 per cent) manifested disturbances of the eyes, while three (21 per cent) did not. Attention is directed particularly to the fact that the three radiogenically oxycephalic and microcephalic children exhibited defects of the eyes. As shown in table 3, nystagmus (a common observation in oxycephaly) was present in two children, while the third child showed abnormal retinal pigmentation. However, none of these children exhibited optic atrophy or amaurosis. The probable connection between fetal irradiation, malformation of the skull and the occurrence of ophthalmic defects is a matter for later discussion.

The anatomic anomalies and errors in development, other than defects in the visual organs, exhibited by the microcephalic children

9. Petenyi, G.: Mikrocephalie nach therapeutischer Roentgenbestrahlung der Mutter, Klin. Wchnschr. **2**:566, 1923.

10. Kupferberg, H.: Bemerkungen zur gynaekologischen Bestrahlungstechnik, Zentralbl. f. Gynäk. **48**:2031, 1924.

are described in table 3. As was concluded in a previous paper,[3] the fetal irradiation probably had a definite etiologic relationship to these defects, but this will not be discussed here.

Through personal communications, I have learned of the recent mental and physical condition of seven children. The information thus secured is included in table 3. It will be observed that one child has already reached the age of 12 years.[6]

COMMENT

It was concluded in previous papers by Murphy and me[11] that the high percentage (approximately 27 per cent) of the disturbances of the central nervous system in children irradiated during fetal life pointed to the irradiation as the causative factor. The etiologic relationship of fetal irradiation to microcephaly and co-existing ophthalmic disorders has also been discussed by several writers, notably Aschenheim[4] and Zappert.[12] I believe that irradiation acts somewhat similarly to toxic factors or environmental influences in arresting fetal development. This is in accord with the view of Mall,[13] who stated that malformations are not due to germinal and hereditary causes but are produced by changes in environment (i. e., faulty implantation) profoundly affecting the development of the normal ovum. Monakow[14] was of the opinion that the pathogenesis of microcephaly lies in two groups of factors: (1) toxic, such as alcohol, syphilis and metabolic toxins, either acting on the embryo, or having affected the germ cells prior to conception, and (2) general pathologic processes, such as meningitis or encephalitis of the fetus.

If irradiation acts as a harmful environmental agent on the fetus, the question to be considered is whether the central nervous system is especially sensitive to this influence or whether it is equally sensitive to any inhibitory influence. The fact that abnormalities of the nervous system, with or without malformation of the head, are the most common defects occurring in the general population[15] would seem to

11. Murphy, D. P.: Ovarian Irradiation; Its Effect on the Health of Subsequent Children; Review of the Literature, Experimental and Clinical, with a Report of 320 Human Pregnancies, Surg. Gynec. Obst. **47**:201, 1928; footnote 1, fourth reference. Murphy and Goldstein (footnote 1, fifth reference).

12. Zappert, J.: Personal communication to the author; Hat eine Strahlenbehandlung der graviden Mutter einen schaedlichen Einfluss auf das Kind? Wien. klin. Wchnschr. **28**:669, 1925.

13. Mall, F. P.: A Study of the Causes Underlying the Origin of Human Monsters, J. Morphol. **29**:3, 1908; On the Frequency of Localized Anomalies in Human Embryos and Infants at Birth, Am. J. Anat. **22**:49, 1917.

14. Monakow, C.: Biologisches und morphogenetisches ueber die Mikrocephalie, Schweiz. Arch. f. Neurol. u. Psychiat. **18**:3, 1926.

15. Mall (footnote 13, second reference).

indicate that the nervous system is readily affected by any external factor. The development of the nervous system begins very early in embryonal life (about the second week), and the rapidly dividing and differentiating cells of this system are therefore most sensitive to inhibitory influences at this time. The fact that fifteen of the nineteen microcephalic children under study were irradiated in the first month or two of pregnancy points to the susceptibility of the embryonal nerve cells to irradiation. Child [16] contended that the region of greatest activity, the head region, is most easily inhibited by external agents, and that microcephaly is a characteristic result of differential inhibition along the polar axis. The occurrence of microcephaly in four children irradiated after the fourth month of fetal development shows that the multiplying nerve cells are susceptible to the effects of irradiation even late in gestation.

There is considerable experimental evidence to prove the high degree of radiosensibility of the immature brain of the young animal. The results of some of the experiments on record are shown in table 5 and, in general, they confirm the view expressed by Child and others regarding the extreme susceptibility of the nervous system to external agents.

In the experiments of Foersterling [17] on rabbits, roentgen irradiation produced a retardation of the growth of the skull. In addition to ocular disturbances, the irradiated animal exhibited a pointed nose in comparison with the round, full, convex line of the nose in the animal not irradiated.

From experimental evidence it is apparent that the development of the brain in the very young animal is seriously arrested both by radium and by the roentgen ray. For all practical purposes, the cephalic irradiation of the young animal may be satisfactorily compared with the human fetal irradiation. The pathologic changes and nervous phenomena induced in the irradiated animal (retarded growth, ataxia, twitchings, etc.) are seemingly analogous to the disturbances (microcephaly and associated nervous symptoms) observed in the child irradiated in utero.

Relationship of Irradiation to Ocular Defects.—It was indicated earlier that of the fifteen radiogenically microcephalic children in whom data concerning the condition of the eyes were available, twelve (80 per cent) manifested defects of the eyes (table 3). Eleven of these twelve children (92 per cent) had been exposed to irradiation very early in

16. Child, C. M.: The Origin and Development of the Nervous System, Chicago, University of Chicago Press, 1921, pp. 272; The General Relation Between Susceptibility and Physiologic Condition, Arch. Int. Med. **32**:647 (Nov.) 1923.

17. Foersterling, K.: Ueber Wachstumsstoerungen nach kurzdauernder Roentgenbestrahlung, Zentralbl. f. klin. Chir. **81**:505, 1906.

intra-uterine existence. The question then arose: Are defects of the eyes peculiar to radiogenic microcephaly, or are they also commonly associated with nonradiogenic microcephaly? The etiologic relationship of fetal irradiation would become evident if these were found to be infrequent in nonradiogenic microcephaly and, further, if the type of defect commonly observed in radiogenic microcephaly was a rare occurrence in the ordinary type of microcephaly.

TABLE 5.—*Recorded Experiments Showing Irradiation Effects on Nervous System and Eyes*

Author	Details of Treatment	Disturbances Produced	Eye Defects
Bagg: Am. J. Anat. **30**: 131, 1922	Injections of radium emanation into gravid rats	Development arrests in brains of offspring	Blindness and microphthalmia in offspring
Birch-Hirschfeld	Radium and roentgen ray to eyes of rabbits	.....	Keratitis and optic atrophy
Brunner and Schwarz: Wien. klin. Wchnschr. **21**: 587, 1918	Roentgen ray to heads of young dogs	Spasms of neck muscles; retarded growth of forepart of body; hyperemia of meninges with hemorrhage, parenchymal cerebral degeneration	.....
Demel: Strahlentherapie **22**: 333, 1926	Roentgen ray to heads of dogs, aged 4 days	Abnormality in gait; reduction in size of brain; cellular degeneration of cortex and ganglia	Degenerative retinal changes
Foersterling: Zentralbl. f. klin. Chir. **81**: 505, 1906	Roentgen ray to heads of young rabbits	Retarded growth of forelimbs, trunk and head; change in shape of skull	Diminution in size of orbits
Hanson: Proc. Ann. Philos. Soc. **62**: 301, 1923	Roentgen ray to gravid rats	.....	Cataracts in offspring; microphthalmia
Hippel and Pagenstecher: München med. Wchnschr. **54**: 452, 1907	Roentgen ray to gravid rabbits	.....	Cataracts in offspring; microphthalmia in several rabbits
Krukenberg: München. med. Wchnschr. **56**: 182, 1909	Roentgen ray to head of new-born dog	Retarded growth of trunk and forelimbs; ataxia, tremors of head	Optic atrophy
Obersteiner: Wien. klin. Wchnschr. **40**: 86, 1904	Radium bromide to heads of white mice	Ataxia; paralyses of forelimbs; hyperemia; round cell infiltration of meninges	.....
Tribondeau and Belley: Arch. d'électric. méd. **15**: 907, 1907	Roentgen ray to eyes of new-born cats	.....	Microphthalmia; cataract, and defect in retina

A statistical survey was made of a number of large institutions for the feebleminded in order to determine the incidence of abnormalities of the eyes in the ordinary form of microcephaly. Information was secured concerning the number of microcephalic children at these institutions, the number of them manifesting symptoms of the eyes and the types of disturbances involved. The data obtained from six institutions are listed in table 6. From this it will be seen that seventy-four of 8,352 feebleminded children were microcephalic, and that thirty-nine of these exhibited various defects of the eyes.

In table 7, the frequency of the ocular defects in these microcephalic children—who, as far as known, were not irradiated in utero—is compared with the incidence of such disturbances in the microcephalic children irradiated in utero. Approximately one half of the institutional microcephalic children exhibited disturbances of the eyes, as compared with 80 per cent of those irradiated in utero. Microphthalmia, optic atrophy and amaurosis were frequent in the latter group, while only two of the children in institutions manifested optic atrophy.

TABLE 6.—*Survey of Patients with Microcephaly in Institutions, with Reports on Ocular Defects*

Institutions	Census of Feebleminded Children	Number of Microcephalic Children	Children with Eye Defects
Institution for Feebleminded, Columbus, Ohio	2,009	6	0
Training School, Elwyn, Pa.	948	6	0
State School, Polk, Pa.	2,368	45	28
State School, Wrentham, Mass.	1,564	9	7
Hospital for Mental Diseases, Byberry, Philadelphia	260	3	2
State Institution, Vineland, N. J.	1,200	2	0
Pennsylvania Department of Welfare	3	3	2
Total	8,352	74	39

TABLE 7.—*Comparison of Incidence of Congenital Eye Defects in Radiogenic and in Nonradiogenic Microcephaly*

Microcephaly	Number of Children with Reports on Eyes	Children with Ocular Defects	Microphthalmia	Optic Atrophy or Amaurosis	Other Symptoms, Including Nystagmus, Strabismus, Cataract, etc.
Radiogenic (children irradiated in utero)	15	12 (80%)	7	5*	10
Nonradiogenic (children in institutions)	74	39 (52%)	0	2†	37

* None oxycephalic.
† Including one case of oxycephaly.

It has already been pointed out that oxycephaly is exceedingly likely to be associated with optic atrophy and other serious ocular abnormalities. Of the children with radiogenic microcephaly and optic atrophy, not one was oxycephalic, while of the two with nonradiogenic microcephaly, one was also oxycephalic.

From this survey it would seem that disturbances of the eyes are much more likely to occur in microcephaly produced by irradiation than in the usual type of microcephaly. Furthermore, microphthalmia, optic atrophy and congenital amaurosis are more frequently observed in radiogenic than in nonradiogenic microcephaly.

Gordon [18] explained the rarity of optic atrophy in microcephaly of developmental origin in the following manner:

> The optic vesicles from which later the optic cups form, begin their development very early in embryonal life and grow very rapidly becoming almost independent of the median forebrain, being attached only by small tubular stalks. The inhibiting factor acting on the embryonal brain produces a general hypoplasia resulting in a global formation, microcephaly, whereas the practically independent optic nerves escape this influence and develop normally. However, optic atrophy and other visual disturbances are frequently associated with head malformations (oxycephaly), in which case the disturbances are propably produced by the increased cranial pressure or traction upon the optic nerve.

The intimate etiologic relationship between embryonal irradiation and developmental anomalies in the visual organs is further exemplified by observations made on the eyes of animals irradiated in utero and of young animals irradiated after birth. Table 5 records the results of several of these experiments. It is my belief that the method of irradiation employed experimentally in the pregnant animal or in the new-born offspring is comparable with the therapeutic method employed in the pregnant woman, especially as the types of defects observed clinically are similiar to those observed experimentally (tables 3 and 5). Table 5 shows that optic atrophy and microphthalmia were produced in the young animal receiving irradiation of the eyes, and that, in experimental studies,[19] cataracts were observed in the offspring of animals irradiated during pregnancy.

The experimental studies recorded in table 5 demonstrate conclusively the facility with which serious diseases of the eye may be produced in young animals, either by maternal irradiation during gestation or by direct treatment of the head or eyes after birth. The eyes of the young animals and the embryonal optic anlage, therefore, appear to be very sensitive to irradiation. If fetal irradiation and irradiation of the eyes of the animal shortly after birth may be assumed to correspond with the irradiation received by the human embryo, it is obvious that the developing optic apparatus is especially sensitive to the injurious influence of radiant energy. This would account for the frequent occurrence of congenital microphthalmia, optic atrophy and amaurosis

18. Gordon, Alfred: Personal communication to the author; Optic Nerve Changes Associated with Cranial Malformations, New York M. J. **92**:7, 1911.

19. Hanson, F. B.: Modifications in the Albino Rat Following Treatment with Alcohol Fumes and X-Rays, and the Problem of Their Inheritance, Proc. Am. Philos. Soc. **62**:301, 1923. Hippel, E., and Pagenstecher, H.: Ueber den Einfluss des Cholins und der Roentgenstrahlen auf den Ablauf der Graviditaet, München. med. Wchnschr. **54**:452, 1907. Hippel, E.: Ueber experimentelle Erzeugung von angeborenen Star bei Kaninchen nebst Bemerkungen ueber gleichzeitig beobachteten Mikrophthalmus und Lidcolobom, Arch. f. Ophth. **65**:326, 1907.

in children with radiogenic microcephaly, particularly since nonradiogenic microcephaly is not associated with these types of defects of the eyes.

SUMMARY AND CONCLUSIONS

1. Records of nineteen microcephalic feebleminded children, irradiated in utero, have been studied with especial reference to ophthalmic defects. Because of the etioloic relationship of irradiation to microcephaly, the term radiogenic is applied to the microcephaly in these children.

2. Fifteen microcephalic children were born of mothers irradiated prior to the fifth month of gestation, whereas the remaining four were irradiated after the fourth month of fetal life.

3. Three of the radiogenically microcephalic children exhibited the oxycephalic type of head, while sixteen exhibited the small round type.

4. Ocular disturbances were found to be more common and more severe in association with radiogenic microcephaly than with the nonradiogenic type. Data were available concerning the condition of the eyes in fifteen children; of these, twelve exhibited more or less serious abnormalities of the eyes. Microphthalmia, optic atrophy and congenital amaurosis were the most commonly observed disturbances.

5. Eleven of the twelve children who manifested optic disturbances were irradiated in the first four months of intra-uterine life, while the twelfth was irradiated after the fourth month of gestation.

6. Although defects of the optic apparatus have been observed in nonradiogenic microcephaly, it is believed that the increased frequency, severity and uniformity of the defects observed in the radiogenic form point clearly to irradiation as the specific cause.

7. A study of the results of irradiation experiments on the eyes of the unborn and new-born animals supports this conclusion.

1717 Pine Street.

ABSTRACT OF DISCUSSION

DR. ALFRED GORDON: In view of the frequency of irradiation of women, pregnant or otherwise, this subject is of special importance. In view of the data given by Dr. Goldstein and statistical data, the evidence is not as yet, in my judgment, conclusive concerning the relation of radiation to microcephaly. One feature, it seems to me, might suggest a somewhat more definite conclusion. If irradiation were given before pregnancy took place and if then the woman became pregnant a long time after the irradiation was given, more definite statements could be made.

Dr. Goldstein has cited more eye defects in experimental work on animals than in living adults or children. Microcephaly may be produced by many elements entering into consideration. This requires more prolonged study. I gather from this report that really serious defects to the eye were few. Microphthalmia was most pronounced, but the serious cases are comparatively few. There is a reason for it. If one refers to the embryologic development and growth

of the brain and the eyes, one knows that the optic vesicles develop almost entirely separately from the vesicles of the brain. The forebrain grows forward and upward and makes a ventral enlargement separate from the optic diverticula, so that there are a median forebrain and two lateral optic vesicles connected only with small tubular stalks. This explains why, in microcephaly, optic atrophy is rare.

DR. LEOPOLD GOLDSTEIN: In a recent review of the literature, we gathered the records of more than 300 cases in which women with various pelvic diseases were given roentgenotherapy and subsequently conceived. More than 90 per cent of the children were healthy, while only one child was a microcephalic idiot. In this earlier study, it was concluded that preconception pelvic irradiation probably has no deleterious influence on the health of children born later.

PERSONALITY FACTORS IN ALCOHOLISM*

HENRY H. HART, M.D.

Assistant Attending Neurologist at New York Neurological Institute; Associate in Psychiatry, Columbia University

NEW YORK

The strong impulse to drink which develops into an irresistible craving can be understood only psychobiologically, namely, as a reaction on the part of an individual organization to an environmental situation. These deeper personal needs expressing themselves in alcoholic sprees or chronic tippling are not always easy to understand, lying, as they often do, concealed beneath a mass of rationalizations and deceptions. My purpose in this paper is to investigate to some extent in a small group of cases available the importance of individual factors in determining the tendency to drink, and to throw some light on the significance of alcohol in the life adjustment of the personality.

The thirty cases here presented for study occurred in patients of the Blythewood Sanitarium who have been under observation for periods varying from a few days to six months. They have been selected on the basis of detailed records available and not on outcome or type of personality. It was not possible to obtain a thoroughly penetrating study of the individual personality, but in most of the patients certain common and prominent features were apparent, which I believe are of importance in the understanding of chronic alcoholism.

The alcoholic patient is notorious for his unreliability and desire to deceive, and this fact has been considered in the interpretation of the material at my disposal. When possible, information has been obtained from sources other than the patient, although it is obvious that when it comes to his subjective needs the patient alone can give sufficient information, however distorted it is by his own bias and rationalizations.

The first factor which one comes naturally to consider is that of race. In this series of cases, all the patients, with the exception of two brothers who were of German extraction (cases 17 and 20), were Anglo-Saxon or Irish. Most of them were typically American, in that they were a blend of the Anglo-Saxon or Irish races. It is noteworthy that not one member of the Jewish race is on the list, which agrees with common experience and observation. The freedom from alcoholism is a definite racial characteristic, in spite of the frequency of emotional instability and of neurotic and psychotic disturbances among Jews. The

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northern and Teutonic races have always been noted for their tendency toward alcoholism; this has been recorded by Pearce, Bailey and Kirby.

The factors of age and sex in this small series can be briefly discussed. Only five of the thirty patients were women and all were married, four of the five having been separated or divorced from their husbands. The patients varied in age from 22 to 62 years, the average being 35.7 years. This seems to indicate nothing more than a tendency on the part of men and women to seek refuge in alcohol during the prime of life and when the battle of life is at its maximum intensity. There were no adolescent or senile cases in the series. Between the ages of 20 and 60 there are expressed in their most active form man's instinctive urges, sexual and egoistic. One might explain plausibly that with the inability to satisfy these strivings, alcoholism plays a rôle of importance, particularly when one realizes that the failure of instinctive satisfaction in one form or another constitutes the most fundamental cause of human restlessness and discontent.

Family traits and molding factors are next to be considered. In reviewing the thirty cases, one finds that the variety of family situations is great. Psychoanalysts trace fundamental personality traits to the relationship of the individual to his father and mother or their substitutes, and speak in terms of fixation and incest complexes. Does one find evidence of this in these cases, and is there any uniformity in the family situation which of itself can help to explain the tendency to excessive alcoholic indulgence?

One cannot expect to discover a deeper repressed relationship of child and parent in the course of a few interviews, but if any profound attachment exists, it should be, as a rule, readily discovered. Of the thirty patients, only nine gave a definite history of paternal alcoholism; in three cases, the father died before the patient had attained maturity (cases 2, 8 and 11). In seven cases, the father was reported by the patient as having been strict, stern or repressive; in six cases (4, 5, 9, 12, 15 and 19) he was said to have been unstable or to have had a nervous breakdown. In the examination of data obtained in all the cases, only seven of the fathers could be looked on as healthy in their life adjustment, and free from alcoholism, repression or emotional instability. In only two cases was there any profound attachment to the father (cases 10 and 22). In case 10 the patient tended to identify himself with his father and to replace him in his control of the younger members in the family; in case 22 no unusual identification was observed.

Turning to the maternal factors, one finds four cases in which the mother played an apparently insignificant or absent rôle in the development of the child, owing to death while the patient was quite young

(cases 6, 11, 26 and 27). In ten cases, however, there was a deep enough attachment to the mother to warrant the term "fixation," a notable case being that of a journalist who, at the age of 37, was financially dependent on his mother. In three cases, no information was obtainable regarding the child-mother relationships or the personality of the mother, but in the twenty-seven cases in which definite information about the mother was available there were only three in which the mother was apparently normal, stable, well adjusted and exercising the influence of her personality throughout the first two decades of the patient's life (cases 20, 22 and 28). In eleven cases there was a definite history of marital discord between the parents, resulting in divorce in two cases, and in the mother killing the father in another. In one serious case of chronic alcoholism, there was a feeling of intense hatred toward the mother, which engendered a positive misogynism because she had tried to abort the patient prior to his birth (case 2). In two cases in women, the mother was definitely alcoholic, and these were the only instances of maternal alcoholism in the series. These facts justify the conclusion that the relationship to and the normality of the mother are of decisive importance in the evolution of the alcoholic patient.

In another case (case 8), that of a single man, aged 31, the domination of a strict mother was so extreme that it prevented him from breaking away from her and made him hesitate to marry a girl to whom he had been engaged for three years. He declared that his mother "tried to keep her children young."

Considering the siblings of the patient, one sees the same abnormality of personal adjustment and personality make-up. Of the whole series, only five cases presented evidence of normal siblings. In these instances, the siblings were not examined by the physician, and their normality or abnormality was deduced from statements made by the patient himself or by relatives and other informants. Those who have investigated the heredity of patients with thoroughness realize how unworthy and inadequate are the statements of the patients or of relatives; when in doubt as to the family's normality, they give themselves the benefit of it. Hence, one may surmise that even in the five cases with apparently normal siblings there were doubtless instances of abnormality of adjustment. While twelve patients gave a history of alcoholism in one or more siblings, only one gave a history of a definite psychosis. This suggests not that the alcoholic refuge is dependent on the need or craving of an otherwise normal person, but that some constitutional quality exists in the stock of which he is part; two patients were only children, and seven were the only members of their own sex in the family. Shiftlessness, irresponsibility, feelings of inferiority, religious fanaticism, homosexual perversion and neurasthenia were symptoms and qualities described in the siblings of these patients.

For a truly comprehensive and thoroughgoing study of the underlying factors in the causation of alcoholism, a detailed study of the childhood of each patient is indispensable. This demands of patients an absolute frankness and sincerity, qualities which unfortunately are hard to obtain in alcoholic patients, as all who have dealt with them realize. Particularly is this true in sanatoriums to which patients frequently go with the deliberate purpose of avoiding anything resembling an investigation, scientific or otherwise, into their nature. When no term of commitment is possible, the patient can always leave if he insists on doing so, and most do after a brief sojourn in which they regain their equilibrium and physical well-being. Of my series of 30 patients, only three showed any disposition to cooperate with the physician in a thoroughgoing study of their personalities or adjustment, and none ever voluntarily sought aid along these lines. This lack of sincere downright concern over their weakness is a good index of the degree of seriousness with which they regard their problem; but it is more than this; it seems to indicate lack of desire on the part of the alcoholic patient to get well, probably because of an organic need for alcohol that he has developed. This need appears to be based on an inferiority of constitution, and, as Janet has pointed out, the alcoholic subject ascends from the subnormal to the normal under the initial effects of alcohol. The painfulness of his affliction is outbalanced in his own mind by the comfort and well-being or sense of relief which he derives from it. This is what makes it difficult to effect any permanent cure in the alcoholic patient without substituting something for the alcohol, such as religion, art or love, which can effect a relief of his ego from its feeling of inferiority to a feeling of equality with a group. It explains why the alcoholic patient does not want to get well, however much he may protest that he does.

The sex life of these thirty patients was studied in a superficial manner, but even this was sufficient to reveal gross disharmony and difficulty of adjustment. Marital harmony, an apparently increasingly difficult social achievement, was rare in all my cases. Of the thirty patients, seven were single and only one of these could be regarded as contentedly single, namely, a bachelor, aged 62, who had enjoyed the companionship of mistresses from time to time (case 30). It is worthy of note that none of these unmarried patients were women. Of the series, only four patients could be certified as free from sexual promiscuity before marriage, and two of these were women. Of the twenty-three married persons, the overwhelming number of twenty presented definite evidence of marital discord, unhappiness, separation or divorce; sixteen of the patients had been separated or divorced from their partners for drunkenness, incompatibility or infidelity. Only one patient was reported as making a happy adaptation to married life, and

his wife withdrew him before any study of him could be obtained (case 17). Of the remaining two who did not show difficulty with their wives, little positive information one way or another was obtainable. Infidelity to the marriage bond was definitely ascertained in twelve of the twenty-three cases.

Disappointment in love is offered in many cases of chronic alcoholism as an original predisposing factor. How important one can rate this it is difficult to say, for in the seven cases in which the history of this misfortune was given one can see other factors more fundamental in the development of the personality than this, which is frequently offered as a respectable and sympathy-getting excuse for a flight from an unpleasant reality. One youth, aged 23, whose wife had treated him with indifference and finally left him, drank, he said, to escape from his wounded pride and chagrin (case 3). Two men, both showing chronic alcoholism, said that their drinking began from disappointment following the breaking of an engagement (cases 1 and 10). In one, the effect of this seems to have been more important than in the other, as he telephoned his former sweetheart in the midst of his debauch. In two older men, alcoholism began after their wives had left them to become attached to other men (cases 13 and 29). In these two instances, alcoholism was probably a substitute, because it reduced their sexual desire.

The psychoanalytic doctrine that homosexuality is an important factor in alcoholism leads one to consider what facts are available in the study of these cases which would support such a theory. If one defines homosexuality as a strong tendency to associate with one's own sex in preference to the opposite sex, then in this series of cases eleven of the thirty exhibited such tendencies. Of the four women, two showed possible evidences of this by drinking frequently with members of their own sex. However, this tendency was more obvious in the male patients, nine of whom drank almost exclusively with men when they did not do so in solitude. Three men expressly admitted that while under the influence of alcohol their desire for women was diminished. One youth, whose mother had always despised him and who had tried to abort him, declared frequently that he hated women (case 2). This particular patient, however, sought a sort of maternal sympathy in older women, while he could make no satisfactory adjustment to self-respecting girls of culture and intelligence. One patient was reported by his wife to have had a homosexual attachment at the age of 18 (case 15); another of effeminate make-up and without any satisfactory heterosexual adjustment suffered a depression on the departure of a young boy who had lived with him intimately for months (case 9). The latter patient was the most outspoken example of homosexuality in the whole series, but his indulgence in alcohol was appar-

ently recent and not excessive. Indeed, his appetite for alcohol was slight, in comparison with that of the more heterosexual types. On considering the group as a whole, there seemed little evidence of any important or overt homosexual tendency.

RELATIONSHIP TO CONSTITUTIONAL DEFICIENCY

One cannot dispose of the sex life in these thirty cases, however, without considering the overwhelming proportion of marital maladjustment, which is probably traceable not so much to some unconscious homosexuality or other sexual abnormality as to a general constitutional deficiency—partly inherited and partly acquired through unfortunate childhood influences and training—which renders the person incapable, not only of adjusting himself to the responsibilty of adult life and to the demands made by another individual, but of adapting himself to society in general, and maintaining a constant, steady purposive life activity. Of the total group, twenty-three patients gave a history of having been pampered by parents and allowed to have their own way in childhood, a trait which can probably explain the general want of adaptability in maturer years.

This pampered group of twenty-three patients corresponds closely to the number who presented definite egocentric, egoistic traits—namely, twenty-five. Of the five women, four were definitely self-centered personalities, while the fifth accused herself of being "no good" and regarded herself as a hobo or tramp. Not all of these exhibited their egoism in an aggressive manner; only four of the entire group of twenty-five had any serious consideration for the happiness and convenience of relatives and those nearest and dearest to them. The egocentricity of the alcoholic patient finds usually an aggressive extroverted expression. None of these thirty patients could be regarded as seclusive, introspective or introverted; they had none of the shut-in qualities characteristic of the schizophrenic type. They presented the personality traits usually found in the spoiled and pampered child or the only child of indifferent or pampering parents.

This extroversion or the capacity to make friends rapidly and to make a quick adaptation to the present environment was shown in a marked degree by ten patients. Some of these patients were popular and attractive socially; eighteen were regarded as being moderately extroverted; only two patients could be looked on as slightly introverted and less aggressive in adaptation to the environment. In the use of these terms, however, I realize that the standards of extroversion and introversion are inaccurate and empiric categorizations of human personalities, and therefore one's estimate of these qualities must be built on the observation of these personalities as they adjusted themselves to society, such as that at Blythewood, or on history obtained from the relatives of the patients.

Two indications of defective personality, deceptiveness and unreliability, were striking in the majority of these thirty persons. Unreliability of statement was found in greater or less degree in all but three of the cases. This trait manifested itself in such ways as distorting the facts of the history, glossing over flagrant and disgraceful episodes, projecting the blame of one's condition on others or on circumstances, exaggeration of personal prowess and achievement, making up yarns to secure early discharge, etc. The trait of deceptiveness, which carries a more deliberate intent, is hard to distinguish precisely from unreliability. Five were free from this trait, but the rest were characterized by more or less desire to create a good impression, or to deceive the physician into permitting them to get alcohol. Important business matters, appointments with dentists, theater engagements, automobile rides, etc., were given as subterfuges or opportunities to escape from the discipline of the institution in order to obtain alcohol. One man ran up great taxi bills and signed bad checks with an air of great importance, caring little about the consequences of such shortsighted behavior. All this deception is but another indication of how difficult it is for the alcoholic personality to face the facts of his own life situation.

Parental responsibility was rare in this group of thirty; thirteen of the married group of twenty-three displayed no such responsibility; three were found to have demonstrated it in an occasional or fleeting manner. Six showed occasional interest in the welfare of their children; only one, a man aged 52, with eight children, showed considerable interest in his family. This man, it is worth mentioning, had indulged in alcohol to excess only since the death of his wife a year previously. The reproductivity of these alcoholic persons is not remarkable when one considers that the total number of children derived from the whole group was only twenty-eight. Eleven of the married group of twenty-three had no offspring.

Absence of parental responsibility one would expect to go hand in hand with absence of social responsibilty, and it is interesting to note that both deficiencies are to be found roughly in the same patients, as one may observe from the accompanying reports. Fourteen of the thirty patients showed an almost complete absence of such sense. They had no regular employment and no authority over others and were largely dependent on their parents or on wives or husbands for maintenance. None of them had any civic interests, organizing ability or charitable outlets, and they showed no interest in anything outside of themselves. One youth gloried in the boast that he could drink himself to death if he wanted to and no one could stop him. Of the five women, all of whom were married, none had any real employment, although one had occasionally written plays that had never been accepted, and another

had been a model occasionally. Among the men, only one had no nominal vocation. Those who professed any regular occupation may be grouped as follows: stockbrokers, five; real estate brokers, four; engineers, two; contractors, three; newspaper correspondents and journalists, two; actor, hotel keeper, lawyer, advertiser, association secretary, clothier, clerk and soldier, one each. It is evident that the speculative, interesting, adventurous, get-rich-quick life of the stockbroker with its thrills and chills is the favorite occupation of this group of alcoholic persons. A gambling tendency is strong in these folk. They demonstrate a certain devil-may-care, restless attitude toward life, and it is not purely coincidental that this is the occupation which attracts them most. Professional or business life and the humdrum routine of the more stabilized business or profession does not attract them; only one was a clerk, and his clerkship was intermittent.

Kirby, Gregory, Schneider and others have considered the relationship between alcoholism on the one hand and schizoid tendencies on the other; it was of interest to me to enumerate from the thirty cases, which were selected only on the basis of record and material available, the relative incidence of such tendencies. This was found to be overwhelmingly in favor of the cyclothymic type. In five of the thirty cases the information obtained was considered insufficient to decide such a matter. In the remaining twenty-five cases, twenty-one showed definite swings of mood—particularly, liability to markedly depressed, gloomy or morose states. Four cases showed no clear tendency in this direction. Only two patients of the group were of a sensitive, shy make-up, although this in itself does not warrant one in regarding them as necessarily schizoid. If one is inclined to consider the presence of acute or chronic hallucinosis as indicative of a fundamental schizoid tendency, twelve of these patients at one time or another in their alcoholic career had hallucinations; these were visual in the majority of cases. Only three patients developed symptoms of a chronic, psychotic character. One had beginning symptoms of a Korsakoff's psychosis, with fabricative amnesia and polyneuritis; another developed delusions of infidelity on the part of her husband, and a third had morbid suspicions and transient states of grandeur. In all of these three cases, the social adjustment between the alcoholic sprees and the disposition to swings of mood was definitely characteristic of the cyclothymic make-up.

Turning attention to the important and inciting factors in the problem of the actual drinking bout, one finds less variety of underlying motives than one might expect with a random assortment of thirty cases. The age of onset could not be determined, but in twenty-nine cases, the average age of onset was about twenty-seven years. The mode of commencement was not ascertained in some cases, but in most

the drinking commenced as a form of social habit. In this connection one can usually differentiate three stages of the development of alcoholic indulgence. The first stage is that of social drinking. In this stage the person drinks alcohol only in company with a group of others and never by himself. He partakes of alcohol merely as a part of his social pleasures, and not as an expression of any great personal need or craving. This is the stage of alcoholic indulgence that is generally regarded as normal, and from which only the total abstainer refrains. As the consumption of wines and liquors has been, since prehistoric days, largely a social activity, just as has the consumption of food, one cannot regard it as abnormal.

The next or second stage is that of dipsomania. Here the person passes beyond the mere desire for drinking alcohol in public to the irresistible craving for alcohol which comes over him at times, with or without his insight into the cause, and which leads him into a public or private debauch lasting from days to weeks. From this spell of intoxication he emerges somewhat ashamed of himself and rid of the desire to drink for an interval varying from weeks to years. Frequently, he describes a feeling of nausea and disgust for alcohol, and he is strong in his resolution never to touch the drink again; but the irresponsible craving recurs and, not without shame, he resigns himself to it.

The third and last stage of alcoholism is the stage of chronicity. In this stage the patient is absorbing alcohol every day in variable quantities. He may acquire the capacity to take incredible amounts of gin or whisky over a period of months or years. His color is florid, his speech is at times careless and slurring, his hand trembles, and signs of deterioration of higher psychic integration show themselves in carelessness of person, irresponsibility, shiftlessness, unreliability and general blunting of the finer sensibilities, with a growing incapacity to make delicate discriminations. Alcohol has become food to him, not merely something for which he has spasmodic craving with a resultant disgust. His will has become undermined, and he can withstand the craving for only brief periods at a time. This stage is not necessarily hopeless; tremendous emotional upheavals in life may transform him into a reformed total abstainer or even into a person fanatically opposed to alcohol. Religious conversion, marriage or some other turning point has been known to produce permanent recovery from even such deterioration as one sees in this stage.

Although, from the practical standpoint, in these thirty cases it is difficult to draw an entirely sharp line between chronic alcoholism and dipsomania, there were eighteen cases that could be regarded as essentially dipsomanic, as the impulse to drink to excess came at irregular intervals. The other twelve cases were of more or less steady, chronic drinkers, who took alcohol almost constantly and daily.

More interesting than the way in which the alcoholic person drinks is his state of mind before the drinking bout. All but two patients admitted a feeling of gloominess or depression of mood antecedent to the drinking, and the various precipitating feelings can perhaps be more profitably studied and compared by consulting the accompanying case reports. Worry, anxiety over business, restlessness, loneliness, fright, insomnia and irritability are all common antecedent complaints. That these complaints are also the expression of definite thwarting of the sexual instinct in its expression is well known. One middle-aged, married man frankly admitted that his drinking attacks occurred whenever he had a quarrel or temporary separation from his wife; another found his wife, who was also a chronic tippler, physically repulsive and incompatible. Being thwarted by their husbands from indulging in social revels was given as an excuse by two married women (cases 6 and 12). One man declared that alcohol overcame his "sex repressions" but made women less attractive to him. Although many of the reasons given are rationalizations, most of the patients, on being further questioned, gave replies indicating a feeling of depression or inadequacy.

Financial losses and business reverses have sometimes been offered by the patients and their relatives as outstanding causes of alcoholism. In ten of these cases worry over money, loss of a job, debts and failure in business were undoubtedly factors of importance. However, it is difficult to determine whether these are fundamental causes of alcoholism or merely the results of it, or whether both are but the result of a more fundamental constitutional deficiency which is responsible for a vicious circle. One man, a building contractor, seemed unable to undertake for any length of time the responsibilities of his business; growing careless in his accounts, he lost much of his business. Alcohol thereon became a refuge from his difficulties until remorse and disgust drove him back to them.

SUMMARY AND CONCLUSIONS

From a review of these facts and the picture of the various personalities studied, there can be derived a composite pattern or type which is common to most alcoholic patients. In the first place, there is a constitutional instability in the stock, with parental discord, alcoholism, want of discipline and lack of sensible direction in the environment of childhood. In some cases, pampering of an only child produces a want of mature sense of responsibility, and a resultant exaggerated sense of importance in the community. Thus, ill adjusted to life, frequently with a marked cyclothymic make-up and exposed on all sides to the opportunity of alcoholic oblivion, the impulse comes to escape the unpleasant relations and situations which the personality has precipitated, and the man finds in alcohol both an exhilaration and a

nemesis. Frequently, religious devotion can provide a simple retreat from the world; in one of these patients, both these means of escape were utilized without any apparent sense of inconsistency (case 16). The drink in some cases seems a definite substitute for sexual satisfaction, because of the diminished libido which results. The need for alcoholic comfort in the advent of no reformative influence in the individual life becomes more frequent. The dipsomanic patient develops chronic alcoholism, and then there appear the deterioration and changes, both psychic and physical, that are effected by chronic poisoning.

Because of its emotional significance, alcoholism cannot be regarded as a disease, such as typhoid or pneumonia, which runs its course and from which the patient is glad to recover. He retains his alcoholic solace, since he can find no substitute that will secure release from the feeling of inadequacy. It is an emotional and moral problem, not an unduly physical one. This has to be considered before one can expect any permanent cure. Only that which can build up the self-respect, the habit organization and the feeling of adequacy, rendering it not only possible but attractive to attain a level of mature responsibility, can be expected to emancipate the alcoholic patient.

REPORT OF CASES

Case 1.—A single man, a broker, aged 22, first began drinking alcohol at the age of 16; since that age he had taken it to excess. The drinking was dipsomanic, the alcoholic bouts lasting from about a week to ten days. The father, a dipsomaniac and heavy drinker, was shot and killed by his wife. The mother was psychotic; she shot and killed her husband, baby and self, and wounded the patient severely in the brain. One sister was psychopathic; she used to run around in night clubs, but became sober after the family tragedy. A brother also was nomadic and alcoholic. The family life was unhappy. The patient showed some unreliability in statements. He was pampered and egoistic, had definite swings of mood, and became depressed before drinking. He was sexually promiscuous. He was engaged to a girl, but the engagement was broken. He drank chiefly in solitude but began to drink in a crowd. When under the influence of alcohol, he became talkative, boasted of aims and achievements and became argumentative and unrestrained.

Case 2.—A single man, aged 23, with no occupation, of English-American stock, began drinking alcohol to excess at the age of 15 and became a chronic alcoholic. The father died when the patient was 19; he had been a successful, able business man. The mother was divorced from the father, and had tried to abort the patient. One brother was also alcoholic. One sister was apparently normal. The patient was extremely egoistic, pampered and deceptive in his statements. He ruthlessly disregarded others in his alcoholic bouts. He took absolutely no social responsibility and had no desire for it. He was extroverted. He stated that he felt depressed before drinking. Frequently, he developed alcoholic neuritis with foot drop, and on one occasion he had epileptiform convulsions. Sexually he was promiscuous. He had never felt comfortable with decent girls, and associated only with prostitutes. He stated that he hated women. He drank both in solitude and in company, usually with other young men. Under the influence of alcohol he

became irritable, self-assertive, profane and obscene. He declared that he took it to brace himself up and he defied anybody to stop him. He declared that he could kill himself with liquor if he wanted to. On one occasion he had delirium tremens.

CASE 3.—A married man, aged 23, of Scotch ancestry, without occupation, took alcohol to excess at 17. The drinking was dipsomanic, the bouts lasting from three to five days. Little information was available about the parents. The father was a broker. There were two younger brothers, who were apparently normal. The patient was extremely pampered and indulged during childhood. He was egoistic and took no social responsibility. He was moderately extroverted. He had slight deafness. Marital discord was present. His wife was indifferent to him and cared only for his money; she went around with other men. The patient's parents sought annulment of the marriage. The patient drank only with male friends, and he stated that he drank to overcome nervousness; he also probably sought to overcome resentment at his wife's indifference. Alcohol made him sullen, defiant and combative.

CASE 4.—A married man, a broker, aged 30, of English stock, began drinking to excess at 23. The drinking was dipsomanic, the bouts lasting from one to three days. The patient was an only son. The father was excitable and scolded the patient. Little was known about the mother. The patient was extremely self-centered and pampered, and assumed no social responsibility. He had no children. He went out with wealthy youths and came back drunk. He had had marked financial losses and debts. The death of his wife was regarded as a precipitating factor. He felt gloomy and depressed before drinking; he stated that after drinking everything looked rosy. He preferred to drink in solitude.

CASE 5.—A married man, aged 30, of English stock, an actor, first took alcohol to excess at the age of 14. Drinking was of the dipsomanic type, the bouts lasting from seven to ten days. The father was egoistic and rather severe, but not alcoholic. The mother was an actress, and very neurotic. The patient was egoistic and pampered, rather seclusive, and inclined to swings of mood; he felt markedly depressed before drinking. He married a woman about ten years older than himself and had constant quarrels with her. The wife was more interested in her cats than in him. He became legally separated from her. There were no children. Before he drank he felt depressed and very much the under-dog. The drinking bouts came on after quarrels with the wife. He said that alcohol gave him confidence. He preferred to drink in solitude. He had had delirium tremens twice and once an acute paranoid state with ideas of infidelity.

CASE 6.—A married woman, aged 30, of Scotch-English-French ancestry, with no occupation, stated that she had been used to alcohol all her life, but began drinking to excess at the age of 29. She had chronic alcoholism. The father was intolerant; after the death of the patient's mother he married again. The patient was quite young when the mother died. Three brothers drank to excess. The patient was an only daughter, considerably spoiled and self-centered. Her statements were unreliable. She assumed no social responsibilities. There was one son, aged 10. She showed a definitely cyclothymic make-up. She was quite extroverted. She was not able to get along with her husband; under the influence of alcohol she consorted with other men and was promiscuous sexually. She went about with a drunken female nurse as a boon companion, but drank chiefly in solitude. She stated that alcohol exhilarated her.

CASE 7.—A married man, aged 31, a soldier, began drinking excessively at the age of 15; drinking was of a dipsomanic type, the bouts lasting one week. The

father was a heavy drinker, divorced from his wife, and was a murderer. The mother was a periodic drinker who let the patient have his own way. One brother was also an alcoholic. The patient was extremely egoistic and pampered, and was unreliable in his statements. He was subject to definite swings of mood. He was extroverted and made friends quickly. He had no sense of social responsibility. After much marital discord, his wife ran away and they were separated. Sexual promiscuity and infidelity had been marked. He had no homosexual attachments. He had two children, but showed no sense of parental responsibility. He declared that before drinking he felt restless and useless, like a bum. Alcohol made him explosive, talkative and bombastic. In 1925 he had acute hallucinosis. He drank both in company and in solitude.

Case 8.—A single man, aged 31, of Irish stock, a civil engineer, first took alcohol at the age of 19 and began drinking to excess at 26. Drinking was dipsomanic, the bouts lasting a week or more. The father died when the patient was 21. The mother was dominating, religious and strict; she tried to keep the patient young and dependent on her. One younger sister was high strung and nervous. The patient was rather quiet and shy; he was frank and comparatively unspoiled; he was moderately extroverted, and had occasional depressed moods without definite swings of mood. Sexually he was promiscuous. He drank chiefly in the company of other men. He was engaged to a girl for three years but could not make up his mind. He gave his worry over business and bad company as an excuse for drinking. Alcohol, he said, eliminated the worry. A week before admission he had acute hallucinosis in which he saw a negro castrated by two white men; he was being followed in subways, and people pointed a finger at him.

Case 9.—A single man, aged 32, of English stock, an advertiser, said that he had used alcohol all his life, and began drinking to excess at 31. He was now a chronic alcoholic. The father had a nervous breakdown at the age of 32. The mother was high strung, excitable and neurotic. One older sister was dominating, unstable, and had been divorced twice. The patient was the only son; he was extremely self-centered, self-indulgent and pampered, and showed considerable unreliability and deceptiveness. In his firm he showed considerable responsibility and efficiency. He felt depressed when alone and was inclined to swings of mood. He was quite extroverted. He had no deep interest in women and he regarded marriage with cynicism. He stated that he had had several mistresses. He had shown a definite tendency to homosexual attachment to men and young boys. Before drinking he had incurred large debts from extravagance. He became disappointed over the departure of a young boy who had lived rather intimately with him and he made an unsuccessful suicidal attempt with gas after this event. Debts and disappointment preceded the period of alcoholism which led to his coming to Blythewood. Alcohol "pepped him up"; he drank chiefly in solitude. He had not used drugs. He had had no hallucinations.

Case 10.—A single man, aged 32, of English stock, a broker, began drinking to excess at 31. He had periodic bouts lasting three months at a time. The father, a man of business, was much idolized by the patient. The mother pampered and fondled him and prevented his growing up. He had two sisters and one brother who lived off his earnings. The patient was extremely haughty, domineering and pampered. His statements were somewhat unreliable and deceptive. He was definitely subject to depressed moods. He had shown some sex promiscuity. Alcoholism followed a disappointment in love. Before drinking he was melancholy, restless and discouraged. Alcohol tended to quiet his nerves. He drank for the most part in solitude and had taken excessive quantities of peraldehyde, phenobarbital, barbital and other drugs.

CASE 11.—A single man, aged 33, of English stock, a clerk, first took alcohol at the age of 14 and had taken it to excess ever since. He was subject to spells of marked debauch lasting from one to four weeks. The alcoholism was chronic. The father, who was self possessed and apparently normal, died when the patient was 7 years of age. The mother, who was sweet, gentle and affectionate, died when the patient was 15. One sister suffered with occasional neurasthenia; the other was normal. The patient was the only son. He was markedly self centered and spoiled; his statements were unreliable and deceptive. He had no sense of social responsibility. He could not hold a job successfully for longer than a year. He was extroverted but showed no cyclothymic tendencies, though he had had occasional depressed spells. He had been sexually promiscuous, but showed little interest in women and apparently had had little sex drive. He failed in the dairy business and fell into debt. He had had a constant feeling of inferiority. Alcohol sometimes made him violent and extravagant with money. He drank chiefly in solitude. He had had auditory hallucinations once.

CASE 12.—A married woman, aged 34, of Irish stock, who worked occasionally as a model, had been accustomed to alcohol since 4 years of age and began drinking to excess at 31. The drinking was dipsomanic, the bouts lasting from one to two weeks. The father was alcoholic and unstable. The mother was placid, selfish and indulgent, yet dominating. One sister was psychopathic; one sister hysterical, and another was hypomanic and had been cured of alcoholism. The patient was tricky and deceptive, particularly when under the influence of alcohol, very self-centered, self-willed and pampered. She had always been subject to violent tantrums of temper. There was a marked tendency to swings of mood. She was extroverted and sociable. She had had marked discord with her husband, who was a somewhat dominating, successful young lawyer. Divorce had been threatened frequently. Under the influence of alcohol, she ran around with strange men and showed no discrimination in her society. No homosexual tendencies had been observed. She had no children and no desire for them. As an excuse for drinking bouts she gave thwarting by the husband, who sought to restrain her from extravagance. Under the influence of alcohol, she became more defiant, irritable and combative. She drank both in company and in solitude. She had not used drugs and had had no hallucinations or delusions.

CASE 13.—A married man, aged 34, of English stock, a newspaper correspondent, began drinking to excess at the age of 32. He had been a periodic drinker, the bouts lasting one week. The father was described as phlegmatic and unaffectionate—something of a Babbitt. The mother was morbid, subject to depressions and showed a marked emotional attachment to younger brothers of the patient who were apparently normal. The patient was somewhat self-centered and showed the effects of considerable pampering. He had an exaggerated notion of his own ability and capacities. He had shown considerable initiative and some social responsibility. He had shown a definite tendency to swings of mood, and was characteristically hypomanic in reaction. He made friends quickly and was markedly extroverted. He was definitely undernourished. His mental life had been constantly unhappy; his wife was unfaithful and ran away with another man, and there was a divorce. The patient was also unfaithful and promiscuous. He drank with men only. He had one child but assumed no parental responsibility. He stated that he drank to overcome sex repression. Under the influence of alcohol women became less attractive and he felt like talking about himself. He drank both in company and in solitude. In November, 1928, during an alcoholic bout, he had hallucinations of men having intercourse with his wife.

Case 14.—A married woman, aged 35, of English stock, with no occupation, began drinking alcohol to excess at the age of 33. Drinking was dipsomanic, the bouts lasting about one week. The father was dead; little was known about him. The mother was alcoholic, and dominated the patient who was much attached to her. One half-brother was impracticable, idealistic and philosophic. The patient was the only daughter. She was of the self-centered type and was considerably pampered in childhood. She was brought up to regard herself as artistic and something of a genius. She had never had any social responsibility to amount to anything. She was subject to moods of extreme depression, with exhaustion and apathy. On the whole, she was definitely extroverted. She talked rapidly but in a rather tiresome manner about herself. Physically she was undernourished, with an extremely rapid pulse rate, low blood pressure and occasional peripheral neuritis. Her marital life had never been happy. Both the first and second husbands were likewise alcoholic. The first husband was divorced. The patient admitted sexual promiscuity between her marriages. No special homosexual tendencies were observed. The patient preferred to be in the company of men and always considered herself attractive to them. She had had no children; there was no desire for parental responsibility. The recent and exaggerated alcoholic indulgence was preceded by an inability to sell Florida real estate after the boom. She felt disappointed with both her marriages. Her present husband was younger than herself, alcoholic, shiftless and effeminate. She stated that she became depressed before drinking and lost interest in life. Alcohol made her do the things she disliked and rendered her more cheerful. She drank mostly in solitude. No drug addiction had been discovered. Her unreliability of statement suggested the development of a Korsakoff's psychosis.

Case 15.—A married man, aged 36, a real estate broker, of English stock, first took alcohol at the age of 23, and began taking it to excess at the age of 32. Drinking was dipsomanic, the bouts lasting from one to two weeks. The father was alcoholic, hot tempered and unstable. The mother was hard working, and occasionally hysterical. One brother was alcoholic and very spoiled. The patient was extremely self centered and assertive and evidently had always had his own way. He gave a history of considerable pampering in childhood. He lost his temper quickly and was fairly frank and straightforward. He had been subject to definite swings of mood, and was extroverted; he made acquaintances quickly, particularly among women. He had been definitely subject to depressed spells. He could not get along with his wife, and they had been separated for many months. He had shown marked sexual promiscuity before marriage and infidelity afterward. The wife stated that at the age of 18 he associated with a homosexual man, but the patient denied this. He had no children and no parental responsibility. Prior to his recent alcoholic indulgence he had lost his job through a fight with his employer. He stated that he became lonely before he drank. Alcohol made him feel happy; more social and confident. He drank in company and in solitude. He did not use drugs. On one or two occasions he had had delirium tremens.

Case 16.—A man, aged 37, divorced, of English stock, who was a journalist whenever he did anything, began to drink when 21; drinking was of dipsomanic type, the bouts lasting from six weeks to two months. The father was a chronic alcoholic and sexually promiscuous. The mother was dominating; she looked on him as her bad child and supported him financially. The patient was the only child by her first marriage. Two half siblings were normal. The patient presented a brilliant and attractive exterior, but in his statements he was extremely unreliable and deceptive, and always carried himself with a great air of importance. He was extremely self centered under a polished exterior. He had always had his own

way in childhood and was much pampered by his mother. He showed slight social responsibility and was of definitely cyclothymic make-up. At times he was subject to definite hypomanic drives. He was extroverted, making friends easily, particularly with women with whom he was popular. He was subject to depressed moods. During the past year he had been troubled with alcoholic neuritis. His sex life was full of marital discord, ending in divorce. He was extremely promiscuous sexually. He stated that he drank for want of women. When drunk he would associate with negroes and the riff-raff of the bootleg joints. He had no children and no indication of a desire for them. Before taking alcohol he felt depressed and restless. Under the influence of alcohol he felt witty and pepped up, and showed an overbearing, argumentative attitude. In this state he would sign bad checks and confabulate. On one or two occasions he had shown paranoid trends.

Case 17.—A married man, aged 39, a contractor, of German stock, could not recall when he began to drink. He drank alcohol steadily. The father was alcoholic and spoiled him with money. The mother was a good woman and apparently normal. One brother was alcoholic (case 20). The patient was moderately self-centered, and had been used to having his own way in youth. He had, however, considerable social responsibility. No cyclothymic tendencies were ascertained. Cirrhosis of the liver was present. Marital discord was present and usually preceded excessive drunkenness. The patient had two children. He stated that he was confused and dazed after drinking alcohol. He drank almost entirely in solitude. There was no history of drug addiction or psychotic episodes.

Case 18.—A married man, aged 40, a contractor, of Irish stock, had used alcohol all his life, but began taking it to excess at the age of 33. The drinking was dipsomanic, the bouts lasting from one to three weeks. The father was strict. The patient was much attached to the mother who pampered him a great deal. He was an only son. One sister was healthy. The patient showed some sense of social responsibility and was moderately extroverted. No history of marital discord was obtained, and the patient was never separated or divorced from his wife. Before marriage he had promiscuous sexual relations. He had four children. As one contributing cause for drinking he gave worry over inability to collect money from clients. Before drinking he was gloomy, depressed and hopeless. Alcohol made him cheerful and confident. He drank chiefly in speakeasies. While at Blythewood he was acutely hallucinary, seeing faces and animals.

Case 19.—A married man, aged 41, a hotel-keeper, of Irish stock, had used alcohol all his life. He began drinking to excess at the age of 27. He had chronic alcoholism, being practically never free from indulgence. The father was a hot tempered but extroverted Irishman. The mother was known to worry over trifles. Two brothers were alcoholic; one was definitely a religious fanatic. The patient was tricky and deceptive in statements about himself. He was not particularly self-centered; however, he always exhibited marked stubbornness. He had been showing a decreasing sense of social responsibility and had gradually lost control of the hotel business through alcoholism and neglect. He showed some tendency to swings of mood. When he was not drunk he was extroverted and made friends, particularly with men. He had considerable friction with his wife. Before marriage, he was promiscuous sexually and since marriage he had been unfaithful to his wife. He was fond of drinking with male guests. He had one child. He gave as an excuse for drinking worry over inability to run his business, He had a feeling of inadequacy. Alcohol made him sleepy and less nervous. He used no drugs. Under alcohol he became muddled. He had had occasional visual hallucinations.

Case 20.—A married man, aged 43, of German stock, a contractor, began drinking to excess at the age of 30. The drinking was dipsomanic, the bouts lasting from two to four weeks. The father was alcoholic, and gave him a great deal of money. The mother was healthy and normal. One brother was alcoholic (case 17). In personality the patient showed the effects of pampering, but showed considerable social responsibility and was fairly frank and not particularly self-centered. He had a slight tendency to swings of mood, and was occasionally depressed. He attacked his wife on one occasion when he was drunk, and divorce was frequently threatened. He was sexually promiscuous and unfaithful. He had two children and showed some parental responsibility. He stated that he drank because he worried over business. After taking alcohol he felt at peace with the world. He said that he did not care for women. He drank chiefly in solitude. He took no drugs and had no cyclothymic episodes, but showed a positive Wassermann reaction of the blood.

Case 21.—A married man, aged 44, an insurance broker of French-English stock, began drinking to excess at the age of 30. He was of the chronic alcoholic type. The father was divorced and unfaithful to his wife. The mother was apparently normal. The home life was unhappy. Friction between the parents led to a divorce. One sister was spoiled. One brother was shiftless, and was kept by his wife. The patient showed some unreliability and deceit in statements. He was not unduly egocentric or pampered. He was subject to definite hypomanic drives, with periods of depression. He was definitely extroverted. His marital life was definitely unhappy, his wife being both unfaithful and alcoholic. The patient was unfaithful and promiscuous. He was said to be "very fond of the boys." He had no children and no sense of social responsibility. He gave marital discord and dissatisfaction as the cause of alcoholism. Under the influence of alcohol he forgot his trouble and had a feeling of well-being.

Case 22.—A married man, aged 44, a broker, of English-French stock, had been drinking alcohol steadily since the age of 28 and to excess. He had definitely chronic alcoholism. The father was healthy and the patient was apparently much attached to him. The mother was likewise healthy and normal. One sister, who was older, took the mother image. One brother was a homosexual pervert and alcoholic. This patient showed relatively few signs of pampering and egocentricity. He had definite swings of mood. He had a considerable though a declining sense of social responsibility. He got along poorly with his wife, and there had been instances of infidelity, promiscuity and separation on his part. One time he lived for several months with another woman. No overt homosexuality was elicited. He had two children and showed some sense of responsibility for them. He stated that before drinking he usually became nervous, sometimes depressed, and wanted to bite his tongue. Alcohol gave him a sense of exhilaration; it made him feel buoyed up and optimistic, and he tended to spend more money at that time. At first drinking was of a social character, but later he drank chiefly in solitude. He had shown definite hallucinosis at various times, and twice he had shown a tendency toward a paranoid state.

Case 23.—A woman, aged 44, divorced, of Irish stock and with no occupation, first drank at the age of 21, but began drinking heavily at 34. The drinking became periodic and dipsomanic, the bouts lasting from one to three days. The father was said to have been normal, sensible and not alcoholic. The mother, on the other hand, was pampering and possessive. One brother died from a tumor of the brain but was otherwise apparently normal. The patient, an only daughter, was unquestionably pampered in childhood. She showed definite unreliability,

egocentricity and deceitfulness and had no sense of social responsibility. She was cyclothymic and markedly extroverted. She had periods of depression. Her marital life was so unhappy that divorce ensued on the grounds of her husband's infidelity. She, herself, was never unfaithful or promiscuous. She gave evidence of a persisting love for her husband who ran away from her. She had one child of psychopathic make-up, whose conduct greatly worried her. Before drinking she became depressed and worried over her daughter's condition and felt more intensely the loss of her husband, and her isolation. Alcohol buoyed her up and made her feel normal. All her drinking was done in solitude and secret. She had occasionally indulged in camphorated tincture of opium.

CASE 24.—A married man, aged 48, a broker, of English stock, began drinking at the age of 20. He drank to excess at 23. The drinking was dipsomanic, the bouts lasting about three or four days. Both the father and mother were regarded by him as religious and excessively strict. Two older brothers were both alcoholic. The patient was unreliable and deceptive in his statements. He was not particularly egoistic or pampered, and showed a considerable sense of social responsibility. He was subject to definite swings of mood and periods of depression. Marked marital discord resulted in separation. The patient was promiscuous sexually and unfaithful. Most of his drinking was done with men. He gave as the cause of the drinking his separation from an extravagant wife, and a feeling of fatigue, over-work and depression. Alcohol tended to "buck up his spirits." At first his drinking habits were social, later more solitary.

CASE 25.—A married woman, aged 50, with no occupation and of unknown stock, began to drink to excess at the age of 26. The drinking was dipsomanic, the bouts lasting three or four days. The father was also dipsomanic. The mother was a religious fanatic. The parents were incompatible. Nothing was known of the patient's siblings. She was deceptive in statements but not especially egocentric or spoiled. She was definitely subject to periods of depression, and was extrovert. Her marital life was filled with discord, perversions and beatings, and ended in separation. The patient acquired syphilis in addition to alcoholism. She was not sexually promiscuous and it is probable that she derived the syphilis from her husband. She had two children and showed some interest in them. Before drinking she felt miserable, irritable and unable to sleep. Alcohol made her profane, slipshod and depraved. She drank entirely in solitude. She took no drugs. There had been frequent attacks of hallucinosis. The patient developed a paranoid trend relative to her husband.

CASE 26.—A widower, aged 52, an engineer, of Irish stock, had taken alcohol all his life, but had drunk to excess only during the year preceding admission to the sanatorium. The consumption of alcohol was more or less daily and chronic. The father was likewise alcoholic at the end of his life. The patient's mother died when he was 2 years of age. He had two sisters, but no brothers; hence he was an only son. He received little pampering, however, and was not self-centered. He showed some social responsibility. He was fairly frank in his attitude toward his problem. He admitted depression. He was friendly and extroverted. He and his wife got on well, but shortly before the onset of his excessive drinking she died of cancer. He had been sexually promiscuous before marriage but not since. He had eight children, and showed definite parental responsibility. As an excuse for drinking, he gave a sense of loneliness after the death of his wife and worry over financial losses. Alcohol overcame worry, but made him dizzy and gave him indigestion. He drank mostly in solitude.

CASE 27.—A married man, aged 53, a clothier, of Irish stock, began drinking to excess at the age of 19. He showed chronic alcoholism. The average period of excessive intoxication extended to one month. The father was strict and harsh. Little was known about the mother who died when the patient was young. He was an only son, but had two sisters, one of whom was alive and well. The patient showed no special pampering or egocentricity but was unreliable in statements. In spite of his alcoholism, his business was fairly successful. He had a definite cyclothymic make-up. He made friends quickly and freely. His marital life had never been very happy. He had probably been promiscuous before his marriage, but not unfaithful since. He drank only with men. Before drinking he was melancholy, discouraged and uncomfortable. Alcohol made him talkative, hilarious and later quarrelsome and boastful. He drank chiefly in company.

CASE 28.—A married man, aged 58, a real estate broker, began drinking to excess at the age of 17. He had definite chronic alcoholism. The father was a firm disciplinarian. The mother was alive, well and apparently normal. Little was known of the patient's siblings. He was extremely unreliable in statements, dishonest and deceptive; he made a great pretence of wealth and responsibility but actually was dependent financially on his father. He had little actual social importance. He made friends readily, principally with women. His married life was unhappy and ended in separation. He was both unfaithful and promiscuous. Before taking alcohol he had a feeling of restlessness, irritation and general misery. Under the influence of alcohol he became exhilarated and spent money freely. He had had delirium tremens.

CASE 29.—A man, aged 60, divorced, of English-German extraction, occupied himself with collecting rents. He began drinking to excess at the age of 30. Drinking was characteristically dipsomanic, the bouts occurring every six months and lasting two to three weeks at a time. The father had drunk to excess and treated the patient's mother badly. The mother died of diabetes. She was much attached to the patient who was an only child. He was brought up in a spoiled manner. He was extremely egoistic and showed little social responsibility or care. He was cyclothymic and subject to depression and periods of exhilaration. He usually made friends quickly. Shortly after his marriage, from which he had two children, his wife ran away with another man, and divorce ensued. He admitted some infidelity, but he declared that alcohol tended to reduce sex desire. He showed some parental responsibility. He stated that he took alcohol when he got frightened. It made him feel more confident and exhilarated. He always drank in solitude to buoy himself up. He used no drugs and had no hallucinosis.

CASE 30.—A man, aged 62, Irish, single, an association secretary, began drinking at the age of 32. Excessive drinking began two years before admission to the sanatorium. Drinking was dipsomanic, the bouts lasting from one to three weeks. Both parents were apparently normal but one brother died of alcoholism. The patient did not show signs of pampering. He had considerable social responsibility. No emotional instability was present. The patient was extroverted, making friends quickly. He was inclined to moderate swings of mood. He had no marked sex urge but had had several mistresses. He gave as a cause for drinking a certain lack of confidence in himself, and self-consciousness in making speeches and giving reports. Alcohol exhilarated and then made him quiet and sleepy. He drank both in company and in solitude.

Clinical Notes

THE DIAGNOSIS OF RACEMOSE CYSTICERCOSIS DURING LIFE

Changes in the Cerebrospinal Fluid *

A. E. KULKOV, M.D., MOSCOW, RUSSIA

The diagnosis of cysticercosis of the brain during life presents great difficulty because of the variability of its clinical course as well as the absence of characteristic symptoms. The symptomatology of cysticercosis of the fourth ventricle is distinct in its characteristics, and the diagnosis during life is more or less probable; much more complicated is the diagnosis in other localizations of cysticercosis (in the presence of a limited process), and diagnosis becomes impossible when a disseminated racemose form of cysticercosis is present.

The difficulty of diagnosis is generally attributed to the rarity of this special form of disease. In the literature treating of this particular problem, one finds a series of reports in which the writers consider the possibility of the diagnosis slight during life, but, nevertheless, attempt to establish a symptomatology on which the diagnosis may be based with more or less probability. Recently, certain changes in the cerebrospinal fluid have been emphasized as of considerable assistance in the diagnosis, although the changes cannot be regarded as pathognomonic.

I have had an opportunity to observe a case of cysticercosis, the clinical course of which showed a series of interesting neurologic and psychic peculiarities as well as a number of changes in the cerebrospinal fluid. I shall emphasize the most striking features of this symptomatology which have the greatest importance in the diagnosis. In the literature only sporadic cases are mentioned in which a correct diagnosis was established during life.

REPORT OF CASE

Clinical History.—R., a man, aged 42, was admitted to W. A. Obuch's Institute on Aug. 11, 1927, complaining of constant headache of rheumatic character, with vomiting, vertigo when walking, a tottering gait, occasional general weakness and fatigue. The onset had occurred a month and a half previously with vomiting, severe headache, giddiness and occasional diplopia. The gait became unsteady, and there was extreme fatigue. Later, the patient was able to walk steadily. He had some difficulty in answering questions quickly, and the headache became more severe, particularly at night and when bending the head forward or to the right. His father was a dipsomaniac and died of cancer of the esophagus. The mother died of an abdominal tumor. The family consisted originally of five persons; two of the children had died. The patient was the fifth child, born at full term, and had a normal mental history. He used tobacco and drank moderately. As a child, he had measles and angina.

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* From the Division for Nervous Diseases of W. A. Obuch's Institute of Moscow, Prof. S. N. Davidenkov, Director.

Examination.—The patient was tall and fairly well nourished. The neck anteriorly showed dark pigmented areas. The pupils were unequal, the right larger than the left; they did not react to light but responded in accommodation. There was facial weakness on the left side and the tongue protruded a little to the right. The cranial nerves were otherwise normal. Active and passive movements of the limbs, as well as the muscular strength and tonus, were fairly well preserved, as were all forms of sensibility. Subjectively, there were various complaints of acute pain, particularly in the right frontal region. Associated movements of the hands were rather disordered (dysdiadokokinesis). The Romberg sign was absent. The triceps reflex was decreased on the right and normal on the left; the knee reflexes were active; the achilles response was normal; the abdominal reflex was more active on the right; the cremasteric reflex was normal. The optic disks showed papilledema; the limits were obliterated, the veins dilated and the arteries contracted. There was hemorrhage near the disks.

The cerebrospinal fluid pressure in the sitting position was 600 mm. of water; the fluid was clear and colorless; lymphocytes numbered 7 per cubic millimeter. The Nonne-Apelt sign was negative; the result of the Pandy test was positive; 9 cc. of the fluid was removed. The colloidal gold reaction was 1122210000; sugar, 0.048 per cent; the Wassermann reaction was negative. The Wassermann reaction of the blood was negative.

The hemoglobin content was 84 per cent; leukocytes numbered 4,650; neutrophilic leukocytes, 56 per cent; lymphocytes, 38; mononuclear cells, 2; eosinophilic cells, 3.

Roentgenograms: The region about the sella turcica was vaguely outlined, and the sella turcica itself was obliterated. The whole picture suggested a tumor.

Disseminated râles were heard over the lungs. The heart was enlarged; the liver was not palpable; the blood pressure was 125 systolic, 65 diastolic.

Course.—February 22: While walking, the patient showed a tendency to fall to the right. There was a distinct Romberg sign; the temperature was 37.2 C. (98.9 F.); there was impairment of the senses of smell and taste; mentality was unimpaired; the pulse was 72 in rate and regular. Anisocoria was present, the pupil being greater on the left than on the right; the pupils reacted sluggishly to light, in accommodation and in convergence. Dysdiadokokinesia was especially noticeable in the left hand. The knee jerk was exaggerated on the left; from time to time ankle clonus appeared. The finger-to-nose test on the left was less well performed than on the right.

February 23: There were attacks of severe headache. The patient left his bed and started for the corridor, but could not reach it on account of sudden giddiness; consciousness was unimpaired; the temperature ranged from 37.3 to 39.4 C.

February 27.—During the night, the patient got up and walked about the room unaware of where he was. He answered questions slowly, the headache was diminished; cranial percussion showed tenderness, especially on the right; muscular strength in the distal parts of the lower extremities was somewhat diminished. The pulse rate was 78, with imperfect filling of the vessels, but rhythmic; the pupils were unequal, the left being larger than the right.

March 1: Sleep was poor; the neck muscles showed tenderness posteriorly, especially when the patient turned his head; there was no rigidity.

March 2: The temperature was 37.8 C.; the patient slept much, and talked little and very unwillingly. He was disoriented as to time. When asked what hospital he was in he answered "St. Paul's." He was euphoric; he said that his headache was distinctly diminished, that he was "better," and that he was

"all right." Slight rigidity of the neck persisted with a distinct Kernig sign. If the facial nerve along the ascending branch of the lower jaw was compressed, pain and contraction of the facial muscles of the corresponding side occurred. This appeared at times spontaneously; paresis of the lower branch of the seventh nerve was present; the knee jerk on the left was exaggerated, as were both ankle jerks. There was incontinence of urine.

March 3: Orientation was clouded; the patient did not recognize his wife; muscular tonus of the lower extremities was increased on the right. There was rigidity of the neck, with euphoria.

March 7: Consciousness was rather clouded; the patient did not know where he was, and he was also disoriented regarding time; he thought that he was at home near his working place. Slight rigidity of the neck and a Kernig sign were observed; he did not recognize persons about him; he, however, discerned surrounding objects, pointed out of the window and apparently considered that the physicians were old acquaintances. He was euphoric and showed some humor. He had no insight into his position. He was given to fantasy formation, confabulation and pseudoreminiscence. His memory was distinctly impaired, especially regarding recent events. He was, however, able to count and his attention was easily held. The knee jerk was more marked on the left than on the right; clonus was noted on the left; the ankle jerk was somewhat more active on the left than on the right.

March 8: There was no rigidity of the neck; the face was drawn to the left. From time to time, there were involuntary facial movements; weakness in the left hand was noted; there was a slight Kernig sign on the right; the knee reflexes were active, more on the left than on the right.

March 9: The pupils did not react to light; the neck was distinctly rigid; the knee jerks were unequal, more active on the left; a tendency to clonus was more pronounced on the right; the abdominal reflexes were more active on the left, and very slight on the right.

March 12: The psychic state continued the same. The tongue deviated to the right; there was no rigidity of the neck; a Kernig sign was present; there was persistent headache with poor sleep.

March 13: The patient was very quiet and showed better orientation. There was slight increase of muscular tonus in the lower extremities. The eyegrounds showed pale disks with lost outlines; the veins were dilated and the arteries contracted.

March 14: Mentally, the patient was more disturbed, disoriented and euphoric; rigidity of the neck and a positive Kernig sign were present.

March 17: There was a change for the worse; the patient brought together his belongings and attempted to go downstairs to the storeroom where he intended to take up his abode; no rigidity of the neck and no Kernig sign were observed.

March 23: Mentally, the patient was better; the tongue deviated to the right; a slight Kernig sign was present; the knee jerk was exaggerated on the left, together with ankle clonus. The abdominal reflexes were more marked on the right; the cremasteric reflex was equal on the two sides; incontinence of urine was present.

March 24: Vision was poor; the fundi showed dilatation of the veins with hemorrhages; the pupils were unequal and did not react to light or in convergence.

March 25: The patient could not be made to answer questions; he was extremely weak.

March 26: The patient was in a state of stupor; he showed irritability, gave strange answers and did not recognize his wife. He often attempted to get out of bed and escape; during the night, he had certain hallucinations.

March 29: The muscles of the neck were rigid. Lumbar puncture in a supine position gave a pressure of 370 mm. of water; the fluid was clear and colorless; a cell count revealed 57 lymphocytes. The following results were obtained in various tests: Nonne-Apelt, +++; Pandy, ++++; Weichbrodt, negative; colloidal gold curve, 565664400; Wassermann, negative; globulin and Kahn, negative. The sugar content was 0.032 per cent.

March 30: The Babinski sign was absent, but there was an Oppenheim reaction on the left.

April 3: The patient was unable to discern surrounding objects; he showed disorientation in time and was irritable. There were slight meningeal symptoms.

April 4: Sight was deeply involved.

April 9: The patient was stuporous; no pathologic reflexes were present; he had had hallucinations.

April 12: There was no rigidity of the neck; a slight Kernig's sign was present; the knee jerk was greater on the right than on the left.

April 14: The patient was irritable and tried to escape; he was controlled with difficulty; he was poorly oriented.

April 17: The patient was drowsy and apathetic. The face was drawn to the left; the left hand was weak, but at times contractions of that hand were noted; no abdominal reflex was obtained.

April 18: There was complete paralysis of the left extremities. The face was expressionless and showed some distortion; speech was normal; the corneal reflex on the left was slow; the periosteal reflexes on the left were increased; the knee and ankle reflexes on the left were diminished; there was a positive Babinski sign; the defensive reflexes (Marie-Foix) were positive; automatic movements were present on the right. On this date, the patient was transferred to the surgical clinic of the First State University of Moscow for operation.

Comment.—A summary of the foregoing clinical history shows a man, aged 42, who was attacked by disease about a month and a half before admission to the neurologic section, followed by vomiting and severe headache, and later diplopia with impairment of gait. During his stay in the hospital, there occurred first a general aggravation of symptoms followed by symptoms of intermittent character which were easily traceable throughout the whole course of the illness. He had anisocoria, with sluggish or no reaction to light, facial asymmetry and deviation of the tongue to the right. Dysdiadokokinesia was noticeable, chiefly in the right hand. The tendon reflexes of the arm were diminished on the right side; the knee jerks were very active; the optic disks showed papilledema, dilatation of the veins and hemorrhage around the papillae. The cerebrospinal fluid pressure was high at the first test, 600 mm. of water in the sitting position, and the fluid showed a positive colloidal gold curve. A blood count revealed lymphocytosis and eosinophilia, 3 per cent. Further symptoms appeared later, occurring after the manner of seizures, from which the patient soon recovered. There were lucid intervals of from two to three days when he was in rather good condition. The gait of the patient during the whole course of the illness showed considerable disturbance; orientation in space and time was poor. Occasionally he showed irritability and made attempts to escape. He was euphoric and at times showed humor. He had a tendency to confabulation and at times to hallucinations; memory was impaired and there were occasional meningeal symptoms; hemiparesis of the left side later developed into a definite hemiplegia with pyramidal tract symptoms. A second lumbar puncture showed high pressure, acute lymphocytosis, a positive globulin reaction and a paretic colloidal gold curve.

The symptomatology as well as the clinical course of the disease suggested the diagnosis of syphilis of the brain, but signs of increased intracranial pressure (spinal fluid, severe headache) established the diagnosis of tumor of the brain. With this diagnosis, the patient was transferred on April 19, 1928, to the surgical clinic of Professor Burdenko for decompressive craniotomy, following which, on May 11, 1928, he died.

Necropsy.—A postmortem examination was made by Prof. I. V. Davydovsky. The dura showed evidence of considerable tension. At the seat of the operation wound, the dura was adherent to the arachnoid; blood was present in the dural sinuses. In both hemispheres the arachnoid was diffusely thickened and whitish. In the region of the chiasm, the arachnoid was adherent to the sella turcica; here it was thickened and dense and somewhat suggested the appearance of soft transparent cysts. In the right hemisphere, there were a number of yellowish areas of various sizes. The brain substance was collapsed. The lateral ventricles were considerably dilated and filled with large quantities of fluid. On section of the right hemisphere, a number of dark soft areas were revealed, spread throughout the whole hemisphere. The basilar artery was thickened, whitish and dilated. On section at a point where the posterior cerebral artery is given off, a large thrombosis was found in the lumen of the basilar artery closely adherent to the vessel wall. At some points the vessels were considerably narrowed.

DIFFERENTIAL DIAGNOSIS

From the pathologic-anatomic standpoint, the diagnosis presents great difficulty, in differentiating between *Cysticercus* cysts and syphilitic leptomeningitis. The changes in the brain, the dura and the arachnoid suggested cysticercosis, although the scolex was absent and there were no changes in the blood vessels. The possibility of a syphilitic origin of the process was likewise considered. Microscopic study of sections of the sclerotic regions of the arachnoid did not support the diagnosis of syphilis. The section of the cyst membrane presented a picture similar to that of racemose cysticercosis. The outer layer of the cyst consisted of a delicate chiten cuticle; the parenchymatous layer contained numerous cell elements. In the cyst walls, there were calcareous corpuscles of various sizes. In the surrounding tissues, the dura and the arachnoid as well as the brain itself showed distinct inflammatory changes. In a number of places, small areas of necrosis, with an inflammatory reaction about them and an accumulation of giant cells, were observed. The brain tissues were edematous. A number of softened areas were present, with a considerable quantity of granular ball-like formations. The ependyma of the lateral ventricles was thickened and revealed a number of small areas resembling granular ependymitis. In the brain vessels, an obliterating endarteritis was noted. The elastic membrane was not involved in the process. The lumen of the vessels was for the most part obstructed by the connective tissues of the internal membrane. The vessel walls were infiltrated throughout, chiefly by lymphoid elements.

In my case, therefore, the clinical diagnosis of racemose cysticercosis seemed to present an insoluble problem. The pathologic-anatomic data did not reveal at once the true character of the disease. As the symptoms gave reason to suspect the existence of a syphilitic process, similar cases are of interest.

The characteristic clinical features of cysticercosis of the brain simulate closely those of tumor of the brain. Cases have been reported in which during the course of the illness no neuropathologic symptoms were revealed (according to Kuchenmeister, 16 of 88 cases). In my case, one's attention is drawn to the fact that

the symptoms showed great variability and were intermittent. The headaches were at times severe, but were followed by intervals in which the patient was in fairly good condition. One of the most characteristic symptoms of the disease is Brun's symptom-complex, namely, sudden dizziness with vomiting, and at times death following a quick movement of the head. Stern[1] enlarged the limits of this sign and spoke generally concerning the connection existing between changes in the position of the head and sudden attacks of severe headache, dizziness and vomiting, symptoms which chiefly concern cysts of the fourth ventricle. This symptom-complex, though not pronounced, was observed in my case, but apparently it concerns the localization of the process — the fourth ventricle — rather than its etiology. In my case further symptoms were: anisocoria with sluggish pupillary reaction; occasional asymmetry of the face, but varying somewhat during the course of the illness; dysdiadokokinesia and a distinct Romberg sign with a tendency to fall to the right; the knee and ankle jerks were very active, but varied in intensity on the two sides. A considerable time after the onset of the disease edema of the optic disks occurred.

Some observers mention that attacks of cortical epilepsy frequently occur during the course of the disease together with hemiparesis or hemiplegia. In my case, only a slight weakness of the left arm and leg was noted, which after a time developed into a hemiplegia with obvious involvement of the pyramidal tract and slight spasmodic twitching of the extremities. The chief changes, therefore, were evidently in the right hemisphere, which the necropsy revealed to be the seat of a number of areas of softening. In this form of the disease, symptoms of excitement prevail; meningeal symptoms are also often mentioned, but here again great variability is noted. In my case, there were on several occasions a pronounced rigidity of the neck and a distinct Kernig sign. When these symptoms disappeared, they were succeeded by lucid periods. Of exceptional interest were the changes in the psychic state that usually accompany this form of the disease: general confusion, disorientation in time and space, occasional clouding of consciousness, hallucinations, excitement, distinct failing of memory, irritable behavior, euphoria, occasional stupor and general impairment of the intellect. These were especially well developed in my case. Attention should be drawn to the fact that the psychic symptoms, like the neurologic symptoms, were unstable and varied from time to time. After a period of deep confusion and disorientation, lucid intervals occurred when the patient enjoyed full consciousness and was in relatively good condition. This observation has been made by a number of writers who have pointed out the importance of psychic changes in cases of cysticercosis of the brain (Gianulli, Salomez, Guillain,[2]). In some of the cases described, a hysterical symptom-complex has developed, usually at the beginning of the illness. It is only in the later course of the disorder that an organic lesion has been demonstrated, induced by *Cysticercus* (Babchin[3]). A distinguishing point in the otherwise very similar symptoms occurring in tumor of the brain is the intermittent course of the disease, which is sometimes of long duration, together with

1. Stern, A.: Ueber Cysticerken im IV v., Ztschr. f. klin. Med., 1907, vol. 61.

2. Guillain; Bertrand, and Péron: Études anatomocliniques d'un cas de cysticercose cérébrale avec méningite parasitaire par cysticercose racémeuse, Rev. neurol. **1**:1018, 1926. Guillain; Périsson; Bertrand, and Schmite: Cysticercose cérébrale racémeuse, ibid. **2**:433, 1927. Guillain; Péron, and Thévenard: Compt. rend. Soc. de biol. **95**:455, 1926.

3. Babchin: Ksimptomatologii i operationomu lecheniyu cycticerkoza mozga, Nov. Khirurg. Arkhiv, 1928.

the variability of the neurologic and psychic symptoms. To this marked variability of symptoms in cases of cysticercosis of the brain, attention must particularly be drawn; this has also been emphasized by others. The second characteristic feature is Brun's sign; if definitely developed, it is of the greatest importance, especially in cases of cysticercosis of the fourth ventricle. In cases of racemose cysticercosis, the importance of this is relative. Sudden death is also considered as one of the most characteristic features of the disease. From the diagnostic standpoint, other symptoms are of relatively little value in differentiation from tumor of the brain.

In my case, the illness was of short duration, about two months. The patient died following an operation. The most striking feature of the disease, namely, the intermittent character of the symptoms, was apparently caused by the varied degree of hydrocephalus as well as by pronounced changes in intracranial pressure. According to Stern, the racemose cysticercosis is particularly apt to show remissions. While the clinical symptoms of the disease do not give information of great importance as to diagnosis, a study of the cerebrospinal fluid may help decidedly. In order to simplify the most striking features of the changes which take place in the cerebrospinal fluid during the course of development of the brain cysticercosis, I am appending in the accompanying table a summary of reported cases in addition to my own. These cases are in some instances not completely reported; yet they present certain important leading points which are of assistance in establishing the diagnosis during life. Not including my case, sixteen cases have been reported in which analysis of the spinal fluid was made. The chief observations were: In all cases the fluid was clear and transparent, with one exception (Morawiecka,[4] 1928), in which xanthochromia and spontaneous coagulation of the fluid were noted. In this case, the presence of *Cysticercus* cysts in the cerebrospinal fluid was revealed. In most of the cases the intracranial pressure was rather high, ranging from 75 to 620 mm. of water. Furthermore, one of the most characteristic features was a sudden dropping of the pressure after a small amount of the fluid escaped, explainable by the absence of free communication between the subarachnoid space and the brain ventricles. The globulin reaction was for the most part positive; the protein content was heightened. One of the most important observations was the cell count. Here, in addition to a distinct lymphocytosis which reached the number of 312 per cubic millimeter (Guillain), eosinophilia was noted which, according to a number of observers, was sufficient to establish the diagnosis of cysticercosis (Rizzo[5]). Eosinophilia in the cerebrospinal fluid was also observed in a series of cases reported by Guillain and Grund[6] in 1913 (from 8 to 10 per cent), and by Rizzo in 1928 (from 45 to 48 per cent), with a corresponding eosinophilia of the blood, in this case, of 2.4 per cent.

Among other characteristic features of major importance in the cerebrospinal fluid must be mentioned the discovery of the cysts. This was noted once in the case of Morawiecka. *Cysticercus* cysts in the spinal fluid were also demonstrated in a case reported by Sterz (1913), in which a fibrin net was present. In a case reported by Hartmann,[7] after lumbar puncture a blood coagulum contained

4. Morawiecka: Méningite spinale à cysticerques, Rev. neurol., 1928.

5. Rizzo: Diagnosi in vita di cisticercose cerebrale, Riv. di patol. nerv. **32:** 952, 1928.

6. Grund: Ueber Eosinophilie in Liquor cerebrospinal Rautengrube Cysticercus, Deutsche Ztschr. f. Nervenh., 1913.

7. Hartmann, F.: Cystericercus cerebri diagnosticiert durch die Lumbalpunktion, Wien. klin. Wchnschr. **15:**547, 1902.

Changes in the Cerebrospinal Fluid in the Presence of Cysticercosis of the Brain

Author	Date	Color	Level of the Fluid in Mm. H_2O	Nonne-Apelt Test	Pandy Test	Weichbrodt Test	Protein, Total	Cells per C.Mm.	Colloidal Reactions	Wassermann Test	Biochemical Analysis, per Cent	Parasite	Comment
Kulkov..... (first lumbar puncture)	2/16/28	None	600 (sitting position)	0	++	0	....	7	Colloidal gold 1122221000	0	Sugar, 0.048	Absent	
Kulkov..... (second lumbar puncture)	3/29/28	None	370 (supine position)	+++	++++	0	....	57	56566440000	0	0.032	Absent	
Guillain,	9/1 and 9/21/26	None	250	..	+	0	0.45	76	Colloidal	0			
Bertrand	10/2 and 10/7/26		(supine)		+	0		78	benzoin				
Péron	10/3 and 10/11/26		530 (sitting)					80	positive				
Guillain	10/24/27	None	140	..	+	+	0.71	160	Colloidal	0	Sugar,		
Périsson	11/ 2/27		320 (sitting)	..	+	+	0.71	312 (eosinophils)	benzoin positive	0	0.52 Chloride, 0.71		
			300	..	..	..	0.71	120		0			
Stern.....	1927 four successive days	None	620 75 230 175	..	..	..	N	20 to 30 +					
Sterz.....	1913	None	...	+	..	..	Heightened	Distinct	.....	...	.....	Twice parasitic cysts	A fibrin net
Grund.....	1913	None	165	..	..	..	....	Eosinophils (polymorphonuclears) 8 to 16	.....	0	.....	.....	Crystals Charcot-Leyden
Rizzo.....	11/15/28	None	...	+	..	..	....	411 eosinophils (45%)	Kafka positive	0			
Rizzo.....	11/22/28	.....	...	++++	..	..	0.4%	56 eosinophils (48%)	M. R. paretic curve	0	.....	.....	Eosinophilia in the blood
Morawiecka.....	1927	xanthochromia	...	..	..	..	....	..	Cysticercus cysts (5)	...	.....	.....	Coagulation
Hartmann.....	1902	.....	...	..	..	..	....	..	Cysticercus cysts in the coagulum (cysticercus cellulosae)				

the parasite. In such cases, the diagnosis of cysticercosis is definitely established. The colloidal reaction (colloidal gold, colloidal benzoin, etc.) showed a strikingly characteristic curve, often similar to those of dementia paralytica, which evidences deep parenchymatous lesions of the brain. In all cases, the Wassermann reaction was negative.

In my case, the observations were: The cerebrospinal fluid was clear and transparent; the pressure was high; at the first puncture, the globulin reaction was slightly positive, and at the second distinctly positive; there was a lymphocytosis of 7 and 57 per cubic millimeter; the colloidal gold reaction gave at first a syphilitic and second a distinctly paretic curve; the Wassermann reaction was negative, no *Cysticercus* cysts were revealed. There was an eosinophilia of 3 per cent in the blood, not obtained in the spinal fluid. The presence of sugar in the spinal fluid (0.048 and 0.032 per cent) should be attributed to the presence of the parasite. Although rather inadequate, these data allow one to point out a certain cerebrospinal fluid syndrome that should help to establish the diagnosis during life. Among the definitely positive signs are: (1) the presence in the spinal fluid of the parasite itself, (2) the more or less distinct eosinophilia. Other features of the fluid, which are not pathognomonic are: the combination of a paretic type of colloidal reaction with a negative Wassermann reaction; distinct lymphocytosis, and positive globulin reaction. These suggest some organic process other than syphilis. Should, however, the last data be considered alone, a diagnosis of cysticercosis would be out of the question. In combination with other clinical symptoms, however, they may have diagnostic significance.

CONCLUSIONS

1. The diagnosis of cysticercosis during life presents the greatest difficulty, since the disease shows no characteristic clinical course. Among other special features which permit one to surmise the diagnosis during life are: the intermittent course of the illness itself as well as of its individual symptoms, both neurologic and psychic; and the characteristic changes in the psychic state, such as general confusion, disorientation, hallucinations, excitement, failure of memory, etc.

2. Important changes in the cerebrospinal fluid are: (*a*) discovery of the parasite; (*b*) eosinophilia; (*c*) increased brain pressure; (*d*) a positive globulin reaction, and (*e*) the paretic character of the colloidal reaction curve.

LILLIPUTIAN HALLUCINATIONS FOLLOWING THE USE OF CAFFEINE CITRATE*

PHILIP WORK, M.D., DENVER

Instructor in Neurology, University of Colorado School of Medicine; Attending Physician (Neurologist), Children's Hospital

In his essay on Lilliputian hallucinations,[1] Leroy cited a considerable number of French references to this condition, but the English and American literature is singularly deficient in similar observations, though the syndrome must be of

* Submitted for publication, Nov. 29, 1929.

1. Leroy, Raoul: Syndrome of Lilliputian Hallucinations, J. Nerv. & Ment. Dis. **56**:325, 1922.

frequent occurrence. Vonderahe [2] recently reported it in a case of acute hyoscine poisoning.

Leroy, who apparently first designated the syndrome "Lilliputian," defines it as . . . "the vision of small people, men and women of minute or slightly variable size either above or accompanied by small animals or small objects proportionate in size. These hallucinations are motile, colored, generally multiple. . . . These microscopic visions give an impression of real life." [1] It should be remembered that these small figures operate in an environment, of normal size, and they are not to be confused with a true micropsia. As in other visual hallucinations, there is seldom associated aural hallucinosis, but if it should exist, the figures speak in an elfin voice.

Leroy conceded and cited references to prove that these hallucinations occur in psychic, hypnagogal and toxic states and asserted that the classic mood is euphoria though he later said that toxic states as a rule produce terrifying visions. Alcohol, however, the most common of the toxic agents, not infrequently induces innocuous or definitely pleasurable hallucinations. The tone of the affective state at the time conditions the mood of the hallucination just as the state of the organic substructure determines the affective state.

"Lilliputian" hallucinations owe their name to Swift's immortal pen, but the syndrome has played a part ever since his time in the literature of the world, vide Anatole France.[3] The occurrence of this syndrome in disease may perhaps be predicated on previous knowledge of such literature, a prior familiarity with the Lilliput theme. It is hard to understand the terror sometimes evoked by these diminutive and presumptively harmless mites.

The works, both lay and medical, collected by Leroy noted the existence of the syndrome in a great variety of psychotic, hypnagogic or toxic states. Leroy himself said: "There are scarcely any mental maladies, toxic or toxi-infectious states where one cannot find the syndrome." In the exogenous toxic group, ether, cocaine, hashish and curare are mentioned specifically, and alcohol stands preeminent. Kraepelin [4] gave a recognizable description of it in discussing alcohol; he called the figures "winzige Männchen." Curiously enough, opium does not seem causative. Maury [5] related his own hypnagogic experience in 1843 following the fatigue incident to a long journey in a stage coach. The minute hallucinosis reported of various saints and mystics can well be attributed to inanition and religious emotional frenzy.[6]

I here report a case presenting a Lilliputian hallucinatory syndrome in a neuropathic negress following prolonged automedication with caffeine citrate. I cannot discover any reports of the effect on the mind of excessive dosage of this drug. Researches in this country have been on the beverages, and none so far bears on the extreme sensorial changes induced by it. The following observations cover two months, beginning fifty-two days after the last dose of caffeine. This patient, mentally brillant, could not recollect that she had any previous knowledge of Swift's creations. Her basic mentality was high and her emotional threshhold was low, as evidenced by her past history of mental disturbance and her present marked instability and hypersuggestibility.

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2. Vonderahe, A. R.: Lilliputian Hallucinations, Arch. Neurol. & Psychiat. **22**:585 (Sept.) 1929.
3. France, Anatole: The Crime of Sylvestre Bonnard.
4. Kraepelin, E.: "Psychiatrie" (Alcoholismus).
5. Maury: Le sommeil et les rêves, 1878.
6. Lives of the Saints.

REPORT OF CASE

Clinical History.—Z. H., aged 20, a full-blooded, unmarried, Mississippi negress, was admitted to the Children's Hospital on June 26, 1929, for "insomnia and nervousness." She was seen in consultation with Dr. Sara Wilcox, who gave me permission to study and report the case. The family history was negative for tuberculosis, psychosis and constitutional disease. The father, mother and one sister were living and well. The patient had mumps, pertussis, varicella and rubeola during early childhood. Tonsillectomy was performed some years previously. She had not had chorea. Menstruation began early and was now normal. The patient said that she had had no sex experience and did not use alcohol or tobacco. Following graduation from high school at the age of 14 years, she had a "nervous breakdown" (details not available) and was hospitalized for four months. That fall she entered a large northern state university and graduated, A.B.cum laude, three years later. She then matriculated in law at the same school, the only negro and one of the few women in the class and again graduated cum laude, in May, 1929, at less than 21 years of age. She characterized the law school as "very interesting and not hard." She had hopes of becoming the Portia of her race, but even in the hospital had insight into the handicaps imposed by sex and race.

Early in the spring of 1929, she became fearful for her academic standing and began intensive systematic study. Unable to keep awake, she resorted to the use of caffeine citrate which she took for seven weeks until commencement, when she stopped abruptly. The dosage varied from never less than 5 grains to 18 (0.32 to 1 Gm.) or more per day. Diuresis annoyed her only for the first two weeks, she insisted. After graduation, she was "keyed up" physically and mentally but could not think consecutively. She was "jumpy" and "could not sleep at all." She came to Colorado to rest before going into a law office, but did not improve. She tried to induce physical exhaustion by walking, mountain climbing, etc., but to no avail. Dr. Wilcox saw her in the middle of June and advised hospitalization.

Examination.—On admission, no organic disease was found. She was plump and not dehydrated, under great physical tension, apprehensive, suspicious and uncommunicative mentally. Laboratory tests: The red blood cells numbered 4,600,000; white blood cells, 6,000; hemoglobin was 65 per cent; differential count was normal. The blood chemistry showed: sugar, 83.3 and nonprotein nitrogen, 38. The Wassermann test of the blood and spinal fluid, vaginal smears and gastric analysis all gave normal results. The urine showed an inconstant trace of albumin, but nothing else. The spinal fluid was under increased pressure (22 mm. of mercury), but was normal in character.

At the time of the examination she was restless, uncommunicative and reacted wildly to all manner of stimuli, light, noise or touch. She was obviously influenced by visual hallucinations and would not stay long in bed. Station and gait were approximately normal, but she had a pronounced generalized muscular hypertonicity, increasing after stimulus to tetany. All deep reflexes were wildly exaggerated, and any attempt to elicit them set off the tetanic spasm, identical in appearance to that seen in strychnine poisoning. She had marked intention tremor of both hands. There were no paralyses or plegias, no Babinski sign or ankle clonus. The cranial nerves were not affected, and the optic disks looked normal, despite the increased spinal pressure.

She was well oriented and had fair insight into her condition, but was continuously preoccupied and apprehensive over visual hallucinations. She later

explained the apprehension as due to fear that "those little people" would overhear her telling on them and would later punish her in some unstated way. The hallucinations did not first appear following the use of any hypnotics and remained uninfluenced by later medication. For ten days before their appearance she had had inconstantly the idea that some vague person was following her. These "little people" were brightly and fantastically garbed male negroes about 4 or 5 inches tall, not resembling any of her acquaintances. They looked like and deported themselves as human beings, appearing only on relatively level surfaces, never on ceilings or walls. They were incredibly agile, jumping from floor to bureau or to bed at will. There were never any Caucasian images.

She first saw them when she suddenly found a group of them in conference on the bureau. They seemed to be talking about and watching her, and while she did not hear them, she "knew" that they were conspiring against her life. Some of the time these little people played about entertainingly, but from time to time they would try to carry off her law diploma (which, incidentally, was not there) and in general spent much time annoying her by various maneuvers, all the time inspiring in her an acute distrust of their motives.

They never attempted any physical harm, but by talking together and making threatening gestures, they inspired and maintained a fear that they would. When she got out of bed to protect her property, which they were trying to carry off, they vanished; they did not run away; they "just disappeared." At times these little figures got in her bed, and she felt that they contemplated a sexual attack, but they never harmed her.

Course.—With the patient under eliminative and reconstructive treatment, encouragement and reeducation, the figures disappeared finally, and the patient was discharged on August 29; she is now working in a law office, it is reported, doing well.

A SUPPORT FOR THE SUPPLEMENTARY TREATMENT OF FACIAL PARALYSIS *

A. R. VONDERAHE, M.D., CINCINNATI

In the treatment for paralysis due to involvement of motor cells of the anterior horn of the spinal cord and the peripheral nerves, orthopedic surgeons have long recognized the value of supporting the affected muscles in such a way that a position of moderate tone is maintained and the dead weight of the muscles is relieved from dragging on the blood vessels and particularly on the diseased nerve. By maintaining a position of partial contraction of the muscle by means of a support, the muscle is in the most favorable position in which to respond to the earliest and slightest impulses to contraction following regeneration. The results of such a method in many peripheral palsies are definite and often brilliant.

In the treatment for facial paralysis of the peripheral type, various supports for the involved muscles have been devised. Jaeger [1] described a method of

* Submitted for publication, Jan. 11, 1930.

* From the Department of Anatomy (Neurology), University of Cincinnati College of Medicine.

1. Jaeger, Charles H.: A Method for Improving the Treatment of Facial Paralysis, Arch. Neurol. & Psychiat. **1**:374 (March) 1919.

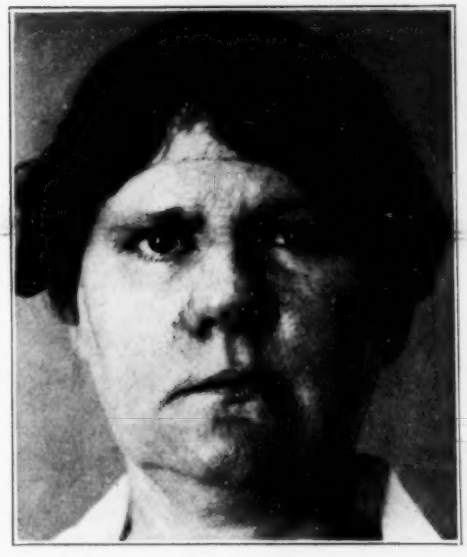

Fig. 1.—Patient with paralysis of the right facial nerve.

Fig. 2.—Support of vulcanized rubber modeled to fit the patient's cheek.

support consisting of a cap which fitted over the patient's head and which formed a point of fixation for strips of adhesive tape applied to the dropping cheek. This method was used in the case of a child, aged 3 years. Yawger [2] devised a method of support, using the scalp at the side of the head as a point of fixation for strips of plaster adjusted to support the cheek. Ombrédanne [3] made use of a hook which lifted up the sagging corners of the mouth by attachment to an upper back tooth. The use of a hook of silver inserted into the angle of the mouth and suspended from the ear is recommended by many.[4] Two objections have been advanced against the latter method: 1. The entire weight of the cheek is carried on a very small area. 2. Irritation of the mucous membrane is caused by the metal. The support illustrated in figures 2 and 3 obviates the first of these difficulties by using a broad smooth base on which the tissue rests, and

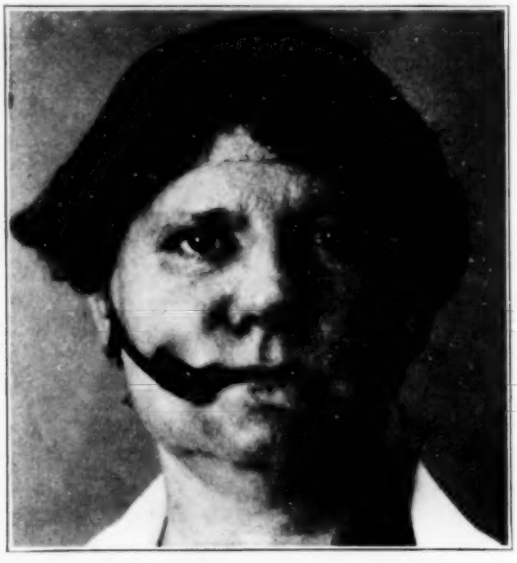

Fig. 3.—Support in place, showing correction of the paralysis.

the second difficulty is avoided by molding the support exactly to fit the configuration of the patient's mouth so that friction is almost entirely eliminated.

The support is best made by a dentist according to the physician's order. A wafer of modeling compound, softened by immersion in warm water, is made into a strip approximately 8 cm. long, 2.5 cm. wide and 0.5 cm. thick. An impression of the patient's cheek and angle of the mouth is taken; this makes an L, the inner limb of which is made to measure 5 cm. and the outer limb 3 cm. When the compound is hardened, it is removed from the mouth. It is then inserted in a flask containing plaster. After heating, the modeling compound

2. Yawger, N. S.: A Support in Facial Nerve Paralysis, Arch. Neurol. & Psychiat. **4**:659 (Dec.) 1920.

3. Ombrédanne, L.: Correction of Facial Paralysis, Presse méd. **29**:636 (Aug. 10) 1921; abstr., J. A. M. A. **77**:1053 (Sept. 24) 1921.

4. Dana, C. L.: Text Book of Nervous Diseases, ed. 9, Philadelphia, William Wood & Company, 1920, p. 117.

is removed and replaced by rubber which is vulcanized at 320 C. for fifty-five minutes. The rubber support is then removed, ground smooth with emery and pumice, and polished with whiting and rouge. With a burr, an opening is drilled into the outer limb for a silk ribbon which is attached as shown in figure 2. The support is inserted into the angle of the mouth, and the free ends of the silk cord are adjusted over the patient's ear and tied in a firm knot after sufficient traction has been made to correct the sagging facial muscles. A ring slipped over the ribbon serves to maintain the adjustment.

The support can be left in place day and night; it is readily removed and replaced by the patient; it is quickly and easily sterilized each day by immersion in any of the common antiseptic solutions. Other modes of treatment are used, the support being intended merely to supplement their action.

SPECIAL ARTICLE

PROGRESS IN PSYCHIATRY

II. THE ACTIVE WORK THERAPY OF DR. SIMON *

FREDERIC WERTHAM, M.D.
BALTIMORE

The fact that the majority of mentally diseased patients are found in institutions is often not sufficiently apparent in scientific discussions of psychotherapy. A great contrast exists between the minute differentiations of psychotherapeutic theories and methods applied or applicable only to relatively few persons and the scarcity of psychotherapeutic attempts for the large proportion of institutionalized patients. The latter, after all, form the bulk of psychiatric patients in need of medical aid. It may be said, therefore, that any advance in the psychotherapy of mental disease which is practicable on a large scale in institutional work deserves careful attention on the part of the psychiatrist. Such an advance is the active work therapy which Dr. Hermann Simon has introduced at the psychiatric state hospital at Gütersloh, Westphalia.

Simon first reported on his methods and their results in a number of lectures,[1] but he has recently published a full account under the title, "More Active Treatment of Patients in the Psychiatric Institution." [2] The foundation of Simon's treatment is occupational therapy, which has, of course, been practiced for many years. By "more active" he refers not only to the greater activity of the patient, but also to the greater activity of the hospital personnel. Treatment for all disturbed patients in institutions is a cornerstone of his system.

Simon described vividly the scenes that occur so frequently, and in many institutions habitually, in the disturbed wards. The patients are unsocial in the most conspicuous fashion; they yell, are obstreperous, tend to interfere with each other, destroy things, etc. In Dr. Simon's opinion, the root of these manifestations of disordered behavior is

* Submitted for publication, Jan. 16, 1930.

1. Simon, H.: Aktivere Therapie in der Irrenanstalt, Allg. Ztschr. f. Psychiat. **81**:425, 1925; Arbeitstherapie in der Irrenanstalt und ihre moderne Ausgestaltung, ibid. **86**:466, 1927; Psychotherapie in der Irrenanstalt, Bericht über den II. Allg. ärztl. Kongr. f. Psychotherapie, Leipzig, S. Hirzel, 1927.

2. Simon, H.: Aktivere Krankenbehandlung in der Irrenanstalt, Allg. Ztschr. f. Psychiat. **87**:97, 1927; II. Erfahrungen und Gedanken eines praktischen Psychiaters zur Psychotherapie der Geisteskrankheiten, ibid. **90**:69 and 245, 1928. (Published in reprint form by de Gruyter, Berlin, 1929).

inactivity. "Life is activity! This is true of physical and of mental life." He quoted the saying of an old psychiatrist: "Man never does nothing—if he does not do anything useful, he does something useless." And Simon added: At least he thinks something which is useless and ill-directed. Successful activity creates satisfaction and inward and outward poise; inactivity, on the other hand, creates ill humor, unwillingness and irritability. The latter leads to conflict with the environment, with its concomitant results and reverberations, and finally there results the terrible atmosphere of wards with excited patients.

Whatever the fundamental causes of mental disease may be, the manifestations are in the mental sphere. To influence these, an active psychotherapy is necessary, the main factors of which are occupation and education. These have been used for many years, but according to the experiences of Simon much more can be accomplished by them than was formerly believed. One of Dr. Simon's main theses is that many more patients can be treated with active work therapy than is usually done. He quoted figures according to which considerably less than 50 per cent of the patients in many German state hospitals have the benefit of occupational therapy; in others the percentage is from 50 to 60, and in a few cases it is as high as 80. In his own institution, to which the rank and file of mental patients of the district is admitted, just as in other German state hospitals, he reached at times an average as high as 99 per cent.

Simon's experiences date back to 1905. At that time, as the director of a new institution (Warstein), he had patients work in the completing of gardens, paths, etc. As more and more patients were occupied with work, it was found that a general improvement in behavior took place. Evidence of irritability and impulsive outbreaks decreased or even disappeared completely. Negativistic and obstreperous tendencies gave way to friendliness and willingness. Patients who had led a generally dulled existence took a renewed interest in their environment. After about nine years, nine tenths of all patients were regularly occupied. It was found that the whole atmosphere of the institution had changed to quietness and order. Simon told with pride of an episode when a visiting psychiatrist said he had seen enough of the "sanitarium," and asked to be shown the disturbed wards—after he had spent two hours in seeing the institution and its thousand-odd patients, including the most difficult wards.

The patients themselves offer little objection to regular occupation. One important factor in the psychotherapeutic situation is the whole atmosphere of working and being occupied which permeates each ward. Regular, serious activity is an ingrained habit of the majority of people. It is something self-understood in normal life; it becomes an actual need. Dr. Simon observed that fitting in some sort of regular work, however limited it might be in the beginning, made it much easier for

newly admitted patients to adjust themselves to the new circumstances of living in the hospital. It often helped to overcome the sometimes painful realization of being confined in a psychiatric institution.

There are, according to Simon, no symptoms of mental disease, from stupor or catatonic negativism to impulsive excitement, that would absolutely preclude attempts at work therapy, although individual treatment of each patient and repeated efforts over weeks and months are frequently necessary for success. With some "old inmates" of institutions it has even taken years for habituation to regular useful work. The main point is to establish slowly sufficient rapport with the patient so that he will do something, however little, for somebody. To lead the patient to regular occupation is inseparable from educative influence; occupation is only a part of the work of reeducation of the patient. Methods of force or any procedures of punishment are never to be used. On the other hand, patients who work regularly earn thereby all sorts of advantages and compensations in the life of the institution. When the patient ceases to work, these advantages are discontinued. In this way, a degree of self-responsibility is created. Dr. Simon made the interesting observation that patients respond well if the hospital personnel that supervises them is at the same time doing similar or the same work as the patients. He found that this was practicable and did not interfere with the "watching" of the patients.

As far as possible, the work should serve some useful purpose. Simon is opposed to letting the patient be engaged over long periods in the creation of schizophrenic or paranoid "works of art." The tendency of his psychotherapy is to oppose consistently and with energy all pathologic manifestations and to direct any tendency to activity which the patient shows spontaneously or which can be aroused in him, in a normal, healthy and useful direction.

It usually requires a great deal of education to habituate the patients to work. The mental state of the individual patient has to be carefully considered both in the choice of work and in the process of training. Some young female manic patients, for example, were successfully set to work washing in the laundry. It is important that the work of the patient should be maintained as near the upper limit of his capacity as possible. The cautious individualization which has to guard against overexertion of the patient while keeping him near the upper limit of his capacity for performance is one of the most important and most difficult tasks of the psychiatrist. It requires that all sides of mental life be taken into account, from the condition of the sensorium to the affective attitude. Simon divided the work into stages of increasing difficulty, which he compared to the grades of a school. He enumerated the different kinds of work that he found useful in the five stages which he distinguished, from the simple to those involving more and more personal responsibil-

ity. The physician has to allot the different kinds of work and keep track each day of the occupation of each patient. The aim is to have all patients do regular work, with the exception of only those who are physically disqualified.

Dr. Simon made a distinction between work therapy and occupational therapy. He advocated work therapy, and by work he meant real work. "The work allotted to the patient should be real and serious." The necessary corollary of this arrangement is that the patient gets paid something for the work he does, however small the amount may have to be. Apart from work there are also occupational and recreational activities. Especially on Sundays and holidays is it necessary to create "activity." Everybody familiar with life in a psychiatric hospital will endorse this last desideratum.

Three important injurious influences threaten the patient in state institutions: inactivity, the "institutional environment" and lack of responsibility. All three belong inseparably together. Dr. Simon gave the following example of the importance of the inanimate environment for mental patients. He observed that if flowers and pretty curtains are put in the rooms of excited female patients, the patients may break chairs or objects of daily use, but they hardly ever touch the flowers or curtains. The social environment is still more important. Dr. Simon described with understanding the unsocial environment that disturbed patients create for each other. He has come to the conclusion that these "unsocial" outbursts of all sorts are hardly ever fundamental symptoms of disease. They are manifestations of the underlying personality, and the "unsocial" behavior may have its root in early childhood influences. There are many behavior disorders of psychotic patients, including those disorders which made confinement in the hospital necessary in the first place, which are open to the influence of systematic education. Formation of a suitable environment is therefore an important principle of the "active" therapy.

Successful work therapy for patients is possible only when through continued effort the whole environment of the patient is influenced to an optimum of helpful circumstances. "Every patient has the right to an orderly, quiet environment which does not excite him." Every effort is made to prevent emotional outbursts or disturbing behavior of any sort. This requires an understanding of the individual personalities of the disturbing patients. Whenever there is any more serious disturbance, it is the physician's task to determine the sources and to find out to what extent they lie in the personality of the patient, in variations of the course of the psychosis or in circumstances of the environment. It can frequently be found that the disturbance could have been prevented if certain factors in the environment had been different. Usually a

generally disturbed atmosphere in the ward is caused by only a few patients. Brief isolations—on an average for from eighteen to twenty, and not more than thirty minutes—have been found useful. But more important are continued educative efforts, not so unlike those applied to children. It is especially important to nip such disturbances in the bud. Often it is possible to prevent them by careful attention to early manifestations of changes in mood and attitude.

Simon emphasized that the most important part of his "active" therapy is the creation of a suitable environment (Milieugestaltung) for the patient, and not simply increased occupational therapy. An atmosphere has to be created in which the patient feels that he is to a large extent responsible for his behavior, especially as far as others are involved, and that he is not completely irresponsible because he is confined in an institution. "The mental patient, like all other living things, exacts from his environment just a little more than the environment will stand for; when he meets really serious and consistent resistance to unsocial behavior, then he can act and learn differently." Direct education of patients is not so expedient as indirect influence through changes in the environment. The introduction of an "impersonal" discipline, such as transfer to a different ward with fewer advantages for certain transgressions, has proved of great value.

To describe a method of psychotherapy and to convey what actually happens is, of course, exceedingly difficult. But Dr. Simon has succeeded well in explaining his point of view and its working out in action. He gives many details about the actual measures to be taken: the best planning of buildings, the types of occupation suitable for different patients, the training of the hospital personnel, the actual handling of difficult situations with disturbed and disturbing patients, etc. For example, he described a disturbed ward: patient A does this, patients C and D that; then patient B joins in, and so on. He then analyzed the different reasons why each patient acts the way he does and discussed the best ways to abolish such disturbed situations. The formation of a suitable environment for each patient is the keynote throughout.

The results which Dr. Simon obtained in his institution at Gütersloh were striking. There are practically no more really disturbed wards in the old sense. The atmosphere in the hospital is one of quietness and order. These results have been confirmed by numerous visitors to the institution (see, for example, Trapet [3] and Gründler [4]). The use of sedatives was diminished, and methods of restraint of any kind have been greatly restricted. (Dr. Simon considers so-called packs a worse kind of

3. Trapet: Die Simonsche Beschäftigungstherapie, Psychiat.-neurol. Wchnschr. **28**:94, 1928.

4. Gründler, W.: Bericht über die Dienstreise nach Gütersloh, Psychiat.-neurol. Wchnschr. **24**:347, 1927.

restraint than the old straight-jacket.) A noteworthy change occurred in the behavior manifestations of psychoses. As might be expected, schizophrenic psychoses were especially influenced. Every psychiatrist who has visited many different institutions knows that the incidence of catatonic phenomena on the whole varies in different places. In Dr. Simon's institution not only the negativistic phenomena decreased, but also many other behavior anomalies, such as impulsive restlessness, combativeness, yelling, untidiness, anomalies of movements and positions and paranoid outbursts. Even schizophrenic scattering and delusion formation and hallucinations were found amenable to consistent active therapeutic influences. There always are, of course, patients who remain refractory. They are regarded not as hopeless, but as "failures of psychotherapy," and renewed efforts are made with just these patients. It is theoretically interesting that in a relatively large number of cases of severe schizophrenic psychoses, active psychotherapy caused the more conspicuous symptoms to retreat further and further into the background, frequently leaving a picture of "simple schizophrenia" with lack of agility, energy and initiative, but with generally orderly behavior. The main point made is that if psychotic patients are consistently regarded as not irresponsible for their acts and behavior, as far as this is possible, they live up to the change in their environment to a remarkable degree.

Dr. Simon realizes that his method makes great demands on the physician and the hospital personnel. He takes for granted that each institution should be directed by a psychiatrically trained physician. He demands at least one physician for every hundred patients. The ideal organization in his opinion would be one in which each employee occupied in any work should have had training in psychiatric nursing.

CRITICISMS OF OTHER WORKERS

Already a large literature has sprung up about this "more active therapy." Dr. Simon had put his point of view into actual practice for almost twenty years before his first public utterance on the subject. At a conference of directors of state institutions he remarked about the harmfulness of "bed treatment" of psychotic patients and about the possibility of having almost all patients occupied in some useful work, in this way effecting a general improvement both in the hospital atmosphere and in the condition of the individual patients. His remarks were challenged, and he explained his methods. Soon after this, he reported on his work before a psychiatric society and aroused a great deal of criticism.

From the literature on the subject it is apparent that Dr. Simon's assertions have been amply substantiated. Critical observers who doubted

the claims made in his papers convinced themselves of their truth by personal investigation of Dr. Simon's institution. More important, the method has been tried out and found successful in other places. Most significant are reports by Thumm and van der Scheer. Thumm [5] introduced the more active therapy in a large institution at Konstanz. He reported that since the introduction of this therapy the changes described by Simon have been noticeable in the institution. The use of sedatives and of continuous tubs were greatly restricted. The number of patients who had to be cared for in disturbed wards was much reduced, and many more patients than before could be occupied with regular work. In the generally more orderly atmosphere it was even found possible to break the rule—usually strictly enforced—of keeping the two sexes always apart. Early discharge of psychotic patients was also made practicable on a larger scale. An especially striking demonstration of Dr. Simon's claims was given when on several occasions large numbers of disturbed patients were admitted from other institutions. They fitted quickly into the new environment and showed a much more pronounced and lasting change in behavior than is usually the case in transfers from one institution to another. Thumm pointed out that it is a question not only of more occupational or work therapy, but of the consistent change in the environment in which the patient finds himself. He described the chief dangers of institutional life: the progress and fixation of autistic tendencies, the emotional isolation, the dissolution of contact with reality, the infection by suggestion from excitement and disorderly behavior in the environment. He made the significant remark that—thanks to Dr. Simon—at last the psychiatrists in institutions are again that which their name implies, namely, psychotherapists.

Van der Scheer,[6] director of the institution at Santpoort in Holland, visited Dr. Simon's institution in a doubting and skeptical frame of mind. After close investigation he convinced himself of the value of the "more active therapy" and began it in Santpoort. He also reported

5. Thumm, M.: Ueber Erfahrungen mit „aktiver Therapie" bei Psychosen, Ztschr. f. d. ges. Neurol. u. Psychiat. **103**:225, 1926; Beschäftigungsgrad und aktive Therapie, Psychiat.-neurol. Wchnschr. **29**:242, 1927; Milieugestaltung im Rahmen der aktiveren Therapie und ihre Auswirkung auf freie Behandlung und offene Fürsorge, Allg. Ztschr. f. Psychiat. **88**:55, 1928; Literaturbericht zur aktiveren (Beschäftigungs-) Therapie nach Simon, ibid. **89**:1928, 1928. (Discussion of literature up to 1928.)

6. Van der Scheer, W. M.: Rückblick über zwanzig Jahre Irrenpflege und Betrachtungen über die aktivere Therapie in der Provinzial Heilanstalt bei Santpoort (früher Meerenberg, Holland), Zitschr. f. psych. Hyg. **1**:161, 1928; Ueber einige wichtige Behandlungsmethoden in unserer Anstalt, Psychiat.-neurol. Wchnschr. **28**:73, 1926; The Occupational Therapy Method of Dr. Simon of Gütersloh (Westphalia), J. Ment. Sc. **75**:203, 1929.

good results. Whereas before the introduction of the new methods 20 per cent of his patients were unoccupied for reasons of mental illness, this number dwindled to 1.75 per cent under the new regimen. Fewer patients than before had to be cared for in disturbed wards. Van der Scheer introduced payment of all working patients by a special hospital coinage. He described the general changes in the hospital in a historical setting, through the different phases of institutional policies and methods from 1849 up to the introduction of Dr. Simon's methods. He regarded Simon, like Pinel, Conolly and Neisser (systematic propagator of bed treatment) as one of the great stimulators of psychiatric progress.

Good results by the "more active therapy" were also reported under different circumstances by Ilberg,[7] Müller,[8] Goetze, Nobbe and Powels,[9] Wickel,[10] Gross [11] and various others.[12] Ilberg, who was at first skeptical, found in his hospital, Sonnenstein, that after introduction of more active therapy the percentage of patients regularly occupied increased from 40 or 50 to 83 in men and 87 in women. Hinrichs and Grabow [13] have reported good results with the active therapy combined with continuous sleep treatment. They modified Dr. Simon's technic by giving the patients much more individual freedom, such as free parole on the grounds of the institution or to the city. Meltzer [14] praised Simon for having systematically introduced into psychiatric hospital practice pedagogic methods which have long been known to educators and are in use in educational institutions.

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7. Ilberg, G.: Erfahrungen mit erweiterter Beschäftigungstherapie, Allg. Ztschr. f. Psychiat. **88**:108, 1928; Ueber den Umfang der Arbeitstherapie, Psychiat.-neurol. Wchnschr. **28**:433, 1926; Anregungen zur Verbesserung der Irrenpflege, Wien. med. Wchnschr. **77**:1232, 1927.

8. Müller, G.: Die Beschäftigungsbehandlung in der Lippeschen Heil- und Pflegeanstalt Lindenhaus bei Brake (Lippe), Psychiat.-neurol. Wchnschr. **27**: 491, 1925.

9. Goetze; Nobbe and Powels: Erfahrungen mit der Beschäftigungstherapie nach Simon in den ostpreussischen Provinzialanstalten, Allg. Ztschr. f. Psychiat. **86**:102, 1927.

10. Wickel: Fortschritte in der Erkenntnis und Behandlung der Geisteskranken, München. med. Wchnschr. **73**:1463, 1926.

11. Gross, A.: Der Umbau einer Zellenabteilung, Allg. Ztschr. f. Psychiat. **84**:183, 1926.

12. The method has also been introduced to South America. See Sicco, Antonio: Sobre laborterapia, el método del Dr. Hermann Simon, Talleres Gráficos de la Penitenciaria Nacional, Buenos Aires, 1928.

13. Hinrichs and Grabow: Erfolge der Dauerschlafbehandlung und Arbeitstherapie in der Landesheil- und Pflegeanstalt Neustadt in Holst, Psychiat.-neurol. Wchnschr. **29**:410, 1927.

14. Meltzer, E.: Muss der Anstaltsarzt wissenschaftlich arbeiten? Psychiat.-neurol. Wchnschr. **31**:405, 1929; Die neuen Ziele der Psychotherapie, Psychiat.-neurol. Wchnschr. **29**:108, 1927; **29**:117, 1927.

Löwenstein [15] attempted to use psychologic experiments as an aid to differentiation of individual patients for the different forms of active therapy. These experiments are directed toward the demonstration of a "reactive-labile constitution," that is to say, a tendency to psychogenic reactions. He found a tendency to psychogenic reactions in 44 per cent of epileptic patients, 54 per cent of healthy persons, 73 per cent of those with paranoid forms of schizophrenia, 90 per cent of catatonic patients and 96 per cent of hysterical patients. He emphasized especially the frequency of the "reactive-labile" constitution in catatonia.

The criticisms of Dr. Simon's claims deserve careful attention. It is noteworthy, however, that so far the principles of active therapy have nowhere been fully tried out without in the main the same success which its originator claimed. But Neisser [16] expressed the criticism that it has not been made sufficiently clear how it is actually brought about that so many disturbed patients are educated to a more social life in the hospital. He also objected to the fact that Dr. Simon left so much to the hospital personnel who have to attempt to stop the first incipient manifestations of unsocial outbursts and join generally in the education of patients. Neisser expressed the fear that often this may interfere with the freedom which should be left to the patient. Dr. Simon, on the other hand, particularly stated that too great freedom given to the patient to do what he pleases is an abuse which should be abolished. Neisser also opposed the policy of having every new patient, after admission and completed examinations, join at once in regular work activities. For the final evaluation of Dr. Simon's claims, he demanded a demonstration that more patients are discharged and that the duration of the average hospital stay is shorter. Reiss [17] defends Simon's method against this last criticism. He claims, although he does not give any statistics, that it ranks at present with the extramural service for discharged patients as the best means of shortening the hospital stay in cases of mental disorder. Kahlbaum [18] stated his opinion that patients who were combative and generally aggressive should not be treated with regular work therapy. He warned against an exaggeration of the value of work therapy.

15. Löwenstein, O.: Ueber einige experimentelle und klinische Grundlagen für die Anwendung der Psychotherapie bei Psychosen mit besonderem Hinblick auf die Arbeitstherapie, Ztschr. f. d. ges. Neurol. u. Psychiat. **110**:50, 1927.

16. Neisser, C.: Bettbehandlung, Arbeit, aktivere Therapie, Arch. f. Psychiat. **77**:663, 1926; Die Weiterentwicklung der praktischen Psychiatrie insbesondere der Anstaltstherapie im Sinne Griesinger's, Monatschr. f. Psychiat. u. Neurol. **63**:314, 1927.

17. Reiss: Die aktivere Beschäftigungsbehandlung der Heil- und Pflegeanstalten, Psychiat.-neurol. Wchnschr. **31**:105, 1929.

18. Kahlbaum: Aktivere Arbeitstherapie bei Psychisch-Kranken, Klin. Wchnschr. **6**:1294, 1927.

Criticism has also been expressed on details of actual hospital management. Courbon [19] and Halberstadt,[20] although they expressed the belief that the introduction of Simon's methods would be desirable in France, doubted whether this is practicable at present on account of the present policies with regard to psychiatric institutions. Gross [21] states as his opinion that Dr. Simon's therapy is only a one-sided reaction to the former therapeutic inactivity brought about by circumstances during and after the World War. Kraepelin ascribed Dr. Simon's successes to the small number of new admissions and to the preponderance of schizophrenic patients. He disapproved of work therapy for depressive patients, as they are painfully aware of their general inhibition. They should be treated in bed.

There is indeed danger that this "more active therapy" may be applied too schematically, according to routine, rather than in the setting of careful clinical examinations and observations. Vocke,[22] an experienced psychiatrist, expressed this well when he remarked that in state hospitals not only "more active therapy" but also "more active diagnosis" is necessary.

Surveying the various reports and opinions, one arrives at the conclusion that judgment as to the scope and extent of the possible results of this "more active therapy" is as yet premature. But the question as to whether Dr. Simon has actually contributed something new to psychiatry can be answered. Taken each for itself, none of his methods are new. He himself modestly stated that in the treatment which he advocates there is nothing fundamentally new. It is not a "system" of treatment; in fact, Simon warned against special systems of treatment in psychiatry. They tend to become rigid, and finally they hinder rather than further the individual treatment of patients. But the principle of a whole concerted attack, using all these methods of work therapy, education, discipline, formation of a suitable environment and active every-day psychotherapy, constitutes not only something new, but indeed a development important for progress in the care of patients with mental disease. The fact that environmental and educational factors can have such a far-reaching influence on the severest types of psychoses has not before been emphasized and demonstrated so clearly. The whole-hearted systematic use of a combination of methods of psychotherapy has shown,

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19. Courbon, P.: Un voyage d'études dans les asiles de Hollande, Ann. méd.-psychol. **86**:289, 1928.

20. Halberstadt, G.: A propos de "l'ergothérapie," Ann. méd.-psychol. **87**: 193, 1929.

21. Gross, A.: Kraepelin's Bedeutung für die Anstaltspsychiatrie, Arch. f. Psychiat. **87**:50, 1929.

22. Vocke, in discussion on: Nachprüfung der Diagnosen bei älteren Anstalts-Pfleglingen, Allg. Ztschr. f. Psychiat. **88**:426, 1928.

as Dr. Simon expressed it, that the picture one sees in the so-called chronic and hopeless institutional cases is only one-third due to disease, while two-thirds is a reaction to an unfavorable and unsuitable environment.

Credit should be given to Dr. Simon for having demanded and inaugurated the ideal of active psychotherapy for all rather than for specially selected, calm and tractable patients. His active work therapy helps greatly to prepare the patient, as Römer [23] points out, for his rehabilitation in social life. And in this connection, Steckel's proposal [24] of "sheltered work shops" outside of hospitals as an intermediate stage before final return to social life deserves careful attention.

But Dr. Simon's achievement lies not only in the practical sphere. Theoretical understanding of psychoses cannot but be greatly advanced by the results obtained by his methods in the treatment of patients with schizophrenia and other mental disorders. His is an important contribution to the great problem of modern psychopathology: the interrelation of personality and psychosis.

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23. Römer: Die Frühentblassung der Schizophrenen, Zentralbl. f. d. ges. Neurol. u. Psychiat. **53**:662, 1929.

24. Steckel, H. A.: The Need of Sheltered Work Shops in the Community Rehabilitation of Mental Patients, Psychiatric Quarterly **3**:404, 1929.

News and Comment

COMMONWEALTH FELLOWSHIPS IN PSYCHIATRY

The Commonwealth Fund of New York has, for the second time, made five fellowships available, in the Department of Psychiatry at the Harvard Medical School, for workers who have already shown evidence of good work in psychiatry, who wish to prepare themselves in a well rounded way for a career in psychiatry and who are willing to devote adequate time to this preparation. Each fellowship may be continued for three years, but continuation will depend on the excellence of the work done. Each Fellow would follow a program determined by his special needs and interests.

Letters of application, with details as to previous training and experience, may be sent to Dr. C. Macfie Campbell, Boston Psychopathic Hospital, 74 Fenwood Road, Boston.

Abstracts from Current Literature

RETROBULBAR OPTIC NEURITIS OF NASAL SINUS AND DENTAL ORIGIN: EVOLUTION AND PRESENT STATE OF THE QUESTION. E. REDSLOB, Rev. d'oto-neuro-opht. **7**:405 (June) 1929.

Retrobulbar optic neuritis is defined as an acute optic neuritis, usually unilateral, characterized by a more or less accentuated and sudden diminution of visual acuity. It is retrobulbar because most often with the ophthalmoscope one sees no change in the papilla, or at most, a slight softness. The portion of the nerve affected is that part extending from the chiasm to the point of penetration of the artery, which is 1 cm. behind the papilla. When the condition extends farther forward, changes in the papilla, often suggesting edema, are seen. Diminution of vision is caused by a central scotoma, due to injury of the central fibers of the nerve which supply the macula, these being the most vulnerable. In severe cases the whole trunk can become involved and then the central scotoma extends to the whole visual field and blindness is the result.

In the great majority of patients, cure occurs spontaneously without prejudice to vision. The benignity of the disease is proportionate to the rapidity of its development. In those cases with pain in the orbit or from pressure on the globe rapid and complete cure will result. However, one must remember that certain cases become chronic, and vision is compromised.

It is relatively frequent in young persons and may be caused either by a general disease or a nearby local lesion. Among the former are the infections such as grip, erysipelas, anginas, syphilis and disseminated sclerosis. It is a very important symptom of the last named disease and occurs early, sometimes even preceding its development.

Otitis, cerebrospinal meningitis and especially dental and rhinologic diseases are among the conditions of contiguous areas causing this malady. It has been known for a long time that suppuration of the posterior group of nasal sinuses can cause optic neuritis. The anatomic relations between these sinuses and the optic nerve facilitate propagation of infection from one to the other. At times dehiscences in the bony walls are found and the wall is always pierced by openings for the passage of lymph and blood vessels as well as vasomotor nerves.

After the publication of Wright and Sluder's observations on the rôle played by latent infections of the sinus in the cause of retrobulbar neuritis and other troubles, much interest in this subject was aroused among European ophthalmologists and rhinologists. To decide the question, it was assumed that, if the retrobulbar neuritis was due to a suppurative or latent posterior sinusitis even without clinical evidence of infection, opening these sinuses would give a palpable therapeutic result. This was done and there was often prompt amelioration of the condition of the eye. Certain operators went so far as to assert that retrobulbar neuritis is often the sole symptom of a sinusitis, in the absence of all nasal symptoms. The case was summed up by Velter and Liébault as follows: "It seems established that there exists latent posterior sinusitis, rarely visible or discoverable, which determines quite a gamut of orbital complications of which the gravest is retrobulbar neuritis."

These clinical results were surprising, and efforts were made to explain these bizarre phenomena, on anatomopathologic grounds, and many theories were proposed. Then a reaction set in, and certain ophthalmologists expressed the opinion that one was misled, not only as to the origin of the optic neuritis, but also as to the efficacy of the surgical intervention; that the natural evolution of the disease was toward recovery and that one was the victim of a therapeutic illusion. Furthermore, the sinus operations were not without unfortunate consequences. Others combatted the conception of a rhinologic origin and suggested the frequency

of multiple sclerosis as an etiologic factor, and still others affirmed that even in sclerosis the sinusitides were of importance by creating a place of least resistance in the optic nerve. One group of surgeons operated on the sinuses, even when they appeared clinically sound, and for the following reasons: (1) very often the disease of the sinus is discovered only after opening the cavity; (2) there can be a hyperplasia of the ethmoid causing a compression of the nerve; (3) whatever the cause of the retrobulbar neuritis, operation exercises a beneficent effect on it. Another group refused to operate except when the clinical examination revealed a frank suppurative sinusitis. A third group operated only under certain variable conditions. Some operated radically, others limited the intervention to resection of the middle turbinate or of the nasal septum. Others replaced operation by the production of ischemia of the nasal mucosa with tampons of epinephrine. While the fact is emphasized that persons with retrobulbar neuritis most often recover spontaneously, attention is called to the further fact that the coincidence of amelioration of vision with operative procedures has convinced many operators of the efficacy of the operations. The author believes that it is not just to deny this contention. But when the integrity of the sinuses is perfect and when other factors, such as disseminated sclerosis, syphilis or an infectious process, are present, excluding a sinus etiology, how can the beneficent action of an operation be explained? One theory is that the neuritis is not due to inflammation but to a lack of aeration of the sinuses. Another is that the amelioration is due to the blood letting, acting as a derivative. This has given rise to a new conception of the nature of retrobulbar neuritis. The sudden development, the almost certain recovery, the beneficial effect of a modification of vascular tone by the bleeding, suggest the idea that troubles of circulation, especially spasm, are at the base of the characteristic symptoms of retrobulbar neuritis. It is known that the central artery of the retina can be affected with spasmodic contractions and one can conclude that the arteries of the trunk of the nerve may likewise be so affected and if the spasm is not complete, it will be the fragile central fibers that will be affected, causing a central scotoma. If the spasmodic obstruction is complete, all the fibers of the nerve will feel the effects of the lack of nutrition, and total blindness will ensue. The papillary vessels will not be influenced because they are a part of a different vascular territory. It is understood that this theory applies to only a certain category of cases of retrobulbar neuritis; in others, the inflammatory theory is still valid.

Other rhinogenic cases of retrobulbar neuritis have been reported: ozena, nasal polyps, maxillary and frontal sinusitis. In Walter Deichler's case, associated with frontal sinusitis, the middle turbinate was removed and the sphenoidal sinus opened without relief; only after an operation on the frontal sinus was there improvement. The explanation is that the neuritis was due not to inflammation but to arterial spasm, the frontal sinusitis representing the irritative point, and this being removed by operation, the spasm vanished.

As to the rôle of dental infection in the etiology of retrobulbar optic neuritis, here again opinion has been divided, one group claiming that the malady is due to the tooth and another believing that the recovery following extraction was coincidental and due to the tendency of the neuritis to get well spontaneously. The reports of amelioration of vision following extraction have been so numerous that it is difficult to deny the relation of cause and effect. The author admits his own skepticism until an illuminating case occurred in his own practice. It concerned a woman, aged 44, who had a central scotoma in the right eye with vision reduced to 1/10. The following day the vision was practically gone. Two diseased teeth in the right upper jaw were removed. Improvement in vision began at once, and within two weeks it was normal. Later on, a general examination revealed that the abdominal reflexes were absent and the patellar reflexes were exaggerated, and that the patient had had for some time a paralysis of the right hand, suggesting a disseminated sclerosis as a factor in the production of the optic neuritis. It is to be borne in mind that both conditions might have been responsible. Admitting the possibility of an infection of the optic nerve by the teeth, it has been suggested

by Fromaget that there is a metastatic infection, the microbes being arrested in the nerve through specific affinity or by finding a favorable environment. The latter may be due to reflex vasomotor troubles from infection or from irritation of the trigeminus. This conception includes the rôle of infection and a reaction of the sympathetic. But is one certain that in what is called retrobulbar neuritis, an inflammatory process always comes into play? In a case with a diseased tooth, the latter could be the irritative point which causes arterial spasm; removal of the tooth relieves the spasm. Thus, it may be neither inflammation nor vascular congestion of the nerve which is responsible. Cases of persistent spasm of the central artery of the retina have been observed which were relieved by extraction of diseased teeth. This could apply equally to spasm of arteries of the nerve trunk. All these questions are far from being solved. An extensive bibliography accompanies the article.

DENNIS, Colorado Springs.

NERVOUS MANIFESTATIONS IN OSTEOMALACIA DUE TO STARVATION (SPINAL CORD CHANGES). TOSIHIO KUROSAWA, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **31**:173, 1929.

CASE 1.—A widow, aged 67, developed at the age of 51, immediately before the menopause, weakness of the right leg which progressed and soon involved the left leg. The weakness was associated with diminished sensibility in the limbs, but it was painless. There were no evidences of syphilis. In 1922, fifteen years after the onset of the illness, there was noted a curvature of the spine, thorax and pelvis, and the patient was unable to get about. The ribs were tender on pressure and the symphysis pubis was unusually prominent. Both lower extremities were paretic and spastic, with increased deep reflexes, ankle clonus and bilateral Babinski sign. At this time she had pains and paresthesias in the knees, but no objective sensory disturbances. As time went on she developed sphincteric disturbances (retention). The author believes that there was no doubt that the diagnosis was osteomalacia because she improved after the administration of cod liver oil and phosphorus, although the paralysis of the lower extremities and the symptoms of the bladder were unaffected. She died of pneumonia in 1924.

Necropsy revealed chronic purulent cystitis, pyelonephrosis, calculosis, chronic tuberculosis of the right upper lobe of the lung, an old pleurisy, fatty degeneration of the parenchymatous organs, cerebral edema and healed osteomalacia with deformity of the pelvis and thorax.

Examination of the spinal cord revealed: With the Weigert stain the white substance of the cervical enlargement showed several areas of sclerosis with edematous margins. The sclerosis was more diffuse in the anterior columns, and although both sides were involved symmetrically, one side appeared denser than the other; it surrounded the gray matter uniformly and was less distinct toward the periphery of the cord. The sclerosis was still more marked in the lateral column of one side; here it surrounded, in bandlike fashion, the anterior horn and reached its maximum density between the spinocerebellar and pyramidal tracts. Lissauer's marginal zone and a portion of the posterior horn were similarly though less severely affected. It was also noteworthy that in cross-section this portion of the cord was somewhat deformed.

In van Gieson preparations there was a moderately chronic leptomeningitis, without infiltration but with definite invasion of glia into the pia. The "Lückenfelder" in the sclerotic areas were also unusually marked in sections stained by the van Gieson method. There were no reactive changes of any kind. The walls of the vessels were thickened and the tissues were filled with corpora amylacea. Except for a moderate lipoidosis, the ganglion cells were relatively well preserved, especially those in the anterior horns.

With the Bielschowsky stain, axons and myelin sheaths could be observed only in the periphery of the sclerotic zones, and even here they appeared swollen and thickened.

In the dorsal and lumbar portions of the cord the sclerotic areas and "Lückenfelder," though present, were not nearly as marked as in the cervical portion of the cord.

Relatively speaking, the secondary degeneration was not very intense; it was most marked in the cervical, less in the lumbar and least in the thoracic portion of the cord.

CASE 2.—A woman, aged 64, developed a severe uterine hemorrhage at the age of 22, which necessitated extirpation of the uterus. Except for an attack of dysentery three years later, there was nothing noteworthy in the history till the World War, when she fractured first the left, then the right, and then again the left thigh. The Wassermann reaction of the blood was positive. Careful questioning revealed that at this time the patient suffered a great deal from hunger, and the osteomalacic process was regarded as a starvation osteomalacia. It was also elicited that prior to the fractures the patient had already been suffering from paresthesias and pains in the lower limbs.

Examination in 1923 revealed: normally reacting pupils, absent patellar and achilles reflexes, positive Babinski sign and objective sensory disturbances, more marked on the right than on the left (general hypesthesia beginning at L2 on the right, and at D12 on the left, merging into anesthesia in the legs, and a zone of hyperalgesia between D10-D12 on the right, and D11-D12 on the left). The muscles were not tender, but owing to the healed fractures motor power of the lower limbs could not be tested. The patient was unable to walk. No definite information could be obtained about sphincteric control. As the disease advanced, the pupils did not seem to react as well as on admission. The clinical diagnosis was tabetiform degeneration of the posterior columns in a patient who was suffering from osteomalacia. Death was due to a pulmonary embolus following thrombosis of the femoral vein.

Necropsy confirmed the diagnosis of osteomalacia. The leptomeninges of the brain and cord, as well as the vessels at the base of the brain, were free from involvement. Only in the thoracic cord did the posterior columns show evidences of gray degeneration. In addition to this, there was also found a slight syphilitic mesaortitis.

Microscopic examination of a cross-section of the lumbar portion of the cord showed a peculiar degeneration, which was by no means radicular in type but seemed to conform to the type described by Bechterew-Trepinski. The ventral portion, the ventral field of the posterior column, the dorsolateral zone and the dorsal zone of the septum were free from changes, but the areas lying between these zones (the mesial root area) showed a moderate sclerosis, whereas the points of entrance of the roots into the cord appeared normal. In the thoracic portion of the cord the degeneration could not be made out so clearly. Here it was found in Goll's column, especially in its paramedian portion, without invading the periphery dorsally or ventrally. The margins of the sections in this portion of the cord were edematous. In the cervical portion of the cord the entire column of Goll showed typical secondary degeneration. In the lateral column there was found on one side, external to the pyramidal area, a narrow strip of light sclerosis, more marked dorsally than ventrally. The meninges showed a chronic inflammatory process with lymphoid elements in the vessels and also slight proliferation of the adventitial cells. These changes were most marked in the thoracic portion of the cord which appeared definitely edematous. The sclerosis was definite and there was no question that the infiltrate was syphilitic in nature. The changes in the cervical cord did not differ much from those in other portions of the cord—always a slight meningitis and a severe involvement of the blood vessels.

In preparations stained by the Nissl method the ganglion cells in the anterior horns appeared unusually well preserved, with no evidences of lipoidosis. In marked contrast to these, some of the cells in the lateral columns were severely degenerated. The anterior horn cells in the cervical portion of the cord were intact, as were the cells in the spinal ganglia.

Both cases then showed evidences of severe changes in the spinal cord in persons affected with osteomalacia due to starvation. In the first case the lesion of the cord was one of polysclerosis and in the second it was tabetiform and probably due to syphilis. In the latter, however, the pathologic picture was not characteristic of classic tabes owing to the associated involvement of the lateral columns. In both cases the cells in the lateral horns were also severely degenerated.

KESCHNER, New York.

THE ASSOCIATION OF OCULOGYRAL SPASM AND THE VESTIBULAR SYNDROME IN THE COURSE OF EPIDEMIC ENCEPHALITIS. PICOT, Rev. d'oto-neuro-opht. **7**:85 (Feb.) 1929.

After citing the articles of Sainton, Euzière, Page and Jean Blum, Picot reports a case. Two and one-half years before, a carver, aged 36, after prolonged close work, was suddenly stricken with a spasm of the eyes. The globes were turned upward until the corneas were partly masked by the upturned lids. The lids could be closed by the patient, but it was impossible during the spasm to change the direction of the gaze. The head was in hyperextension accompanied by trembling of the lids, chin and lower lip. The duration of the spasm was ordinarily from fifteen to thirty minutes but at times it would last from two to six hours. The attack was not painful but the ocular fatigue and the attacks themselves, which were repeated several times daily, made work difficult. Visual repose and sleep caused a disappearance of the symptoms. The condition is now much improved, but a close and prolonged fixation on any object is sufficient to provoke ocular spasm. The hereditary and past history were unimportant.

On examination, the eye movements and visual fields were normal. There was no apparent strabismus but there was a crossed diplopia, discovered only in the dark room and by the use of a small light. The diplopia was revealed between the near point and 80 cm. Beyond this point it disappeared. It also disappeared on looking downward. Accommodation was intact; the near point was at 10 cm. The anterior chambers and cornea were normal. The pupils were dilated, equal, a little irregular and oval, the pupils of the two eyes together having the form of a circumflex. The light reflex was very feeble as was the reflex of accommodation; there were no synechiae. There was an astigmatism of 2 diopters in the right eye and of 1 diopter in the left eye which was corrected by cylinders at 0 degree of 1.5 diopter in the right and of 0.5 diopter in the left eye. There was no abnormality of the lens, vitreous, eyegrounds or ocular tension.

In spite of the lack of any history of acute illness, this ocular syndrome is attributed to a spent epidemic encephalitis as is the slight parkinsonism.

Numerous medicaments have been used with more or less success, but even without treatment the attacks diminish in frequency and duration. The most useful drug in this case was sodium salicylate.

The pupillary disturbances suggest syphilis but a negative Wassermann reaction and the failure of specific treatment ruled it out. Disseminated sclerosis, meningeal tuberculosis and cerebral softening could likewise be ruled out.

The cochlear function was intact and there was no tinnitus, vertigo or spontaneous nystagmus. Quix's tests were normal. The Romberg test gave negative results. Von Stein and Babinski-Weil tests were negative. Galvanic, turning and caloric tests showed hypo-excitability of the horizontal canals with slight and transitory vertigo. There was no reaction from the vertical canals. The fistula test was negative.

The majority of writers on this subject have found hypo-excitability of the vestibular apparatus, although sometimes normal excitability or even a hyper-excitability may be met with.

An attempt is made to explain the ocular and the vestibular symptomatology and their relations to each other. It is a difficult task because most researches on the pathologic physiology of epidemic encephalitis have ended only in hypotheses. 1. The cause of the oculovestibular syndrome is extralabyrinthine.

2. The cause of this syndrome does not appear to reside in the oculomotor nuclei; in fact, in the course, or in the sequelae, of epidemic encephalitis, the ocular observations confirm the integrity of the oculomotor nuclei. 3. It is then necessary to go back to the regulating centers of the oculomotor functions; on the one hand to the corpus striatum and on the other to the vestibular nuclei. 4. Now, pathologists have found in epidemic encephalitis lesions of the subependymal gray substance and remarkably constantly in the vestibular nuclei and the striate body or striopallidal system.

The corpus striatum, made up of the caudate and lenticular nuclei, is a true center, containing Golgi type II motor cells, afferent fibers from the cortex through the thalamus and efferent fibers to the red nucleus (Luy's body), locus niger and Darkschewitch's nucleus. The impulses received, via the thalamus, from the cerebellum (sensory tract) and the cerebral cortex (corticothalamic tract) are transmitted by the motor cells of the pallidum to the extrapyramidal motor tract of the bulb and spinal cord, which receives the rubrospinal and vestibulospinal fasciculi. Thus, the striate body dominates reflex action and tonus. The phenomena of subcortical automatism and the parkinsonian syndrome are explained by an interruption of the cortico-thalamostriatal tract.

5. A second center for regulating the oculomotor functions is the system of four pontile vestibular nuclei. This center has extensive connections. Through the column of Clark and the cerebellum it receives cenesthetic fibers from the whole body and can thus transmit to the superior centers images of somatic identity and of segmental attitude; they have numerous subcortical relations: a descending fasciculus (vestibulospinal), homolateral, from Deiter's nucleus to the motor pyramidal tract; an ascending fasciculus (vestibulomesencephalic), homolateral, from Bechterew's nucleus to the nucleus of the third nerve, and a heterolateral fasciculus which extends from the three principal vestibular nuclei to the opposite posterior longitudinal bundle, furnishing vestibular influence to all the motor nuclei of the bulb and of the pons up to the optic layer.

If the lesion is transitory, this will explain the transitory tonic ocular spasms and also variations of labyrinthine excitability noted by various authors.

Vestibulostriatal and strio-oculomotor connections render possible the association and the parallel variations of the vestibular and oculomotor syndromes.

DENNIS, Colorado Springs, Colo.

CANCER PROBLEM IN LIGHT OF MODERN VIEWS. GUSTAV KOLISCHER, J. A. M. A. **94**:625 (March 1) 1930.

Gustav Kolischer asserts that progress in cancer therapy has undoubtedly been impeded by the tenacious clinging of the profession to traditional concepts. A tendency to search for a single cause of cancer has prevailed, without discrimination between essential and contributory factors. Two conceptions have been considered axiomatic, and all conclusions have been based on them. It has been accepted that cancer in its initial stage is a strictly local disease and that therapeutic efforts may be successful only by virtue of complete regional eradication of the tumor. Clinical observation and consideration of the physiologic facts make it probable that cancer is produced by the collaboration of several factors. It is easily understandable that persistent chemical or mechanical irritation may furnish a contributory factor in producing cancer, especially between structures within which the normal equilibrium between stimulating and regulating forces is rather unstable. This consideration leads to establishing the concept of predisposition. A distinction must, however, be made between general and localized predisposition. Food for thought is furnished by a contention of Boerstrom, who spent many years in investigating cancer. He asserts, and his reasoning is rather convincing, that the traditional belief in the production of cancer by the transformation of epithelial cells is no longer tenable in the light of his investigations. He is ready to prove that a once highly differentiated cell never will change its established characteristics. He states that cancer is produced by the transformation of the

not specially differentiated cells of the reticulum around the capillaries. This would help to explain the production of cancer mainly by constitutional influences in areas that are not exposed to any mechanical irritation, such as cancers of the pelvic fibrous tissue. One may be entitled to the statement that, in view of the enumerated instances, cancer even in its initial stages is never a genuine local disease but only the local manifestation of a general derangement. Definite therapeutic results obtained by partial irradiation or incomplete mechanical destruction of malignant tumors help to support belief in the importance of the constitutional factor. It also has been found that the functional capacity of the defensive cells of the reticulo-endothelial system is raised by their receiving certain proteins or metallic compounds. This fact may explain how the administration of proteins, such as the metabolic products of bacteria, or the injection of some metals, such as gold or lead, in rare instances have been instrumental in producing favorable results. It also must be considered that any localized increase in metabolism attracts these cells to the area of stimulation. The rather favorable results of electrocoagulation in cancers are in harmony with these concepts. It has been found that electrocoagulation in short order is followed by a striking abundance of macrophages in the area surrounding the coagulated structures, this area being called the perithermic zone. There is another item not yet absolutely convincing because the number of cases is too small to permit of definite conclusions. In several patients with cancer in whom a stain test has proved a deficiency of the reticulo-endothelial system, all therapeutic efforts have proved to be futile. The therapeutic intervention will have to be divided into two distinct items: (1) the mechanical removal of the accessible growth, and (2) the biochemical attack on the causative factors. Surgery cannot ever be entirely dispensed with because, even if chemotherapy should succeed in producing absorption of the intact bulk of the tumor, the already degenerated part of the growth will always have to be removed by mechanical means. The other aspects of the therapeutic problem will be the changing of the relation between stimulating and regulating forces. The essential feature of such a therapeutic effort is a biochemical one. By electrocoagulation and radiotherapy in some instances it is possible to produce a beneficial biochemical system reaction, but this is accomplished in an empiric way. It will be the task of the biochemists to find an exact way of bringing about the necessary reaction. They will have to determine and isolate the compounds carrying the stimulating and regulating impulses. The next step must be to develop the proper reagents necessary to guarantee a desirable overproduction and control of the regulating and stimulating ferments. While it is known in a general way that the intake of some proteins or metallic compounds will stimulate the functional capacity of the reticulo-endothelial cells, it will be necessary to determine exactly which particular substance is most apt to increase the carcinophagic activity of the macrophages, which materials will stimulate the production of defensive regulating ferments, and which compounds will temporarily neutralize the stimulating ones. Along these lines it may be possible to develop a rational chemotherapy of cancer, cure without extensive mutilation comprising a guaranty against recurrence.

[EDITOR'S ABSTRACT].

EARLY AND DIFFERENTIAL DIAGNOSIS OF BRAIN TUMORS. RUDOLPH SCHMIDT, Med. Klin. **26**:6 (Jan. 3); 45 (Jan. 10) 1930.

This article, while rather elementary, presents some interesting reminders in the differential diagnosis of tumors of the brain. Headache, especially if no previous headaches have existed, must always be interpreted as a possible symptom of tumor of the brain. At onset, headaches associated with tumor of the brain are usually insidious; they gradually tend to become closer and more severe. Nocturnal headaches and early morning headaches are common in tumors of the brain and are not to be considered as a sign of syphilis, as is so frequently suggested in textbooks. Localized headaches should always be looked on as of localizing value. Percussion tenderness may also have a localizing value, especially

in indicating the side of lesion rather than the exact site. In cerebellar tumors it is frequently observed that the patient will lie on the side of the tumor. Lying on the well side will produce pain, vomiting and vertigo. Mechanical influences, as bending, exerting pressure, or suddenly changing posture, will increase or even precipitate a headache. Severe headaches may be associated with a slowing of the pulse. Absence of headache does not rule out the possibility of tumor.

Choked disk, while very frequent, may be a late sign, and its absence therefore must not be taken as a definite contraindication to the diagnosis of tumor.

The author stated that emotional disturbances are especially common in children. Memory disturbances, especially for recent events, are also common. "Witzelsucht," while considered a frontal lobe manifestation, may be simply an expression of a previous neuropathic make-up. The writer expressed the belief that if mental changes are early and persistent, a frontal lobe significance may be interpreted. If they occur late, it is probably only a manifestation of severe general pressure. Lethargy may be a focal symptom and indicate a lesion of the third ventricle.

Jacksonian attacks are naturally of great importance. Trembling attacks have been noted in tumors of the cerebellum and the thalamus. Rhythmic twitching has been observed, both in cerebral and cerebellar lesions. The author does not look on hemiplegic attacks as being of localizing value.

In cerebellar lesions there is a distinct tendency to a fixed position. Cerebellar ataxia is usually an early symptom, while frontal ataxia is usually a late symptom.

Corneal anesthesia should always be searched for. It may be of special importance in the early diagnosis of angle lesions. Slowing of the blinking of the eyes, with diminished corneal reflex, appears to be not infrequent in tumors of the cerebellum.

Eosinophilia should suggest the possibility of a cysticercus. The author reported two cases of polycythemia with tumor of the brain. Leukocytosis is not rare and must not be accepted as indicating an inflammatory lesion. Tuberculosis with secondary tubercles is always a pitfall. More often one finds tubercles in a torpid tuberculosis and the fact that a patient has no fever must not mislead one. Foci of infection, as indicating the possibility of an abscess of the brain must also be kept in mind. One must consider especially otitis media, bronchiectasis and subphrenic abscess.

Under the heading of differential diagnosis one should consider cholesteatoma, which may have inflammatory changes and cause fever. Pleocytosis in the spinal fluid does not rule out a tumor of the brain and need not indicate either an abscess or syphilis. An apoplectic onset is not uncommon in tumors of the brain and may even be mistaken for hemorrhage into the capsule. It must be recalled that tumors of the brain may have remissions and, in cases in which tumor is suspected, the author warns against assuming that the complaints are hysterical. Encephalitis should always be a consideration, especially if the patient shows any marked lethargy. Roentgen examination should never be neglected, as frequently calcification may be observed. Calcification of the falx or of the choroid plexus must not be interpreted as tumor. Changes of the sella are not always related to pituitary tumors but may indicate a generalized pressure. Finally, localizing help may be obtained by the aid of ventriculograms.

MOERSCH, Rochester, Minn.

THE PSYCHIATRIC USE OF THE METHODS AND RESULTS OF EXPERIMENTAL PSYCHOLOGY. CHARLES E. SPEARMAN, J. Ment. Sc. **75**:357 (July) 1929.

This article is the tenth Maudsley Lecture of the Royal Medico-Psychological Association. While conceding freely that they are merely different abstract aspects of one and the same real event—mental experience—mental processes may still with advantage be divided into cognition, affection and conation. It is especially important to separate cognition from the other two. "Failure to observe this line is responsible for many aberrations, I believe, both in psychology and psychiatry." If one accepts the view that all knowing must be given in sensations and their

complex combinations, all disorders of cognition are reducible to disturbances of sensory characters and properties. "To study all these systematically would be laborious and of doubtful value."

Though it is true that "faculties" are taboo, the conception is still retained under various forms as powers, capacities, etc.—the most important being apperception, attention, memory, imagination, intellect or intelligence and reason. In the use of these terms there is wide variation of meaning by different authors. " . . . the immense majority of the supposed faculties have no real foundation; that is to say, when an individual shows a high degree of any mental power, this gives little or no information about his power for operations of the same form but different material." There are, however, "a few precious exceptions," which are largely the outcome of the application of mathematical methods to the study of results of experimental tests.

Three especially important results follow from these studies of cognition: (1) The measurement of general ability, *g*, by a statistical study of performance in tests of numerous mental abilities, so numerous that any special abilities, *s*, cancel one another and leave a final average *g*. This factor is regarded by the author as in reality an expression of cortical energy, even though one does not know at all what is being measured. This has as much practical value as measurements of electricity when nothing was known of its actual nature. (2) The measurement of perseveration, *p*, the general tendency of a state of action to persist for a longer or shorter time as a residual excitement. This has been shown by mathematical study to have a certain general value in addition to special variations in particular fields. If *g* measures the quantity of psychologic energy "then perhaps *p* may measure its mobility." (3) The measurement of oscillation (Flugel), *o*. This as yet has been little studied clinically; it is regarded by Spearman as the steadiness with which the *g* is applied.

As yet psychologists have done little with the affective sphere. Conation is being measured, by methods as yet primitive, in animals; for example, measurements are being made of such features as the amount of electrically produced pain that is needed to prevent a rat from forcing its way through a barrier to reach a mate. Little has yet been done in this field with human material; some measurements have been made of such factors as trustworthiness and truthfulness, and experiments of this type suggest that there is a unitary factor, comparable to *g* and deducible by similar mathematical studies, to which already the designative letter *w* (derived from the extinct notion of "will") has been allotted.

The principal purpose of the lecture, however, was to suggest that there is need for close cooperation between the clinical psychiatrist and the experimental psychologist; each has something to contribute to the work of the other. The article concludes with a proposal that the Royal Medico-Psychological Association should establish the means for a definite liaison of this kind.

SINGER, Chicago.

THE HYPOPHYSIS IN OLD AGE. M. LUCIEN, Rev. franç. d'endocrinol. **6**:441 (Dec.) 1929.

After making a series of studies of the hypophysis in the aged Lucien draws certain conclusions. He does not believe that one should speak of senile atrophy of the hypophysis, since in the anatomic study of the gland there is no evidence that there is reduction in its size or weight during the process of ageing. On the other hand, it is of note that the largest and heaviest pituitary glands have been found in old people. Microscopic study reveals areas which might be described as sclerosis, areas which are localized especially in certain parts of the gland, for example, at the hilus and near vascular pedicles, do not take on a different staining reaction and therefore should not be the cause of atrophy of glandular elements. He considers that the glandular parenchyma does not seem definitely reduced.

The author expresses the opinion that histologic changes observed are evidence of a special functional state, which is peculiar to the hypophysis of old age. He calls attention to the fact that changes in the gland noted in old age have certain characteristics in common with changes occurring in some chronic nervous disturbances, such as chronic encephalitis and dementia paralytica. In the latter, there is evidence, besides a sclerosis, of a tendency toward enlargement of the acidophil cells. He considers, therefore, that the changes seen in the hypophysis of old age seem to be partly the result of an inflammatory process. This theory presupposes a new functional state of the hypophysis in the decline of life which is characterized by the accumulation of colloid substance in the lobes of the gland where pseudo-acinous forms are numerous; by the development of colloid vesicles around the hilus, and by the presence of a considerable amount of colloid in the pars nervosa. Thus, it would seem that there is in the hypophysis of the aged a true retention of colloid substance in the different parts of the gland. Secretory phenomena are in general slowed up, the acidophil elements being the only parts of the gland which preserve evidence of true glandular activity.

The invasion of the posterior lobe by the basophilic cells of the pars intermedia is a contradiction of the hypothesis of slowing up of secretory phenomena. The migration of the acidophils into the pars nervosa would seem to indicate an active multiplication of these elements. The author interprets it in a different fashion. In normal function of the hypophysis, cells of the pars intermedia emigrate to the pars nervosa where they slowly disappear by granular necrobiosis. The enigmatic bodies of Soyer represent these elements in the process of disintegration, and a collection of colloid in the neuro-hypophysis should be considered the result of this cellular piling up. This theory has been verified by Collins from experimental observations on the hypophysis of the cat. This author has seen islands of cells from the pars intermedia which have emigrated almost to the level of the infundibular recess.

In the aged, emigrating cells, of the type found in the pars intermedia, progress little by little to the pars nervosa where their degeneration later shows itself. There is a slowing up of the cellulolytic processes and an accumulation of basophilic cells in the neuro-hypophysis. Changes shown in the hypophysis of the aged may be an expression of a general slowing up of secretory function and a marked reduction of excretory function, of which the collected colloid is the most characteristic manifestation.

WAGGONER, Ann Arbor.

ANATOMICOCLINICAL CONTRIBUTION TO THE STUDY OF THE SYNDROME OF FOERSTER. G. MARINESCO and S. DRAGANESCO, Encéphale **24**:685 (Sept.-Oct.) 1929.

After reviewing the original case of Foerster in 1909 and several other similar reports, the authors present the clinical observations on a male child aged 6. With normal development up to 4 years, this child suffered an attack of malaria which was inadequately treated. There followed convulsions, generalized rigidity and trismus. This lasted for ten days interrupted by periods of incomplete relaxation. Then gradually the contracture receded, leaving the child incapable of movement or of articulation.

At the time of examination, the conditions observed were substantially as follows: physically, the child was well developed, with some diminution of muscular volume; he had no voice, but responded with a crying monotone when pinched; there was an expressionless face; hearing was conserved; there was some strabismus; he was incontinent; he was completely confined to bed; irregular but almost constant oscillations of the head were present. In decubitus the position was one of slight flexion of the knees, thighs and toes; the abdomen was supple; the vertebral column conformed to the plane of the bed. On raising to the vertical posture, there ensued immediately a remarkable degree of hypertonia — rigidity, increased reflexes, positive Babinski sign, etc. The analogy to the "Liftbewegungen"

of Magnus and de Kleijn is discussed. After six months' observation, the child died of septicemia, during which the hypertonia and athetotic movements disappeared.

At autopsy, there was found a generalized fatty degeneration which was especially marked in the liver and myocardium. In the former it approached that occurring in acute phosphorus or chloroform poisoning. Macroscopic examination of the cerebrum gave generally negative results, but a cross section showed brownish discoloration and reduction in volume of the putamen and caudate nuclei bilaterally. Microscopically, there was bilateral and symmetrical degeneration of the same nuclei, together with degeneration of the cells and fibers of the pyramidal tracts. A very light demyelinization process existed about the globus pallidus and also in the capsulae externa and extrema. With Nissl stains there was disappearance of almost all the nerve cells of the putamen and caudate, while those of the pallidus were well conserved. An abundance of microglia and astrocytes replaced the lost nerve cells. As a consequence of the lenticular lesion there was an almost total disappearance of the striopallidal fibers. In the cortex there was a chronic leptomeningitis, with hyperplasia, infiltration and weakening of the vessel walls. In the motor zone was rarefaction of the Betz cells and also of certain tangential fibers. The same was true of the Purkinje cells of the cerebellum.

In considering the case, the authors found it difficult to view the hepatic degeneration as simply a terminal state. It might be a case of Wilson's hepatolenticular degeneration, but does not show the cirrhotic changes of that condition. The marked changes in body attitude on change of plane likewise vary from the reports of Wilson's disease. They believe that the lesions of the striatum constitute the anatomic base for the dystonia described. Bilateral symmetrical lesions of the caudate and putamen liberate the automatic mechanisms of subjacent zones and permit the rigidity. This is not viewed as a decerebrate rigidity, except in that probably justifiable sense of looking on hemiplegia as a partial state of that condition. It is probably an expression of a so-called "standing reflex." The pathologic changes can be best explained in part on the basis of vascularization and in part on a concept of "laminary Pathoklise."

ANDERSON, Los Angeles.

PATHOPHYSIOLOGY OF THE OPTIC TRACT AND VISUAL SPHERE. O. FOERSTER, J. f. Psychol. u. Neurol. **39**:463, 1929.

It is generally accepted that the area striata (area 17) represents the primary end station of the optic radiation. It has not, however, as yet been demonstrated that tracts of the afferent optic radiation also find their terminal stations in other regions of the cortex. Destruction of the entire area striata is followed by blindness, and unilateral destruction of this area by a hemiopic visual field defect with sparing of the macula. In lesions of the upper calcarine lip, only the lower quadrants of the visual field are affected, whereas in lesions of the lower calcarine lip only the upper quadrants are affected. Lesions of the posterior portion of both areae striatae affect the macula, and lesions of the anterior portion produce only eccentric defects. Irritative pathologic processes involving the area striata give rise to photomes similar to those produced by faradic excitation. Foerster was able to produce similar photomes by mechanical irritation, such as compression of the occipital lobe, but he never observed ocular movements following faradic irritation of the area striata. This is in marked contrast with the results obtained by Sherrington, who in the course of his experiments with animals found, after electrical stimulation of the area striata, ocular movements toward the opposite side. It is noteworthy that hemiopic photomes have also been observed in association with pathologic processes of the convexity of the occipital lobe, although it is impossible to determine whether these are due to the direct irritation of the area striata or to the optic radiation. It became necessary, therefore, to determine whether optic photomes could be produced by direct faradic irritation of the cortex of the convexity of the occipital lobe. Foerster was able to make such determinations, and he found this to be actually the case in many instances.

As a result of his experiments with stimulation, the author is inclined to believe that faradic stimulation of area 19 (Vogt) gives rise to an irritative process of the entire cortex which produces much more vivid and more complex visual experiences than stimulation of area 17 (area striata). This, however, must still be taken with a great deal of reservation, at least until further proof with more material can be had. In this connection, it must also be recalled that photomes and even hallucinations may be produced by irritative pathologic processes and by the electric currents applied to the peripheral portion of the optic apparatus (optic nerve, optic tract).

In conclusion, Foerster points out that although faradic stimulation of area 17 in man never gave rise to ocular movements, stimulation of area 19 invariably produced ocular movements. It would therefore seem that area 19 represents the occipital center for ocular movements. These differences on stimulating areas 17 and 19, respectively, would also seem to confirm the hypothesis of O. and C. Vogt that the morphologic (architectonic) structural differences of the various cortical areas also correspond to the various physiologic differences of these areas.

KESCHNER, New York.

PARADOXICAL HYPERMOTILITY IN PARKINSONISM. I. I. RUSETZKY, Rev. Psychiat., Neurol. & Reflexol. (Leningrad) **4**:14, 1929.

Rusetzky discusses in this article the protean manifestations following encephalitis. In 1921, Souques noted that some patients with parkinsonian conditions would occasionally exhibit a marked overactivity for short periods of time. This phenomenon is discussed, and illustrative case histories are given.

A tailor, aged 48, was admitted to the Neurological Clinic of the Army Medical School, with the classic symptoms of parkinsonism. He was rigid, extremely slow, drowsy and apathetic, with marked evidences of involvement of the vegetative nervous system. Suddenly, after another patient had made some derogatory remarks about him, the patient jumped up in bed, became very excited, swore and threatened to shoot the offender, but quieted down within a short time and went back to bed. On the following morning, he was extremely dull and apathetic and seemed to have lost all interest in the affair of the preceding night. In another case, the patient, who was suffering from marked parkinsonism, was told that he would have to leave the hospital on account of a shortage of beds. He became furious, swore, left his bed, ran down the corridor in his underwear, jumped out of the window and threw himself on the street car rails with the intention of committing suicide. When reassured about his discharge, he came back to bed and within a short time again became rigid and immobile. A third case is that of a young man with a marked parkinsonian syndrome, who before the illness with encephalitis had been a fine athlete and a good ice skater. While in the hospital he was extremely slow in all movements, rigid and tremulous, and speech was indistinct. After he was discharged from the hospital, the author happened to see him on the skating field. It took him a long time to put on his skates, and at first his motions on the ice were very slow and clumsy. Prompted by a desire to show what a fine skater he had been before, he gradually began to skate rapidly and gracefully, performing difficult stunts and pirouettes on the ice. However, in ten minutes he again became clumsy and rigid and fell.

Jarkowski (Kinésie paradoxale des parkinsoniens, 1925) explained this phenomenon by the fact that the muscles in parkinsonism become hypertonic when they act as antagonists. This explains the cog-wheel movements in flexing an extremity. The affective motor reaction is decreased in encephalitis, but a very strong affective stimulus produces a normal reaction which, of course, becomes paradoxic. However, this explanation is too simple, because a great many other factors are involved; one is dealing with both the cortical and the subcortical apparatus and also with complicated biochemical phenomena. In encephalitis, the extrapyramidal system is not involved; under emotional stress it becomes active,

together with some of the coordinating centers of the cortex. In other words, parts of the nervous system which are not affected by the disease in cases of emergency assume a directing influence over the muscular system.

KASANIN, Boston.

A NEUROLOGICAL TEST FOR STUTTERERS. ROBERT WEST, J. Neurol. & Psychopath. **10**:114 (Oct.) 1929.

The author believes that some unknown factor is present in stuttering (dysphemia). He devised a test whereby the speed of the facial and mouth muscles could be recorded when simple repetitious movements were made. Normal males showed a greater speed and variation then normal females. Normal males and females respectively were faster then stuttering males or females. The variation in speed between normal persons as compared to stutterers of both sexes was similar to a high degree. In charting the data, the number of movements of some normal persons and some stutterers were found to overlap; therefore, West believes that the factor he tested is the most important single factor of dysphemia. To have a low rating would be rather presumptive evidence of a neuromuscular etiology in a case under consideration, but a low rating does not so much increase a subject's chance of stuttering as a high rating decreases it.

The speedy cerebral mechanism of shifting inhibition from one group of muscles to its antagonists is one of the factors limiting the speed of rapid repetitious muscular acts. The tapping test, no doubt, demonstrates the speed of this mechanism. In adults, neither the speed of the tapping test nor the "jaw-brow" test of this experiment is increased with practice.

Hand movements, as shown by unilateral tapping tests in normal persons, are more speedy than the tested facial or jaw movements. When both hands are used in tapping, the rate is reduced approaching that of the facial muscles. This may be due to two possibilities: (1) The necessary route of the nerve impulses may cause time loss. The hand, being contralaterally innervated, has but a simple pathway. The decrease in speed in bilateral movement is due to the additional time-consuming synapses through which the nerve impulse must travel. (2) This is based on the intimate connections of the musculature of the face, mouth and throat with subcortical centers for the control of certain emotional patterns. The rôle of the thalamus in laughing and crying, and of the medulla in breathing, coughing, swallowing, etc., is cited. The investigator believes that the test may be a measure of the ability to maintain an efficient cerebral control over the lower motor neurons to the face, mouth and throat in opposition to subcortical control of the same neurons.

Thus, dysphemia can be defined as a "hair-trigger" balance between the cerebral and subcortical centers with certain emotional factors intermittently shifting the dominance from one to the other. This shift is characterized by tonic or clonic spasm that is known as stuttering. The greater incidence of stuttering in males may be partially explained by the greater range of rate in the "jaw-brow" test. This gives a wider range of the possibilities of imbalance among the brain centers.

The conclusions appended, although interesting, do not deal with problems on which this series of experiments has definitely shed light.

BECK, Buffalo.

THE SOMATIC BASIS OF NEUROSIS. PAUL SCHILDER, J. Nerv. & Ment. Dis. **70**:502 (Nov.) 1929.

Every neurosis must have an organic basis because normal psychic life has such. Freud connects neuroses with metabolic changes. Eppinger and Hess, the first to approach the neuroses from the somatic point of view, developed the concepts of vagotonia and sympathicotonia, the symptoms of which are usually combined in neurotic persons. There is no vegetative organ that is unaffected in one neurosis or another. Vasomotor instability is present, however, in many

nonneurotic persons. For Kraus, the neurosis is a problem of balance between calcium and potassium in the tissues. Although a neurotic person can develop symptoms of hyperthyroidism, the latter condition is different from a true neurosis. Variability in temperature itself, probably due to vasomotor lability, can be found as marked in nonneurotic as in neurotic persons. Hofman and Hartmann have studied electric excitability in neuroses without positive results. Jaensch has attempted to distinguish constitutional types among normal and neurotic persons by what he terms "eidetic" pictures, which are something half way between an optic image and an optic perception. The author believes that psychologic interpretation of neuroses is impossible by a study of changes of the vasovegetative system. Every emotion has characteristic reactions in the system. Basal metabolism can be influenced to the extent of 30 per cent by psychic influence. Allergic processes can also be psychically modified. The quality and extent of the disturbance of the vegetative system vary according to the special content of the suggestion, however, and one has to find the special situations of life that are connected with these changes. It is possible that the neurotic person is one who in early childhood was under the influence of marked emotions which created an entirely different state of the vasovegetative system; hence, with subsequent emotional states a special autonomic response is evoked. Weinberg has discovered an alternation of sympathetic and parasympathetic reactions with every increase of attention, which illustrates the close connection of the autonomic system with every action in life. The author's observations on the conditioned reflex throw less light on the neuroses, in his opinion, than the neurosis throws on the conditioned reflex. Psychic influence may provoke a shifting of hyperkinesis from one limb to another. Hyperkinesis and organic tic of the labial muscles frequently provoke coprolalia. This interaction of psychic and somatic is expressed, according to the author, in interesting fashion by the complicated interrelationship between cortex, diencephalon, midbrain and medulla, which serves to emphasize the interpretative power and action of the organism. Psychic problems must be approached from a dynamic and not a static point of view. Psychoanalysis can afford insight into the whole organism with psychic and somatic manifestations.

HART, Greenwich, Conn.

A METHOD OF INTEGRATING PHYSICAL AND PSYCHIATRIC EXAMINATION: WITH SPECIAL STUDIES OF BODY INTEREST, OVER-PROTECTION, RESPONSE TO GROWTH AND SEX DIFFERENCE. DAVID M. LEVY, Am. J. Psychiat. **9**:121 (July) 1929.

Twenty extensive case histories are reported, with charts and tables summarizing the observations. By taking advantage of rapport established in the physical examination of the children, the interview was elaborated to include four groups of responses: (1) anatomic variations, (2) height, weight, strength and appearance, (3) body growth and maturity, (4) knowledge of sex difference and sex activity. The group studied included fourteen boys and six girls from 5 to 15 years of age, ranging from dull to superior in intelligence, and referred to the clinic as mild "behavior problems." Eighty responses indicated special interest in or sensitivity to some part of the body. The factors suggested in explanation were parental oversolicitude, history of illness or injury, exposure to illnesses and bodily variations in the patient. Over-protection in the parent was investigated and described. Response to crooked teeth showed its effect in speech. Sensitivity among boys was related to competition in terms of strength and size, among girls in terms of physical attractiveness. Over-dependency was shown in seven, who did not wish to grow up. In the others, unhappy conditions in childhood reinforced the wish. The wish for marriage or its rejection was apparently influenced by similar considerations plus parental influence, loneliness and lack of affection. Revulsion of body hair was frequent among the boys. Fallacy in sex differences was common even after observation of naked bodies. None had correct ideas

about the navel, and more were ignorant of coitus than of birth, and more were ignorant of birth than of pregnancy.

Responses to scenes of sex activity were confirmatory of the psychoanalytic observations. Masturbation was reported by eight of fourteen boys with a median age of 10 years. The effect of knowledge about sex on personality, the writer avers, is a difficult problem to demonstrate by direct study, but in some cases it was shown that excessive curiosity was closely related to a tabu against nakedness. It was inferred that very early knowledge of sex differences is necessary to prevent false generalizations, that early knowledge of sex processes tends to prevent distortions of fact and their later vulgarization, that training of girls to be constantly aware of aggressive sex tendencies in the male may help create excessive sex interest and homosexual tendencies and that the child's attitude toward sex activity is derived from the details and setting of the act and from his attitude toward the people who first reveal it, besides his previous experience.

IRISH, Philadelphia.

OBSERVATIONS ON THE GROWTH, FUNCTION, AND NERVE SUPPLY OF LIMBS WHEN GRAFTED TO THE HEAD OF SALAMANDER EMBRYOS. S. R. DETWILER, J. Exper. Zool. **55**:319 (Jan. 13) 1930.

The transplantation of an anterior limb rudiment to the region of the otic vesicle in embryos of *Amblystoma (A. punctatum, A. tigrinum* and *A. mexicanum),* regardless of the orientation of the graft, has resulted in the production of 99 per cent single limbs. The asymmetry of the resultant limb depends on the orientation of the graft with respect to the anteroposterior axis. When this is reversed, the limb is disharmonic; when it is not reversed, the limb is harmonic. A greater percentage of cases with limb growth was obtained in embryos of *A. punctatum* than in *A. tigrinum* or *A. mexicanum.* When two limb rudiments are grafted to the same side of the head and in such relationship as to be practically contiguous, both rudiments develop into separate single limbs. Limbs grafted to the ear region may exhibit a remarkable degree of functional activity. The function is typically coordinated with either swallowing or gill movements or both, depending on the nerve supply. Limbs from *A. tigrinum,* when grafted to the ear region on punctatum embryos, function just as vigorously as do punctatum limbs in the same position, even though they grow to enormous size as compared with the punctatum limbs.

When two limbs are grafted to the same side of the head, so that the anterior one lies dorsocaudal to the eye and the caudal one ventral and caudal to the ear, both limbs may exhibit function, which may or may not be associated. The function of the anterior one is typically associated with gular movements; that of the caudal one, with gill action. No cases were observed in which the limbs functioned independently of gill or gular movements. When a limb is grafted to the ear region, regardless of species, it is typically innervated by nerve trunks from the fifth to the seventh, the geniculate seventh and the ninth to the tenth ganglionic complexes. A much greater degree of function is obtained in limbs grafted to the head region than in those grafted to heterotopic positions on the body wall, when the latter are supplied by spinal nerves other than normal brachial nerves. The association of limb function with either gill or swallowing movements or both is correlated with the nerve supply. Cranial ganglia, when in connection with a grafted limb, undergo cellular and volume increases as do spinal ganglia. The magnitude of hyperplasia is relatively not as great as has been found, typically, in spinal ganglia. The volume increases are also of less magnitude, with the exception of ganglia which are connected with large tigrinum limbs. Here the volume increase is notably large. Increase in size of the elements appears to be a more important factor than cellular hyperplasias in the regulatory response of the ganglia to the greatly increased peripheral needs in the case of the tigrinum limbs.

WYMAN, Boston.

DIABETIC TABES. RALPH A. REYNOLDS, Jahrb. f. Psychiat. u. Neurol. **46**:267, 1929.

A woman, aged 58, developed diabetes mellitus at the age of 49. The urine contained 2.2 per cent dextrose (the amount of sugar in the blood is not stated). During the next eight years, she was in the hospital several times for iritis, headaches, diarrhea, vertigo and exhaustion. She was careless about her diet. One year before death, she developed "tearing" pains in both lower extremities followed by paresthesias extending from the toes to the knees. Examination, two months before death, revealed pupils stiff to light and in accommodation, edema of the lower extremities, and moderate sclerosis of the peripheral vessels. The tendon reflexes were present. There was a dark discoloration of the tip of the second toe and sole of the left foot. Wassermann and Sachs-Georgi-Meinicke reactions of the blood were negative. (Spinal fluid examination is not reported.) Muscle tonus was apparently normal, but motor power was diminished (it is not stated in which muscles). There were: hyperesthesia from the toes to the knees; hypalgesia from the toes to the seventh dorsal, only anteriorly; hyperalgesia and thermohyperesthesia in the fourth and fifth sacral, with thermohypesthesia from the knees to the toes. The most distal portions of the lower extremities were analgesic. A slight Romberg sign was present, with disturbances in deep sensibility of the toes and fingers. There was no tenderness on compressing the nerve trunks. As the disease progressed, the patellar and ankle jerks were abolished, and the patient became ataxic and developed hallucinatory confusion, but there were no evidences of paresis. The patient died suddenly.

Macroscopic examination of the brain revealed only hydrocephalus. The vessels of the cord, especially in the posterior columns, were markedly sclerosed, and there were evidences of perivascular sclerotic disintegration, which ran parallel to the vessels without invading the nerve fibers. The posterior nerve roots and their prolongations into the cord showed evidences of degeneration. It was evident that one was dealing not with an inflammatory, but with a purely degenerative, process. In this case, in the absence of evidences of syphilis, it would seem that some sort of a toxic process had occurred which affected the nerve roots, giving rise to a bilateral symmetrical radicular process not unlike that observed in tabes. The author believes that in diabetes one is dealing with a neurotropic toxin which has a predilection for white nerve substance and which may in some cases affect the nerve roots and in others the peripheral nerves.

KESCHNER, New York.

EXTRAMEDULLARY TUMOR. ARACHNITIS FIBROSA CYSTICA ET OSSIFICANS, GLIOSIS OF THE MEDULLA. FRANCIS HARBITZ and INGVAR LOSSIUS, Acta psychiat. et neurol. **4**:51, 1929.

The authors report the case of a woman, aged 26, who at 16 had developed a short period of diplopia. This was followed by paresis of both arms which disappeared after a few months. Ten years later, she gradually developed stiffness and paresis of both legs, but no root pains. Occasionally, she had headaches and dizziness. On examination, she showed spastic paresis of both legs and intention tremor of all extremities. Pain and temperature sensations were normal; joint sensation was impaired in both legs and touch was absent below the sixth dorsal nerve. Cerebrospinal pressure and examinations with iodized oil revealed a partial spinal fluid block. As the symptoms were so indefinite, laminectomy was not advised. Later, the headaches became more severe, she developed choked disks, a marked degree of spastic paresis of her right arm and a slight degree of paresis of the left. A decompression was done to relieve the choking of the disks, but no tumor was found at the operation. Following the decompression she died.

At autopsy, four pathologic conditions in the spinal cord and medulla were found: (1) a large endothelioma behind the medulla and upper part of the spinal

cord; (2) a tumor-like fibrous pouch with liquid contents on the front side of the dorsal region, most pronounced between the seventh and eighth dorsal nerves, an arachnoiditis chronica fibrosa cystica; (3) a gliomatosis with central necrosis in the middle of the spinal cord, most pronounced between the sixth and seventh cervical nerves, a gliomatosis with incipient syringomyelia; (4) numerous bone lamellae deposited in the arachnoid, an arachnoiditis ossificans.

The authors discuss the etiology of these conditions. Apart from the fact that the endothelioma may have been present for ten years, there is nothing noteworthy in its occurrence. The gliomatosis was probably congenital. The arachnoiditis ossificans is a moderately common observation. The presence of the chronic meningitis serosa cystica is more difficult to explain. None of the usual supposed etiologic factors — trauma, acute or chronic infectious diseases — existed in this case. The most probable explanation is that the slow growing tumor, producing an obstacle to the free passage of cerebrospinal fluid, caused a piling up of fluid and a gradual encapsulation in the form of a cystic accumulation with a cystic capsule.

PEARSON, Philadelphia.

EXPERIMENTAL STUDIES ON THE DEVELOPMENT OF THE EYE: I. THE EFFECT OF THE REMOVAL OF MEDIAN AND LATERAL AREAS OF THE ANTERIOR END OF THE URODELAN NEURAL PLATE ON THE DEVELOPMENT OF THE EYES (TRITON TENIATUS AND AMBLYSTOMA PUNCTATUM). HOWARD B. ADELMANN, J. Exper. Zool. **54**:249 (Oct. 5) 1929.

The removal of a median circular disk or a median strip from the anterior end of the neural plate does not prevent the formation of two eyes. The width of the pieces removed was from one-fourth to one-third the width of the widest portion of the neural plate of *Amblystoma*. After such removal the median regions between the eyes are restored, and both brain and eyes are regulated to form a more or less harmoniously proportioned "whole," the size of which depends on the amount of material available. It is concluded, therefore, that the materials of the anterior end of the neural plate of *Triton* or *Amblystoma*, in the stages used, are more or less equipotential in the direction of the transverse diameter of the plate. The experiments do not support the view that there is a mosaic of eye-forming substances in this region, or that the chiasma is strictly determined in the median regions and that a deficiency of median tissue is the cause of cyclopia.

The removal of a lateral third of the neural plate results in the formation of a small rudimentary vesicle or nodule on the side of the operation. If the antero-posterior extent of the defect is not large enough, an eye of practically normal proportions may be formed. In order to prevent completely the development of an eye such a lateral defect would have to extend almost or quite to the midline. Posteriorly, such a defect must extend as far as the level of the broadest portion of the neural plate. When two thirds of the anterior end of the neural plate is removed, the remaining lateral third will give rise to an eye which in many cases approximates and in a few cases equals the normal proportions. No eye is formed if the defect extends posteriorly as far as the widest region of the neural plate. The experiments indicate that there is present in the anterior end of the neural plate material possessing more or less general eye-forming potencies, any part of which is capable of forming any part of the "optico-ocular apparatus."

WYMAN, Boston.

NATURAL SELFISHNESS AND ITS POSITION IN THE DOCTRINE OF FREUD. HELEN WODEHOUSE, Brit. J. M. Psychol. **9**:38 (May) 1929.

The author admits that persons interested in education are in debt to Freud, but she tries to investigate the position in Freud's teaching of a certain doctrine that some form of self-love is the only love man can feel and that he can work for no interests but his own, which she styles that of natural selfishness, which,

while ostensibly connected with Freud, does seem to be bringing real danger into educational psychology. She has searched through Freud's works in order to elucidate two points: (1) what he actually says about the matter, and (2) whether his system as a whole depends so much on that point that any success of the system, theoretical or practical, must be taken as helping to prove the point.

She contends that Freud believes in the predominantly selfish character of human nature, though he is not so rigidly consistent in the question as some of his statements seem to indicate. His use of the term egoism is broad and popular. The most important part of his doctrine of selfishness consists in the assertion of a state of primitive narcissism, the complete form of which he places sometimes in early infancy, sometimes vaguely in childhood and sometimes before birth, but which is never permanently given up. This assertion, prominent in the writings of 1913 to 1917, is there put forward as something scientifically established, but the author suggests that the arguments when examined prove entirely unsatisfactory and that Freud's real foundation is a mental picture with several ambiguities in its interpretation. (The abstractor believes that the author's own arguments are as ambiguous as she claims Freud's to be and indicate a lack of personal experience with the situations from which Freud has developed his theoretical concepts.) She suggests further that the doctrine of selfishness is in no way necessary as a basis for the other and valuable part of Freud's teaching, and that it does not follow in any way from these as a necessary result. His system would be the stronger for discarding it.

PEARSON, Philadelphia.

THE INFLUENCE OF THE p_H ON THE HEMATO-ENCEPHALIC BARRIER. E. L. ROMEL and C. A. GUERTSCHIKOWA, Med.-biol. J. (Moscow) **2**:104, 1929.

In order to increase the p_H of the blood, a solution of sodium hydroxide was injected intravenously. In extreme cases the p_H reached to from 7.72 to 7.78. To decrease the p_H of the blood monosodium phosphate (NaH_2PO_4) was used, or else the animal was made to breathe ordinary air containing from 37 to 40 per cent of carbon dioxide. This was done by having the animal's mouth attached to an outlet of the Kipp's apparatus. Rabbits and cats were used for the experiments. The substances were injected intravenously and blood was drawn from the carotid artery. In order to test the permeability they used crystalloids in the form of sodium iodide or sodium ferrocyanide and colloids such as trypan blue and hemolysins, which, under ordinary conditions, do not pass into the cerebrospinal fluid. The determinations of the content of the fluid were made according to the method of the director of the laboratory, Dr. Stern. All the substances were tested for chemically in the spinal fluid with the exception of trypan blue, which was determined macroscopically in the fluid and in the brain.

The conclusions are: (1) marked variation in the p_H of the blood is accompanied by a decrease of resistance of the hemato-encephalic barrier; (2) the permeability of the barrier for crystalloids, colloids and hemolysins varies with a degree of change in the reaction of the blood; (3) decrease of the p_H below from 7 to 7.1 in the majority of cases results in a decrease of the resistance of the barrier; (4) increase of the barrier above from 7.6 to 7.7 also results in a decrease of the resistance of the barrier; (5) even after the p_H becomes normal, as a result of the previous experimentation with lowering and raising the p_H, the resistance is decreased.

KASANIN, Boston.

ENDOCRINE AND BIOCHEMICAL STUDIES IN SCHIZOPHRENIA. KARL M. BOWMAN, J. Nerv. & Ment. Dis. **65**:485 (May) 1927; 585 (June) 1927.

In twenty-four cases of schizophrenia, tests were used which had special relationship to endocrine function. The following studies were made: complete roentgen study, basal metabolic rate, blood sugar curve, galactose tolerance test, spinal fluid examination, gastric analysis, renal function test and cardio-ocular reflex.

The following observations appear to be of significance: Abnormally low basal metabolic rates were found in half the cases, with a tendency to low or minus readings in nearly all other cases. Nearly one half of the cases showed an abnormal blood sugar curve, all but one being of the sustained type. Over one-third showed a positive galactose test. Roentgen examinations and gastric analyses showed a definite functional disorder of the gastro-intestinal tract in about half of the cases. Roentgen examinations further revealed infected teeth in about 40 per cent of the cases, with questionable infection in 10 per cent more. The roentgenograms also revealed "dropped" hearts in 30 per cent of the cases, questionable pulmonary tuberculosis in 13 per cent and healed pulmonary tuberculosis in 4 per cent.

In the author's opinion, the observations are not consistent as to the constant presence of any definite endocrine disorder, and do not suggest that a simple glandular dysfunction of a constant type is an etiologic factor in schizophrenia. Rather they suggest that many functional disorders closely linked up with the endocrine system are frequently found and that schizophrenia is not a specific endocrine disease but may arise in a number of different cases.

HART, Greenwich, Conn.

THE CYTOLOGY OF THE CEREBROSPINAL FLUID IN DEMENTIA PARALYTICA TREATED BY MALARIA. HENRI CLAUDE, and ALFRED DIMOLESCO, Encéphale **24**:528 (June) 1929.

A technic developed by Ravaut and Boulin, for staining the cells of the spinal fluid, was utilized by these workers. The dye employed was methyl pyronine, which, when properly applied, yielded differential colors of the protoplasm of the mononuclear and plasma cells. In examining the spinal fluid by these coloration methods they found a great resistance of the cells to the dye, indicating a recent accession of the cells to the fluid or a certain inherent vitality. An accentuated polymorphism, i. e., a great variety of the elements in the fluid, is a diagnostic sign almost pathognomonic of dementia paralytica. However, it is the authors' belief that this diversity can in general be catalogued into two formulas—one with a predominance of lymphocytes and the other with a predominance of mononuclears. The other elements are of lesser quantity and importance.

The malarial attack steadily diminishes the cellular elements, until on the days immediately following interruption of the paroxysms there may be only occasional worn-out cells. At this stage polymorphism and cellular vitality have disappeared. Simultaneously there is a marked tendency for the colloidal and serologic tests to be considerably reduced. For several months after the attacks one does not observe an augmentation in the number of cells, their polymorphism or their vitality. If the patient remains in a state of remission this situation is likely to continue; if recidivism occurs cellular activity is probable. However, it is admitted that negative cytology is not an ever faithful expression of clinical remission.

ANDERSON, Los Angeles.

INJURIES TO THE OPTIC CHIASM. H. COPPEZ, Arch. franco-belges de chir. **31**:476 (June) 1928.

Coppez describes the results of injuries in and about the optic chiasm. When the skull is subjected to trauma, under certain conditions, the eyeball is subjected to pressure tending to force it from the orbit. There is a direct pull on the optic nerve. Coppez believes that the optic nerve is made up of tougher material than is the optic chiasm, so that when the nerve is subjected to stress the chiasm is torn at the same time.

He describes two cases in which, following a fracture in the frontosphenoidal region, bitemporal hemianopia resulted, due, he believes, to injury to the cross fibers rather than to the chiasm itself. He reports a second case in which avulsion of the eye resulted from an automobile accident. There was for a while complete loss of vision in the opposite eye, which eventually disappeared leaving a partial

temporal hemianopia. He believes that the temporal field defect in the opposite eye was due to injury to the chiasm at the time of the avulsion. He quotes a number of cases from the literature in which demented persons have avulsed their own eyes. This injury is always accomplished by trauma applied to the external part of the orbit. He states that it is practically impossible for an injury in the neighborhood of the internal canthus to result in complete avulsion.

GRANT, Philadelphia.

NEW CLINICAL ASPECTS OF ALCOHOLISM. J. L. RICHARDSON and M. A. BLANKENHORN, Am. J. M. Sc. **176**:168 (Aug.) 1928.

The authors analyzed 198 cases of alcoholism occurring between 1921 and 1926, for the purpose of studying the differences in those cases found before prohibition. Of fifty-one patients with acute alcoholism, 63 per cent were admitted in an unconscious condition, whereas before prohibition only 31.5 per cent of fifty-one patients were seen in this condition. A large number of the cases showed a febrile and a transient albuminuria. In twenty-four cases of chronic alcoholism, it was surprising that there were so few signs of deleterious effects and only five cases of delirium tremens, showing a decreasing incidence since prohibition. Chronic alcoholism has changed in the respect that the place of the daily "tipster" has been taken by the person who indulges in frequent sprees, but not in daily drinking. In eighteen cases of alcoholic neuritis, it was interesting to note that exquisite hyperesthesia was the most outstanding symptom found in contrast to the symptom of motor weakness noted in the past. The average number of patients with alcoholic cirrhosis was essentially the same as that before prohibition. There is a good probability that modern alcoholic intoxication is complicated with poisoning from other foreign substances.

MICHAELS, Detroit.

PATHOLOGY OF PROGRESSIVE MUSCULAR DYSTROPHY (SPINAL CORD CHANGES). J. M. NIELSEN, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **31**:158, 1929.

The case reported was diagnosed clinically as pseudohypertrophic muscular dystrophy and hydrocephalus. Although the patient was only 28 when he died, the body showed at necropsy remarkable evidences of senescence in all systems. There were: vascular sclerosis and lipodystrophy of the ganglion cells in the cord, varying in intensity from atrophy with swelling to complete destruction. The lesions, as was to be expected, were more severe in the anterior horn cells in the lumbar segments. There was very little glial reaction anywhere. Stilling's cervical nuclei and the posterior horns were fairly well preserved. The myelin sheaths showed marked destruction in the ventral horns and efferent roots. There was no acute degeneration of the nerve fibers. The author raises the questions whether the dystrophy is a sequela to the spinal process, whether the spinal process is secondary and due to the muscular disease or whether both the spinal and the muscular processes are due to a common cause (noxa). He makes no attempt to answer any of them.

THE OCCURRENCE OF SPIROCHETES AND "MINUTE BODIES" IN MULTIPLE SCLEROSIS. R. NISHII, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **31**:153, 1929.

Nishii searched for spirochetes near the ventricular ependyma and in uninvolved parts of the brain in four cases of multiple sclerosis. He was careful and followed strictly Steiner's technic but was unable to find the slightest evidence of a spirochete in any of the cases. He does not doubt Steiner's claims that he found spirochetes in multiple sclerosis but he believes, as Marburg had already stated, that Steiner's cases of multiple sclerosis were probably complicated by syphilis.

The same cases were also utilized by Nishii for the study of granules—"inclusions"—in the ganglion cells. In sections stained by the Mann method granular

deposits were found in the cell plasma but these, Nishii believes, are not unlike Negri bodies as far as their staining is concerned, but there is no reason to believe that these granular "inclusions" have anything to do with the infectious agent at the basis of multiple sclerosis.

KESCHNER, New York.

DIABETES MELLITUS: PATHOLOGIC CHANGES IN THE SPINAL CORD AND PERIPHERAL NERVES. H. W. WOLTMAN and R. M. WILDER, Arch. Int. Med. **44**:576 (Oct.) 1929.

Ten cases of diabetic peripheral neuritis are presented with a view to determining the pathologic physiology of this complication. The authors review the literature and divide the neurologic symptoms into three groups: those secondary to cachexia, those secondary to acidosis and those occurring while the diabetes is under control. They present the various theories suggested to account for the latter group, and after pathologic study conclude that the nerve and cord complications of diabetes are not comparable to the combined degeneration of pernicious anemia, nor are they due to glycosuria or acidosis, nor even to infection. They believe, rather, that atherosclerosis is the underlying cause. The paper is illustrated with nine unusually good and instructive photographs of sections of the nerves.

DAVIDSON, Philadelphia.

HYPOTHALAMUS OF THE OPOSSUM. F. J. WARNER, J. Nerv. & Ment. Dis. **70**:485 (Nov.) 1929.

Nissl, Malone, Johnston, Huber and Crosby, Gurdjian, Van der Sprenkel and others have described and enumerated various nuclei of the hypothalamus in alligators, rats, opossums, primates and man. The author, in toluidine blue sections of the hypothalamus in the opossum, describes the following group of cells as nuclei: (1) magnocellularis nucleus; (2) anterior hypothalamic; (3) lateral hypothalamic nucleus; (4) anterior periventricular hypothalamic; (5) dorsomedial; (6) posterior; (7) filiform; (8) mammillary nuclei: (*a*) medial, (*b*) lateral, (*c*) ventral. Most of these nuclei are well defined and triangular in shape. The hypothalamus of the opossum is more complex than that of the alligator and resembles that of the rabbit and the albino rat.

HART, Greenwich, Conn.

PSYCHICAL TREATMENT OF ORGANIC DISEASE. GEORG GRODDECK, Brit. J. M. Psychol. **9**:179 (Aug.) 1929.

Factors in the psyche of the patient have an important bearing on mental and physical symptoms. Treatment of organic conditions usually neglects entirely the working out of unconscious emotional factors which may be not only causing the organic disturbance, but creating a condition which does not respond to any of the methods of physical therapy. Three cases are described in which relief from the physical condition followed the working out of unconscious conflicts. Psychotherapy is a mode of treatment that should be combined with physical treatment; in the field of surgery this combination is most essential.

ALLEN, Philadelphia.

A CASE OF RETINAL EDEMA ATTRIBUTED TO ELECTRIC CURRENT. HENRY JOURDAN, Ann. d'ocul. **166**:725 (Sept.) 1929.

Jourdan reports a case of retinal edema due to the passage of an electric current through a man's body but not through his head. The moment that the shock occurred the man noted that his right eye was blind. The following day, when he was first examined, vision had improved to 2/10. There was marked edema of the retina; the field was contracted above, and there was a central

horizontal scotoma above the point of fixation. The condition gradually subsided and vision returned to normal. The only treatment consisted of a few injections of strychnine which Jourdan did not consider the cause of the improvement.

BERENS, New York.

HEMORRHAGES IN THE FUNDI IN HYPERTENSION. FRITZ LANGE, Arch. Ophth. **2**:551 (Nov.) 1929.

Urging that a more careful distinction between hypertension and arteriosclerosis be made by clinicians, Lange studies retinal hemorrhages with a view to discovering their relationships to these two conditions. He discovers that in 95 per cent of his cases of hemorrhages in the fundi there was hypertension, and in 85 per cent arteriosclerosis; none of the cases showed arteriosclerosis without high blood pressure, while 10 per cent showed high blood pressure without arteriosclerosis. From this he concludes that it is the hypertension and not the arteriosclerosis which is the factor in retinal hemorrhages. These, he believes, are usually from capillaries. He finds no part of the retina especially favored as a site for hemorrhage.

DAVIDSON, Philadelphia.

PARATYPHOID-ENTERITIDIS MENINGITIS: REPORT OF AN ADDITIONAL CASE DUE TO BACILLUS ENTERITIDIS. FRANK B. LYNCH, JR., and SAMUEL A. SHELBURNE, Am. J. M. Sc. **179**:411 (March) 1930.

Only fifteen cases of meningitis due to bacilli of the paratyphoid group have been reported in the literature; all except one occurred in Europe. The sixteenth case, that of a female child, aged 12 months, is reported. The only symptoms were convulsions. Except for retracted head and increase of the reflexes, there were no other meningitic signs. The child died in twenty-three hours. Spinal fluid revealed 140 cells, of which 80 per cent were neutrophils and 20 per cent lymphocytes. As a result of cultural, serologic and agglutination tests, it was concluded that the organism was identical with *B. enteritidis* of Gaertner.

MICHAELS, Detroit.

GLAUCOMA AND NEVUS OF THE FACE. A. HUDELO, Ann. d'ocul. **166**:198 (Nov.) 1929.

Hudelo reviews the literature of nevus of the face in association with glaucoma and adds the case of a patient, aged 20. In this patient, who had a congenital venous facial nevus, glaucoma, gastric hemorrhages and signs of cortical irritation developed. The glaucoma complicated a sudden increase in the general vascular process. Roentgenograms were of value in this case. Of particular importance to neurologists is the frequency with which cortical cerebral signs are associated with nevus of the face, so that an angioma of the skin should make one suspect an intracranial angioma.

BERENS, New York.

HYPERPLASIA IN THE BRAIN OF AMBLYSTOMA. H. S. BURR, J. Exper. Zool. **55**:171 (Jan. 13) 1930.

The implantation of an additional nasal placode results in hyperplasia of the cerebral hemisphere. Such implants frequently result in the production of aberrant nerves which establish connection with adjacent neural mechanisms. The ingrowth of an aberrant nerve into the diencephalon produces hyperplasia in the pars dorsalis thalami. The centripetal union of an aberrant nerve with the ophthalmic division of the fifth results in hyperplasia of the ophthalmic ganglion. The substitution of a tigrinum for a punctatum olfactory placode similarly results in hyperplasia.

WYMAN, Boston.

SPONTANEOUS INTRACRANIAL HEMORRHAGE FROM A VASCULAR TUMOR. R. J. REITZEL and P. BRINDLEY, Am. J. M. Sc. **178**:689 (Nov.) 1929.

A case of fatal intracranial hemorrhage from a cerebral vascular telangiectasis in a colored man, aged 26, is reported with autopsy observations. Two main groups of blood vessel tumors are discussed: (1) new and proliferating blood vessels spoken of as angiomas, and (2) dilatation and metamorphosis of preexisting vessels considered as telangiectases or hematomas. Insufficient attention has been paid to hemorrhage in cerebral vascular tumors and, on the other hand, one wonders if the association of nevi of the skin with vascular tumors of the nervous system has not been somewhat exaggerated.

MICHAELS, Detroit.

THE NATURE AND SIGNIFICANCE OF SENILE PLACQUES. M. CRITCHLEY, J. Neurol. & Psychopath. **10**:124 (Oct.) 1929.

Critchley gives an excellent and complete critical review of the entire subject of senile plaques. The successive steps in the accumulation of knowledge concerning these structures are well presented. Again the value of the Spanish impregnation methods in the unraveling of long debated neurohistologic problems is shown, for by means of the Hortega stain the relationship of microglia to the formation of senile plaques was observed.

BECK, Buffalo.

KAHN REACTION WITH SPINAL FLUID. R. L. KAHN and E. MCDERMOTT, Am. J. Syph. **13**:557 (Oct.) 1929.

After pointing out the difficulty originally experienced in making the Kahn precipitation test applicable to spinal fluid, the discoverer of this reaction points out some ways to make the test more sensitive. He suggests that the globulin be saturated to 50 instead of 40 per cent as heretofore, the saturation being carried out with ammonium sulphate. He also suggests the use of sensitized antigen.

DAVIDSON, Philadelphia.

NEUROPATHY AND THE NEUROPATHIC CONSTITUTION AS PREDISPOSING FACTORS IN PARALYSES OF THE OCULAR MUSCLES FOLLOWING SPINAL ANESTHESIA. N. BLATT, Wien. med. Wchnschr. **79**:1391 (Oct. 26) 1929.

Several cases of paralyses of the ocular muscles are referred to by Blatt and he adds cases of his own. He points out that these paralyses usually occur in patients with neuropathic constitutions.

BERENS, New York.

A NOTE ON URINARY REACTIONS IN RELATION TO INTESTINAL TOXAEMIA IN PSYCHOTIC SUBJECTS. S. A. MANN and H. L. SHIPP, J. Ment. Sc. **75**:420 (July) 1929.

From a series of studies, with various tests, the authors conclude that there is no warrant for the assumption, based on urinary reactions, that the presence of bodies arising from protein putrefaction has any particular significance in psychotic subjects.

SINGER, Chicago.

A CASE OF TRIFACIAL NEURALGIA, CURED BY POSTGASSERIAN RADICOTOMY. A. JIANU, D. E. PAULIAN and LAZARESCU, Rev. d'oto-neuro-opht. **7**:32 (Jan.) 1929.

This is a detailed case report of tic douloureux, lasting sixteen years, in a man, aged 66, who was operated on by division of the sensory root.

DENNIS, Colorado Springs, Colo.

Society Transactions

CHICAGO NEUROLOGICAL SOCIETY

Regular Monthly Meeting, Dec. 19, 1929

LOYAL DAVIS, M.D., *President, in the Chair*

NARCOLEPSY: "GELINEAU'S SYNDROME." DR. H. A. PASKIND.

L. W., aged 18, single, with no occupation, was first seen in the Neurology Clinic of the Northwestern Univeristy Medical School on May 27, 1929. The father, who had been "very nervous," "highstrung" and seclusive, died at the age of 35 of pneumonia. The mother, aged 48, was "nervous," "highstrung," and hot tempered; up to the age of 46 she had headaches about once a week which lasted twenty-four hours and were severe enough to keep her awake at night, although there were no prodromal symptoms, no scotomas and no vomiting. A brother, aged 20, had had similar periodic headaches since the age of 13, and for many years had had frequent attacks of epistaxis. The paternal grandmother, the maternal grandmother and a maternal uncle had periodic headaches. A maternal aunt was extremely nervous.

The patient was the second child, and was born at full term by natural delivery. There were neither postnatal asphyxia nor convulsions. She was breast fed for fourteen months. She cut the first teeth at 6 months, and walked and talked at 11 months. There were no temper tantrums. Up to the age of 5 months the patient slept almost continuously day and night. At times she suckled during what appeared to be sleep, but she was not apparently ill during this period. She started to school at the age of 6 and left when 17, finishing two years of high school. The grades ranged from good to excellent. Menstruation began at 13, and was always regular, with a twenty-eight day interval and three-day duration. She had always been nervous, highstrung, quick tempered, overly sensitive to criticism and pain and diffident. She had chickenpox at 2, mumps at 10 and several attacks of sore throat. When 14 she was sick for two weeks. This illness was not remembered by the patient or her mother, but an intimate friend reminded the mother recently that the patient had influenza at that time. The present illness started at the age of 16. In the late fall of the following year, she was ill for two weeks with coryza, rhinitis and general malaise. During the first week of this illness she felt weak and somewhat sleepy, and was somewhat restless at night. There was no diplopia. Between the ages of 13 and 16 she had occasional pain in the right ear, and at the age of 16 pus drained from the ear; she had had no similar trouble since.

One morning in January, 1927 (at the age of 16), at about ten o'clock, while sitting in an algebra class, alert and interested, she began suddenly to experience a peculiar "light" feeling in the frontal region, which lasted a few seconds. With this she felt herself becoming drowsy. This lasted for a few seconds and the next thing she remembered was being aroused from sleep by a blow on the back from a classmate. He told her that the teacher had "called" on her, but this she had not heard. She woke up, feeling refreshed, and thought that she had slept for about five minutes. On the same day she had five or six similar attacks at about hourly intervals; all began with the "light" feeling in the head and drowsiness; all lasted from five to ten minutes and from all she awoke refreshed. That night she retired at 10, slept well and had no difficulty in awakening in the morning. On the next day she had six or seven similar attacks and since had had such spells almost

every day, ranging in number from one to five. The longest period free from such diurnal spells was one week. Almost all the attacks were preceded by a "light" feeling in the frontal region and drowsiness lasting a few seconds; sometimes she fell asleep suddenly without any prodromes. The attacks lasted from five to ten minutes. She could be aroused; if so, she did not fall asleep again, and she felt refreshed. With almost every attack there were from one to three coarse jerks of the arms and legs in rapid succession. These occurred early or late in the period of sleep. At other times, during the attacks she puckered the mouth and rotated the pucker. There were no changes of complexion during sleep. She had no memory of events that took place in these slumbers. Frequently there were dreams in the spells, usually of a terrifying nature, of animals and men chasing her, etc. These diurnal slumbers came under any circumstances—sitting down, standing up, in noise or in quiet, or while eating. Sometimes the attacks were preceded for a few seconds by a feeling of fatigue or drowsiness; at other times these were absent. They were never preceded by yawning. She thought that the tendency to diurnal sleep was greater if she was bored or in a warm place, or after a meal, especially after a heavy meal. Fatigue after play or exercise seemed to play no rôle. Sleep was not brought on by mirth, amusement, joy, smiling, laughing, anger, noise or surprise. She retired, as a rule, at 10 p. m., slept practically continuously until 8 a. m. and had no difficulty in getting up in the morning. About three or four times a week during nocturnal sleep there were from one to three coarse jerks of the limbs in rapid succession. The mother had seen these jerks; there was no change in complexion, no salivation, no enuresis and no tongue biting. At night she frequently had terrifying dreams.

About six months after the onset of the diurnal sleeping attacks, she noticed when laughing that her knees became weak and "sagged"; at the same time the head drooped and the eyelids partially closed. This attack lasted about five seconds. Since then she had had such attacks about once or twice a week. She had fallen only once; usually she grasped a table, chair or other support and made no corrective movements with the trunk. These attacks were brought on by laughing, smiling, mirth, joy, surprise, excitement, annoyance or teasing, but they did not invariably follow such experiences; sometimes one of these states might be present and the phenomena not appear. It is important to note that the mimicry of laughing or smiling was not necessary to cause such an attack; a feeling of mirth, amusement or joy, without laughter or smiling, or with conscious and complete suppression of them, might bring on an attack, but in these instances only the head and eyelids were involved. She was aware of what went on about her. At times she became slightly pale during the attack. In these attacks of weakness she felt the same peculiar empty feeling in the frontal region that sometimes preceded a period of diurnal sleep. The heartier the laughter, the worse was the attack of weakness. Recently she had tried to smile and laugh as little as possible. These attacks were never followed immediately by sleep, nor were they brought on by trying to prevent sleep. She had had no headaches, or vomiting; the digestion and bowel action were good. Constipation had no effect on the symptoms. She had had no epistaxis, no changes in vision and no recent gain in weight; the appetite was fair. The cranial nerves were normal. Motion, sensation, reflexes, station, posture and gait showed nothing abnormal.

The Wassermann test of the blood gave negative results. A spinal puncture had not been made. Water intake and output over a period of one week, measured grossly by the mother, were both from two to three pints. The basal metabolic rate was —19.1. The urine was normal. A roentgenogram of the skull was normal. The results of sugar tolerance tests showed a normal curve. The results of a blood count were: red cells, 5,720,000; white cells, 10,850; hemoglobin, 80 per cent; polymorphonuclear neutrophils, 69 per cent; small lymphocytes 28, large mononuclears 2 and basophils 1 per cent.

Adie has stated that it was thought at one time that true narcolepsy does not occur in the female, but cited cases of his own to disprove this view. Wilson found forty-three cases in the literature, thirty-seven in males and six in females,

to which he added four cases of his own in males. Levin subsequently found in the literature fifty-five cases in males and eleven in females to which he added three of his own in males and two in females making a total of fifty-eight in males and thirteen in females. In the most recent literature that I have been able to find, cases in males are reported by Willis, Pearce, Thrash and Masse, Freeman and E. A. Bennett, and in females by Cloake, Bostock, Kluge and Cohen. In several cases, there was a history of epidemic encephalitis.

An interesting feature in my case was that the experiencing of a mirthful emotion, without the mimicry of laughing or smiling, was sufficient to produce an attack of tonelessness. Regarding this point, Wilson wrote: "The act of laughing provokes the attack more readily than any other cause, but we must note particularly that in this case the emotional stimulus itself, unless it manifests itself in laughter, does not suffice; in other words, while anger, and excitement generally, will precipitate the attack in spite of the patient's efforts to frustrate it, a stimulus arousing laughter will not produce the attack should the patient be able to inhibit the motor act of laughing. A consideration of the clinical data undoubtedly bears this contention out." But this case does not bear out this contention.

There seems to be some association between narcolepsy and epistaxis. Wilson cited four cases, including one of his own, in which epistaxis preceded or followed the occurrence of narcolepsy. Epistaxis occurred in a case report by Fulton and Bailey. In the present case there was no epistaxis, but the patient's brother had periodic attacks of epistaxis.

NARCOLEPSY: FOUR CASE REPORTS WITH THE TREATMENT OF ONE PATIENT WITH THE KETOGENIC DIET. DR. ALFRED P. SOLOMON.

CASE 1.—E. D., aged *27*, a sheet metal worker, for the past five years had experienced two kinds of attacks: (1) spontaneous sleeping spells of short duration, and (2) attacks of tonelessness occurring only on emotion. The patient did not recall the exact onset but remembered that the sleeping spells preceded the cataplectic seizures by several months. In the first year after the onset, the attacks were much more frequent than in subsequent years. The patient's childhood and early adulthood were characterized by a rather marked tendency to sleep in a predisposing environment. His disposition had always been that of an easy-going, cheerful person, except that since early life he had been subject to severe outbursts of temper over trivial things; as he stated it, he "burned up easily."

The narcoleptic attacks appeared several or many times a day. They occurred under predisposing conditions or under decidedly untoward environment. He had fallen asleep on a ladder while working, at a meal, when he slumped into a chair while in the act of putting food into his mouth, and frequently when in the midst of a happy group of companions. Some attacks were preceded by an aura of drowsiness and others come on suddenly without warning. He could ward off an attack for a time, but this caused a feeling of distress in the epigastrium, so that he was forced to give in. There were two kinds of attacks, one in which he definitely retained consciousness, was aware of everything that was going on about him and yet had no inclination to participate, and the other in which he was wholly engrossed in dreams. He awakened spontaneously within a few minutes or seconds or was easily awakened by a slight touch. In either case he did not feel refreshed following the attack but instead suffered a feeling akin to drowsiness for a period of about an hour. Excessive sleep at night caused him to have more sleeping spells on the following day, but a short nap of true sleep after working hours seemed to ward off attacks. Following attacks of tonelessness he was likely to have sleeping spells.

The cataplectic seizures consisted of attacks of loss of muscle tone accompanied by rhythmic twitching phenomena and were brought on only by emotion. A great variety of emotional stimuli might be the inciting agents: anger, particularly when aroused over a challenge to the veracity or authenticity of his statements; eagerness

to return a chiding statement with a "snappy comeback"; anticipation, as when opening a telegram or letter; an impulse to make a sudden exclamation, as when he would see a friend passing by in a machine; satisfaction at seeing his jokes or remarks appreciated, and response to a humorous statement or situation when the humor was unexpected. In these latter instances, as in Dr. Paskind's case, he did not laugh audibly but was aware of an inward sensation of mirth. Unexpectedness and unpreparedness were elements of the emotion if an attack was produced. Subsequent repeated similar stimuli on a given occasion did not produce attacks after he had had one or two. When he had withdrawn from a heated argument or had laid aside a letter to combat an attack, he might return to the same environment with apparent immunity.

The major attack was sometimes preceded by prodromes of a few seconds' duration, which consisted of an uncomfortable feeling in the epigastrium or cardiac region. They persisted as a subjective sensation of the attack, accompanied by a feeling "as if my whole body had stopped." His head would begin to bob to and fro, his tongue would feel thick and numb; he became speechless. Rhythmic twitching of the muscles of either side of the face or lower jaw ensued; the eyes tended to close, the limbs felt powerless, the knees gave way and he slumped into a chair or against any support to save himself from falling. Because of the latter characteristic the patient termed his attacks "kinking spells." Consciousness was retained throughout. The completeness of the attack was in direct relation to the intensity of the stimuli, so that all degrees of the features described were seen. His usual milder response was simply a rhythmic twitching of the lower jaw or facial muscles, during which speech was intact. I personally observed a number of these and one of the complete seizures.

The previous medical history was without significance. A younger brother had multiple exostoses of the long bones. General examination gave negative results except for the presence of a severe keratosis pilaris. Neurologic examination gave negative results. The basal metabolic rate was + 5. A roentgenogram of the skull showed a normal sella. The Wassermann reaction of the blood was negative.

The patient had been on a ketogenic antiketogenic diet for six months. He had exhibited a rapid, marked improvement coincident with its use. He now had an average of four sleeping attacks a month and had cataplectic attacks only on excessive stimuli. During a recent vacation when he avoided the ketogenic diet there was at once a return of the attacks. In addition, the patient had been instructed to take a short nap in the afternoon and to avoid deliberately the sources of exciting emotional stimuli. These measures, instituted before the use of the ketogenic diet, resulted in some independent improvement. The use of sodium bromide (10 grains [0.64 Gm.]) and phenobarbital-sodium (¼ grain [0.01 Gm.]), three times a day, caused the sleeping spells to become more frequent and had no apparent effect on the cataplectic attacks.

Three other cases may be summarized briefly.

Case 2.—M. C., aged 18, a nurse at a hospital for three years had experienced frequent typical narcoleptic spells and occasional attacks of loss of muscle tone, with laughter as the only inciting agent. Fatigue was conducive to a greater number of sleeping attacks. The family history and history of previous illnesses were without significance. The patient was irritable, aggressive and domineering.

Examination revealed an obese, yet muscular woman, with masculine secondary sex characteristics. The external genitalia were normal. There were no menstrual irregularities. The basal metabolic rate was —21 and —16 at different examinations. Sugar tolerance was normal. A roentgenogram of the sella showed a normal configuration.

The sleeping attacks were so frequent as to interfere with work and when she had these attacks in the operating room, she was asked to discontinue training.

I was able to present this patient to S. A. Kinnier Wilson when he was a visitor at St. Luke's Hospital. He confirmed the diagnosis and suggested the use of thyroid extract, one-half grain, three times a day. The patient lost weight, but the symptoms did not improve on this therapy.

CASE 3.—Mrs. E. R., a housewife, aged 30, presented typical symptoms of narcolepsy. The sleeping spells were frequent and attacks of tonelessness less frequent, occurring only on laughing. She came to the clinic at St. Luke's Hospital in great distress, saying that her husband was about to divorce her because of the sleeping spells. She was seen only once and the examination was incomplete.

CASE 4.—A chauffeur, aged 22, a patient of Dr. George W. Hall, presented the symptoms and signs of a mild postencephalitic syndrome, with a behavior disturbance. He experienced frequent momentary attacks of sleep, occurring independently of fatigue or other factors and not accompanied by consciousness of dream states. He did not have cataplectic attacks.

The usual therapy for the parkinsonian state appeared to have no effect on the sleeping spells.

DISCUSSION

DR. PETER BASSOE: Efforts have been made to induce sleep in patients with this condition by intravenous injections of calcium chloride. In a case presented at a meeting in Denver, injections of calcium chloride brought on sleep immediately. In that case, and I think in others in which it has been estimated, the blood calcium was unusually high; it is thought that perhaps the sleep centers in the midbrain may be affected in such a way that there is a disorder of the calcium metabolism. It seems to me that, since this syndrome is a combination of sleep with cataplectic attacks provoked by laughing, it is a clearcut picture that must stand for some definite disturbance. The great increase in the number of these cases since the appearance of epidemic encephalitis makes it likely that this disease has something to do with it.

DR. A. B. YUDELSON: I saw the patient presented by Dr. Solomon in 1928; the history given then was as Dr. Solomon recited it now; in addition, the patient stated that in 1923 he had a "cold," with fever, that he could not get rid of. The condition had been diagnosed as "the grip" and the patient was in bed for a couple of days. The disorder subsided; about eight months later he experienced a "kink" in the back, as if he would break in two; then he would fall forward, his head would droop and he would fall asleep. About a year afterward, he experienced a sensation which he described as "loosing of the jaw." The jaw would drop, then the head would fall forward and he would fall asleep. At first these attacks were accompanied by mild frontal headaches. Later, the headaches subsided, but the narcoleptic attacks succeeded them. During the summer, when he was working on a roof, he would fall asleep. At that time his weight was much greater than at present. My opinion then was that he had a mild attack of encephalitis in 1923, and this opinion was sustained by the fact that he had rhythmic tremors of the jaw and of both hands. I could not elicit from him a history of violent emotions, nor could I precipitate an attack by telling him funny stories or showing him funny things. I believe that this was a case of chronic encephalitis.

DR. PASKIND: I did not try to induce sleep by the method mentioned by Dr. Bassoe.

NEUROLOGIC CHANGES FOLLOWING CARBON MONOXIDE POISONING. DR. R. P. MACKAY.

Following an acute carbon monoxide poisoning in a previously normal person, parkinsonism developed with increased tonus of the skeletal musculature, "cogwheel phenomenon," masked facies, monotonous speech, rhythmic tremor of the head and hands, mental decline and early scleroderma. A study of the literature on the clinical and experimental aspects of the condition reveals that the nervous system is the seat of highly variable and complicated degenerative changes, and that these changes are the result of functional and organic vascular disturbances dependent on anoxemia. It is suggested that oxygen deprivation also produces parenchymatous neurologic alterations by direct action on the nervous tissue.

DISCUSSION

DR. W. D. MCNALLY (by invitation): The illuminating gas in the city contains about 29 per cent carbon monoxide. Each year from 330 to 550 deaths are due to carbon monoxide poisoning; deaths occurring in garages from carbon monoxide are over ten a year. They are on the increase and are absolutely preventable deaths. As has been published in the papers on many occasions, anyone attempting to repair a car should open the windows and doors of the garage and in that way dilute the carbon monoxide.

In regard to differentiating automobile deaths from illuminating gas deaths, all are due to carbon monoxide. Until it has been proved that there is an impurity in the gas that causes death one must still say carbon monoxide. Some years ago I was working with ethylene, long before it was used as an anesthetic, and it was thought that some of the deaths might be due to this; but its action is narcotic only, although in plant life it has a blighting effect on carnations. One finds sugar in the urine in many cases. Frequently, in patients who have lived over a period of twenty-four hours, one finds carbon monoxide in the blood. In two cases, I found it in the blood on the fourth day and in one on the fifth. Carbon monoxide is a poison that is of more concern to the physician than any other poison except alcohol. Alcohol is now causing about 570 deaths in Cook County each year. Many thousands of cases of carbon monoxide poisoning probably never come to the physician until severe results have occurred. The cases are difficult to diagnose because they may simulate diseases of almost any organ of the body. A case may remind one of arthritis, or of disease of the brain or lung, and one should study the history carefully to be sure that carbon monoxide is not the basis of the trouble.

DR. G. B. HASSIN: I wish to suggest a change in the title of the paper—to omit the word encephalitis. Inflammatory changes in the brain are not known to occur in carbon monoxide poisoning, and for this reason the name encephalitis is not proper.

DR. ROY R. GRINKER: One patient whom I studied also had a parkinsonian syndrome, which was limited to the upper portion of the body. Correspondingly, the anterior part of the pallidum was necrotic. Subsequently I saw another patient who survived and in whom choreiform movements persisted for several weeks after revival from carbon monoxide poisoning. I think that study of the literature will show that the effect of carbon monoxide poisoning on the brain is not direct but indirect, by means of tissue and vascular anoxemia. I believe that the anoxemia is not due to the carbon monoxide directly but to a damage of the local vasomotor mechanism. It produces a stasis and this results in a deprivation of tissue oxygen.

Dr. MACKAY: No sugar was found in the urine of this patient when he came to the hospital or afterward. I agree with Dr. Hassin in regard to the change in name. Manifestly, this is a degenerative disorder and probably not due to an inflammatory condition in the brain.

Whether stasis produces the anoxemia, as suggested by Dr. Grinker, there is an interesting piece of work by Dr. J. B. S. Haldane, of Great Britain. He subjected rats to a pressure of three atmospheres of pure oxygen and one of carbon monoxide. The rats lived perfectly well in this carbon monoxide and suffered no deleterious effects. He then examined the blood and found that the hemoglobin was saturated with carbon monoxide and could not act as a carrier of oxygen. Nevertheless, none of the rats had the characteristic symptoms which patients show. What had happened was that the blood plasma carried enough oxygen in simple solution, due to the oxygen tension to which the animals were subjected, to carry on the work of the body. I think that it is safe to say that the vascular congestion is the result of a physiologic attempt to compensate for lack of oxygen in the blood.

CIRCULATORY DISTURBANCES AND ORGANIC OBSTRUCTION OF THE CEREBRAL BLOOD VESSELS, WITH A CONTRIBUTION TO THE PATHOLOGY OF PERTUSSIS ECLAMPSIA. DR. FRIEDRICH HILLER and D. ROY R. GRINKER.

This paper was published in the ARCHIVES (**23**:634 [April] 1930).

DISCUSSION

DR. G. B. HASSIN: The problem in this paper is interesting. My impression was that case 1 resembled closely one reported by Dr. Bassoe and me under the title of "A Case of Multiple Softening." Another such case was published by Diamond. In both cases numerous areas of softening were present without changes in the blood vessels; that is, the changes were not of vascular origin. We named them degenerative, as neither the vascular lumen nor other microscopic changes in the blood vessels were present to account for the foci of softening. My impression was that in the cases mentioned the softened areas were due to a septic condition, to septicemia.

There is a condition spoken of as spontaneous meningeal hemorrhages. Some consider them as a vasomotor, that is, a functional vascular disturbance. In his studies of the pathology of the latter conditions did Dr. Grinker take into consideration the spontaneous cerebral hemorrhages which are so little understood? I could not find any reasonable explanation of their pathogenesis; there was no rupture of the blood vessel, and no stasis, and recovery is not uncommon.

DR. THEODORE T. STONE: Were there other changes in the blood vessels in any part of the brain aside from the two mentioned? Is it not true that the type of histopathologic changes seen are dependent on the time at which the brain is examined? Dr. Grinker stated that in case 1 practically all signs of pertussis had vanished; out of a clear sky the child developed first a right-sided and then a left-sided hemiplegia. Is it not possible that this started with the pertussis, and that it required four or five weeks to produce this picture?

I think that the pathologic picture is that of multiple infected emboli, which is often seen in inflammatory states.

DR. GRINKER: The functional vascular changes about which I have been speaking are not due to vascular occlusions. These have not been found. The cerebral changes are due to stasis in the circulation, and such vascular changes as might occur are secondary only to the surrounding tissue damage and anoxemia of the vessel walls. In my patient, even after three months, the wide gaping appearance of the vessels is incompatible with an organic occlusion.

The lesions in both cases were undoubtedly focal and limited to a relatively small area. No evidence of mycotic emboli was found. The lesions themselves fulfilled all the criteria of noninflammatory softenings, conditioned in one example by an organic occlusion of a large blood vessel, and in the other by stasis.

POSTVACCINAL ENCEPHALITIS: REPORT OF A CASE. DR. WILLIAM A. BRAMS and DR. HENRY WOLF.

Encephalitis following vaccination is of importance; we found only two references in American literature, while more than 150 cases, with a mortality of 41 per cent, have been reported from European clinics. A further point of interest is the occurrence of cases with the clinical manifestations of tetanus, with trismus, while histologic examination proved them to be postvaccinal encephalitis; the anatomic changes of tetanus were absent.

There are three views concerning the cause of this form of encephalitis. Some believe that neither the vaccine nor the cause of epidemic encephalitis has any direct bearing, but that a third and independent factor is responsible. This view has few supporters and there is as yet little experimental evidence in support of it.

Levaditi and other investigators believe that either the vaccine or the lowering of the general resistance by vaccination activates the dormant virus of epidemic encephalitis. Another group, McIntosh, Lucksch and others, consider postvaccinal encephalitis a distinct clinical entity, caused directly by the vaccine virus and producing characteristic histologic changes in the central nervous system. Each gives experimental evidence.

All observers are agreed that the severity of the local or general reaction has no bearing, and that there is no evidence of contamination of the virus employed.

The neurotropic qualities have been found not abnormal. The virus itself has not been at fault, because it was obtained from various sources and from different countries; an additional point of interest is that virus from the same source produced postvaccinal encephalitis in some localities and not in others.

The pathologic changes considered characteristic for postvaccinal encephalitis have been described in detail by Turnbull and McIntosh, Ledingham, McIntosh, Perdrau, and McIntosh and Scarff. The essential changes are constant and are found in the brain and cord. Perivascular softening or demyelination is especially significant, as this change is found in postvaccinal encephalitis and not in the epidemic form. The lesions are mainly perivascular, but the sheaths of the vessels are only slightly involved. Hemorrhages are slight and not often present. Vaccine virus was recovered from brains showing these changes.

The clinical manifestations were usually ushered in abruptly, about ten days after vaccination, with no unusual symptoms during the period of incubation or any particular change at the vaccination wound. Headache, vomiting, fever, convulsions and somnolence are frequent, but the most important are the nervous manifestations which may resemble those seen in serous meningitis, encephalitis or myelitis.

Somnolence is the chief symptom in the encephalitic group, but vomiting, paralysis and a positive Babinski sign are frequently present. Stiffness of the neck is common, but ocular palsy is rare. The spinal fluid is usually clear; the cells are sometimes increased and the Pandy reaction is occasionally positive. The entire course lasts from one to two weeks, and sequelae are not common.

A small group of patients with postvaccinal encephalitis showed a clinical picture of tetanus with trismus and some were treated with antitetanus serum. Several of these came to autopsy but showed the characteristic changes described for encephalitis.

A case was observed at the Cook County Hospital. The patient was a laborer, aged 23, who was vaccinated two weeks before admission to the hospital on July 30, 1929. He complained of headache, fever and vomiting, but a detailed history could not be obtained because of the somnolence. Examination revealed temperature 100 F., respirations 24 and pulse rate 96. There was marked somnolence and the patient could hardly be aroused. All the cranial nerves were normal. The pupils were equal and reacted to light, and no strabismus was present. Slight lateral nystagmus to both sides was noted. The corneal and pharyngeal reflexes were diminished. There was moderate rigidity of the neck and a suggested Babinski sign on the left side, but the Kernig and Brudzinski signs were absent. The superficial and deep reflexes were normal and no paralysis was found. The spinal fluid was clear and under slightly increased pressure; the cell count was 50 and the Pandy test was positive. No organisms were found in a smear or culture medium, and the Wassermann reaction was negative. Nothing abnormal was found in the urine, and the blood chemistry showed urea nitrogen 15 mg., and blood sugar 155 mg. per hundred cubic centimeters. No other abnormal physical observations were obtained. The rise in temperature, always moderate, persisted for four days and then became normal. The patient made a complete recovery and returned home in eight days without sequelae.

1. Postvaccinal encephalitis is perhaps more common in this country than the literature would lead one to suppose. 2. Cases have been reported in which the clinical symptoms resembled those of tetanus, but autopsy proved them to be encephalitis. 3. A case is reported with somnolence and rigidity of the neck following vaccination and ending in recovery.

DR. G. B. HASSIN: The term postvaccinal encephalitis can conveniently be replaced by "cowpox" encephalitis, so that in addition to the epidemic, hemorrhagic, poliomyelitic and typhus fever types, one must consider a cowpox variety. In January, 1926, I had the opportunity to study a case of postvaccinal (cowpox) encephalitis (ARCH. NEUROL. & PSYCHIAT. **23**:481 [March] 1930) in a boy, aged 7, who developed a clinical picture of tetanus eleven days after vaccination. He died within twenty-three hours after the onset of the nervous symptoms. While

recovery in postvaccinal encephalitis occurs in 60 per cent of the cases, patients with a clinical picture of tetanus almost always die. The recovery is complete, leaving no sequelae. However, at the Cook County Hospital there was a girl, aged 18, with postvaccinal myelitis, who developed the signs of myelitis about nine days after vaccination. The arm became swollen the day following the vaccination; nine days later, a flaccid paralysis of the arm and of both lower extremities set in. Within four or five weeks the paralysis of the arm disappeared, but the paralysis of the extremities remained stationary. Three years after the onset it resembled poliomyelitis. A report of this interesting case was published by the interns of the neurologic service of Cook County Hospital (Perritt, R. A., and Carrell, R. C.: Postvaccinal Myelitis, *J. A. M. A.* **94**:793 [March 15] 1930). As to the pathologic changes in cowpox encephalitis, they are much more marked and abundant than in the epidemic variety and it is rather remarkable that they usually clear up, leaving no changes in the brain.

Dr. Hans Reese (Madison, Wis.): The interesting paper of Drs. Wolf and Brams brings up the question whether one is justified to diagnose clinically, even with the aid of serology, the picture of postvaccinal encephalitis or encephalomyelitis. I doubt that one can diagnose these conditions correctly, because I do not know of any postvaccinal syndrome clinically.

In November, I saw a boy, aged 14, with a negative history of previous catarrhal infections, who had been vaccinated previous to my examination. On his left arm there was still an enlarged, brownish scab. He complained of paresthesias and muscular weakness in the legs, with urinary retention since the tenth day after vaccination. On the sixteenth day, he presented a distinct transverse myelitis at the level of the eighth thoracic segment. The spinal fluid was under no increased pressure; the Queckenstedt phenomenon was normal and serologic studies revealed a slight increase in globulin, reduction of the colloidal gold curve and 8 cells. He made an uneventful recovery within three weeks. Repeated examinations did not reveal any residuals. Here again the question comes up whether one is justified to bring the myelitic syndrome in relation to the vaccination. I consulted with Dr. Bassoe and we came to the conclusion that one should not connect the vaccination with this clinical picture.

The great number of cases of postvaccinal encephalitis in England may be the result of the lymph that is used. Drs. Myer Coplans, W. J. King and Sir William Simpson state that in eighteen of twenty-five cases of postvaccinal encephalitis that have occurred in England, the lymph was a product of the vaccination of calves with a lymph derived from the rabbit. The possibility of contagious encephalomyelitis observed in stock rabbits involves a possible risk of the leporine virus. The fact that true vaccine lymph was used by the Army Vaccine Institute in England from 1880 to 1910 without disturbing accidents in 2,000,000 cases, shows that it is tolerated by subjects of all ages. This lymph was used not only in men, but also in women and children. The frequency of encephalitis, in comparison with the number of vaccinations performed yearly, remains exceedingly small. While one should recognize the risk, it should not be magnified into an objection against universal vaccination.

Acute, Toxic (Nonsuppurative) Encephalitis in Children. Dr. A. A. Low.

This paper was published in the Archives (**23**:696 [April] 1930).

DISCUSSION

Dr. Roy R. Grinker: I was glad to hear that Dr. Low found so many changes that I have described in acute toxic encephalitis. His work corroborates the suggestion that the damage is largely exerted on the blood vessel walls and that the parenchymatous changes are to a great degree secondary. I had an opportunity to examine the slides of Brown and Symonds and was interested to

see that what they termed perivascular edema was not artefact. Were studies of the pear-shaped cells made with reference to their lipoid content? An increase in intracellular lipoids similar to that found in amaurotic idiocy has been found in tuberculous meningitis and other infectious diseases of the brain.

The edema of the glia reticulum is interesting. Its presence in Dr. Low's cases and its absence in mine is probably accounted for by the fact that life was prolonged for a longer time in his patients. It seems conclusively proved that the anatomic changes are due to a circulating toxin from a distant focus of infection rather than to an invasion of the brain by micro-organisms. The meningeal proliferation, the predominatingly proliferative changes in the vascular endothelium and the absence of mesodermal infiltrations, all resembling those found in heavy metal poisoning, speak for a toxic reaction.

Dr. G. B. Hassin: I followed the work of Dr. Low closely, saw his specimens repeatedly, and wish to state that the slides he demonstrated do not do justice to the pictures seen under the microscope. It is difficult to obtain suitable microscopic pictures for demonstration. I am glad that Dr. Low emphasized the importance of the work of Lotmar in Alzheimer's laboratory. It is the finest work done on encephalomyelitis produced experimentally by injecting into animals various toxic substances, such as the dysenteric virus. The changes found by Lotmar resemble essentially those outlined by Dr. Low. The differences are due to the fact that Lotmar, who worked on small animals such as rabbits, actually flooded the blood with the toxins. The changes thus produced were for this reason more striking; the time during which the animals could be kept alive also varied. Of course, aside from the time element, the virulence of the toxins injected likewise affected the pathologic picture. All these factors were instrumental also in the cases studied by Dr. Low.

NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY

Regular Meeting, Dec. 9, 1929

Moses Keschner, M.D., *President, in the Chair*

Narcolepsy Associated with Chronic Encephalitis. Dr. Walter Bromberg.

A man, aged 39, was admitted to the Manhattan State Hospital in September, 1929, with a history of a psychotic episode with persecutory trends, from the Metropolitan Hospital, where he had been a patient on the chronic neurologic service. He was a Hungarian, resident in this country for twenty-six years. The family and personal history was entirely unimportant for mental or nervous disease, or for any abnormality. The patient said that he did not use alcohol and that he had never had syphilis. A personality study gave normal results except for periodic outbursts of anger which lasted two or three days, leaving no permanent effects. In March, 1920, he had an attack of acute epidemic encephalitis (Bellevue Hospital) in which somnolence was a marked feature. The patient seemed to recover until 1921 when he began to fall asleep at inopportune moments — while talking or eating — in response to an irresistible impulse to sleep. These attacks lasted from five to fifteen minutes; he could be easily awakened from them. Eight months later, in 1922, the patient developed attacks in which he would suddenly fall to the ground when "aggravated" or surprised. At these times he would slump to the ground, unable to speak or move his extremities, but conscious of his surroundings. Later, he observed that when he laughed loudly he would fall to the ground suddenly, so that he schooled himself not to laugh heartily.

In 1923, the patient was observed at the Fifth Avenue Hospital where he gave the impression of having myasthenia gravis, but a final diagnosis of chronic encephalitis was made. From that time until the onset of the psychotic condition, he was at the Metropolitan Hospital; during the intervening years the cataleptic and sleeping attacks continued, and the parkinsonism increased. On admission to the Manhattan State Hospital, a diagnosis of symptomatic narcolepsy was made (September, 1929). Examination then showed bilateral ptosis, muscular rigidity, hyperactive reflexes, clonus on the left, tremors of the hands, legs, jaws and face muscles, irregular pupils reacting sluggishly to light but not in accommodation, difficulty in upward gaze, and convergence difficulty.

Numerous sleep attacks were observed. Although he had the objective appearance of one who sleeps, he was conscious of the environment but was powerless to move his arms. Cataplectic attacks were noted and were increased by emotional excitement, but they also occurred spontaneously. In these attacks he was not in a state of areflexia but he was unable to move and would slump to the ground helplessly. Several variations were noted, combining characteristics both of the sleep and falling attacks. The patient was presented.

The scope and meaning of the term narcolepsy was briefly sketched. It originated with Gélineau, in 1880. The division into idiopathic and symptomatic types was noted. The physiologic, pathologic and psychopathologic views on the etiology of narcolepsy were stated. In a search of the literature only nine cases following definite encephalitis were found, only four having coexisted with parkinsonism. The case presented followed a definite attack of epidemic encephalitis, encephalitic parkinsonism coexisting with the narcolepsy.

DISCUSSION

DR. SAMUEL BROCK: I had occasion to study an instance of idiopathic narcolepsy, cataplexy and catalepsy associated with an unusual type of hallucination (*J. Nerv. & Ment. Dis.* **68**:583, 1928). In my patient an encephalitic etiology was fairly definitely ruled out. With Dr. Bromberg's case, there are some sixty-seven cases described (Levin, M.: ARCH. NEUROL. & PSYCHIAT. **22**:1172 [Dec.] 1929) of which eleven may be regarded as due to encephalitis. The other fifty-six are allocated in the idiopathic group. Narcolepsy and cataplexy were associated in this group. I cannot escape the firm impression that we are faced with an organic disease in the idiopathic group. Just as epidemic encephalitis has been the basis of many types of peculiar tics (which hitherto had been regarded as purely functional), so this inflammatory disease with its protean manifestations has conditioned a narcoleptic-cataplectic symptom-complex. In this connection the recent paper by von Economo on the sleep mechanism and its center lends further weight to the theory of organic causation. In fact, von Economo prophesied the demonstration of structural changes in the upper periaqueductal region of the midbrain. No case of the idiopathic variety has as yet been subjected to pathologic investigation.

PROFESSOR SCHILDER: At the present time many patients are seen with a condition between neurotic and organic sleep disturbances. It seems that narcolepsy belongs also within the interesting group of diseases that can be considered from the psychogenetic side as well as from the organic side. One of the postencephalitic patients I observed showed typical narcolepsy and at the same time had a case of monocular diplopia in connection with hysterical spasms in the eye muscles. It was interesting that the father of this patient also had sleeping attacks.

In such a case there are three converging factors; the psychogenic factor, the factor which is in connection with the encephalitis and some hereditary inferiority of the sleeping center. If one sees a great number of patients with narcolepsy, one will find that in many other cases these three factors can be found. One factor will be prevalent in one case, another factor in an other case. In the present case I think there cannot be any doubt but that the organic encephalitic

factor is the most important one; but there are many cases which stand between. We know that in cases of narcolepsy psychotherapy has sometimes a marked effect; therefore I would always advise looking for these three factors; heredity, psychogenesis, and organic disease.

SOME ERRORS IN THE DIAGNOSIS OF SCHIZOPHRENIA. DR. C. MACFIE CAMPBELL.

There are some factors which stand out in the examination of the schizophrenic patients at the Boston Psychopathic hospital. The occurrence of a low basal metabolism is of interest in a certain number of cases. The suggestion that an endocrine disorder is at the basis of these polymorphic clinical pictures is important, but with the present methods for studying the efficiency of the endocrine system one cannot say definitely that this or that gland is especially at fault. One worker at the hospital has investigated the question of auto-intoxication from the intestinal tract, and the results of these investigations will be published later. In this research, one is in a most difficult field in which a great deal of preparatory work must be done before one can apply the results to our clinical problems.

In our investigation of cases of schizophrenia we have paid attention to other aspects than the somatic aspect of the disorder. If the sick person with his queer attitudes, beliefs and experiences is not physically sick, if these symptoms are not due to some undermining physical disorder, is it not possible that it is the reaction of especially vulnerable or especially sensitive persons to some of the more complex stresses and strains of life; that schizophrenia is not a reaction to *Bacillus coli* or the streptococcus, but to the environment and to strains of a somewhat more complicated nature? So we have studied the immunity of the individual to social stresses and strains and have tried to see whether our patients were of a type that was more likely to break down in the face of environmental strains than the average. We have reviewed the environmental factors which seemed to be involved in the difficulties of the patient, and have learned much from intensive studies of individual cases. In addition to these intensive studies, it seemed of value to make more extensive studies and to utilize the extensive material of the Boston Psychopathic Hospital; with the help of Prof. E. D. Wilson, Professor of Statistics in the Harvard School of Public Health, we undertook a statistical investigation of our material, aided by a generous grant from the Laura Spellman Rockefeller Memorial.

Our problem was to see whether, if we took a large number of cases (or what we consider a considerable number of cases in Boston where we have comparatively small hospitals and comparatively small numbers) and organized that material in such a way that we could run through the usual statistical correlations, we might get confirmation of one or the other theory; either that the disorder is primarily due to a physical condition or that it can be correlated with some type of constitutional weakness, or that it is partly a response to environmental stresses or strains. So we put in motion the ordinary statistical machinery. I want to speak of some of the difficulties. Before you cook your hare you have to catch it, and before you can statistically analyze patients with schizophrenia you have to get hold of them. What is a patient with schizophrenia? How do you know him when you have him? What are the criteria of schizophrenia?

When we studied the records of cases that were listed as schizophrenia, we looked at some with humility and others with critical interest and felt that it was a difficult matter to be quite sure in all cases that bore this label that they really represented an underlying unity. It was interesting to see how cases which in one attack were diagnosed as schizophrenia, in another attack were diagnosed as something else. Perhaps even in the course of the same attack one might find a change of diagnosis — different physicians looking at the same problem in a somewhat different way and affixing different labels without making it clear on what basis the change of label was made.

First one might call attention to the number of cases in which the difficulty was in placing the individual case in the group of schizophrenic as against the affective disorders.

Those of you who have worked in a state hospital are familiar with these classic discussions. One physician emphasizes certain symptoms which, according to the textbooks, warrant the admission of the case into the manic-depressive group; another physician emphasizes some other symptoms which are considered to indicate dementia praecox. Professor Bleuler has given the key to such a situation; he has shown the futility of such discussions by pointing out that it is not a question of either manic-depressive insanity or schizophrenia, but rather to what extent the patient is a manic-depressive, and to what extent the patient is a schizophrenic type. One type does not necessarily exclude the other.

This means that we are thrown back on the problem of constitutional types of reaction. Types of reaction remain fairly pure. The hysterical individual, as a rule, does not pass over into a state of psychosis, although in a few cases one observes such a transformation. Then one has the interesting problem of why there was this progression of the case or why there was this regression to a still deeper level. It looks as if the hysterical patient has a safety valve and a temporary resting place in his symptoms; and by means of his invalid symptoms, whether they are mental or physical, manages to attain something of a working adjustment. His hysteria enables him to get along; perhaps it preserves him from further stresses and strains which might be more crushing than those to which he has already been subject. Every one does not have the hysterical safety valve. That is part of the special endowment of certain persons. We cannot all faint or vomit with the same facility; and some of us have to carry on without getting the relief and the shelter which physical invalidism offers to some. Some persons have other modes of reaction, and we find in the manic-depressive group the man who throws off his responsibility to normal social values and conventional restrictions and lets himself go in his exuberant way, or who, on the other hand, retires into his shell with his depression and lets the storm of this world blow over his head. In the manic-depressive person you find that the reaction may remain perfectly pure and without complications.

But perhaps that reaction does not prove a sufficient adjustment in the individual case. Perhaps there are still other difficulties, and the manic-depressive person may use his human equipment of thought and fantasy and his special sensory endowment. He may withdraw into an inner world which he creates in virtue of his special imaginative endowment and then one has a case which is similar to that of schizophrenia. The schizophrenic person may have neither any hysterical safety valve nor this special affective condition, which allows a certain amount of relief under situational pressure, but he meets a situation by developing a special picture of the outside world, by building up his inner experience in a form which makes existence tolerable to him, and for him this is more or less of an adjustment, although it seems to the outsider to be very far from a social adjustment.

We, therefore, need not be surprised if we find that the manic-depressive patient, the patient with this special type of affective instability, may also show a variety of symptoms which have been more emphasized in the setting of the schizophrenic disorder.

I might make this discussion a little more concrete by referring to a case, some details of which have already been published. The patient was a woman, aged 29, who was admitted for the fifth time to the Boston Psychopathic Hospital in 1926. A study of the record of her third admission (fourth attack) was of considerable interest. The physician had been much impressed by the schizophrenic element in the picture. The patient had been hearing her own thoughts; she heard God's voice; she knew that people could read her mind; her behavior in the ward was apt to be impulsive. The physician was impressed by these elements in the picture, and with the more serious diagnosis in the back of his mind laid little stress on the fact that there was definite depression at times and

exhilaration at other times, and that much of her activity seemed to be in keeping with this exhilaration.

If the physician had been looking for the diagnosis of manic-depressive insanity he would have found material for it in the records — laughter, loud, boisterous pleasantries, coarse jokes, periods of overactivity and apparent playfulness. But with the formulation of dementia praecox or schizophrenia in mind, these various reactions had to be given a rather different interpretation. The laughter, therefore, was referred to as "silly," and the euphoria was qualified as "apparent." To quote from the records: "At times the patient laughs, jumps, and hits the nurses and other patients, which resembles playfulness. But there often seems to be a good deal of impulsivity in it."

The physician reviewing the two earlier attacks of the patient in which the diagnosis of manic-depressive insanity had been made saw no good reason for this diagnosis, although at times a typical manic syndrome had been present.

It is this loose use of terms, and the lack of accurate objective observations carefully controlled and measured, that render psychiatrists the object of amused interest to our colleagues, the biochemist and the physiologist, and their derivative, the internist. We psychiatrists must plead guilty to the charge that the terms used in psychiatric records often represent rather hazy concepts and carry with them subtle suggestions. Our concepts are not cleancut and definite with an exact connotation.

Thus, in the case quoted, which impressed the staff as having a bad prognosis, we find in the record emphasis laid on apathy, indifference, unmotivated behavior, silliness and shallow emotional tone. Each one of these terms is good enough for a novel, but when one is dealing with science each one of these terms is a challenge. Is it sufficiently specific to know that the patient shows apathy? Do you know what the physician observed? As a matter of fact, the condition which might be described as apathy may be an expression of reduced physical vitality due to some infection or metabolic change. On the other hand, this so-called apathy may represent a mild depression of obscure origin, or it may be a reaction caused by an extremely discouraging life situation. Or again, it may indicate the fact that the patient is a day-dreamer, absorbed in a subjective world of his own and little interested in the concrete demands of his work-a-day environment.

It is of great importance in the interpretation of the case to know which of these attitudes is really before us; to refer in a general way to the patient showing "apathy" is absolutely inadequate. This is true of this "unmotivated impulsive" behavior of the patient, the sudden activity without a purpose obvious to the physician helps to give that impression of the odd and unexplained, which influences the diagnosis of the case. But the activity may be associated with undercurrents of exhilaration, the chief expression of which may be in these sudden acts. Or, on the other hand, there may be an underlying mood of resentment or anger. The underlying mood may be related to certain topics of preoccupation; the apparently impulsive act may be the response to a stimulus which has touched a very sensitive spot. In other cases, the so-called unmotivated action may be emotionally intelligible. It may be the reaction to pain from an abscessed tooth or to pent-up resentment at personal failure, and this discharge of pent-up emotion may to the physician seem an absolutely unintelligible part of a generally disorganized activity, and therefore schizophrenic.

We know that the queerer, the more bizarre the behavior of the patient the more serious the case, but we must reduce our observations to much more precise terms. With regard to silliness and "shallow emotional tone," if we wish to paint the picture dark we call the laughter of the patient "silly"; on the other hand if we are optimistic, we call it a mild degree of exhilaration. Thus conscious or unconscious bias in favor of a certain diagnosis may color the description of behavior and utterances and the understanding of the mechanisms is made more difficult. The use of the term "negativism" may obscure the meaning of behavior which otherwise is fairly intelligible. A case like the one reported has to be

utilized in discussing the problem of the affective disorders. It also has to be utilized in a discussion of schizophrenia. From the point of view of statistics and of official classification, it is somewhat embarrassing to give a patient two or three diagnoses. The official emphasis on classification weighs so heavily on the mind of many physicians that they would much rather have the docket clear and have each case neatly classified than leave a lot of cases over which there is no definite label. The enforcement of a rigid diagnostic schema not only gives a bias to the observation of the case, but also to the description of the condition after the psychosis has run its course. Thus a patient may be looked on as still having "a residual defect" when the real condition is some mild change in the affectivity. The term "defect" is used because it ought to exist if the diagnosis made was that of dementia praecox. To a person not warped by too rigid a psychiatric creed it might appear not unnatural that a patient who has been perhaps for years out of normal social life and who has seen a lot of trouble in that time, might not have the same elasticity and joy in life as he had before the breakdown.

In the record of another case, a woman with a recurrent psychosis, there was reference to hallucinations and delusions; obviously a great deal of weight had been laid on the presence of these hallucinations and delusions, without the recognition that these are summary terms for the most complex and the most heterogeneous experiences. This patient had thirteen attacks, and in many of them showed a typical manic picture. She was considered to show typical schizophrenic reactions by one physician, who paid no attention to the fact that the patient as a young woman had always been rather a fantastic dreamer with a somewhat poetic endowment, and that in her psychosis these subjective creations came up without having any profound significance.

I have spent a lot of time discussing some of the problems which come up when one tries to find out which cases one is entitled to look on as schizophrenia. Numerous cases of other types occur. In many cases of an acute transitory psychosis of somewhat unexplained nature, the physician has put down the diagnosis of schizophrenia apparently by exclusion. With no suitable standard term for these heterogeneous psychoses, the physician finds the term schizophrenia always available and with regard to which there is the least likelihood of criticism.

Thus a bachelor, aged 45, who was making a rather poor show of running an office and whose finances were running down, suddenly shot off a gun one day. At home later he told his sister that he had been defending his castle. He tore down the curtains, tore out the telephones in the office, and got up on a chair and orated against the fickleness of woman.

He was brought to the clinic and after two weeks he quieted down and soon was as well as ever; for two years was doing admirably. He was looked on as a schizophrenic patient because his behavior was queer, apparently impulsive, and lacked relationship to the regular plan of his thought and work.

An irritable bachelor who has been losing money for some years may not be in a placid mood; when harrassed by business he tears out the telephones, jumps up and orates on the fickleness of woman; it may be impulsive, but it is not hopelessly unintelligible. At the present moment it seems a better procedure to exclude such a case provisionally from the schizophrenic group.

In other cases the question of alcoholism has complicated the clinical picture and the history; one physician diagnoses schizophrenia, when another physician would prefer to diagnose the case as an alcoholic psychosis. Here again, Professor Bleuler has shown the way to deal with the problem, for he has demonstrated that many persons are latent schizophrenics, and many would remain latent schizophrenics were it not for the reduction of their vitality by chronic alcoholism, in which case the schizophrenia becomes manifest.

Another case which illustrates the complexity of our material was that of a man who offered adequate material for supplying a diagnosis of schizophrenia if one works with counters and symptoms and considers that with enough symptoms to make up the count to so many points the diagnosis of schizophrenia is justified.

In this case, the patient, aged 36, had odd ideas about the cosmos and about life in general. He liked to sleep with his feet to the north, as he was easily affected by the vibrations of the earth, and he was rather impressed with the fact that he had been born under the sign of Aries. He claimed to have mental pictures of conditions on other planets. He had at one time felt he could see into the future. He could heal people, drawing up disease as blotting paper takes up ink. He was admitted to the hospital as he had behaved oddly toward a young lady whose face he wanted to analyze. It was easy to place such a clinical picture in the schizophrenic group, and to suggest a hypothesis as to the rôle of constitution, but we had to take into account exogenous factors of two types, impersonal and personal.

Before he was 15 or 16 he had already been having telepathic communications with his mother, who brought him up in that atmosphere and drenched the boy with telepathic beliefs. At 18, he once happened to hang his coat on a gas bracket; the next morning he was found unconscious, because he had turned on the gas with his coat. During the next six weeks he was seriously ill; he slowly built up his assets and lived in a simple way; he was no longer the fellow he had been before, but lived an odd, nomadic life. In the course of that nomadic life he met a Californian astrologer who further complicated his outlook on life.

Whether you do justice to such a case by referring to it as schizophrenia is a question.

My choice of topic this evening may perhaps require some justification. You may feel that the difficulties experienced at the Boston Psychopathic Hospital are personal troubles and should be kept discreetly in the domestic circle. With a complicated and difficult problem like schizophrenia, it may be of some interest in one clinic to know how people think in another clinic, and what they mean by the words they use. If you want to know what we mean by schizophrenia in Boston, we would have to tell you that we were uncertain ourselves about it.

In any attempt to trace the general laws which are at the basis of the schizophrenic psychoses, we first of all wish to take a material which is more or less homogeneous, to specify the clinical criteria and to work with small groups very carefully selected. One criterion for admission to this group should certainly be barred, viz., the condition that the patient should be deteriorated, or that one should feel sure that he is going to deteriorate, for in making that condition the criterion of admission to the group we are begging the most important question that is connected with this whole topic. It is as if in discussing the whole problem of pulmonary tuberculosis we were only going to take into consideration those cases with cavities in the lungs or which we felt sure were running a downhill course.

The difference between benign and serious schizophrenic cases is one of the central problems in psychiatry. We no longer have quite the same fatalistic attitude toward schizophrenic cases as we had when we were faced with the enigma of dementia praecox. The work which we now carry on is carried on in a much more optimistic spirit and on a broader basis, owing much of its inspiration to the genius of Professor Bleuler, of Zurich, who in his work on schizophrenia has done more than coin a new name; he has given us a new inspiration and a broader outlook.

DISCUSSION

PROFESSOR ERNST KRETSCHMER, Marburg: The speaker is right in pointing out the important rôle of the endocrine glands in the determination of the endogenous psychoses. In the realm of schizophrenia, for example, there are certain endocrine groups differing widely in prognostic value. We find, for instance, that young men with eunuchoid development present a picture of schizophrenic disorders during puberty. These develop into hallucinations and acts of violence, but show no strong disposition toward deeper psychic disturbance.

Quite different is the course of certain psychoses accompanied by endocrine fatty degeneration. This condition occurs particularly in women who at puberty

develop obesity, and at the same time exhibit psychoses, which are first recognized by a psychic violence, sometimes accompanied by catatonic, sometimes by epileptic seizures. Such psychoses may lead to serious enfeeblement.

SUGGESTION AND SUGGESTIBILITY. PROFESSOR E. BLEULER.

Suggestion and suggestibility (as well as hypnosis, one of their special forms) are still frequently considered to be unintelligible peculiarities of the human psyche; particularly their influence on vegetative functions puzzles many people. The purpose of this paper is to show that they can be explained easily on the basis of elementary functions.

The close relationship of the suggestion phenomena with the affectivity can be demonstrated easily. There are two important qualities of the affectivity which have to be considered. Every affect influences all other psychic functions in such a way that everything that tends in the same direction is furthered, while everything else is inhibited. For example, a depressed person can think only of depressing things. The furthering or the inhibiting of other functions by affects concerns all psychic and physical functions and also our vegetative centers, making us blush, influencing our respiration, our metabolism, etc. This mechanism guarantees the uniformity and the energy of our will and of our actions.

Then there is an elementary tendency in people with the same interests to react with similar affects to affective utterances and among enemies to react with opposite affects. The courage of one individual awakens the courage of his friends and the fear of his enemies. These phenomena can be followed in every social relationship, for instance, in that of mother and baby. But they are also true among animals and can be studied in herds or flocks. These affective influences are not even limited to species, but creatures of the most different species can be observed to react to the signs of the affects of other species ("interbestial language" is Neutra's expression).

The affective tendencies (to influence all other psychic and physiologic functions and to awaken affects in other individuals) which have been described can explain all suggestion phenomena.

Frequently, in animals, information in a simple form is transferred along with affects. On account of language men are able to suggest a great variety of ideas, which often no longer have direct connection with the affectivity. A careful study, however, always shows the rôle of the affectivity.

Similar mechanisms produce common types of hallucinations. Under the influence of the irradiation of an affect, the memory pictures of sensory impressions become conscious. (They are usually inaccessible to consciousness, because we have to react to abstractions of the sensory impressions and not to their original forms.)

As highly differentiated mechanisms as the suggestive phenomena are usually considered to be merely psychic. It has been shown, however, that they can be explained by elementary functions which are not specific for the human psyche, but which are also true for vegetative functions and for animals. Psychic functions, as shown by the example of suggestion, cannot be separated from vegetative and biologic functions. This coincides with our conception of the psyche as a specialization of functions which in primitive form are in every living cell, and which I have called "psychoid."

DISCUSSION

DR. PAUL SCHILDER: I shall mention some neurologic symptoms which may sometimes be of great use in the diagnosis of schizophrenia: Rigidity and changes of the shape of the pupils can be observed in some patients. This rigidity changes very quickly, partly by psychic influence, partly under the influence of bodily exertion. These symptoms occur also in acute cases, but are more common in chronic cases. In my material the following family is of special interest:

The mother had a hypochondriac-paranoid psychosis. She felt that she had been ruined and poisoned by an arsenic treatment. The one daughter had an

acute schizophrenic psychosis in which manic features prevailed. She would have rigid pupils for three or four weeks, but there were sometimes changes in the pupillary reactions in the course of hours and even minutes. This patient recovered almost completely. Some years later, I saw her older sister with a neurotic condition (neurasthenic headaches and anxiety). She had incomplete and changing pupillary reactions. These pupillary changes made another neurologist suspect syphilis, but the serologic examination in her case, as well as in the case of her sister, gave completely normal results. There is seemingly an hereditary factor in these pupillary reactions.

Another group of symptoms concerns the motility. One often finds changes in the postural and righting reflexes, especially in catatonic states, in cataleptic states and in hyperkinetic states. Hoff and I have studied these phenomena in normal people. When a normal person stretches out his hands, there is a slight divergency. This divergency reaction, as we call it, is increased in almost all cataleptic and hyperkinetic states. This increase in the divergency reactions sometimes goes so far that the patients open their arms widely and believe that they are Christ and are crucified. Such an extreme divergency is possible only when the postural change is increased by the psychic tendencies of the individual. But some of the delusions of the patients may have motor phenomena of that kind as their basis.

The neck reflexes in catatonic patients are also often much increased. If one turns the head of this type of patient to the right side, the arms and trunk follow to an extreme degree. Sometimes a turning around the longitudinal axis is noted which almost reminds one of organic cerebellar or parieto-occipital cases.

The postural attitudes in all such cases are more like those on children. We are justified in seeing in this phenomenon a sign of a regression to a more primitive state of motility, and in this respect a study of the motility of the schizophrenic patient leads to the same result as the psychoanalytic study of the schizophrenic patient. Certainly it would not be justifiable to believe that in schizophrenia we find only changes in the subcortical apparatus. I should prefer to say that the accent goes from the cortical part of the innervation to the more primitive subcortical apparatus.

An interesting phenomenon I have studied in the last year is the reaction toward pain. The reaction of such patients toward pain reminds me of a symptom I have observed (with Stengel) after a parietal lesion of the brain, the gyrus supramarginalis, near the Wernicke center. These patients feel the pain but not in the right way. The condition is an agnosia for pain, and a quite similar lack of appreciation for pain can be observed in catatonic cases.

The last phenomena are both psychic and physiologic: the phenomena of sleep disturbances in the schizophrenic patient. There are sometimes short attacks of sleepiness; attacks in which thinking seems to stop belong to a similar group. The sleeping center is neutral from a psychophysical point of view; it reacts as well to physical agencies as to psychic ones to the wish to sleep. One has not the right to draw a borderline between psychic phenomena and physiologic phenomena. Phenomena of that kind are always at the same time both psychic and physiologic.

DR. ABRAHAM BRILL: Dr. Campbell's enumeration of the dilemmas in the diagnosis of schizophrenia reminded me unconsciously, I would say, of the story of two detectives who were shadowing a French Canadian crook. Suddenly the crook dodged around the corner and disappeared. As they were trying to locate him, they saw a restaurant with a sign in the window which read: "Ici on parle Français." Said the first detective, "Here is where we get him, being of French descent he surely would go into a French restaurant." "No" said the second detective, "knowing that we know he is a French Canadian he would purposely avoid any French-speaking place." And then it was found that the crook knew no French.

To be sure, Dr. Campbell is right in trying to tell you how difficult it is to diagnose a case of schizophrenia, but I am sure he does not find it nearly as

difficult to make the diagnosis as he was trying to make us believe it. I admit that if one wished to take all the academic implications of schizophrenia into consideration, it would be difficult to eliminate the element of manic depression, or a great many other characteristics frequently found in this malady, but after all we are interested only in finding those special features which are characteristic of schizophrenia. As Dr. Campbell himself said in quoting Dr. Bleuler, it is a question to what extent a person is a schizophrenic type, and to what extent a person is a manic type. Professor Bleuler formulated this concept very clearly, especially after he elaborated on Professor Kretschmer's concept of schizothymia and cyclothymia. For as you know, Professor Kretschmer was the first investigator to broaden the entities of dementia praecox and manic-depressive psychosis, by telling us that there are patients who represent mild examples of dementia praecox which he called schizothymic patients, and mild cases of manic-depressive psychosis, which he designated as cyclothymic patients, who also show corresponding somatic characteristics. Professor Bleuler extended the concepts further by saying that there are normal persons who show schizoid and cycloid, or, as he preferred to call the latter, syntonic reactions to life, and that it is really a question to what extent a person is a schizoid or a syntonic type.

Now, if one looks at the problem in this broader sense, it really makes very little difference whether a person is altogether a schizoid or syntonic type, or altogether a schizophrenic or manic type. We take into consideration the main elements or the main reaction of the picture and classify him accordingly. After all one must admit that the average person is made up of so many complex somatic and psychic elements that it would be strange indeed if we could not find almost every type of character reaction in every person. However, to make a diagnosis it is quite sufficient to consider the elements of the morbid picture that are most preponderating, for it is these characteristics that stamp the patient as a schizoid or syntonic type in everyday life and as a schizophrenic or manic type in the hospital.

I can hardly discuss Professor Bleuler's interesting address. As a former assistant and pupil of Professor Bleuler, I naturally agree with everything he told so lucidly and so instructively. Fearing that some of the auditors did not follow Professor Bleuler, I will repeat that like the other speakers Professor Bleuler stressed the fact that there is little difference between the soma and the psyche, that function is first to appear and then specialization, and that suggestibility is a question of affectivity. Professor Bleuler's remarks show clearly how difficult if not impossible it is to describe a case of dementia praecox in terms of words. For, first comes affectivity, emotions and feelings, and then come words to describe those feelings. Now, I do not believe there is a single word that could appropriately describe the affective state underlying the term "apathy" which Professor Campbell was trying so hard to describe to us. In fact, words can never describe feelings with any degree of adequacy and those of us who have worked in asylums and speak of apathy and similar terms, understand them only through our own affectivity. Through empathy we have learned to sense the patient's emotional states.

During my service in Burghölzli, Professor Bleuler once told us at an after-dinner discussion that even the most inaccessible schizophrenic patients, the catatonics, could be influenced by suggestion, and to prove his thesis he related the case of a chronic catatonic patient whom he wanted to move from one place to another but without the use of force or medication. He decided to try suggestion, and finally, after a number of hours of labor, succeeded in influencing the patient to follow his suggestions and obey all his requests; she dressed and followed him without any resistances.

Thereupon I became ambitious and tried the same experiment with a chronic catatonic patient selected for me, and after a few hours' work this patient, who had been inaccessible for many years, reacted normally for three days. This case taught me a lesson which I have never forgotten. It not only taught me some-

thing about the affectivity involved in suggestion, but I also obtained a new orientation about the morbid process of dementia praecox. It impressed on me the fact that even the most hopeless and disintegrated patients retain the capacity for integration, and that even profound mental disturbances can be influenced by mental factors.

Professor Bleuler has been praised by our chairman, Dr. Keschner, for having brought a number of new terms into psychiatry. My feeling about Professor Bleuler's great achievements is that he has given life to psychiatry. Up to the time of his advent, psychiatry was a sterile and monotonous science, even to those who were, paradoxic as it may seem, interested in it. I know that in spite of deep interest and hard work I gradually became more and more wearied of descriptive classifications, and I know that this feeling was shared by many other psychiatrists. When I came to the clinic of psychiatry at Zurich, I sensed an altogether different spirit for Professor Bleuler instigated his assistants to drop the old descriptive methods and look at psychiatry dynamically. The patient was not merely diagnosed and relegated into a niche of the chronic service; Professor Bleuler had the foresight to recognize the value of Freud's discoveries and to apply them to psychiatry. Unlike all other psychiatrists who rejected Freud's discoveries without examining them, Professor Bleuler knew how to evaluate and utilize them. The works of the Zurich school, as well as the present trend in psychiatry and psychopathology in general, amply show that Professor Bleuler gave life, and hence, interest to psychiatry. This, in my opinion, is Professor Bleuler's greatest accomplishment.

DR. LOUIS CASAMAJOR: It has been said that in a psychiatric discussion one can take one's choice; one can be either vague or wrong. I think we have seen something of that tonight, and with a vagueness which is really right, Dr. Campbell outlined many of those points wherein the concept of schizophrenia has varied from the older concept of dementia praecox and he has impressed us with the greater value of the newer concept.

Dr. Brill has already brought out one of the points I meant to make by calling attention to the fact that these terms of Professor Bleuler's have added a great deal of light to psychiatry, for it has given us a newer and a fresher point of view on the subject. Professor Bleuler has added more to his concept of schizophrenia; anyway he has taught me more, and in stressing the nature of affectivity, he brought out exactly the difficulty which I have in formulating my own ideas about schizophrenia.

Dr. Campbell spoke about the inadequency of such terms as apathy and negativism, and pointed out how frequently the use of those terms indicates the affective state of the observer much more than they do the affective state of the patient. Professor Bleuler stated one of the reasons for this in his remarks about the affective life. Professor Bleuler has laid more stress than anyone previously on the affective life in the psychoses; and therein lies our difficulty. The difficulty is that the affective life has no language. The words that we use are the words of the intellect, and the affective life itself has no words that we can use. When we endeavor to place purely affective things in the symbols of words, as Dr. Campbell pointed out, we often place our own affects in the fore much more than we do the affect we are trying to describe. Professor Bleuler's concept brings us away from those old hard, fixed categories, the manic-depressive psychosis and dementia praecox and the earlier ones they supplanted, primary and secondary dementias, paranoias, etc. These new concepts, being more vague, leave an opportunity for some glimpses of light to come in and leave us the hope that some day there may be a vocabulary of the affective life with which we will be able to translate affective states into words. The words Professor Bleuler has given us, "schizophrenia" and "syntonic" are useful words; they are valuable because they leave much unsaid. They are words that we can use today. They mean so much and they say so little that we can use them, and not be in danger of becoming enslaved by them.

NEW YORK NEUROLOGICAL SOCIETY AND THE NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY

Joint Meeting, Jan. 14, 1930

Chairman of the Section, MOSES KESCHNER, M.D., *in the Chair*

SPASMODIC LATERAL CONJUGATE DEVIATION OF THE EYES: A CLINICO-PATHOLOGIC STUDY. DR. CHARLES DAVISON and DR. S. PHILIP GOODHART.

This paper will appear in a later issue.

THE HISTOLOGY OF EXPERIMENTAL DIABETES INSIPIDUS. DR. FRANCIS JAMES WARNER (by invitation).

Lesions in the hypothalamus were produced experimentally in two dogs in which a complete hypophysectomy had previously been done. A complete hypophysectomy was performed on dog M32 by the transpalatosphenoidal route. A transient polyuria was noted after the hypophysectomy, which subsided. A puncture of the tuber cinereum was done about six weeks after the hypophysectomy. A transient polyuria developed and subsided in six days. A week later a second puncture of the tuber cinereum was performed and another transient polyuria developed. Seventeen months after the hypophysectomy, a double puncture of the hypothalamus was again performed with a subsequent transient polyuria which also subsided in a few days.

At death this animal showed a gain in weight of about 4 Kg., but no atrophy of the ovaries, vagina or uterus could be observed. No abnormal accumulation of fat in the panniculus nor in the mesentery or omentum could be observed. In the case of this dog there were two lesions in the hypothalamus, the lowest and largest being situated in the area of the preoptic and anterior hypothalamic nuclei. The smaller lesion was situated dorsal to the first lesion, but lying also in the anterior part of the hypothalamus. It appeared to involve the filiform nucleus as well as the dorsomedial nucleus.

Dog 283 was operated on on Nov. 27, 1928. Both the anterior and posterior lobes of the hypophysis were extirpated at that time and a puncture of the hypothalamus was done, the lesion being placed just anterior to the infundibular stalk. This dog developed polyuria twenty days after the operation, which lasted until the animal was killed on May 8, 1929. At autopsy, the changes noted were a marked atrophy of the testes as well as a marked increase in fat around the heart, peritoneum and along the anterior abdominal wall. The entire diencephalon of this dog was embedded in celloidin, sectioned serially and stained with the Nissl method. On microscopic examination, extirpation of the hypophysis was found to be complete. The lesion was situated anterior to the infundibular stalk, lying in the midline and involving the nucleus tuberis. Anteriorly, the lesion extended to the level of the anterior hypothalamic nucleus. With a high power lens, an enormous proliferation of gitter cells could be seen in the vicinity of the lesion. There seemed to be a marked reaction of the microglia in the area adjacent to the lesion.

Dog M32 survived eighteen months after a complete hypophysectomy, while dog 283 survived five and one-half months after a complete extirpation of the hypophysis. Both dogs showed no marked metabolic disturbances, in spite of the absence of the hypophysis. With regard to the polyuria, in dog 283 a permanent polyuria was produced by an injury to the hypothalamus at the time of the hypophysectomy, while in dog M32 it was impossible to produce a permanent polyuria. These observations are in accord with those of Leschke and Camus and Roussy who extirpated the hypophysis in dogs and observed no polyuria following the first operation, while a puncture of the hypothalamus some time

after a hypophysectomy induced a polyuria which lasted in many cases a considerable length of time. In our work we attempted to determine the exact anatomic localization in the hypothalamus of the lesions which produced a polyuria. In the case of dog 283 it was possible to localize the lesion, which seemed to involve the nucleus tuberis and the anterior hypothalamic nucleus. In the case of dog M32 it was not possible to determine the exact localization of the lesions in the hypothalamus owing to the fact that the diencephalon was not stained with the Nissl method. It seems from our observations, at least, that the anterior part of the hypothalamus is connected in some way with the production of these experimental polyurias. It is probable that the nucleus tuberis and the anterior hypothalamic nucleus are in some way connected with the metabolism of water.

DISCUSSION

DR. SMITH ELY JELLIFFE: The diencephalic nervous mechanisms, as Dr. Warner says, are very intricate; there is much dispute about the nuclei themselves and a great deal to be said about the fiber tracts. The only point which I will raise with the paper is the old point, that one cannot talk about centers any more. One may talk about stations in a complicated dynamic chain, but centers as such are of secondary consequence. In stations in a complicated chain, there may be those of more or less pre-eminence for greater or lesser regulation at different levels, and it may be that in the various hypothalamic nuclei some important links in the chain may be more or less inaccurately located. There can be no localization from purely histologic studies of the cells. The Nissl method does not enable one to check up the various parts of the chain, and until this type of work is supplemented by further fiber studies one cannot settle either the localization or the function or even the regulation until the whole nexus is brought together in some comprehensive bit of energetic machinery. In other words, to speak of the function of a nucleus is not good engineering, and it seems to me that if an electrician happened to be in the audience, he would try to point out that if this or that particular light went out, it might be due to a great many different types of lesions all along the chain, from the mechanism of central protection, which in my mind is the cosmos, to all types of peripheral manifestations. Whether or not there is a water-regulating mechanism in the diencephalon, I am disposed to believe that the "wisdom of the body" which has accumulated for millions of years in autonomic function structure mechanisms knows how to regulate the distribution of water. It is not all regulated centrally, however, under all types of stimulus. There may be some sort of a steering center, as von Economo prefers to call the sleep center, and there must be some such wisdom located in some central areas. Until more links in the chain of an extremely complicated mechanism are elucidated, I doubt if we can speak of localization in the sense that Dr. Warner was speaking.

DR. LEO M. DAVIDOFF: This is an important contribution. I wish the speaker could have given more data about his experiments. I should be interested in knowing how many animals were operated on, and in how many the results were uniform; and the explanation of how three punctures produced temporary polyuria in one animal and a single puncture produced permanent polyuria for five months in another.

DR. IRVING PARDEE: In the first dog the hypophysis was removed, the slides showing that the stalk of the pituitary was involved in the experimental lesion. Of course, under these circumstances, I cannot believe that he has produced only a lesion of the hypothalamus. Biedl, when here a few years ago, voiced the belief that the stalk of the hypophysis has a function, metabolic in nature. In the second dog the hypophysis also was removed, which again makes a lesion not only of the hypothalamus but also of the pituitary. Dr. Warner very easily, and I believe very carelessly, dismisses the pituitary as a nonfunctioning organ. I do not see that he has done anything to prove such a radical statement. Because his animal lived after hypophysectomy, therefore he says it has no function. If

a man lives after splenectomy — and he does — does it mean that the spleen has no function? As long ago as 1892, Pierre Marie described acromegaly and proved that the pituitary has a function. One cannot yet deny that the hypopituitary syndrome of obesity, acromikria and small genitalia has an origin except in the anterior lobe of the hypophysis. Take, for example, Dr. Warner's experiments; the first dog lived eighteen months, and following its death it was stated that there were marked changes in the gonads. There was also a considerable deposition of fat. What further hypopituitary signs do you need? I suppose he will say that the puncture of the hypothalamus produced these changes also. I think that the pituitary has a definite function, and to my mind no experimental work of this type can destroy this belief. The contention that it has a function is firmly rooted not only in a great mass of clinical presentations, but in the large amount of experimental work that has been done. Then again I will ask Dr. Warner to explain why pituitary extract controls diabetes insipidus. All are familiar with the fact that injections of pituitary extract will cut down the water output in cases of diabetes insipidus, whether they are produced by experimental lesions or are found in clinical conditions. I for one do not agree with the validity of Dr. Warner's experiments because he removed the pituitary before he did the experiments. I also believe that certainly not only his work, but also that of Leschke, Camus and Roussy, and Bailey and Bremer is open to criticism on the same ground; namely, that it is impossible to make an experimental lesion of the hypothalamus which does not also involve the pituitary gland. Conclusions therefore that diabetes insipidus, Froelich's syndrome and the like can be caused by lesions in the tuber cinereum and the hypothalamus are yet to be definitely proved.

DR. S. PHILIP GOODHART: After removal of the pituitary, did Dr. Warner examine any of the other endocrine glands. I am much interested in the function of the pituitary for the reason that in several patients it has seemed to me strange that, even when the symptoms of pituitary involvement had been very pronounced, at autopsy little or nothing was found on histologic examination. I have particularly in mind the case of a girl who showed all the symptoms of pituitary involvement. She grew very thin at first, then very stout, and showed skeletal, metabolic, visual field and disk changes. At autopsy, the body showed little, aside from a tuberculoma of the corpus mammillarium with absolute intactness of the pituitary gland. It seems to me that probably Dr. Jelliffe's suggestion of involvement of the organs rather indirectly may bring one to an interpretation of these changes. The hypothalamic area and adjacent structures remain problems.

DR. WARNER: I will first answer Dr. Jelliff's question as to whether the histologic methods used in this work were supplemented by studies of the fiber tract connections of the injured nuclei. The brain stems of two animals, having lesions in the anterior part of the hypothalamus, were sectioned serially in the sagittal plane stained with the Marchi method and we found no traces of fiber tract degeneration. This is to be expected, because these are visceral fibers. There is no method by which one can trace the degeneration of unmyelinated fibers, and if any one can discover one I shall be very glad to use that method. I am not presenting this work as in any way conclusive, but it is suggestive of what may be found by applying the method in an attempt to determine the function of the hypothalamic nuclei, which comparative anatomists dismiss as being merely secondary olfactory centers. As far as localization of function in the hypothalamus is concerned, other workers, including Ott and Isenschmidt and Krehl, have also found thermic centers in the mammalian hypothalamus. One does know that lesions in certain hypothalamic nuclei produce marked metabolic disturbances and the only conclusion one can draw is that there must be some connection between the lesion of the hypothalamic nuclei and the disturbances of function in these animals.

In reply to Dr. Davidoff as to how many animals were operated on, I may say that it is a very difficult area to approach and there is a considerable mortality connected with experimental work of this nature. It is difficult to secure an animal in which a complete hypophysectomy has been performed and a lesion placed

in the hypothalamus of the same animal. I am, therefore, presenting these animals because they represent two of the best animals on which we have complete metabolic data. Why one gets a permanent polyuria in one animal and a temporary polyuria in another is not known. Bailey and Bremer and also Curtis reported the same observation, that is, regardless of the location of the puncture, in some animals they would get a permanent and in others a temporary polyuria. In this connection I may note that Bailey places most of his punctures behind the infundibular stalk, while some of his animals had punctures of the hypothalamus anterior to the infundibular stalk.

In reply to Dr. Pardee concerning the remnant of the stalk of the pituitary gland in dog 283, I may say that it was about as complete an excision of the pituitary body as one can possibly secure surgically. As for the function of the pituitary body, the work of Leschke, and more recently of Roussy, has shown that an animal can live without a pituitary body for a considerable length of time without any marked metabolic disturbances. A puncture of the hypothalamus in these hypophysectomized animals produced a polyuria and in many cases the development of the adiposogenital syndrome. However, in my work I was concerned mainly with the problem of localizing, by means of serial sections, the exact nuclei which are involved in animals developing an experimental polyuria. My observations as to the function of the pituitary body in the adult animal were incidental to my work on experimental polyurias. However, the fact that the two animals studied lived as long as these did, and developed no marked metabolic disturbances, is suggestive that the function of the pituitary body in the adult animal, at least, is problematic.

In reply to Dr. Goodhart's question as to whether any changes were noted in the ductless glands of these animals, I may say that a complete autopsy was performed on all the organs of these animals. There was no evidence of hyperplasia of the thyroid or of any other endocrine glands in either of the dogs studied.

THE LEGEND OF THE PREVENTION OF MENTAL DISEASE. DR. ISRAEL SPAUER WECHSLER.

The belief has become all too prevalent that a loud voice gives importance to ideas, that error sufficiently often repeated ultimately becomes truth, and that folly spread over wide regions somehow turns into wisdom. Such is the nature of propaganda. On more sober consideration, it becomes evident that the trumpet has no inherent virtues, that the radio imparts no fundamental attributes to ideas, that the printed word is endowed with no special magic, and that neither reiteration nor dissemination is capable of giving substance to a thought which is devoid of it. Nevertheless, the popular mirage exists, and there is sufficient reason to suspect that science too, at least popular medical science, has been fascinated and has succumbed.

Lest the title of this paper convey a wrong impression, let me make the trite observation that every physician, myself included, is firmly convinced that the future of medicine lies in scientific prevention. But the loud fanfare about prevention has created the curious opinion that we are dealing with a modern concept or a modern discovery. As a matter of fact, even the savage evolved what for him were definite methods of prevention. That he made use of prayer, taboos, sacrifice, incantation and various sorceries does not alter the fact that by means of white magic he sought to prevent disease and actually believed that he did. He may even have attained some measure of success in the field of mental disease, the old wizard comparing not unfavorably with the modern cultist and quack. In some instances the efficacy of the more ancient measures was neither greater nor less than many heralded so confidently in our own day.

The fundamental difference in the methods of prevention employed then and in some of the modern methods lies in the scientific recognition of causes. It is by this standard, by the knowledge of specific causes, of the mode of transmission, of the manner of evolution of disease, that we must test our ability to prevent, and

it is by these criteria that I would test the claim so glibly and so confidently made as to the prevention of nervous and mental disease.

Before proceeding, it may not be irrelevant to stress another fact, namely, that treatment, cure and prevention are entirely different matters. There are diseases which we know how to prevent but not to cure; there are others which can be cured but not prevented; there are a few which can be prevented as well as cured; there are many which can neither be prevented nor cured; and finally, there is that large group which can be treated but not cured.

Sad as it is to make the confession, the fact remains that despite the accumulation of knowledge, the ultimate cause or causes of nervous and mental disease are unknown. There are a great many theories but few facts. Their very profusion is not only in inverse ratio to our knowledge, but is an actual confession of ignorance or merely a verbal cloak for it. Nor do we know very much concerning the rôle of heredity in the causation of illness and mental diseases, although admittedly it is a very potent factor despite the loud claim of some that the superstitution ought to be uprooted. Nevertheless, we see medical men issue in print or proclaim from the rostrum statements which cannot for a moment withstand the test of scientific investigation; we hear men speak with assurance that they can prevent social and individual ills whose ultimate cause is utterly unknown. They arouse false interest and vain hopes and enthrone themselves as popular idols in the guise of honest servants of an accurate science.

Enthusiastic mental hygienists tell us (and I am quoting from actual statements) that they are concerned with the prevention of mental deficiency, criminality, the psychoneuroses, the psychoses, antisocial traits, family unhappiness, divorce, prostitution, alcoholism, sexual perversion, epilepsy and other such simple matters. All one can do is to gape and wonder, or perhaps ask a few questions. How much is actually known of the causes of mental deficiency? Or, considering the question of criminality, admittedly it depends on ill defined but undoubted hereditary factors, on mental deficiency, on diverse psychic and emotional states, on complex familial situations, on educational standards, on the social structure, on moral or ethical principles, on intricate matters of law and statutes, on politics, on concepts of justice, on religious views and on a host of interrelated problems which will require generations to study, let alone to solve.

Despite these elementary truths, endocrinologists do not hesitate to state that mental deficiency and criminality can be prevented by the simple process of juggling a few extracts of the glands of internal secretion. It is not an exaggeration to say that endocrinology is but a medical fledgling which has hardly emerged from the womb of experimental medicine, and that it can be applied clinically in only a few limited conditions with any degree of accuracy or success. Such is the uncertain, if not confused, domain of clinical endocrinology which one begins to suspect as a recrudescence of alchemy. And yet the claim is made that glands will cure and prevent disease and almost perform miracles. No less modest are the claims of some of the mental hygienists.

Without indulging in destructive criticism, one may ask in all sincerity, what great advance has been made in psychiatry in the past twenty-five years, what new scientific discoveries have been made to warrant the sweeping generalizations and confident promises which are given to the effect that mental diseases can be prevented? What do we know of the ultimate cause of dementia praecox? What do we know of the causes of organic psychoses that justifies this boastfulness? What specific disease of the nervous system can we actually prevent, and have the psychoses diminished actually or relatively within recent years?

One enthusiastic exponent uttered a repetitious formula, derived from a purely hypothetical notion, spun out of fantasy, without relation to clinical experience or laboratory facts. He explains, claims to cure and prevent disease, and promises all sorts of panaceas with — words. It is all so very simple. Inferiority accounts for neuroses, psychoses, perversions, criminality, greed, love, ambition, cowardice, bravery, alcoholism, marital infelicity, divorce, general morality, economic distress,

wars, panics, revolutions, religion, history, philosophy; in short, every conceivable social and individual phenomenon. A notion which does violence to all accumulated knowledge (which, if dignified with critical analysis, is found to approximate the novel and evolution-defying concept that out of a biologic defect comes a biologic purpose) has been popularly accepted because it is easy, so simple that it requires no effort to think for full comprehension. It is positively axiomatic, and rests on the irrefutable evidence of — I say so.

It is no discredit to psychiatry to acknowledge that it has barely emerged from the descriptive stage, that the practice of it is still considerably more of an art than a science, that it is perhaps less rooted in anatomy, physiology, pathology and all the laboratory sciences than most other branches of medicine. But instead of due regard for fact there is almost a groveling worship of formulas, an infatuation with fetishes of words. New shibboleths are coined, and the illusion is created that new facts have been discovered.

One must also confess that what passes for social psychiatry is barely out of the religious or supernatural stage and quite deeply immersed in the speculative or metaphysical. Sociology has not yet discarded medieval concepts and scholasticism is not quite dead; nor is human thought altogether freed from the baneful influence which magic, religion and metaphysics have exerted on the development of the positive sciences. We still speak of ultimate purposes and vital ends, and call them by the high sounding name of teleology without recognizing that they are religious and not scientific concepts.

It is not my object to speak against the legitimate and well established efforts of prevention. (I am purposely leaving out the important question of eugenics and its relation to mental deficiency and mental disease.) I would not decry the honest but few achievements of mental hygienists in the study of mental problems, in the invaluable surveys of institutions for the insane and of prisons, in the establishment of child guidance and court clinics, in the humanizing efforts in the treatment of patients with mental diseases, in the focusing of attention on modern aspects of social psychiatry, in the impetus it has given to the study of mental diseases inside and out of medical schools. Nor would I overlook the fact that not only has the study of child psychology, based on sound psychoanalytic principles, resulted in valuable insight and moderate contributions, but the dissemination of knowledge among physicians, parents and educators holds considerable promise for the ultimate understanding of many of the problems of the neuroses.

Without, therefore, impugning legitimate efforts and valuable results, I would still call attention to the sensational claims made in behalf of a budding science, and venture the opinion that much of the mental hygiene stock foisted on the lay as well as the medical public has been watered beyond reason and that it stands in urgent need of deflation. I do not know of any other branches of science, not even excepting philosophy, which relies so much on words, so much on arm chair thinking, and so little on accurate knowledge. I do not know in what other fields speakers and writers say so much about so little; talk for hours on subjects which can be explained in a few minutes; issue volumes on what can be said in paragraphs, and succeed in giving the momentary impression that they have said something when in reality they said nothing at all. I am not aware of any other branch of learning in which the future and painfully slow progress has been discounted in advance to such a degree and in so great a hurry.

For example, we have before us a shameful spectacle in the propaganda of cancer prevention. It is admitted, without exception, that we have no knowledge of the real cause of cancer and that we grope blindly in all our methods of treatment. We employ the knife, radium and the x-rays because we obtain some temporary and few permanent results with them, and because as yet, we have not found a better method of treatment. We are quite ignorant of the reason why in two persons having apparently similar types of cancer in exactly or approximately the same parts of the body, one profits from treatment and the other does not, but of true prevention or even control we know nothing. Still that does not deter propagandists, or medical propagandists, from literally frightening people,

even school children, out of their wits and making vain promises and arousing false hopes which are so often followed by a tragic awakening. Again I venture the opinion that, whatever the motive, science is not served thereby. But to revert to our own topic, one shudders to contemplate that since we already have fire-prevention week and cancer-week, we may soon read of criminal-week, dementia praecox-week, imbecile-week, and what-not other week.

In conclusion, I realize that much of what I have said, and perhaps the way in which it was said, will arouse great resistance no less than vehement criticism. My remarks have been animated solely by a desire to serve what I conceive to be scientific truth. It is my opinion that progress is hindered rather than advanced by exaggerated claims, spectacular methods, and sham research. Knowing how abysmal ignorance is, I feel that one should be extremely modest before knowledge. One can pursue truth to greater advantage in silent retreats than in the noisy market place. Useful discoveries can be well disseminated without the aid of trumpets. The honest scientist does not announce his investigation before verifying his results. To be a true scientist one must possess absolute intellectual integrity. The great scholar is not only modest in his statements and constantly beset by doubts, but can well afford the luxury of saying, "I do not know." Only the fool and the ignoramus deludes himself into thinking that he has taken out a patent on wisdom and a copyright on knowledge. All of us still are children before the great unknown, and it ill befits us to play the rôle of high priests. The day of omniscience, whether human or divine, is gone forever. To insure progress we must honestly and courageously seek to avoid the dangerous paths of folly as well as the lurking pitfalls of error.

DISCUSSION

DR. SMITH ELY JELLIFFE: It appears to me that Dr. Wechsler's conception of prophylaxis is, prophylaxis without prophylaxis. It is very difficult to discuss this essay. One of the things that upsets Dr. Wechsler, as it upsets most of us, is the fantasy or autistic thinking which is so prevalent in the community. He spoke of it in terms of white magic and black magic, various types of superstition and the spread of misinformation of all kinds, and we are all thoroughly in accord with the case which Dr. Wechsler has made out. Nietzsche's phrase, the "transvaluation of values," was pertinent as an opening suggestion, but I did not learn just where Dr. Wechsler arrived with these transvaluated values. I am altogether in sympathy with much that he said, but twenty-five years ago I was much more in sympathy with everything he said. Maybe when Dr. Wechsler is older and has reached my gray-haired stage, he will not be quite so out of patience with the community as his paper would indicate. Many a time I have been out of patience with the community, and have been more or less the champion of much the same kind of phraseology, although perhaps not so well expressed as by Dr. Wechsler. There are, however, one or two things which might be thought of, taking perhaps a side-track from what Dr. Wechsler emphasizes. It is more or less absurd to talk about the prophylaxis of "mental diseases." There is no such entity as "mental diseases." This term is an abstraction, and a very poor abstraction at that. No one should try to prophylact something that does not exist. A man may acquire syphilis and then develop dementia paralytica. If one is trying to prevent the individual mental disease called dementia paralytica the concrete problem arises: How can one stop the man from getting syphilis? That is but a sample illustrative of the concrete type of question which should come under consideration in a discussion of this kind. Otherwise we have, as Dr. Wechsler said in reference to other people, nothing but words, words, words, and my apology for this discussion is that I may not join with him in a good many words, words, words.

DR. ABRAHAM A. BRILL: I am in full sympathy with what Dr. Wechsler said, but I am not as pessimistic as he is. The accusations which he hurled at some of the groups dealing with mental diseases are justified, to some extent, but I believe that many of the evils mentioned by Dr. Wechsler cannot be prevented.

My experience as a pioneer in psychoanalysis taught me that it is not possible to do anything without attracting a great many so-called friends whose enthusiasm for the cause often mounts to dangerous heights. Dr. Wechsler was somewhat guarded in mentioning names, but I presume that he referred to endocrinologists and mental hygienists. I am pleased that he enumerated the very excellent results brought about by the mental hygiene movement in the various activities of mental diseases. I believe that the leaders of the mental hygiene movement know that some of the people who carry on propaganda in the name of mental hygiene indulge in the absurd cant of the patent medicine vender; as Dr. Wechsler puts it, they are just words and words. One hears a lot of nonsense about panaceas, and, with Dr. Wechsler, I feel that they do some harm, but nothing can be done for any cause without running the risk of doing some damage. In the long run, however, the scientific members of the group know how to control the situation. Dr. Weschsler tells us that, strictly speaking, one has no right to talk of prophylaxis in any branch of medicine and surely not in nervous and mental diseases. Here too, I do not quite share his pessimism; I feel that it is better to think and talk about it than have no hope at all. It naturally pleased me to hear Dr. Wechsler pay tribute to psychoanalysis, as taught by Freud, but Freud started us on the road to prophylaxis in nervous and mental diseases.

Freud has always been very modest in his assertions about treatment and prophylaxis; he simply keeps on showing facts and urges us to investigate them. I have no more patience than Dr. Wechsler with those who splatter high sounding words and magic formulae, such as the omnipotent cure-all, the "inferiority complex." Here again Dr. Wechsler was careful not to mention names. We know, of course, that he was referring to Dr. Adler's followers, to whom everything is so simple. In order to be straight one must first be a cripple; in order to talk well one has to begin as a stammerer and stutterer, etc., and yet this system gained such popularity that one of our greatest medical schools saw fit to flirt with it. This system is popular and acceptable to the crowd because it is so simple; all one has to do is to put together a piece of superiority with a piece of inferiority, and one gets a mediocre person. Yes, it is just words and expressions.

In a recent letter to me, Professor Freud, speaking of the popularity of psychoanalysis in this country, expressed the view that this is not due to broad-mindedness or better understanding of the subject, but to a tendency to take things lightly. If the populace really understood there would be more serious discussion and perhaps less of the ready acceptance and enthusiasm. Notwithstanding this carelessness, I do not believe that there is as much harm done as Dr. Wechsler believes. In the long run people come to their senses; they are not fooled all the time. Thoughtful people soon find out that neither mental hygiene nor glands nor any other form of therapy is a cure-all.

In brief, I think that Dr. Wechsler courageously called attention to an important situation, which all of us realize and deplore. I am sure that Dr. Wechsler knows that no member of this society is guilty of uttering the extravagant claims he described. It is the camp followers, the good ladies and gentlemen who, ill prepared to understand the serious imports of mental hygiene or endocrinology, become enthusiastic for the cause and gallop on where learned physicians move carefully. As far as I know, the mental hygienists and endocrinologists who are occupying themselves with the subject scientifically, are decrying these extravagances just as much as Dr. Wechsler.

Dr. J. Ramsay Hunt: When Dr. Wechsler asked me to discuss his paper on "The Legend of the Prevention of Nervous and Mental Disease," I was like some of the rest of you, rather curious to know just what he had in mind, although I thought I knew judging from certain criticisms which I had felt myself I am sure we all agree with him as to certain exaggerations which have appeared in the course of the mental hygiene movement in this country, and the exploitation newspaper notoriety and the lay interest in all matters pertaining to psychology of the modern advances in abnormal psychology. This is no doubt due largely to newspaper notoriety and the lay interest in all matters pertaining to psychology

and especially to its abnormal phases. Some medical authorities in "talking down" to lay audiences have made statements that are highly colored and which they would not have made to a group of medical men. I think, notwithstanding these exaggerations, that the mental hygiene movement and the lay interest in psychology are most important and have done and are doing a great deal of good, both in educating the community and in advancing the higher aims of psychiatry. The words, mental hygiene, are something that the public can readily understand and sympathize with; they encounter no resistance and place mental health on the same level with physical health. If it is important that we have a physical hygiene, which no one can deny, the thing we call mental hygiene is no less important. If one is advantageous to the human race, the other will also confer great benefits. All know that certain phases of physical hygiene from time to time are carried to an extreme degree by some people and some organizations, and the same is true no doubt of mental hygiene, but that should not detract from its great usefulness. This whole movement received a tremendous impetus during and after the war. Then psychiatrists, neurologists and psychologists became, as it were, socially conscious, in a way that they had not been before, and perhaps it is not surprising that in their earlier efforts with the public there has been some overemphasis, exaggeration and even romance, inherent in the propaganda methods that have been employed. But think of the great good that has come out of mental hygiene and all this public interest in the problems of psychopathology. Consider the guidance clinics for children, the guidance departments in schools and universities and the psychopathic and psychiatric institutions that have been founded, and are now doing splendid practical work. The various surveys in the psychiatric field, the fundamental researches that have been made in certain social aspects of psychiatry, the fellowships in psychiatry and the efforts to produce young psychiatrists to fill important places in the future, are all closely associated with the so-called mental hygiene movement. Men of wealth can now be approached confidently, for here is something that they can understand, something that is popular and practical; money is flowing in for these purposes, and will flow in more and more.

DR. FRANKWOOD WILLIAMS: I think that Dr. Wechsler has done a very healthy thing. There are many people, probably most of the people here, who agree in general with his points of view, or at least feel some degree of dissatisfaction with the situation that exists. The situation should, therefore, be discussed. Dr. Wechsler expressed the fear that there might arise a great opposition to his ideas, or that feeling might be engendered. I think that the last group in which there would be any feeling at all would be the mental hygiene group, particularly the organized mental hygiene group, the National Committee for Mental Hygiene, because I think that all associated with the National Committee will very thoroughly agree with what Dr. Wechsler has said, and what has been emphasized by those who have discussed his paper. We regret, as much as does Dr. Wechsler, these exaggerations and overstatements which are made from time to time. My difficulty with his paper would not be with these details, with which I agree, but rather with his concept in general. Dr. Wechsler has complained of armchair psychiatrists. It looks as if a neurologist had sat down in an armchair and written a paper. In other words, while Dr. Wechsler has some knowledge, he apparently has little understanding of psychiatric material and of what is actually going on in the psychiatric field and child guidance clinics. What is most surprising is the concept which Dr. Wechsler has carried over, an old concept which is now largely given up in the field of psychiatry, and which I am sure that Dr. Wechsler himself does not hold in regard to other physical matters, and that is, these bothersome notions of categories, long discarded by physicians in psychiatric clinics because untenable, but still bothersome to people like Dr. Wechsler. Dr. Wechsler in his medical cognizance, I am sure, is past the point of thinking in terms of limited, circumscribed diagnoses, a method of thinking better adapted to the work of plumbers and automobile mechanics than to physicians. He realizes that the

physician must deal with the person as a whole rather than with any particular part. I am sure Dr. Wechsler is past the stage of thinking in terms of circumscribed, unrelated heart disease or kidney disease or any other organ disease. He thinks at least in terms of systems and of relations, even though for the moment his attention may be directed at a particular part. In other departments of medicine he has stopped thinking in these small categories. When it comes to mental disease, however, he immediately goes back to the old method of thinking in terms of definite, circumscribed disease entities. Here is a disease, a mental disease; and this mental disease must have a certain etiology; if we discover this etiology, then we can do something in regard to the treatment, cure and prevention of this disease. If it worked out this way it would all be very nice. Psychiatrists once thought it would; Dr. Wechsler insists that it should, in fact shall, if he is to be satisfied. But psychiatrists who have made a somewhat closer examination of the clinical material have a different view. We do, of course, speak of mental diseases and names are given to different clinical groups, but I think that Dr. Wechsler will find that most modern psychiatrists do not deceive themselves in regard to what they are dealing with. These names do not necessarily represent actual diseases. We are getting further and further from any such concept. It is a question whether some of these conditions can be called diseases in the ordinary sense. In the sense in which Dr. Wechsler uses them, I do not think the modern psychiatrist thinks of them at all. So he sets up an untrue situation when he says we have this disease and know nothing about its etiology.

He carries over the same concept into his social thinking. We can all smile with him at the gentleman from abroad, who with his inferiority complex material is ready to cure all the things which Dr. Wechsler has mentioned. We will smile with him at that; but on the other hand, Dr. Wechsler makes almost as naive a mistake by trying to think of social problems as existing in neat little compartments. He names delinquency, prostitution (I do not know whether he included dependency, but he might have done so), the neuroses and psychoses, and so forth, as rather separate, different things with presumably each a cause as yet undiscovered. These problems are, therefore, to be dealt with in these small, sharply bound categories. I think that those of us who have had some experience not from the armchair, in dealing clinically with these various types of situations, recognize a situation not so neat, but complex, interknit and with many factors entering in. Sociologists, medical and nonmedical, have given up thinking in such limited terms, except for purely practical purposes, and so far as the mental factors go I am inclined to think that it is pretty generally agreed that we have to do not so much with a series of sharply defined things as with a variety of manifestations (symptoms) of certain underlying conditions. At least we cannot think of these problems any more in terms of sharply defined categories. Some people would search in each disease for some specific cause, and then for a preventive. The thing is in one sense much more complicated than that, and in another probably simpler. At least it cannot be dealt with in that way.

Although I have not yet reached Dr. Jelliffe's age, I agree with him and with the others in what they have said in regard to the comparative harmlessness of popular foolish talk. I was younger once, and these things disturbed me more then, but my experience in the past twenty years in a more or less community type of work has aged me more rapidly perhaps than Dr. Jelliffe, so that even at my age I can have somewhat of his philosophic view on the matter. And I am not so alarmed by the Coués or by the Greenwich Village type of psychoanalysis which was so popular for a time. Such things are not very disturbing. One is more disturbed by what would seem to be Dr. Wechsler's social, or perhaps, medicosocial point of view. What I would call attention to is this, here again Dr. Wechsler carries over this old idea of categories in his thinking on medical-social relationships. He has not actually said so here, but it is implied in his paper, that he thinks in terms of doctors who deal with disease, and perhaps teachers who deal with education, of lawyers who deal with the courts, and of penologists who deal with prisons, etc. The physician's work is the study of

disease, and after he has found the cause of disease and the methods of prevention, then he is to announce this to the world, and the world is to accept this and put it to work at once and cure itself of this particular ill. Until the physician speaks, everyone else must keep silent. I think if we are going to handle medical problems in a social way, we have got to give up this particular concept, which is again nothing more than an old compartment concept, because life is not quite like that; the community is not organized quite that way but like an individual body. So does the community act as a whole and not alone by special organs.

I am sure Dr. Wechsler would resent the assumption of any person, and I know I would — although his grandfathers did not and mine did not — resent the assumption of any person who thought he had any personal responsibility to save his soul. I won't admit that it is anybody's duty to save my soul, or anybody's responsibility, nor any group's responsibility. I hold my own right to consider matters which concern my soul and to come to my own conclusions in regard to that question so far as it pertains to myself. I reserve the right even to be wrong. I am a physician, and I am not any too well informed perhaps about matters in this field, I am willing, therefore, to listen to those persons in the community who do devote themselves to this study and who may have something to tell me that would be important to me in regard to spiritual matters. I look to them as leaders in their field, but I reserve my entire right to accept or decline anything they say. I recognize no particular authority in that respect, and I know too that I must do this, because in this field as in all others there are quacks and charlatans, and there are those who although sincere would deceive, being deceived themselves, so I must reserve this right to myself. This is equally true in regard to any other social group, medicine included.

Dr. Wechsler cannot say, and I cannot say, nor can any other physician here say that he is personally responsible for saving the life of any other person, nor can we insist that any person shall permit us to allocate such responsibility to ourselves. That is presumptuous and is to be resented as much when it comes from medical men as from spiritual guides. It is the other man's business, after all. We do know more about it than he does, because it is our business to know. He has a right to look to us for information, but he has a right to reserve to himself any decisions in regard to the information which we may choose to give him. We are not responsible for his health. When it comes to his ill health endangering the health of others, there is some reason for stepping in, but so far as his own health goes he must retain responsibility and the right to decide for himself. He has, further, the right to such information as we have, and not only the information that we have completely. He does not necessarily have to wait and there is no essential reason why he should wait until Dr. Wechsler and the eugenists and the endocrinologists have come to the end of the story. Most of us will be dead by the time the end of the story is reached. He has the right to such partial information as we have. He has the right even to the theories which we have. Even though he had no such rights it would be to the interest of the community and to the interest of the medical profession itself that he have as free access to information as possible. He can be fooled if he likes. He may even have to learn that he can be fooled. Or, he may not be fooled, and not nearly so many are fooled as is sometimes thought. Medical men are not the only intelligent persons in a community; medical men are not the only ones who are disgusted by foolish talk. Intelligent people can spot foolishness just about as quickly as we do. If Meyer, Kirby, Jelliffe, Hunt and Brill, being eminent authorities, should tomorrow announce in *The Times*, that by standing on one's head for five minutes every day one could assure absolute mental integrity for the rest of one's life, they would not of course fool this organization, and neither would they fool many intelligent people in New York. There is no question that they could organize a group of their own, and they might raise some money and form a committee, but it would not injure greatly the community life of New York nor the eventual arrival at truth in these matters. Intelligent people are capable of understanding medical ideas, facts and procedures. It is well that they should

discuss them, and that such information as we have, complete when complete, partial when partial, and so announced, theory when theory, and so announced, should be available for them. This makes for intelligence in medical matters on the part of the community. Greenwich Village psychoanalysis, or inferiority complex panaceas, or Coué's publicity may be far below the level one would desire, but there are people living and thinking at that level and its promulgation need not disturb one. First, it does no very great damage; these things are little more than a seven day wonder; in the second place, they do a certain amount of good in that they draw attention to and make people familiar with certain fields of work in which they should have an interest. They may find they have wasted five dollars in purchasing somebody's foolish book, but nevertheless the book is likely to have interested them in its subject; they have not been damaged by it, and they will likely read another. The general idea becomes familiar to them; the topic finds a place in their minds. Eventually, they may be ready for some real information on the subject. If we are to progress in this field, the progress will come, not alone from what we do as a group but from what we are permitted to do by the community. There has been a lot of twaddle in the public press, for example, in regard to crime, but even though it was twaddle it has done more good than harm because it has kept the matter to the fore. Psychiatrists have for some time realized that there is an important relationship between mental condition and delinquency. Even though this idea gets down to the level of the *Daily Mirror,* it is extended through the community, it is kept to the fore, and it eventually will work around to the time when the community does wish to know, after all, what is in the proposition and be willing to consider the matter seriously. A few years ago you saw what sharp reaction there was against any reference to the possible mental condition of a delinquent. Now responsible men come forward with programs that far exceed what was then angrily rejected, and there is no sharp reaction. The American Bar Association at its 1929 meeting, passed a resolution that could not have been passed at the American Psychiatric Association ten years ago. We would predict this. Not long ago, it became necessary for a very honorable and very dignified organization in our own field to raise a large sum of money for its work. The public had not been prepared as I have indicated, and what was the result? That organization, dignified and sound as it is, had to go out and beat drums until it made a very unpleasant noise, and had to issue publicity that would make almost all the men in the organization cringe. It was apparently necessary to do this in order to raise the fund needed. That would not have been necessary if the things I have been speaking about had obtained over a longer period of years. As people become better prepared, in the sense of a better understanding and therefore a greater interest in these things, when large sums are needed to do important things, it will not be necessary to prostitute ourselves or to be humiliated by the rot-fund raisers, put out in order to obtain the amount required.

DR. WECHSLER: I am very grateful to the gentlemen who have come here to discuss the paper, and particularly to Dr. Williams.

First, I am not at all pessimistic; in fact, I am a little too enthusiastic. What I particularly liked in the discussion is that everyone agrees with me, and every speaker promptly neutralized what he assented to. I want particularly to emphasize that I have come here to talk to neurologists and psychiatrists, to physicians and not laymen; not to people who have axes to grind for purposes of their own, and I tried to lay down certain medical principles which I think hold good. I do not believe that all the accumulation of knowledge of centuries can be discarded by the formulation of a few theoretical principles or by the catering to popular tastes. Nor have I any quarrel with words; it is the misuse of words that I quarrel with. It is the bad use to which words are put that is dangerous.

I, too, have practiced in neurology and psychiatry, two decades or more, and I am not unfamiliar with psychoanalysis; it is just because of my long association with psychiatry that I was led to say the things I have said.

Dr. Williams has made so many general statements that it would take hours to discuss them. I do not know what he means by the individual as a whole, though I hear the words used with emphasis. The concept is tremendous, and most be reduced to smaller factual terms. Science advances by attacking small problems first, building one fact on top of another, by using the inductive method. Dr. Williams would use the deductive method. He first sets up a large structure, and then derives certain vague truths from it.

I am familiar, of course, with the philosophic conception of categories, but I do not know what Dr. Williams means by them. I tried to emphasize that there is no such thing as a simple delinquency, or insanity or criminality. It is just because they are complex that I quarrel with those who attempt to deal with them in such a glib and easy fashion. I see in that a real danger to neurology and psychiatry and mental hygiene, the danger being all the greater since it is based on unscientific method. Incidentally, I hold that every man has an inalienable right to go to heavon or hell in his own way. The fundamental question is, are we using scientific methods? Is all the knowledge accumulated until now worth while or not? If the methods are faulty, then let us, so far as psychiatry is concerned, give up the study of the anatomy of the brain, and chemistry and biology, and if that is the thing to do, it seems to me all departments of neurology and psychiatry ought to shut their doors, so that we may speak of large concepts, of individuals as a whole, etc. I do not agree that twaddle does no harm; the only thing that twaddle can do is to beget more twaddle. Nor would I be deluded by those dubious methods which are called practical. I must insist that making extravagant claims is incorrect, that indulging in armchair speculation is a mistake. We all want to foster mental hygiene, but we can do it better by building honestly on sound scientific foundations. Whether what I have said tonight will leave its impression or not, I do not know. Probably it will not.

Book Reviews

ANATOMICAL STUDIES OF THE CENTRAL NERVOUS SYSTEM OF DOGS WITHOUT FOREBRAIN OR CEREBELLUM. By L. RAYMOND MORRISON. Pp. 108. Haarlem: de Erven F. Bohn, 1929.

Young dogs, operated on by Rademaker in the laboratory of Magnus, were kept alive long enough to permit secondary degeneration to occur according to the principle of von Gudden and von Monakow. The operations were performed in stages, and in each dog the cerebellum and both cerebral hemispheres were removed; in addition, in one dog the corpus striatum was removed on one side, while in the other dog the corpus striatum was completely removed on one side and partially removed on the other side. These are the first dogs described in the literature that have survived for any length of time such extensive extirpations; the dog of Pavlov, in which the cerebellum was intact, and the dogs of Goltz, Munk and Rothmann, in which the cerebellum or parts of the cerebrum remained, are compared with them. In these two dogs, "Robbie" and "Duimelot," the clinical symptoms were different because of the differences in the order of extirpation; in "Duimelot" the cerebellum having been removed first, and in "Robbie" last, and so on. Physiologic notes are given on the conditioned reflexes, supporting tonus, etc.

The differences in the severity of the degeneration, occasioned by the differences in the sequence of the extirpations, serve as a good control between one dog and the other; so the degenerations in both animals are capable of being reviewed together.

Because of the removal of the cerebellum, best seen in the dog that lived one hundred and thirty-six days without it, there were cell as well as fiber degenerations. All cells in the nuclei ventralis pontis disappeared. Cells in the columns of Clarke degenerated or fell out, especially in the lower cervical level. In the nuclei olivares inferiores practically all cells disappeared and the occasional isolated remaining ones degenerated. All cells in the nuclei funiculi lateralis vanished, and the nucleus proprius corporis restiformis was not to be seen. The nuclei gracilis and cuneatus showed but few cells.

The fiber degenerations due to the cerebellar extirpations were also pronounced. The tractus spinocerebellaris dorsalis was still to be seen in the area ovalis corporis restiformis, probably because of its great length. But around it were the degenerated olivocerebellar tracts, the tract from the nucleus proprius corporis restiformis to the cerebellum, as well as the fibers from the nucleus funiculi lateralis to the cerebellum. All were greatly degenerated. There were no transverse pontile fibers to be seen. Outside the area ovalis could be found the fibers of the corpus juxta-restiforme, including the cerebellar connections, but it was much atrophied. The tractus uncinatus of Russell was invisible. The brachium conjunctivum had almost entirely disappeared. Coming from the nucleus gracilis and nucleus cuneatus, the fibrae arcuatae externae, intermediae and internae were practically completely degenerated. The lemniscus medialis was degenerated entirely. The fibers of the pons were among the most conspicuous of all degenerations, the fibrae transversae superficiales being slightly better retained than the others.

Owing to the removal of the cerebrum there were extensive degenerations of cells and fibers. All the cells of the thalamus, except those of the medioventral nucleus, disappeared, as well as those of the lateral nucleus of the corpus mammillare. The dorsoreticular part of the nucleus ruber was free from cells. In the corpora quadrigemina antica and postica and also in the corpora geniculata laterale and mediale there were great losses of cells.

Fiber degenerations could be traced from the capsula interna through the pes pedunculi and decussatio pyramidium into the contralateral tractus pyramidalis. There was a loss of fibers from the thalamus to the cortex, and the fornix was conspicuously degenerated.

Photodrawings from corresponding levels of both sides of the mesencephalon, showing shrinkage of the tegmentum on the left (upper figure).

Because of the removal of the corpus striatum there was loss of cells of the medioventral nucleus of the thalamus, loss of cells in the lateral and frontal parts of the corpus luysii and a disappearance of the entire substantia nigra, except the paleonigrum. The fiber loss, due to the removal of the corpus striatum, consisted of degeneration of the tractus centralis tegmenti and the "Kammsystem" of Edinger.

Most of these observations are completely conventional and confirm the work of many other investigators, but there are a few additional things which are apparently new. Among them may be mentioned the cell connections of the medioventral part of the thalamus, the lateral part of the corpus luysii and the composition of the tractus centralis tegmenti. Evidently all the cells of the thalamus do not send their axons to the cortex, for those in the ventral part of the medial nuclei and those in the medial part of the ventral nuclei send theirs to the corpus striatum. This is proved by the fact that on the side where the corpus striatum is present, these cells are present, and on the side where the corpus striatum is absent, these cells are absent, the cerebral hemispheres having been removed in both cases.

It was also due to this unilateral absence of the corpus striatum, other things being equal, that the connections of the tractus centralis tegmenti were discovered. The fibers of the tractus centralis tegmenti, because of their atrophy on one side, caused a shrinkage of the entire tegmentum on that side (as can be seen from the accompanying figure). These fibers could be followed in serial sections from their earliest appearance in the mesencephalon to their various terminations, and as a result of this the constituent parts of the tractus centralis tegmenti have been renamed according to their connections, as the tractus pallido-olivaris and the tractus pallido-reticularis. Besides that, arising from the cells of the tegmentum, there were fibers extending in a centripetal direction, the reticulostriatal bundles. It is stated that Professor Winkler will show in volume 9 of his "Opera Omnia," that the nucleus ruber also contributes to these bundles and that they are really the tractus pallidorubro-reticularis, tractus palidorubro-olivaris, as well as the rubroreticulostriatal tract.

With regard to the corpus luysii, the lateral nucleus lost practically all its large cells on the side where the striatum was taken away, showing that these cells, like the cells of the medioventral nucleus of the thalamus, send their axons to the corpus striatum.

This important piece of anatomic work from the laboratory of Professor Winkler gives to the student of the basal ganglia new facts which help in understanding the function of this part of the brain. Heretofore no one has demonstrated a clearcut efferent tract from the thalamus to the striatum. Scarcely less important is the discovery of tracts to the formatio reticularis, for physiologists have been prone to speak of the rubrospinal tract as of great importance, although in man it is a very small bundle. The tractus pallido-olivaris gives another link in the chain of coordinating mechanisms associated with the olivopontocerebellar complex.

THE ADOLESCENT—HIS CONFLICTS AND ESCAPES. By SIDNEY I. SCHWAB and BORDEN S. VEEDER. Price, $3. Pp. 365. New York: D. Appleton & Company, 1929.

This is one of the most stimulating and valuable books on the adolescent that the reviewer has had the pleasure of reading. Its main theme is that adolescent behavior is the result of the struggle toward social and environmental adaptation. The authors have approached the problem from their respective interests. Dr. Veeder, from his interest in pediatrics, constributes the first three chapters on the physical framework of the adolescent. Dr. Schwab, as a psychiatrist, contributes the remainder of the book on the more complex situations presented by the adolescent in his struggle with his environment. The point of view presented in the whole book, however, is shared mutually.

In the first chapter, Dr. Veeder discusses the physical growth and development at adolescence, but instead of beginning his discussion with puberty considers also the prepubescent period. As other authors have done, he stresses the resemblance between the growth acceleration at pubescence and during early infancy in contrast to the slower rate during childhood. This rapid growth requires an enormous amount of food intake—"half as much again as a farmer at his daily work

requires." It also throws a severe strain on the heart. He is not certain as to the reasons for puberty, except to indicate that the endocrine glands have some regulating influence on growth and the development of secondary sex characteristics.

In the second chapter, he discusses puberty and sex. He draws attention to the need of considering adolescent activities in relation to not only the chronologic age but also the physiologic age. The need for adequate instruction in the phenomena of puberty for both sexes is discussed adequately, and the author points out that it must be recognized that many of the sex problems of adolescence have their origin in the artificial and arbitrary social environment built up around the subject of sex in opposition to biologic laws. The conflict between the inherent sex urge in the adolescent and the environment, which forces him to hold the instinct in check or else to satisfy it in an unnatural manner or in one that transgresses the custom of society, is the basis of the sex problems of the period. No matter how efficiently or effectively, from a practical standpoint, the control of the sex instinct during adolescence has been accomplished by social or ethical codes and the substitutive physical outlets, it must not be forgotten that these measures are but substitutes at best. In the discussion of sex education, he stresses the highly important view that adolesence is not the time to begin such instruction, that if there is a proper parent-child relationship the subject has already been covered, and the adolescent will need only an elaboration of what has been said previously. Furthermore, the problem of sex at adolesence is not merely one of knowledge. Masturbation is discussed also in the same adequate manner.

In the third chapter on school and play, attention is drawn to the danger of overstraining the physical organism, the chief source of the tendency to overstrain lying in the complexity of the present-day environment. In this relation there is a practical discussion of the desirable and undesirable types of physical activities.

Dr. Schwab begins his section of the book with a discussion of the methods of approach to the study of the adolescent. The method he has followed is that of trying to view the subject in as detached a manner as possible, making guarded use of any methods which seem to promise accurate information and avoiding reading one's own problems into those of the subject. Chapters V, VI and VII are devoted to a consideration of the adolescent conflict, and are particularly good. The adolescent conflict is one between the youth, his needs and desires, and the hostile external world of the present day. It arises with the sudden enrichment of the inner mental life which gives an increased awareness of a rather awkward, stupid and misunderstanding world. "The difference between the adolescent of our times and that of the primitive races lies simply in the fact that then there was no obstacle to the carrying out of impulses but that now there is." (The reviewer takes some exception to this last statement). The revolt so characteristic of adolescence is simply the state of antagonism to the existence of a conflict and the state of mind that desires something different. In order to study adolescence it is necessary to investigate both sides of the conflict, the individual and the external world, and to follow the processes by which the former attempts to adjust himself to his environment. The author then proceeds to discuss his subject from these points of view. In chapters VIII to XIII he analyzes certain factors in the present-day environment, the state and the family, education, work, religion and sex as environment. These chapters are the poorest in the book. The one on religion could be improved. Such statements as "Religion to the average adolescent is a social matter—planned to fit the adolescent more securely into the social fabric of which he is becoming more and more a part" at best are inadequate, if not definitely misleading, concepts of the place of religion in the psychology of the adolescent. The reviewer does not agree with the author's conception that entrance into industry is detrimental to the adolescent. Such a sudden transition from a childhood environment to an adult one at adolescence is no different from that which has occurred in the human race at all times. Certainly it occurs more

definitely among primitive peoples than in present-day civilization (see Miller, N.: The Child in Primitive Society, chapter X). This transition bears something of the imprint of a folk rite and as such its genesis and its effects are topics for study rather than for criticism.

Although in the apologia the author acknowledges the debt psychology owes to Freud, in both chapters XIII and XVI his discussion of the freudian theories indicates that he is laboring under many misconceptions of what those theories really are. There is also some doubt as to the accuracy of the statement in chapter XIII that "in early society the female became the protective object of laws, customs and traditions—and the male the aggressive factor against which these institutions were devised." This is in direct opposition to the studies of Crawley (The Mystic Rose), who concluded that many of the taboos were developed to protect the male against the dangerous female.

Chapters XIV, XV and XVI are devoted to a study of the adolescent himself. The next two chapters discuss the ways in which the adolescent attempts to solve his conflict with his environment, by conformity, running away, rationalization, cultivation of a special interest, industry, dreams and phantasies, delinquency and suicide. These chapters are well worth while as is the succeeding one on mental derangement. In chapter XX, adolescence is considered as a process of social adjustment, and in this chapter and the next the need for a less rigid environment and for the development of a more tolerant and understanding attitude of both the adolescent and his environment are emphasized. "A nice balance ought to be struck between the two and the merging into a sort of common interest in the world of ideas as such brought about. The adolescent will as a result begin to feel free to bring into the open of mutual discussion such ideas as he thinks are worthwhile—thus avoiding the easy refuge of repression and the feeling of antagonism. The consciousness on the part of the environment that adjustments of ideas are as important as the adjustment of conduct should be all that is necessary to free this period of the harmful influences of conflicts, neither perceived, understood, nor indeed realized."

In chapter XXII, the author gives some practical applications of his concepts. The applications regarding religion and sex are good, the latter particularly so, whereas those regarding school and school difficulties are not so valuable. To the authors, adolesence is a process of social adjustment to a difficult environment. This is largely true, but it is true also that many of the difficulties of adolescence arise out of a conflict within the individual himself as a result of his preadolescent experiences. Except for this reservation and the few points of disagreement already noted, the reviewer is heartily in accord with the views expressed by the authors. The book should be read carefully by every neurologist and psychiatrist and is particularly valuable to those whose interest lies in the problems of children.

BRAIN MECHANISMS AND INTELLIGENCE. A QUANTITATIVE STUDY OF INJURIES TO THE BRAIN. By K. S. LASHLEY. Price, $3. Pp. 200. Chicago: The University of Chicago Press, 1929.

This monograph, the first to be issued by the Behavior Research Fund of the State of Illinois, takes up a topic of the utmost importance. It begins with an excellent discussion of the theories of intelligence held by the psychologic and neurologic schools. From this it is apparent that from both standpoints the problem is in confusion. The author attempts to solve the problem by experimentation with rats. The experiments include the training of rats in a variety of problems, either before or after destruction of parts of the cerebral cortex, to determine the influence of lesions on previously performed habits or on retention of habits formed after an injury.

From these experiments the conclusion is reached that the capacity to form habits is reduced by destruction of cerebral tissue, and that the reduction is roughly proportional to the amount of destruction. The same retardation in learning is produced by equal amounts of destruction in any of the cyto-architectural

fields. Hence the capacity to learn the maze is dependent on the amount of functional cortical tissue and not on anatomic specialization. Additional evidence is presented to show that the interpretation of association or projection paths produces little disturbance of behavior as long as cortical areas supplied by them remain in some functional connection with the rest of the nervous system.

In a separate chapter the author attempts to review the cerebral functions of the dog, monkey and man. He makes an interesting criticism of neurologic methods of examination, uttering the warning that one must be wary of accepting clinical literature at its face value. He states that in the whole literature of neurology there is scarcely any attempt to measure the actual capacity of a patient with an injured brain to perform such a varied assortment of tasks as is included in a modern intelligence test. The statement that patients may suffer loss of specific capacities as a result of injury to the brain without some general intellectual disturbance thus lacks any adequate proof. He has even less respect for the anatomic studies. He states: "I know of no attempt in the whole literature of neurology to determine what proportion of any anatomical area was destroyed by a lesion; and in many cases which are cited as evidence on the localization question, the delimitation of the lesions is woefully inexact." He calls attention to the fact that frequently conclusions are reached from lesions in patients who have survived. His third criticism is one of logical method. "The evidence for separate localization of two functions is always nothing more than the survival of one after a lesion which abolished the other." He cites the confused point of view held regarding cerebral localization of vision and then calls attention to the chaotic state of knowledge regarding the localization of aphasia.

To a neurologist, to attack cerebral localization is heresy, and yet Lashley's criticism of neurologic methods of examination is on a firm basis. He is equally right in attacking the pathologist, for it is true that rarely does a pathologic report give the full extent of the lesion. No one is better aware of the limitations of clinical and pathologic methods than those who are attempting to work on the problem, but to attack the whole theory of localization and to say that it does not exist on the scanty evidence furnished by the author is, to say the least, unsound. Many attempts have been made to arrive at physiologic conclusions from experimental data on lower animals, but to say that what happens in the rat is necessarily bound to occur in the human being is flying in the face of accepted knowledge; every one knows that one cannot apply the same principles to both, for after all a rat is a rat and a human being is an entirely different species. However that may be, this book has served a useful purpose in that it has opened up the problem of localization from a new angle and has called attention to obvious weaknesses in neurologic concepts.

THE VISITING TEACHER AT WORK. By JANE F. CALBERT. Price, $1.50. Pp. 235. New York: The Commonwealth Fund, Division of Publications, 1929.

This book is a sequel to the earlier publication prepared under the auspices of the National Committee on Visiting Teachers, the Problem Child in School. Its aim is to follow up that presentation by presenting in simple and practical form a discussion of the professional standards, procedure and administrative relationships of the visiting teacher; the author has fulfilled that aim admirably. In the introduction she discusses the status of the visiting teacher. This professional group has evolved in response to the recognition by educators of the need to shift the emphasis in education from the school to the child and by social workers from a new conception of the responsibilities of social work. The causes of bad behavior and truancy, or the underlying physical and mental maladjustment, are linked so closely with the totality of the child's experience that the school must share along with the home and the neighborhood the responsibility for the cause of those maladjustments. The school cannot with complacency require that a child shall conform to its established order without first inquiring whether or not that order is suited to the needs and experience of that particular child. The

visiting teacher, because she is both social worker and educator, is specially fitted to make this inquiry at home and in the neighborhood and on her observations to base suggestions concerning beneficial changes in the child's life in school and without. Her integral connection with the school facilitates her approach to the two groups of adults who have the most influence on the child, parents and teachers.

The author then describes the manner in which the visiting teacher works with the child, the school, the home and the community. Much emphasis is laid wisely on the use of other community organizations as treatment factors and on the need for psychiatric help in cases presenting serious personality maladjustments.

The second part of the book is of equal value. It deals with professional relationships within the school system and stresses as one of the important aspects of visiting teacher work the need to develop a constructive working relationship with all branches of the school organization. Chapter VII is devoted to a discussion of the professional preparation and personal qualifications of a visiting teacher.

It is the reviewer's opinion that the visiting teacher will come to play a more and more important rôle in community mental hygiene. She is in a strategic position for investigating cases of maladjustment when the signs first become easily recognizable and can plan treatment either to be undertaken by herself or if the seriousness of the case warrants it, the referral to a psychiatrist at a time when it will be of greatest value. As a consequence, the book can be heartily recommended to all visiting teachers, school officials — superintendents, school principals and teachers — and social workers (the latter will find in it much food for thought as to both case work and professional relationships). It is of interest also to the pediatrician and to the psychiatrist that they may get a better understanding of the scope and activities of another professional group who can be of great service to them in the treatment of their patients. The appendix contains a number of record forms which will be useful as guides to the worker beginning work as a visiting teacher.

SOME EXPERIMENTS OF PERIPHERAL VISION. By MYER SALAMAN. Special Report 136, Medical Research Council, Reports of the Committee Upon the Physiology of Vision. Price, 2 shillings and 6 pence. Pp. 47. London: His Majesty's Stationery Office, 1929.

This is the sixth of a series of special reports. It is a contribution to the study of some phenomena of vision in the peripheral parts of the retina, and is divided into two sections. The first is concerned with the perception of movement, and, in particular, with the conditions under which the successive exposure of the peripheral retina to two discrete stimuli arouses an illusory perception of movement. The second section, which deals with the perception of form, gives an account of some experiments designed to determine the conditions under which simple geometric shapes can be recognized when viewed peripherally.

Part 1 is based on Riddoch's observation that during recovery from injuries to the occipital cortex which have given rise to restrictions of the visual field, a moving object may be perceived over considerable areas where there is no perception of the form of stationary objects, and that the perception of moving objects recovers first, beginning in the periphery and extending toward the fovea. Riddoch believes, with others, that the perception of moving objects, together with a crude light sense represents a more primitive form of vision, distinct from other visual perceptions. Salaman used two different experiments for this part of his thesis, the second, in which he used lighted points in a darkened room, being the more accurate. This experiment depended wholly on the so-called illusory perception of movement produced by the successive stimulation of adjacent retinal points of the peripheral retina. This work had been done before by others, but only with the central portion of the retina.

Part 2 deals only with the visual perception of lines and simple geometric figures. Horizontal, vertical and oblique lines were used, as well as equilateral triangles with their apexes directed toward different points, an equilateral pentagon and a square. These objects were exposed to view on plates, the observer viewing them first by direct vision, and then peripherally only; at that time he was asked to describe them. The results obtained included both object discrimination and object recognition. The former was a simple statement of the object seen; the latter was a required description of the object seen. The results are given in great detail. Both experiments are interesting investigations of the physiologic processes of peripheral retinal stimulation and should be read with care.

LE LIQUIDE CÉPHALO-RACHIDIEN. By RISER. Price, 28 francs. Pp. 250. Paris: Masson et Cie, 1929.

The subject matter of this book is divided into four nearly equal parts. Part 1 concerns the methods of obtaining fluid from different loci and a consideration of the various tests. It is noteworthy that in this chapter the various colloidal reactions find no place, brief discussion of them being relegated to the chapter on neurosyphilis.

Parts 2 and 3 form the significant part of the monograph and deal with the physiology of the cerebrospinal fluid, from the point of view of both animal experimentation and observations on man. In these chapters the work of earlier workers is discussed, which includes many references to the American literature, and also the presentation of the author's observations. He accepts the choroid plexus as the chief source of origin of the fluid, and admits the perivascular origin as probable though not proved. Personal experiments prove the lack of deep penetration into the brain or spinal cord of substances introduced into the fluid spaces. Meningeal permeability, he believes, is in fact vascular permeability. He thinks that substances enter the cerebrospinal fluid spaces from the blood through the vessels at all points.

The diagnostic application of puncture at different loci is well covered, and indications are given for differential punctures of various kinds, the use of the Queckenstedt test, injections of air and the use of dyes, especially phenolsulphonphthalein. Naturally, iodized oil is given a prominent place, but one infers that he considers studies with iodized oil less easy of interpretation than has been stated by some, and admits that both false arrest and failure of arrest may be misleading.

Chapter 4, a short chapter on the clinical interpretation of observations on the fluid, is somewhat disappointing, probably because it is not and hardly can be very different from that of other books on the subject.

Riser's book should certainly be owned by those interested in the cerebrospinal fluid.

STERLIZATION FOR HUMAN BETTERMENT. By E. S. GOSNEY and PAUL POPENOE. A Publication of the Human Betterment Foundation. Price, $2. Pp. 202. New York: The Macmillan Company, 1929.

This volume is a plea for the wider use of the method of sterilization legal in California—vasectomy in the male and salpingectomy in the female—to prevent the propagation of defective stock—mental defect, psychoses, etc.—and possibly as a method of birth control. It is based on the results of a study of more than 6,000 operations performed as a legal measure in that state and of about 100 performed mostly on men as a voluntary contraceptive measure. The purpose of the operation was accomplished except in seven instances, three men and four women. The authors point out that there is little objection to the operation—seldom any decrease in sexual ability or desire (in fact the latter is often increased). Also they do not attribute any change in behavior to the operation per se, although more defectives can be paroled because the danger of their producing defective children has been removed. The reviewer agrees with this measure and with the

definite benefit which may result in certain family situations from a wider use of adequate contraceptive measures. But he does not agree with the sweeping semistatistical generalizations concerning the influence of heredity in adult maladjustments of the psychotic and marital types on which the plea for a more extensive use of the operation is based. Also, although the authors apparently did not find any severe psychic disturbances as a result of an emotional reaction to such a procedure, the psychiatrist cannot be so certain that these would result infrequently.

The book makes interesting reading for the psychiatrist as an exposition of a rather biased point of view, but the reviewer has many misgivings of the advisability of placing it in the hands of the many professional and lay groups now interested in the question of mental hygiene, because of the fallaciousness of its premises and the fact that it leaves the impression that vasectomy and salpingectomy are the saviors of the race.

GRENZ RAY THERAPY. By GUSTAV BUCKY, M.D. Translated by Walter J. Highman, M.D. Price, $3.50. Pp. 170. New York: The Macmillan Company, 1929.

Bucky has been the leader in the development of the "Grenz" or "borderline" rays. These rays are described in the book from a physical standpoint by Otto Glasser, who says: "It must be understood that there is no sharp boundary between radiations which are called ultraviolet rays and those called roentgen rays. From a physical point of view, this boundary is a relatively broad band."

Bucky claims that the radiation beams of from 1 (0.00000001 cm.) to 3 Angström units have biologic effects different from those produced by the shorter roentgen rays or the longer ultraviolet rays. He therefore calls these "Grenz rays" (border rays). Glasser describes the apparatus and method of producing and measuring these rays, using from 4 to 12 kilovolts. The average absorption in skin tissue was found to be approximately from 0.35 to 0.69 mm. half value layer, allowing about 14 per cent to pass through 2 mm. of skin tissue.

The rays have been found of service in the treatment for certain skin diseases, especially those that ordinarily yield to soft roentgen rays. Some constitutional diseases have also been affected as a result of the action of the Grenz rays on the skin.

As a whole, the book is a more or less comprehensive preliminary report on the nature, development and use of the Grenz rays. It is of special interest to the dermatologist.

SYMPTOMS OF VISCERAL DISEASE. A STUDY OF THE VEGETATIVE NERVOUS SYSTEM IN ITS RELATIONSHIP TO CLINICAL MEDICINE. By FRANCIS M. POTTENGER. Fourth edition. Price, $4.50. Pp. 426. St. Louis: C. V. Mosby Company, 1930.

That visceral neurology is attracting increasing attention from medical men is evidenced by the appearance of the fourth edition of Pottenger's "Symptoms of Visceral Disease." The first edition appeared in 1919. The fourth edition, which is here reviewed, has been so revised as to take advantage of the increasing knowledge of the last five years, during which period considerable advances have been made in the physiology of the vegetative nervous system.

The book is divided into three parts. The first deals with the anatomy and physiology of the vegetative nervous system; the second part considers the relationship between the vegetative nervous system and the symptoms of visceral disease, and the last part deals with the innervation of important viscera with a clinical study of the more important viscerogenic reflexes. The book contains thirty-four chapters. The subject matter is well presented and up to date. The illustrations, particularly the color work, are excellent. The book should be of equal interest to neurologists and internists.