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THE COMPARATIVE ANATOMY OF THE AFFERENT SYSTEM OF THE HEAD*

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PHILADELPHIA

Pain is the most common expression or indication of disease. Constant as it is and has been, there are still elements of uncertainty and doubt as to its nature or as to how it shall be defined. Concerning this, there has been much metaphysical discussion. Meyer [1] queried whether pain was a true sensation or only a perception and interpretation of a sensation to which, from experience, conscience has learned to give the name pain. Is it true that there is such a phenomenon as "memory pain"? Sometimes, in attempts to explain certain pain pictures, this term has been used loosely after the apparent cause of the original pain has been removed. It is true that some of the stimuli of painful impressions may be transmitted to memory centers, which in the future may be the source of a pain, subjective in consciousness and characterized as "subjective pain"?

In the apparatus for receiving and conducting sensation, one acknowledges the existence of receptors for touch, pressure, temperature and vibration, as well as for pain. But little or nothing is known about the differentiation of the recording apparatus for different expressions of pain. Why, in some instances, is pain described as cutting and in others as tearing, throbbing, or burning? Means of distinguishing by practical tests different forms of tactile sensation, such as epioritic and protopathic, are available, but as yet there is no means of proving the existence of specific receptors for different kinds of painful stimuli.

Unlike other forms of disturbed sensation, no registering apparatus to estimate degrees of pain are available. With the esthesiometer one can determine degrees of tactile sensation, but it is impossible to record externally the results of the physicochemical events in the nerve substance expressed by the patient as pain. In an attempt to evaluate degrees of pain in the patient, it is a serious handicap to have no recording apparatus. One must rely altogether on the patient's statement, often so misleading as to eventuate in errors in diagnosis or in proper methods of treatment. Nothing is to be learned from a study of pain

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1. Meyer: Der Schmerz; Eine Untersuchung der psychologischen und physiologischen Bedingungen der Schmerzvorganges, Wiesbaden, 1906.

in lower organisms which, though they may have the same pain sensation as man, react differently to pain stimuli. Norman [2] found that the shark could be cut and operated on without the slightest movement. One must study vertebrates of a higher scale before pain phenomena, as expressed by man, can be analyzed.

These and many other unsettled problems as to the phenomenon pain have been forced to the fore in my mind because of my daily contacts with patients whose outstanding discomfort is pain in the face. On my files are spread the records of 1,854 such cases. It has been impossible for me to escape the patient with the neuralgic syndrome. The problem is ever before me, and of necessity I find myself daily confronted with it in one phase or another. Facial pain is often baffling as to its origin, varying in expression, characterized by similarities and contradictions, differing as to its conformity to and its extraordinary departure from type, as to its response to remedial measures in some instances and its rebellion against all efforts at relief in others.

COMPARATIVE ANATOMY OF CERTAIN SELECTED SPECIES

How can one account for the pains or paresthesias that are sometimes recorded after the radical operation for major trigeminal neuralgia, when all forms of sensation except pressure have been abolished? How can one explain certain pain syndromes in the trigeminal zone that are influenced not one iota by destruction of the one nerve commonly accepted as exclusively supplying the face with different forms of sensation? These two observations, apart from others, would seem to demand an intensive survey of the sources of afferent impulses in this region, and it seemed reasonable at this juncture to pause and review what is known of the afferent system in the cranial nerves in lower forms of life, to make a phylogenetic study of the cranial nerves and their distribution in different species in the evolutionary scale. The literature is vast, too vast for a comprehensive review, and one must be content with a sketch of the comparative anatomy in certain selected species, especially of fish, amphibians, reptiles and various mammalians.

Fishes.—The trigeminal tract in the species of fish shows certain striking differences from that of man. There are two ophthalmic divisions, among other things, the ophthalmicus profundus and the ophthalmicus superficialis. The former has an origin in the brain remote from that of the trigeminus, a separate and distinct ganglion, a separate and distinct peripheral system. The trigeminus facialis group, which includes the acusticus, constitute the principal nerves of the head. In the fish there is also a different arrangement of the cranial ganglia. Instead of separate and distinct ganglia, the gasserian ganglion, the

2. Norman: Am. J. Physiol. **3**:271, 1899.

geniculate ganglion and the two lateral ganglia of the facialis are fused into a common mass. From this and other anatomic relations, one is impressed with the fact that in this species there is a much closer and more intimate relation between the trigeminal and facial systems.

There are but two divisions of the gasserian ganglion, the ramus ophthalmicus superficialis and the maxillomandibular issuing from the ganglion as one instead of two separate units, to which is added a large contingent from the geniculate ganglion. The maxillary and mandibular divisions are always in close association with the facialis. The maxillary division is distributed chiefly to the mucous membrane of the mouth and nose. The mandibular division has a proportionately larger motor representation and a comparatively small sensory territory.

In this species there are two cutaneous nerves not represented in man, acusticus *a* and acusticus *b*, originating in the medulla and running free from all entangling alliances from there to the skin. Thus, in this species there is a subdivision of function between the trigeminus and the acusticus.

As to the relationship of the trigeminus and facialis in fish, the geniculate and gasserian ganglia are so fused as to be indistinguishable as separate units. There is an intimate connection between the two systems, trigeminal and facial, through the ramus palatinus of the seventh and the ramus maxillaris of the fifth. There is also an anastomosis between the facialis and glossopharyngeus and between the facialis and the vagus.

The cutaneous sensory element of the facialis, the various anastomoses between the fifth, seventh, ninth and tenth cranial nerves, the fusion of the cranial ganglia, the cutaneous representation of the facialis and acusticus seem to be the outstanding points of distinction in the distribution and function of the cranial nerves of fish and man.

Amphibians.—Passing to the species next higher, the amphibian, one finds a most detailed description of the cranial nerves of *Siren lacertina* in the work of Norris.[3] As in many other studies in comparative anatomy of the cranial nerves, the purport of this contribution has to do chiefly with the differentiation of species or types rather than with specific nerve function. But in the amphibian one finds a pattern in many respects comparable to the ganoid fish, the next lower form of life. There is the same clustering of the cranial ganglia of which there are six, the dorsolateral line, the gasserian, the geniculate, the general cutaneous of the seventh, the ventrolateral line and the auditory, together forming a continuous ganglionic mass.

One of the essential distinctions between the fish and the aquatic amphibian, on the one hand, and terrestrial vertebrates, on the other,

3. Norris: J. Morphol. **24**:245, 1913.

is the suppression in the latter of that extensive system of external sense organs for the reception of pressure stimuli, so essential to aquatic life, the disappearance of the acusticolateral line system common to aquatic animals but lost in the terrestrial vertebrate. In the loss of this lateral line system the facialis loses three important branches, the superficial ophthalmic, the buccal and the external mandibular.

In the siren the component parts of the trigeminus are practically the same as those of the ganoid fish, except perhaps that the mandibular division has a larger sensory representation. The innervation of the special sense organs or neuromasts by several branches of the facialis (the ramus mentalis internis and the ramus mentalis externis) persists. As in earlier species, there is a general cutaneous element of the facialis, which, without any connection with the trigeminus, finds its way into the facialis through a communicating branch from the vagus. In the so-called ramus jugularis of the facialis, there are many cutaneous fibers.

Noteworthy, too, in this species is the sensory element in the vagus, chiefly through its ramus supratemporalis. If I may summarize in a general way the anatomic features of the cranial nerves in the amphibian as compared to those in man, I should lay emphasis on the relatively large sensory function, exclusive of the trigeminus, as represented by the afferent system of the facialis, the acusticus and the vagus.

Reptiles.—As in the more primitive species, one finds in the reptile the ophthalmic ganglion with its deep ophthalmic division persisting as a separate entity.[4] The superior labial, one of the two main divisions of the maxillary ramus, in combination with the palatine branch of the facialis, supplies the mucous membrane of the palatine and maxillary regions of the mouth. As in other species, too, there is this intimate relation between the gasserian and geniculate ganglia, the two being indistinguishable, and from the geniculate ganglion is given off the ramus palatinus passing cephalad and the ramus hyomandibularis passing caudad. The latter eventually joins the ramus mandibularis of the fifth and, although composed of fine unmyelinated sympathetic fibers, it would seem to correspond to the chorda tympani of man.

The Mouse.—The ramus cutaneus facialis in the mouse is a nerve solely of sensory function, as demonstrated experimentally by Rhinehart[5] and by the clinical observations of Hunt.[6] This nerve supplies the auricle and a part of the tympanic membrane. In the amphibian (*Amblystoma* larva), Herrick[7] described fibers from the geniculate ganglion which are generally cutaneous in function, and the same

4. Willard: Anat. Record **11**:435, 1916-1917.

5. Rhinehart: J. Comp. Neurol. **30**:81, 1918.

6. Hunt: Brain **38**:418, 1915.

7. Herrick: J. Comp. Neurol. **24**:361, 1914.

observer[8] found in bony fish a general cutaneous component in the facial nerve for the region of the operculum.

COMMENT

The sphenopalatine ganglion has been a center around which there has been much controversy as a source from which painful syndromes may emanate. Never have I been able to identify the so-called nasal ganglion neurosis of Sluder. To me, this syndrome seemed to bear no relation to the trigeminal nerve and it is not without interest in this connection to note, as Rhinehart observed in his experiments, that the sphenopalatine ganglion belongs more to the facial than to the trigeminal system. This ganglion receives its supply from the facial nerve through two branches, the nervus petrosus profundus and the nervus canalis pterygoidei. Both of these branches carry abundant sympathetic fibers, so that there is in this central nerve station in the mouse—the sphenopalatine ganglion—a very complex structure with three contributing units, namely, the facialis, the trigeminus and the sympathetic system, in which the facialis element predominates. One is wont to regard the nervus facialis as playing as its major rôle a motor function. While this, in the main, is true, one must not forget that it has definite sensory contacts.

In the ascending scale from the lower forms of *Gnathostomata*, from the fish to the terrestrial vertebrates and mammals, the most striking change in the cranial nerve distribution is the disappearance of the lateral line system, that extensive system of external sense organs for the reception of pressure stimuli. This whole system, known as the acusticolateral line system, disappears; the ramus profundus ophthalmicus disappears as a separate unit; the mandibulary division develops a more extensive sensory function; the facialis becomes more essentially motor and loses proportionately its sensory function; of the various ganglia, the lateral line and the ophthalmic disappear and the remaining continue as distinct units and not as a conglomerate mass. The facialis loses three branches and their communication with the trigeminus, the superficial ophthalmic, the buccal and the external mandibular. Its palatine branch becomes the large superficial petrosal and its internal mandibular branch the chorda tympani. Jacobson's nerve appears as a communicating branch from the glossopharyngeus to the otic ganglion. Of the sympathetic system the four ganglia, ciliary, sphenopalatine, otic and submaxillary, become a part of the trigeminal system. The acusticus, formerly an integral part of the facialis, becomes a separate nerve of special sense.

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8. Herrick: J. Comp. Neurol. **9**:153, 1899.

A review of the morphology of the gasserian ganglion from reptile to man is intriguing. Perna,[9] in 1914, studied alterations of the ganglion after extraction of teeth from one half of the upper or lower jaws of dogs and monkeys and found segmented areas of degeneration in the ganglion, which distinguished the maxillary from the mandibular portion. More recently, Allen,[10] recording the process of degeneration of the ganglion in the cat after section of its several divisions, made interesting observations, observations difficult to harmonize with the earlier conception of the morphology of the ganglion. He concluded that in the cat there were two separate and distinct segments of the ganglion, a large ophthalmic-maxillary and a smaller mandibular portion. Furthermore, he found definite mixing of the cells of the ophthalmic and maxillary portions, but no mixing of the cells of the maxillary and mandibular portions. Whereas in lower forms in the evolutionary scale the ophthalmic ganglion is separate and distinct from the remaining portion in the cat according to Allen, the ophthalmic-maxillary portion is more or less interwoven, and the mandibular portion has no admixture of cells with the adjacent maxillary portion.

Allen's conclusions are not at all in accord with mine, which are based on embryologic studies in the human embryo and clinical observations in the human adult. I am convinced that though fused with the gasserian ganglion as a whole in man the ophthalmic portion may still be regarded as a more or less independent unit, and this conviction is in keeping, not only with the embryologic and clinical facts but with the knowledge of the phylogeny of the ganglion from which, as previously mentioned, in earlier species, the ophthalmic ganglion is separated as a distinct unit.[10a] On these premises, I proposed subtotal rather than total section of the sensory group as the approved procedure in the radical treatment of trigeminal neuralgia.[10b]

I have referred elsewhere to the different types of pain impulses that are transmitted over a general cutaneous nerve. Perhaps it is too much to expect such refinement of technic ever to distinguish between types of pain fibers, but Allen,[11] in his studies of the cells concerned in innervating the teeth of dogs, thought that he had discovered a difference between the fibers of cutaneous sense and those of pain. He found in the inferior alveolar nerve small nonmedullated or sparsely medullated fibers, on the one hand, and large and medium sized medullated fibers on the other. In the ganglion, too, there was a corresponding

9. Perna: Ricerchen. lab. di anat. norm. d. r. Univ. di Roma **17**:81, 1914.

10. Allen: J. Comp. Neurol. **38**:1, 1924-1925.

10a. Frazier, C. H.: Brain **48**:458, 1926.

10b. Frazier, C. H.: Subtotal Resection of Sensory Root for Relief of Major Trigeminal Neuralgia, Arch. Neurol. & Psychiat. **13**:378 (March) 1925.

11. Allen: J. Comp. Neurol. **39**:325, 1925.

variation in the size of the cells. Of the total number of cells undergoing degeneration, three-fourths were of the large or medium sized variety. Assuming that the cutaneous nerves supplying the teeth function mainly in conducting pain impulses, it appears highly probable that these impulses are conducted over the large or medium sized medullated fibers and through the corresponding cells in the ganglion.

Nittoni,[12] after section in the rat of two peripheral branches of the trigeminus on one side, found not only signs of degeneration and regeneration on the affected side, but similar changes in the corresponding nerves of the intact or contralateral side. This degeneration was present not only in the divisions of the ganglion, but in the ganglion itself and even in its root. Nittoni attributed this phenomenon to the possible influence of specific neurolysins, coupled with disturbances of the circulation, the capillaries becoming less visible and the blood vessels decreasing in diameter. This observation, so far as I know, has never been made in man. It might be noted, however, that in man [13] there is a connection between the trigeminal system of both sides through a reflex path passing between the sensory and motor nuclei of the same and opposite sides, and from this collaterals to the descending fibers of the radix mesencephalica.

One is amazed at the number of researches into the origin, course and function of the mesencephalic root from the time of Meynert's first contribution, in 1871.[14] Johnston reviewed the literature of the subject to 1909, including the work of Kölliker (1896), van Gehuchten (1895), Cajal (1896) and Wallenberg (1904). The prevailing view up to that time was in favor of a motor rather than a sensory function. Johnston, however, from an investigation of the origin and course of the mesencephalic root mostly in vertebrates, concluded that the root was sensory in function, basing his conclusion partly on the fact that he had seen the root leave the mesencephalon with the sensory root and partly on the fact that the bundle lies in the dorsal half of the brain.

Allen,[15] in 1918, took up the question anew and after referring to the work of May and Horsley, Willems and Kosaka, recorded the results of his own experiments in the guinea-pig. His views are in accord with those who recognize in the root both ascending and descending fibers, the former originating in the gasserian ganglion and the latter in the alar plate of the mesencephalon. The fibers pursue a cephalic course between the trigeminal motor and sensory nuclei and here intermingle with the motor fibers which follow the sensory root

12. Nittoni: J. Anat. **35**:133, 1923-1924.

13. Spalteholz: Handbook of Human Anatomy, Philadelphia, J. B. Lippincott Company, 1906, p. 698.

14. Meynert, quoted by Johnston: J. Comp. Neurol. **19**:593, 1909.

15. Allen: J. Comp. Neurol. **30**:169, 1918-1919.

through the ventral surface of the gasserian ganglion eventually to form the motor components of the nerves of mastication.

In other words, the mesencephalic root is composed of both ascending and descending fibers. The ascending fibers originate in the gasserian ganglion and on entering the pons join the descending fibers. The descending fibers join the motor root.

As to the function of the descending fibers, there has naturally been much discussion. Allen, concurring with Johnston and Willems, postulated that though these mesencephalic root fibers enter the motor root they are not necessarily motor in function. The distribution of these descending fibers through the branches to the muscles of mastication is such that they can hardly carry cutaneous sensations. Hence, he concluded that if sensory in function the mesencephalic root must function as the muscle sense component of the trigeminal nerve.

Five years later, Thelander [16] came to somewhat similar conclusions. He succeeded in tracing the descending mesencephalic fibers continuously from their exit in the pons into the mandibular branch and on into the masseter, deep temporal and pterygoid nerves.

This confusion as to the precise function of the several fibers making up the mesencephalic root, especially those which join the motor root (and they are in the majority), tempts one to speculate as to whether certain of these atypical sensations in the face may not be transmitted through the motor root. Certain it is that there is now substantial evidence that there are afferent fibers in the ventral spinal roots. Whether or not the premises justify the deduction, I have of late abandoned my previous practice of conserving the motor root in radical operations for trigeminal neuralgia.

As to the origin of the paths of the autonomic system, it seems probable that the ganglion cells migrate from the central nervous system, from the neural tube and spinal ganglia. Morphologically speaking, the autonomic ganglion cells are said to be equivalent to the motor cells of the anterior horn of the spinal cord. The preganglionic sympathetic fibers of the head originate in the upper thoracic nerves. Of the four autonomic cephalic ganglia, the ciliary is the only one described in species below the reptile. In the hog, fish and lamprey (*Cyclostomata*), the autonomic system is diffuse, and in the elasmobranchs multiple ciliary ganglia frequently have been observed.

Historically speaking, the separation of the sympathetic ganglia began in the head, where, as in the fish, the autonomic system is more advanced than that of the trunk. I have already referred in a description of the anatomy of species between the fish and the mammal to the presence of cranial ganglia corresponding to the ciliary, sphenopalatine

16. Thelander: J. Comp. Neurol. **37**:207, 1924.

and otic ganglia in man. An elaborate study of the sympathetic elements of the trigeminus was made by Takeda [17] in various vertebrates (dogs, rabbits, rats, cats, guinea-pigs and cows); in all he found collections of multipolar cells in no way connected with the cells of the semilunar ganglion. These cellular groups were variously situated; in the ophthalmic and maxillary divisions (cows); between the first and second divisions (guinea-pigs); medial to the first division (cats, rats and rabbits), and central to the ganglion (dogs). Takeda also found these ganglion cells in the motor root of the trigeminal nerve, an observation confirmed by Allen [18] who found similar cells in other motor cranial nerves, notably in the third, fourth and sixth. Allen said that it is apparent that all the cranial nerves with motor roots contain proprioceptive sensory cells which retain the primitive condition of the sensory nerve cells of the spinal roots of the amphioxus and other vertebrates.

What one knows of the autonomic system of the head has to do chiefly with the efferent fibers and, widely diffused as they are, there is still reasonable doubt as to their function. Of the afferent pathways little is known and since, in connection with the interpretation of the various types of sensation in the face, one is more concerned with the afferent system, there is here a tremendous gap and a fertile field for research.

To comprehend the functional transition of the cranial nerves from the lower forms to the higher vertebrates, mammals and man, one must visualize the division of the mesoderm originally into nine segments or myotomes, each with its ventral and dorsal root corresponding to the ventral and dorsal roots of the spinal nerves. In the first segment, the ophthalmicus profundus represents the sensory and the oculomotor, the motor root; for the second segment the trigeminus is the sensory and the trochlear the motor root; for the third segment the facialis is the sensory and the abducens the motor root; for the fourth segment the acusticus is the sensory, but without a motor unit. In the remaining five segments, the sensory elements are supplied by the glossopharyngeus and the vagus, and the motor elements are supplied by two branches of the hypoglossus.

In lower forms, the gill arches are derived from the splanchnic mesoderm, a part of the alimentary system to be supplied by the fifth, seventh, eighth, ninth and tenth nerves. One of the gill slits eventually becomes the mouth of the higher vertebrate; the pretrematic sensory branch becomes the maxillary nerve to supply the upper jaw and the post-trematic branch the mandibular to supply the lower jaw. The

17. Takeda: Folia Anat. Japon. **2**:297, 1924.
18. Allen: J. Comp. Neurol. **38**:349, 1925.

first gill arch is the origin of the external ear, to supply which the mandibular nerve gives off its auriculotemporal branch.

In lower vertebrates the taste buds of the mouth and skin are supplied by branches of the facial, palatine and internal mandibular, both arising from the geniculate ganglion, a visceral afferent function in which the trigeminal nerve plays no part. Eventually, the palatine branch of the facial nerve becomes the great superficial petrosal in man and the internal mandibular the chorda tympani. The association of the latter with the trigeminal system through the lingual nerve is a secondary aquisition, purely topographic in nature.

If it is true that the relationship of the chorda tympani to the trigeminal system is a secondary acquisition, the same may be said of the great superficial petrosal nerve. The latter, developing embryologically from the geniculate ganglion, at first has no association whatever with the trigeminus. What association it develops later in man, therefore, must be a relatively late phylogenetic acquisition.

Possessed of a component so definitely sensory in function, this sensory portion of the facialis is considered by some anatomists as a separate cranial nerve, composed of the palatine nerve, the sphenopalatine ganglion and the pars intermedia.

In the lower organisms, the most important channel of information was the snout through its sense of smell and sense of touch. Therefore, the olfactory and trigeminal nerves were the two most important of the cranial group. In the process of evolution, as in an arboreal existence, the snout loses its importance as a tactile organ and, as Wood Jones pointed out, this important tactile function was transferred from the trigeminal to the median nerve. Not only in function, therefore, but, relatively speaking, in the territory supplied, the trigeminal nerve has lost rather than gained importance. Whether, as Thompson [19] suggested, the existence of trigeminal neuralgia as a disease in man may be accounted for on the basis that structures in process of evolutionary regression are more susceptible to pathologic processes is a matter of speculation.

CONCLUSIONS

Certain obvious rearrangements and reassignments of function in the cranial nerves have been concurrent with evolutionary alterations in gross patterns, as well as with the gradual development of special senses. As one unfolds the evolutionary history of the cranial nerves, the major rôle which the facial nerve played as a nerve of afferent impulses seems to stand out in bold relief. The universality of sympathetic elements, not only represented in the recognized cranial ganglia but discovered

19. For these observations in comparative anatomy I am indebted to Dr. J. MacLaren Thompson (Eye, Ear, Nose and Throat Month. **4**:22, 1925-1926).

also as groups of ganglion cells in the gasserian ganglion, peripherally in its several divisions and centrally in its motor root, arrests one's attention. From the great storehouse of information, the numerous contributions to the minute anatomy of the cranial nerve system in species lower than man, one derives little of practical moment in the solution of the clinical problem with which primarily we are concerned, since many of these researches have to do predominantly with the differentiation of type rather than with the demonstration of special function. Before further light can be shed on any disturbance of sensation now of uncertain origin, an investigation must be made, far more fundamental than any hitherto undertaken, on the afferent portion of the preauditory system.[20]

20. Preparation for this has already been made under the direction of Professor Coghill at the Wistar Institute of Anatomy in Philadelphia.

POSTURE WITH SPECIAL REFERENCE TO THE CEREBELLUM *

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EVIDENCES OF DECEREBRATE RIGIDITY IN ANIMAL AND IN MAN

The description of decerebrate rigidity by Sherrington was an important event in neurology. Sherrington observed, in 1896, after a transsection through the midbrain between the anterior and the posterior corpora quadrigemina, an increased tonus of the extensor muscles of the neck, the back, the tail and the legs. All the muscles that secured the erect position of the animal were overinnervated. It is the function of standing which appears in such a way. Richter [1] has shown that in the sloth, an animal in which the normal attitude is that of hanging from trees with flexed limbs, decerebrate rigidity is a flexor rigidity. The muscular tension in connection with decerebrate rigidity is a special one. Sherrington discovered the shortening and lengthening reaction of muscle: The muscle adapts itself to any length given to it without change in tension. Sherrington and Lidell also described the stretch reflex or myotatic reflex: an increasing tension when the muscle is passively stretched. Magnus and de Kleijn have shown that in the decerebrate animal a change in the position of the head provokes regular changes in the tonus of the extremities and the trunk, which they termed "Lage-reflexes," reflexes of position. These postural reflexes are partly in connection with the position of the head in relation to the trunk (neck reflexes). Such is the typical picture of decerebrate rigidity. However, Schaltenbrand [2] and others pointed out that there may be variations in this picture; thus, there may be flexor rigidity instead of extensor rigidity (Beritoff), or the Magnus and de Kleijn reflexes may be absent (Bazett and Pennfield). A similar decerebrate rigidity may be obtained by sections made through deeper parts of the brain stem. Even sections through the medulla oblongata provoke a similar picture if the section is made above the calamus scriptorius.

It is difficult to find a true analog to decerebrate rigidity in man. One finds such pictures mostly in cases with gross destruction or com-

* Submitted for publication, Jan. 30, 1929.
* Read by invitation at a meeting of the Philadelphia Neurological Society, Dec. 21, 1928.

1. Richter and Bartemeier: Decerebrate Rigidity of the Sloth, Brain **49**:207, 1926.

2. Schaltenbrand: Theorie der proprioceptiven Lage- und Bewegungs- reaktionen, Deutsche Ztschr. f. Nervenh. **100**:165, 1927.

pression of the brain stem. Here, however, the arms are often not in extension but in flexion. The Magnus and de Kleijn reflexes may be absent. Pronation and extension in the arms may be found. The legs are in extension. If there is no shortening and lengthening reaction, one should not speak of the condition as decerebrate rigidity.

Walshe considers that typical hemiplegia is a close analog of decerebrate rigidity, and the interesting experience of Russell Brain,[3] that a quadrupedal attitude in hemiplegic persons changes the flexion contracture of the arms to an extension contracture, seems to point in the same direction. But in hemiplegia the shortening and lengthening reaction is not present, at least not in the same way as in decerebrate rigidity. The stretch reflex plays a much more important part.[4] Simons and Walshe have found the Magnus and de Kleijn reflexes in hemiplegic patients. The arm toward which the chin is turned shows an increased extensor tonus, whereas the flexor tonus of the opposite arm increases. These changes, however, can often be obtained only by provoking associated movements, and the latter are present only in cases in which a tendency to such movements exists. The results of the change of the position of the head in space are rather uncertain. Although Pette reported positive observations, Hoff and I[5] have often missed the Magnus and de Kleijn reflexes in older hemiplegic persons. They are at least not constant in older arteriosclerotic patients. Simons' material consisted chiefly of younger people with traumatic lesions of the brain. When the reflexes are present in older people, they are less marked than in younger people.

The differences between hemiplegia and decerebrate rigidity are so great that there is no justification for identifying the two states. Schaltenbrand considered as decerebrate rigidity not only hemiplegia, but the rigidity of paralysis agitans. But this theory presents a marked difficulty. The stretch reflex is, at least in some cases, not well marked (that a stretch reflex exists in some cases, Mayer, Gerstmann and I have proved). The position of the limbs in cases of paralysis agitans is different from the position in decerebrate rigidity. In typical cases of paralysis agitans, there is a semiflexion in all joints. As a rule there are no righting and postural reflexes. There is also no reason to consider the muscular state of Parkinson's disease or of a parkinsonian case as an analog of decerebrate rigidity.

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3. Brain, Russell: On the Significance of the Flexor Posture of the Upper Limb in Hemiplegia, Brain **50**:113, 1922.

4. One may identify the characteristic elastic resistance of hemiplegia with the stretch reflex.

5. Hoff and Schilder: Die Lagereflexe des Menschen, Vienna, Julius Springer, 1927. Our common experiences there reported are the basis for these remarks. This book contains a complete bibliography other than the one cited in this paper.

In my own experience, I have observed a close resemblance to decerebrate rigidity in persons whom I etherized. In them one sees an extensor tonus in all extremities and the arms show a considerable degree of pronation, but so far I have not obtained Magnus and de Kleijn reflexes. After all, experiences with man show that an absolute analog to decerebrate rigidity does not exist. An organism in which the pyramidal tract plays such an important part is too unlike organisms in which the pyramidal tract is of only little importance. One must describe, therefore, the clinical facts as such, and one cannot always reckon that identification with physiologic experiments will be possible.

POSTURAL AND RIGHTING REFLEXES IN ANIMAL AND MAN

The decerebrate animal has retained the possibility of standing, but it has no longer the possibility of getting back a lost position. It has lost the righting reflexes. The animal with righting reflexes brings the head back into the normal relation to the trunk and brings the head and trunk back into the normal position in space. According to Magnus and Rademaker, the righting reflexes have an important center in the red nucleus, but the question is a rather open one since Mussen, after experimental destruction of the nucleus ruber, did not find lasting changes in the righting possibilities.

(*a*) *Neck Reflexes.*—In normal animals Magnus has observed postural as well as righting reflexes, and these reflexes are also present in human beings. Schaltenbrand saw them in children, but he thinks that in the normal adult the neck reflex has disappeared completely. Hoff and I, however, found that in about 90 per cent of normal adult persons passive turning of the head influences the arms as well as the trunk. Turning the head to the right, for instance, provokes a deviation of the stretched arms and of the trunk to the right. The right arm rises, whereas the left arm remains either in the same horizontal plane or sinks a little. The subject must close his eyes during the experiment. One deals here with a neck reflex which resembles a postural as well as a righting reflex.

(*b*) *The Pronation Phenomenon.*—There are also some other postural reactions in normal people. If a person stretches his hands in a position of strong supination (with the eyes closed), pronation sets in, the extent of which varies in different people. The subjects are not aware of the pronation. There is a tendency to go from the inconvenient posture of supination to a more convenient position of a minor degree of supination.

(*c*) *The Divergence Reaction.*—Another reaction of that type is the divergence reaction. If a normal person stretches both arms straight forward, the arms will diverge in a more or less pronounced degree.

Only if the arms are outstretched so that they are at an angle of from 45 to 60 degrees outward from their parallel position does no divergence take place. Also, no outward movement can be observed if only one arm is stretched forward and the other hangs down. The divergence reaction and the pronation tendency are the expression of a tendency to a comfortable and "normal posture." This tendency is at least closely akin to the postural and righting reflexes.

(*d*) *Persistence of Posture.*—Another experiment that I shall stress is a phenomenon in normal people which I call persistence of posture (Lagebeharrung). If I passively raise (lower) the arm (M = moved) of a subject who has stretched both arms forward, with closed eyes, 60 degrees above (below) the horizontal line and leave the raised (lowered) arm in this position for thirty seconds, and then request the subject to put the raised (lowered) arm at the same height as the quiet arm (Q = quiet) the M arm stands above (below) the Q arm. The distance varies between 2 and 10 cm. I emphasize the fact that the reaction has nothing to do with the persistence of muscular tension (experiments of Kohnstam). It is a persistence of postural influences. The subject knows nothing about his mistake, and the erroneous posture of the limb becomes for him the normal posture. If, for instance, the raised M arm is brought passively to the same level as the other arm, the subject feels as if the Q arm was higher and the M arm lower. In other words, the postural scheme of the body is distorted in such a way that the position is felt as the normal position which the limb would reach when following the muscular tendency. The true normal position of the limb is now felt as a position different from the normal position in a direction opposite to the direction of the persistent tone. The postural model of the body is therefore changed, while the persistent tone goes on. The position of the limb into which the muscular tendency tries to pull the limb becomes the normal position of the limb, and all other postures are now judged in relation to this "normal" position.

The same is true for all other types of tone which are in connection with postural tone, the tone of the righting reflexes, or, in brief, the tone of the Magnus and de Kleijn reflexes. If, for instance, from a homolateral cerebellar lesion the right arm shows a tendency to go upward and both arms are placed passively at the same height, the patient will perceive his right arm lower than the left one. If there is an outward tendency, when the arms are put in a parallel position (passively) the subject will get the impression that the right arm stands more inward than the left one. The tone of persistence of posture can be taken as a model for postural activities. It is always present in normal people and is absent only in about 20 per cent of the cases of

paralysis agitans and parkinsonism. It is also absent in those groups of cases which often show also a lack of other postural and righting activities. I have failed to find the persistence of posture in one case of symmetrical pallidal lesion and in one case of a tumor of the midbrain.

(*e*) *Reflexes in Extrapyramidal Lesions.*—In Parkinson's disease and parkinsonian cases, turning of the head has no effect at all. Sometimes it seems as if the arms and trunk would deviate in the same direction in which the head is turned. But careful investigation shows that the limbs and the trunk are carried out with the head in a mechanical way by the rigidity. A normal person, when getting up from lying on uncertain ground, such as on a springy mattress, feels uncertain and makes use of a more infantile way of getting up, by turning around his trunk. Most patients with paralysis agitans are not able to make use of these primitive righting reflexes; they get up from a mattress in the straight way, without turning around. The postural and righting reflexes are not absent in all cases of paralysis agitans and parkinsonism. Two phenomena here described are always present in the parkinsonian cases: the pronation phenomenon and the divergence phenomenon. There is, however, a peculiarity about the divergence phenomenon in cases of paralysis agitans. It is obtained only if one fixes the elbow joints rigidly with bandages. If one does not do so there is a marked convergence of the stretched hands, a sign which has proved of diagnostic value. It is based on the tendency to flexion in the elbow joint in paralysis agitans. It is worth while to remark that tonus of paralysis agitans and parkinsonism does not influence the postural scheme of the body. It is different from the tone in the righting reflexes and in the postural reaction we dealt with in this paper.

Comment.—In normal and pathologic cases one should study the following reactions: (a) the reaction to turning the head; (b) the divergence reaction; (c) the pronation phenomenon, and (d) the persistence of tone.

POSTURAL AND RIGHTING REFLEXES IN CEREBELLAR DISTURBANCES

(*a*) *The Cerebellar Reaction.*—In cerebellar cases the neck reflexes are increased on the side of the lesion. One can see this clearly in cases of hemichorea, in which there is at least a strong cerebellar component. In rare cases the neck reflexes are so increased that there results a spontaneous rotation around the longitudinal axis (André-Thomas, Hoff and myself, Gerstmann). If, for instance, the head is turned to the left after a cerebellar lesion on the right, the trunk and arms may deviate in a paradoxic way to the right. There is here an increased tendency to turn to the right in the presence of a lesion of the right side of the cerebellum.

(*b*) *The Pronation Tendencies.*—Wilson [6] has drawn attention to the fact that cerebellar patients often shown an increased pronation and has brought this observation in relation to the pronation posture of decerebrate rigidity. Hoff and I have stressed the fact that this is an exaggeration of the normal tendency. Gierlich expressed the opinion that supination is philogenetically a younger function than pronation. In pyramidal lesions one also finds the pronation tendency. This pronation tendency, however, occurs not only in connection with a change in tone, but also in connection with a change in strength. One comes, therefore, to the conclusion that the cerebellum helps in the assumption of an uncomfortable position which is at the same time a philogenetically younger position.

(*c*) *The Bárány Test.*—Bárány's past-pointing test probably belongs in the same group. Goldstein has emphasized that most cerebellar patients point outward. This is an exaggeration of the divergence reaction. If one examines with the divergence test one finds that the divergence is better marked on the side of the cerebellar lesion. Whereas in a normal person the outward deviation is to be obtained only when both arms are stretched forward (and not when only one arm is stretched forward), in cerebellar cases the deviation also takes place if only one arm is stretched out. This deviation, as Fisher and Wodak have emphasized, is the basis of past-pointing. Past-pointing is the result of a tonus which interferes with active movement. A cerebellar lesion increases the outward tendency of the whole half of the body. Turning the head to the side of the cerebellar lesion increases the outward tendency of the arm to a considerable degree. Often the elevation of the arm is also increased. It also happens sometimes that turning the head to the opposite side will provoke an increased outward tendency of the affected arm. I speak of this then as a unilateral paradoxic reaction. As I have pointed out, these different types of cerebellar hypertonus influence the postural model of the body. When only the outward move ments of the arm are increased, one deals with an anisosthenia (André-Thomas) or, as I should prefer to say, with an anisotonia.

In cerebellar cases, one finds also another interesting homolateral phenomenon of anisotonia. If a cerebellar patient is lying on an even support and the healthy leg is brought into semiflexion with the heel resting on the support and the patient is asked to place the other leg in the same position, he imitates in such a way that the knee on the side of the cerebellar lesion is higher than that on the healthy side. That is not hypermetria. The same mistake is made if the leg on the side of the lesion is brought to an extreme degree of flexion and the subject

6. Wilson: On Decerebrate Rigidity in Man and the Recurrence of Fits, Brain **45**:490, 1922.

is asked to imitate the semiflexion of the other leg. The leg on the side of the cerebellar disease will be in a state of hyperflexion again as compared with the other leg. One is therefore dealing not with hypermetria but with anisotonia. The lesion of the cerebellum makes the flexors prevalent. In the lower extremities the cerebellum protects the influence of the extensors and is opposed to the influence of the pyramidal tract. I want to emphasize that the patient knows nothing about his trouble; if the leg of the diseased side is passively brought into the same position as the other one, the patient will feel that the leg of the diseased side stands lower than the other. One reaches the general conclusion that the tonus of this cerebellar symptom influences the postural model of the body. It is difficult to say whether or not these disturbances of tonus are in connection with a special localization of the lesion. The facts point to a different localization in the vermis (trunk) and in the hemispheres (extremities). Goldstein denied that there is ever a cerebellar lesion with which only the leg or arm is affected. The problem is not yet settled. I have seen localized muscular disturbances of a type different from the one here described, but in this case no autopsy was made and it was impossible to exclude a lesion of the medulla oblongata. That the cerebellum has some connection with postural reflexes was pointed out first by clinicians, especially Goldstein. Afterward, Rademaker adduced experimental facts which give the same evidence. No one believes today that the primary reflex arc of tonus goes over the cerebellum. But the cerebellum has a great influence on this primary reflex arc.

Comment.—Rademaker denied that a cerebellar lesion can ever provoke loss of tonus. From the point of view of a clinician, I emphasize the possibility that loss of tonus can occur in cases of cerebellar lesion. Patients who have no palsy let the hand sink at the joints whether the hand is supinated or pronated. There is an incapacity to maintain the posture of the hand against gravity. Those patients, too, do not react with sufficient resistance against passive movements, for instance, shaking. I have also seen patients in whom the ear irrigation test of Bárány did not provoke past-pointing and deviation, and I have also seen patients in whom the fall reactions were absent. I believe, therefore, that the function of tonus can be inhibited by cerebellar lesions.

One finds also marked changes of postural tonus in cerebellar lesions. One cannot suppose that such a complicated organ as the cerebellum has only one function; besides the postural changes of tone, one finds hypermetria and hypometria (bradyteleokinesis). Intention tremor is only one type of this insufficiency of the cerebellar brake system. Not enough is known about the localization of these symptoms. I am inclined to relate them to the nucleus dentatus-ruber system. I believe that the imitation phenomenon, which can often be found in cases of combined

sclerosis perhaps has a relation to the spinocerebellar tracts. But all these problems are still unsettled. Another cerebellar system has to do with the classic symptoms of Babinski's asynergia, consisting in a lack of coordination between the active motor tendencies of the arms and legs and the innervations of the trunk. That is a disturbance quite different from the one described here. I believe also that adiadokokinesis is a special trouble with its mechanisms. I consider it, according to my investigations with Gregor, a kind of central myasthenia.

But whatever the theoretical aspect of these problems may be, I stress the practical and diagnostic value of the following symptoms: increased divergence (deviation test); pronation phenomenon; increased righting and postural reflexes; paradoxic bilateral or unilateral deviation; the imitation phenomenon. Finally, I stress the fact that the change of the postural tone changes the postural model of the body. (Goldstein's opinion also should be compared.)

In tabes one sometimes finds one or another of these symptoms, either in connection with the fact that the loss of afferent impulses changes the function of the postural apparatus, or in connection with some primary changes in the postural apparatus and the cerebellum. However, little is known about the changes in the postural apparatus in tabes. Clinical facts, however, emphasize that not only is there a loss of coordination but there are also characteristic changes in the postural apparatus.

The primary reflex arc of the postural apparatus is under the influence not only of the cerebellum. I have described with Hoff, and partly also with Gerstmann, a syndrome occurring after a lesion of the parietal-occipital lobe (gyrus angularis or field 19 of Brodmann). In these cases I found not only increased postural and righting reflexes but also spontaneous turning of the body around the longitudinal axis. In these cases there occurred also changes of the postural model, and optic hallucinations were present. The postural tone also influences optic perception (Goldstein's opinion should be compared with this, too).

Goldstein and Riese, Zingerle, Wodak and Fischer have found similar movements and changes of perception in normal people who were given the order to follow their motor impulses freely. Many investigators have suspected that in these experiments psychogenetic factors might play an important part. Indeed, it is not easy in normal persons to distinguish between voluntary impulses and postural reflexes. But the results in organic cases show clearly that there is at least some truth in the experiments of Goldstein and others. The phenomena that I have described here are by no means psychogenic. They have been proved valuable from the point of view of diagnosis. The problem of tone and posture is complicated. The results are not final, but I consider them a beginning of an important investigation.

DISCUSSION

DR. T. H. WEISENBURG: While Dr. Schilder's discussion includes the question of tone as related to structures other than the cerebellum, my remarks will be limited to the latter organ only. I wish, however, to point out that the behavior patterns of animals following decerebration differ greatly, according to whether or not the animal is intact, as was shown by Mussen, whose results were different from those of Rademaker.

It is obvious that Dr. Schilder looks on the cerebellum as an organ which always acts as a whole and that the same symptomatology results no matter where the lesion is. I agree with him that the cerebellum has one function, but I do not agree that it acts as a whole, i. e., that a lesion in the right lateral lobe of the cerebellum will give the same symptoms as a lesion in the vermis or in the left lateral lobe. The argument against localization of function in the cerebellum has always been based on the theory that there are not enough well recorded cases to substantiate such a point of view. As a matter of fact, there have been enough cases reported to support localization. Moreover, the fiber tract connections and development of the cerebellum in the different species all show that the cerebellum must have localization. If it does not, then it is the only part of the nervous system which does not conform to the uniform rule.

Dr. Schilder makes certain statements such as that in cerebellar cases the neck reflexes are increased on the side where the cerebellar lesion has its seat; that there is a pronation tendency; that Bárány's past-pointing test is an exaggeration of the divergence reaction and is the result of a tone which interferes with the active movement; that the function of tone can be inhibited by cerebellar lesions and that there are marked changes of the postural tone. He then discusses hypermetria, hypometria, asynergia and adiadokokinesis, which he considers special functions, the last being a kind of central myasthenia.

Many years ago, in a series of studies made by moving pictures, many of the symptoms described by Dr. Schilder were indicated. The original pictures were shown at the International Medical Congress in London before the Neurological Section, and since that time have been reported. It is easy to theorize about all of these symptoms, and especially easy to give each one a special name and to say that they are the result of disturbances of tone or of postural tone somewhere in the cerebellar mechanism. All this, however, does not help the cerebellar problem. What does help is the adequate description of the kind of disturbances which occur in cerebellar lesions, with an adequate check-up of the pathologic changes. As it is now, there is no uniform method of describing symptoms of cerebellar deficit, and it is difficult after reading the literature to understand how much cerebellar disturbance there really is and the nature of it. It would be much better for everyone to use a uniform method of description.

My own view is that the function of the cerebellum is to synergize whatever motor activity is initiated, not only by the motor level in the precentral convolutions, but also by the psychomotor levels and by the striatal levels, for all of them have their own separate function to perform. Most authors, in discussing the cerebellum, give the impression that the only motor activity which the cerebellum synergizes is that which is initiated in the precentral convolutions. Cerebellar activity may be described in terms of synergy and deficit in terms of asynergy. If this plan were adopted, as has been said before, with an adequate pathologic check-up, something would be accomplished.

DR. W. G. SPILLER: The study of cerebellar function is difficult; it has occupied the attention of many men over a long period of years, and the results obtained leave much to be desired. Thomas was carrying on his investigation in

1895 when I was studying in the Salpêtrière. The cerebellum has been shown to have a minor part in reflex movements. The most important function of the cerebellum, it seems to me, has been acceptably expressed by Walshe in the following statement: "The cerebellum is an organ through which the cerebral motor cortex achieves the synthesis of coordinated units which go to make up voluntary movements, or the cerebellum is the organ by which the cerebral cortex achieves integrative synergia in voluntary movements and cerebellar ataxy is the expression of a defect of integrative synergia in voluntary movement."

Walshe remarked that those who refer to cerebellar function are invariably thinking of the quite distinct problem of the localization of symptoms of lesions within this organ. This is true, and yet this has been the common method of clinical approach in determining functions of the central nervous system.

Dr. Schilder has stated that another important function of the cerebellum is inhibition of those movements which phylogenetically are older, and these are movements of flexion in the lower limbs and of pronation in the upper limbs. In regard to the lower limbs, especially, he uses the term "imitation phenomena." By these phenomena he shows that from a cerebellar lesion flexion of the lower limb at the knee or pronation of the upper limb in the forearm and hand on the side of the lesion is greater than in the corresponding limb on the unaffected side. This inhibitory function of the cerebellum is an important contribution to knowledge of the cerebellum. The fact that when the upper limbs are held extended in front of the body the hand on the same side as a cerebellar lesion assumes a position lower and in greater pronation than does the other hand was described by Dejerine and may have been recorded by Thomas, but the interpretation of this phenomenon given by Schilder seems to be new.

It seems to me that the inhibitory function of the cerebellum over the flexion of the upper limbs may be demonstrated by having a person with unilateral cerebellar lesion attempt to bring the extended hands elevated above the head down (i. e., flex the limbs at the shoulder) to the same level in front of him with the eyes closed. The hand on the side of the cerebellar lesion probably will assume a position at a lower level.

Localization of function in the cerebellum is still a matter of great dispute. This is not surprising. Much has been learned about localization of motor function in the cerebrum, but even this is still a matter of dispute. It is not known even now whether the motor fibers for an upper limb are distinct from those for the lower limb of the same side below the internal capsule or not. Many believe that they are, and Foerster has placed definite limits in the pyramidal tract for the fibers of the limbs and trunk on the same side. Several years ago, Mellus demonstrated before this society that after he had removed the motor cortex for a single limb in the ape the degeneration in the pyramidal tract was diffuse throughout this tract and not confined to a separate area. Others have also borne testimony to similar observations, and my own investigations have been confirmative of them. If such discrepancies exist regarding the best known portion of the central nervous system, it is not surprising that localization in the cerebellum has remained largely an unsolved problem.

I cannot accept the views of those who hold that each limb or a portion of a limb has been shown definitely to be sharply limited in its cerebellar representation. If one argues that from analogy such definite representation ought to exist, I accept his statements so far as he acknowledges he is presenting theory and not proved fact. I believe that there may be likewise separate representation of function in the corpus striatum or even in the substantia nigra, but at present this is only theory. My own conclusions are similar to those of Gordon Holmes and

Schilder, and are the result of personal observation over a period of more than thirty years, for a paper by me on the cerebellum was published in 1896. Holmes has had extraordinary opportunity for studying the function of the cerebellum, and his conclusions appeal to me. He says that precise localization of function in the cerebellum at present is not possible. Each lateral lobe influences the motor function of the same side of the body only. When the vermis is injured or both lateral lobes are involved, articulation and the postures and movements of the head and trunk are more affected than when one lateral lobe only is damaged. No local lesion affects only or exclusively the one limb or a portion of a limb.

I believe that future investigations may greatly increase the knowledge of cerebellar function and that more definite knowledge of cerebellar localization may be obtained.

DR. PAUL SCHILDER: Dr. Spiller and Dr. Weisenburg have not, as far as I can see, a fundamentally different point of view concerning the localization in the cerebellum. Dr. Spiller, with whom I agree as well as with Dr. Weisenburg, believes that the facts point to a localization in the cerebellum. Dr. Weisenburg is more optimistic about the matter. The only elaborate trial for determining the cerebellar localization in men is the work of Bárány, and in this point, as far as I can see, it is a failure. I do not want to say that a topical localization in the cerebellum will never be determined, but as yet, it has not been.

Dr. Weisenburg would like to speak generally about cerebellar asynergia without making special distinctions. I cannot agree with him on this point. Asynergia means only that something is wrong with the synergia. It is better to distinguish between the phenomenon of asynergic gait, which consists, indeed, of an inadequate synergia, and the release phenomena and postural peculiarities that I have described here. These types are well defined, and one should not apply the same name for such different entities.

It is true that French writers have described an overactivity of pronators in movements. But they took it as a sign of hypermetria and overlooked the fact that this hyperactivity is only in the pronators and not in the supinators. Hypermetria has nothing to do with the pronation phenomena.

I know that this problem is not settled yet, but I am glad to have had this opportunity of expressing my point of view.

CEREBRAL LOCALIZATION AND FAILURES IN WRITTEN LANGUAGE*

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My remarks will be limited as far as possible to the subject of cerebral localization and failures in the acquisition of written language as discussed by Dr. Samuel T. Orton in various presentations and publications, but some excursions in the form of illustrations necessarily will be taken. I shall indicate here only a few points necessary to be kept in mind for a complete comprehension of the subject on hand. Dr. Orton has confined his remarks to failures in the acquisition of written language, in which he includes reading, writing and spelling. These chiefly show themselves in a tendency to reversals or confusion in words, syllables or letters and to sinistrad progress in reading. These mistakes seem to interfere with the process of prompt association.

In his studies, Dr. Orton found many instances of mirror reading and writing. He believes that his data as a rule do not show mental inferiority, but a failure to acquire a clearcut unilateral dominance.

The question which arises from a study of the shortcomings of language as here presented is on the one hand largely that of evolution as applied to language, and on the other hand of devolution, retardation or modification as similarly applied.

Keeping closely to the question of cerebral localization in its relations to written language, I need recall only that the brain is supposed to be divided into numerous centers and areas which are associated anatomically and functionally.

The brain develops in accordance with the use to which it is put. The acquisition of brain power and control follows, according to Darwin, certain laws of inheritance which he includes under the term pangenesis. According to this theory of pangenesis, inheritance is the result of the transmission of cellules or gemmules which exist and persist in all parts and in every part of the body.

The subject of reversion is extremely important in this connection. Certain anatomic and functional acquisitions may be dormant for several generations, then be lost and later reassert themselves. It cannot be asserted too frequently that such reversion may take place after many

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years and many generations. As the brain is the dominating influence in transmission, this means in effect that a form of cerebral localization is at the root of the matter.

Some extraordinary illustrations of the tendency to return to ancestral languages might be adduced by me. One such illustration, which perhaps properly might be termed paleozoic, has come to my observation. To comprehend fully this example it may be necessary to consider important changes in the earth's surface and connections during the paleozoic and perhaps earlier or later periods. France, or at least Brittany, in ancient geologic times was terrestrially connected with Ireland, Wales, Scotland and England. A mountain chain called the Amorica extended from northwestern Ireland to Brittany.

As is well known, the language of Ireland has been much changed during the progress of time, especially in comparatively recent periods. St. Patrick brought to Ireland Roman language and ideas about 500 A. D. and changed the original Gaelic in a large degree. Many modifications have since occurred, especially under the English influence. The original or primitive language of the people is, however, to this day retained to some extent in parts of the island, and a revival of the old language has been attempted by Yates, Lady Gregory, Donn Byrne and others.

To return to the example to which reference has been made, a peculiar illustration of the language used by one from a certain part of Ireland and its comprehension by one born in Brittany has come under my observation. An intelligent woman of Breton birth had an Irish servant from Cork or the immediate neighborhood; this woman and the servant reciprocally could understand each other's language but others could not. Other Irish servants, as those from Dublin, spoke the usual brogue but understood nothing of Breton.

With regard to language reversals to which Dr. Orton refers and of which he gives many examples, I have had numerous illustrations. They are evinced both in oral and in written language. These reversals may be of the entire word, of syllables or even of letters, but it is especially shown in what might be termed syllabication. Examples in the cases to which I allude are the use of "onimus" for ominous, "resort" for restore, "restore" for resort and the reading or writing of "bab" for bad. These and many other examples which have come under my observation are not, as Dr. Orton has shown, the result of serious brain impairment or destruction but of a tendency to confusion in associating the mental images of words or of their constituent parts.

Manifestly it is of the greatest importance to teach children to recognize syllables as separate entities, and one of the best ways to do this is by repeatedly reading aloud. This as usually practised in

schools is not taught as it should be, although the fault may not be due to the teacher. Commonly, it is because the teacher has too many pupils to instruct.

What have been humorously called "blab schools" are schools of the worst sort. Children are urged to repeat after the teacher certain words or phrases and to read in concert words and phrases that are put on the blackboard. Instead of this, as far as possible defective pupils should be taught individually or in small groups.

Some of the evils both of writing and of pronunciation or of enunciation can be overcome only by frequent and correct repetition. The evils of confusion in language of school children and in later years of adults have been, as I have pointed out in other articles, the outcome of the so-called "see and say" methods.

Some of the efforts of ambitious innovators and would-be reformers in elementary and high school education undoubtedly are responsible for some of the shortcomings of the grammar and high school graduates. Personally, I believe with Dr. Orton that one has departed too much from what is sometimes spoken of as the old-fashioned methods of teaching. Spelling books and etymologies have been banished in large part from the schools, and instead an imperfect method of teaching has been substituted.

Phonetic methods are to a certain extent necessary, as for instance teaching children the proper pronunciation of such combinations of letters as "ch" and others which cause difficulties in enunciation and pronounciation. The derivation and definition of the meaning of words should be taught if the use of words and phrases is to be understood. A child or an adult cannot pronounce and use properly a word the meaning of which is not understood, or he can do this only by imitation without comprehension.

I have made numerous experiments in language with secretaries from the high schools and normal schools of Philadelphia. In one such recent case I found that the only way to bring about improvement was to have the secretarial reader keep a list and look up the meaning of all the words on which she stumbled, this list being considerable in an hour's reading. When the words occurred in later reading periods she had no difficulty in remembering their sound associations.

One reason why in older civilizations like that of France children are better informed in matters fundamental to language is that first they are drilled thoroughly in the rudiments of the language.

Formal conversation should be included in the curriculum of primary and secondary schools, private, public and parochial. The boy or girl who under the discipline and close scrutiny of the school room is taught the use of correct language both in speech and in writing often is inclined

to use slang or otherwise incorrect language as soon as he is relieved from observation and control. He revels in slangy expressions and foolish profanity.

Not long ago I was struck by the unusually correct method of conversation by a man and his wife following a humble but respectable vocation. Curiously, I never heard a word of slang in the business office of these people except as introduced to explain what some one else had said. The answer to my inquiries with regard to this matter enforced on me the important part played in home instruction. The father of the family in the case of the woman referred to was one of the rare examples who insisted on propriety of language.

As exemplified in this case, correct speech is not always used by those of scholastic surroundings or inheritance. While traveling abroad, I found that the English children as a rule were more precise and careful in their use of language, but this did not in any way indicate that they were intellectually brighter than the American children. The atmosphere with which the English boys and girls were surrounded was one that encouraged naturally the use of correct language. The teaching of Latin in English schools probably exerts an early formative influence on the speech of both children and adults.

Something has been done and more could be accomplished by the encouragement of the Boy and Girl Scout organizations as agents in the improvement of the proper use of language.

It has been objected that too much attention to methods of speech among children might lead to a form of pedantry and stilted expressions. When, however, speech becomes more fluent and fluid the result is not only desirable, but agreeable.

In the explanation of the disorders or difficulties of language exhibited in the children whom he studied, Dr. Orton lays great stress on the effects of interference between the activities of the two cerebral hemispheres. This is especially emphasized in the case of mirror writing and left-handed subjects. Possibly, it seems to me, he has not laid as much stress as he might have done on the extremely complicated cerebral mechanisms of language. The left hemisphere in the ordinary right-handed person is the leading hemisphere for speech, writing and reading. In the left-handed person, the right hemisphere has a tendency to take the leading part. Because of the manner in which the ordinary school education is conducted, the two hemispheres struggle with each other for dominance and give confusing results.

The subject of the dual activities of the two cerebral hemispheres is an important one in discussing the remarkable disorders of language exhibited by the school children studied by Dr. Orton. The two halves of the brain, as he has shown, differ little in weight and dimensions, and

yet that the left hemisphere is the dominating one as regards language in a great majority of people is one of the best known facts. If the right cerebral hemisphere is more or less dormant or inactive, in what way does it maintain its weight and general proportions?

Speaking broadly, the human being is both emotional and intellectual, the creature of feeling and of reasoning. Many facts seem to indicate that the right cerebral hemisphere takes a larger part than the left in emotional life. Even the most extreme localizationist recognizes that the brain acts as a whole as well as in its separate parts; to borrow a political term, it acts as a central federation as well as a state or local subdivision. One part of one hemisphere may be dominant in particular cases rather than the whole of one hemisphere. Either the sensory or perceptive, the association or conceptive, or the emitting or motor subdivision of the brain may be most affected in cases which are abnormal.

The question of so-called morons is highly illuminated by the observations of Dr. Orton. It is necessary only to call the attention of members of the society to the definition of a moron as one whose mentality is several years behind what it should be normally according to one's age. The child of 14 or 15, for instance, is mentally equipped as one of 9 or 10 years. Some of those to whom the term moron is loosely applied may be permanently defective, but they are not all of this class. In the cases of some morons, as in a number of children examined and recorded by Dr. Orton, the mentality of the child is delayed rather than destroyed. Many of the so-called morons may be possessors of sound brains after all. Not infrequently precocious children eventually are left behind by their slower classmates.

I have in former times paid much attention to different schemes of cerebral localization. I have shown in my presentation of some of these schemes that all percept or projection centers take part in language recognition, spoken or written. However, the concrete concept or association areas and the higher psychic regions of the brain concerned with perfecting language both by synthesis and by final analysis have a dominant influence in preserving language from impairment or destruction.

The whole of the dual and complicated cerebral apparatus may be involved more or less in strephosymbolia or the twisted language group. Any one concerned with researches such as that discussed in this article needs to have a thorough idea of the manner in which parts of the brain are segregated in centers or areas and also are united and associated when the organ functions as a whole.

Closely related in the cerebrum to the isolated centers for words, letters and numbers, all of which are separately represented, is an area for object imagery sometimes spoken of as the area of architectonic

form. Some of the cases studied by Dr. Orton have illustrated the presence in the cerebrum of this object area. Those patients whose general vision was clearly good recognized the form of objects presented whether these were words or objects in the more general sense. The form, however, seemed to be present in the minds of the patients either in the normal or the sinistral or in some other position.

Studies like those made by Dr. Orton, while of world-wide or international interest, are particularly of value to this conglomerate land, representing as it does the nations and subdivisions of nations of all parts of the world. Reversals to ancestral language may play their confusing part as in the case already given regarding the language of southwestern Ireland and Brittany.

One of the best signs of the times is the interest which the subject of education is taking. This interest is shown not only in teachers, in parents and in educational administrators but even in the children themselves. Systems of progressive elementary education have been discussed, advocated or opposed in whole or in part.

Recently this question of elementary education was presented in an article by Eunice Fuller Barnard in the *New York Times* (Sunday, April 2, 1929, Special Feature Section, p. 14). The advocators of educational reform naturally arrange themselves into two classes. Those in the language of political assemblies or chambers might be classed as radicals or the left and the conservatives or the right, with, as is always the case, an intermediate class tending to the left or the right.

The radical revolutionary reformers would make the children suggestors or leaders in experimentation. The instructor or teacher, instead of being an active director who cajoles or coerces the child, becomes a consultant to whom the child applies for guidance in the skilful technic necessary to accomplish the end in view.

From my knowledge of school children, it seems to me that conservative thinkers on education have the right point of view, who hold that children in the formative or reaching forward stage of development, for the majority of children say between the ages of 8 or 9 and 11 or 12, will be most helped by some guidance in fundamental matters. In other words, children need the guidance of older and wiser heads until they have passed or while they are passing through the difficulties which surround the acquisition of the three R's. After having mastered these the children can be allowed more freedom of thought and action in completing their elementary education.

Much has been said in the article to which I refer and in other similar articles on children making use practically of the education after which they are striving; in other words, they can play with advantage at being bankers, or merchants, or cooks and thus obtain and grasp

some knowledge on discount and cube root and domestic economy by using the tools which have been placed in their hands.

After all, discussions of this kind are those with which Henry Pestalozzi and his pupils enlightened the world more than 100 years ago.

ABSTRACT OF DISCUSSION

DR. BERNARD SACHS, New York: We as a group of men who are more or less interested in the education of the young should take exactly the point of view that Dr. Mills has taken. There is a great tendency in the present day to trust altogether to the experimental side of education and to do away with the guiding influence of the older teacher.

Personally, I was delighted to hear Dr. Mills express those views, and I hope that those members of the association who have an opportunity to exercise their influence in educational matters will take the advice Dr. Mills has given, or accept the views that he has given, that, after all, the child of the present day in school should be given the benefit of experience and not be subjected entirely to the modern experimental method of education.

DR. S. T. ORTON, New York: I have some hesitancy in discussing this particular topic because it is no near to my heart that I am afraid I shall not be able to stop in proper time, but there are several points which Dr. Mills has brought up which have interested me keenly. One of them, particularly, is the question of the relative importance of the hereditary patterns and of use or practice in education.

How much does one actually modify the inherent, hereditary patterns in a child when one educates him contrary to his natural physiologic bent? Can one make the other hemisphere take the lead in any one particular function or does one always merely precipitate difficulties when one educates him contrary to the native tendency?

That I can not answer. I have been very much interested in hereditary patterns, and at the end of this week shall make a report before the Eugenic Research Association concerning a series of families showing an intimate interrelation of left-handedness, stuttering, delayed speech, defective writing, very poor spelling and reading disability. Apparently, all of these things are closely interrelated genetically and occur in the same families in slightly different settings, so that they all seem to be expressions of this major problem of variations in cerebral dominance.

From working with these cases in considerable number, I believe that one must recognize that in the individual one is often dealing with a striking variant. In other words, one can train one left-handed child by standard methods with no difficulty, but with another one meets difficulties with exactly the same type of training. One must estimate the individual capacities with the greatest accuracy before one can say that this or that should be the method of education. And that bears somewhat on our criticism of the methods of teaching in vogue today. With some persons apparently the more modern method of teaching, the look-and-see method or the flash method of teaching reading, seems to be fairly satisfactory. I believe that those are children who already have their cerebral patterns fairly well established before they are subjected to reading, but with others this method seems to constitute an almost insuperable obstacle. I have published figures to show that the elective use of the sight method of teaching reading probably triples the number of reading disability cases in a school system

in which no phonetic training is permitted. In other words, there are probably three times as many cases blocked in reading if the sight method is used exclusively. On the other hand, many of the normal children of that group may learn to read more quickly and better by that method.

When one finds a child who has been taught by the sight method of learning to read but who has made poor progress, one very often finds curious things cropping up in his language. Thus a whole word may be planted in lieu of a sound, so that he knows only "you" for the sound of the letter "u" and he spells words with the "you" written into them; for example, "education," spelt "egyoucation," and "documents" spelt "dockyouments." On the whole, word plants of that sort are frequently found.

One small boy in attempting to read for me the word "hostility" pronounced it "hostlostility." On questioning as to how he got "hostlostility" out of it, he said, "I never saw the 'h-o-s-t' before, but I did know 'l-o-s-t.' I took the 'l' off 'lost' and put 'h' in front of it." Obviously, after he made that start with "host," the "lost" was still reverberating and when he came to the "l" later in "hostility" it brought back again that "lost" sound, and he produced the "host-lostility."

As a whole, I believe one must go rather cautiously in either one's acceptance or one's rejection of various methods. We have a highly individual problem, and I believe that before he is damned as a defective each child of this nature, who is not learning in a specific part of the language zone, should be submitted to a careful analytic test to find out whether his educational processes in the past have been adequate to give him the materials that he needs to learn language and its proper usage.

DR. BERNARD SACHS: May I request that the gentlemen present take a serious interest in this matter in their various communities?

I have personally known this matter of teaching children to learn by the sight method to be responsible for the fact that some children are supposed to be defective. So many of us are faced with the problem of determining whether a child in the early school years is defective or not. And I wish to say in this connection that what is supposed to be the newest educational method is doing mischief throughout the country, and this group of men can be helpful in applying a sober criticism to the educational methods, particularly as regards the learning of reading. It is really an important duty that we have to perform, individually, in the various communities in which we live.

PARESIS TREATED WITH MALARIA

THE RELATIONS BETWEEN CLINICAL, SEROLOGIC AND HISTOLOGIC OBSERVATIONS, WITH SPECIAL REFERENCE TO PERMEABILITY DETERMINATIONS *

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During recent years, the methods of research in the treatment for paresis as a syphilitic disease of the central nervous system have been associated with concomitant studies on the serology and pathologic histology of this disease with two main purposes in view: (1) to determine the mode of action of the different therapeutic agents, and (2) to establish some tangible guides of prognostic value. For the latter purpose, the different methods of examination of the blood and cerebrospinal fluid seemed to promise particularly good results. If the serologic tests would prove as valuable guides to the progress in cases under treatment as they did as aids in differentiating some neurosyphilitic diseases from others and neurosyphilis as a group from other organic diseases, one would have definite criteria for the management of each case under treatment. The advent of the malarial and allied nonspecific methods of therapy has given a new impetus to this work. On the one hand, these methods have shown definitely that paresis is not necessarily a hopeless condition, and that in a surprisingly large percentage of cases not only short respites, but even lasting remissions (even if it is too early to say complete cures) can be effected during which the patient may return practically to his previous social level. On the other hand, however, this form of treatment was accompanied by more dangers and taxed the patient's strength to a greater extent than any previous method. These two factors made it imperative to find methods whereby one can tell whether the disease process has been arrested, so that the patient can be allowed to recuperate from the previous effects of the disease, as well as from the effects of this rather drastic method of treatment. If it has not been arrested, a course of the same or some other treatment could be undertaken so as to prevent further damage to the patient's constitution.

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Three fields of study offered possibilities for the discovery of such criteria: (1) observation of the clinical course before, during and following the treatment for the possibility of detecting certain features in it that would help one to foretell the probable course in other cases; (2) as already mentioned, investigation of the blood and cerebrospinal fluid before, during and after treatment to discover the relation between changes in these and the outcome of the disease; (3) the histologic method, in which the central nervous system of patients who have died at different stages of the disease is studied to discover the actual occurrences in the body during the treatment with a view of creating rational bases for standards found in the first two fields. It goes without saying that no matter where one might find such indicators of the course of the disease, their practical value and reliability could be definitely established only on the basis of a study of all these fields and that no definite conclusions could be drawn on the basis of isolated studies in any one.

It soon became apparent that the clinical picture following malarial or any other treatment for paresis was far from offering any constant features. Many times, cases that showed practically the same picture before treatment showed different reactions following it. Some would show almost immediate improvement, which would gradually increase to complete recovery and lead to a lasting remission. Others would show no signs of any appreciable improvement for several months, and then possibly the patients would begin to improve, while still others with immediate improvement would suffer a relapse and return to the pretreatment level. The clinical picture in itself, therefore, is not sufficient as a prognostic criterion. This variability in the clinical course naturally led to an investigation of the serology of these patients. The rather discouraging observations reported by Viennese investigators[1] in the early days of malarial treatment were subsequently shown to be unfounded, and as time went on different observers reported definite changes in the serology of patients with paresis treated with malaria. At present, most investigators are of the opinion that practically all patients with paresis treated with malaria show changes in the serology. This fact, however, would leave one in practically the same place as if there had not been any serologic criteria. For unless these changes could be shown to be in any way related to the outcome of the treatment, one could not utilize them. The reports of investigators in this field differ. Bunker,[2] for instance, found that some of the serologic changes which occur after malaria are of negative

1. Gerstman, J.: Ueber den jetztigen Stand der Malariatherapie der progressive Paralyse, Ztschr. f. d. ges. Neurol. u. Psychiat. **81**:255, 1923.

2. Bunker, H. A., Jr.: Influence of Malarial Treatment on the Spinal Fluid in General Paralysis, Arch. Neurol. & Psychiat. **19**:478 (March) 1928.

importance; that is, when certain tests (cell count and total protein) do not change following the treatment, the prognosis is poorer than in others. On the other hand, Ferraro and Fong [3] found that although changes in all tests occur ultimately in all patients treated with malaria, they are not definitely related to the clinical outcome. They cited cases in which the serology improves concomitantly with clinical improvement, as well as cases in which the reverse is true. (Incidentally, if there are cases in which clinical improvement is accompanied by serologic tests remaining as before treatment, then the generalization that in all cases following malaria there is a serologic improvement is not true.)

It must be remembered that in studying the changes in the blood and cerebrospinal fluid in these cases one must be careful not to accept the possible resultant of several methods as the effect of any one method of treatment. Bunker's stipulation (this was also suggested by Ferraro and Fong) that only cases in which no other treatment has been administered following the course of malaria should be used in such investigations is, of course, a distinct advance in this line of research, because the effects of the other agents undoubtedly change the situation a great deal. We would state further that it would be still more desirable to utilize for such research only cases in which no other treatment has been administered either before or after the malarial therapy. This would make it so much more probable that whatever observations are made are the effects of the treatment in question.

Attempts to approach this problem by means of histologic investigation have also not led to any definite or universally accepted conclusion. As in the case of serologic investigation the facts that are generally agreed on seem to be of little help, as they do not bear any practical relation to either the clinical or the serologic picture. Thus, the only change in the histologic picture which according to most observers follows malarial treatment is that of inflammation of the mesodermal elements.[4] Conclusions based on histologic changes in cases of paresis that are fatal at any stage during or after malarial treatment are even more risky than those in serologic studies. Primarily, most of the cases reported showed no improvement, and clinically the paretic process was apparently not influenced. Secondly, in all cases one is dealing with the end-process without any possibility of stating just what the condition was in this particular case at any

3. Ferraro, A., and Fong, T. C. C.: The Serology of General Paresis in the Malaria Treated Cases, M. J. & Rec. **124**:562, 607, 682, 1926; The Malaria Treatment of General Paresis, J. Nerv. & Ment. Dis. **65**:225, 1927.

4. Ferraro, A.: The Pathology of Paresis After Treatment with Malaria, Arch. Neurol. & Psychiat. **21**:69 (Jan.) 1929.

stage before death. One must remember that the histologic picture in paresis is far from being always the same and, as Spielmeyer especially has shown, is not invariably related to the clinical picture. Both the mesodermal and the ectodermal components may be intense in cases that show a comparatively mild disease process clinically, and vice versa.

If one wishes to utilize the knowledge gained by histologic studies for a better understanding of the course of the disease and the efficacy of any method of treatment, one must be able to find some definite correlation between objective manifestations observable during life and data obtained from a postmortem examination. Purely clinical data apparently fail here just as they do in indicating the probable prognosis. Therefore, in this aspect of the problem too, we feel forced to search for such manifestations in the blood and in the cerebrospinal fluid. In other words, the solution of the problem depends on the discovery of some serologic reactions that could be carried on at different stages of the disease and that could be shown to be related to some components of the histologic picture. If, furthermore, fluctuations in such reactions could be found to be related in a definite form to the clinical course and prognosis, one would thus obtain not only a practical guide in treatment, but a link between the clinical, serologic and histologic components of the disease. In the present communication, we propose to discuss the practical value of the examination of the passage of chemicals from the blood into the cerebrospinal fluid as such a test.

CLINICAL DATA

Our material consists of the first fifty-four cases of neurosyphilis in which treatment at this hospital was given with tertian malaria. Forty-four of the patients were males, and ten females; forty-eight had paresis and six, other forms of neurosyphilis. Of these, thirteen patients received no other treatment either before or after the malaria treatment; eighteen were previously treated by other methods but received no other treatment following the malarial method; in twenty-three the malarial treatment was followed by some form of chemotherapy, in most cases tryparsamide. In our report on the serologic investigations, we will therefore have an opportunity to study the results as they occur with or without additional treatment. All of these patients were observed for periods ranging from six to twenty-seven months. (In only eight of the fifty-four cases was the treatment discontinued less than twelve months before the conclusion of this study.) According to the clinical course and final outcome, we have divided our cases into five groups.

1. In group 1, consisting of fourteen cases (26 per cent), are included all patients who improved practically to a predisease level,

and who on discharge took up positions they held before the onset of the present illness.

2. In group 2, consisting of sixteen cases (30 per cent), the patients showed definite improvement of different degrees without reaching their prepsychotic level. A number of these were discharged, and some of them are holding positions that they had before the onset of the psychosis.

3. In group 3, consisting of four cases (7 per cent), the patients showed no definite improvement, but the progress of the disease was apparently arrested.

4. In group 4, consisting of fifteen cases (28 per cent), the disease showed further progress. This group includes a number of patients who at first showed definite improvement (to an extent that enabled them to be discharged from the hospital and to work for a period of time outside), but who later suffered a relapse. In these cases, which showed especially pronounced oscillations in the clinical course, a search for concomitant fluctuations in the serology was particularly indicated, and we will return to them in our discussions of the serologic data. Three patients in this group (cases 4, 5 and 13 in table 4) died. As they were cases in which the malaria clearly had nothing to do with the outcome and we felt that we were dealing with deaths due to paresis, we have not included these in the next group.

5. In group 5, consisting of five cases (9 per cent), the patients died during or shortly after the malarial treatment.

As numerous communications have been made on clinical observations of patients with neurosyphilis treated with malaria and as excellent reviews and discussions of these are available in the literature, we thought it best to confine ourselves to data observed. Our patients were all inoculated with tertian malaria directly from other patients, and practically all by the intravenous route. The number of chills ranged from nine to twenty. Jaundice in three cases and intense vomiting in one were the only outstanding complications. The heart action, blood pressure and blood picture were checked up as indicators for early discontinuation. We experienced no difficulty in checking the fever by oral administration of quinine sulphate, 10 grains (0.6 Gm.) three times daily for a week. Our indication for a second course of malaria or the carrying out of other treatment following the malaria will be discussed later. It is important to realize that discrepancies between the proportions of cases in which improvement occurred in the reports of different authors may be largely dependent, as Geratovitsch [5] has recently pointed out, on the material that one has to

5. Geratovitsch: Ueber Malariatherapie bei Paralyse, Arch. f. Psychiat. **78:** 64, 1926.

deal with. Thus, for instance, our material, representative as it is of cases in state hospitals, includes a large number of patients with old chronic forms of the condition of the simple dementia type, as well as some who belong to the stationary group who have been treated in different ways for a number of years and in whom the ectodermal elements have been damaged to such an extent that little improvement can be expected. Similarly, the choice of material here is limited; all one can hope in some of these cases is an arrest or slight improvement. These facts tend to raise the percentage of the last three groups as compared, for instance, with cases from clinics in which fresh material is generally available. One must remember, however, that from a practical point of view the fact that one can change the clinical picture from that of untidy, excited, confused, and at times violent behavior to that of a type which would be compatible with the patient fitting into the hospital group or even carrying on some work about the hospital is in itself a desirable result.

SEROLOGIC OBSERVATIONS

In the charts we have tabulated the results of tests on the blood and cerebrospinal fluid carried out and reported as follows:

1 and 2. The Wassermann Reaction of the Blood and Spinal Fluid.—These tests were not done at the hospital but were made by the Massachusetts Department of Public Health and were reported to us in terms of plus, doubtful or minus.

3. The Cell Count: The cell count was carried out in a Fuchs-Rosenthal chamber.

4. Cerebrospinal Fluid Albumin: The amount of albumin in the cerebrospinal fluid is reported in terms of degrees of intensity of reaction to trichloracetic acid. A normal opalescence is indicated by +; ++++ indicates that there is a heavy precipitate, and ++ and +++ ranges between the two.

5. Cerebrospinal Fluid Globulin: The globulin in the cerebrospinal fluid is reported in terms of degree of intensity of reaction to ammonium sulphate. When there was no change of color, the test was reported as negative. A slight suggestion of a change was reported as "s" and a heavy ring reported as +++, + and ++ ranging in between.

6. Cerebrospinal Fluid Colloidal Gold Curve: The colloidal gold curve was reported as usual in terms of the change of color in the ten tubes.

7. Permeability Index: In the previous communication, we[6] reported our technic and the results of an investigation of the blood-cerebrospinal fluid barrier by means of Walter's bromide method. This consists in administering to the patient by mouth 0.015 Gm. of an aqueous solution of sodium bromide per pound weight over a period of five days, at the end of which the blood and cerebrospinal

6. This paper is the second communication on the study of the barrier between the blood and the cerebrospinal fluid, the first communication being by Malamud, William; Fuchs, D. M., and Malamud, N.: Changes in Permeability in Mental Disease, Arch. Neurol. & Psychiat. **20**:780 (Oct.) 1928.

fluid are taken at the same time and examined for the amount of bromides they both contain. Normally, the blood contains about three times as much bromide as the cerebrospinal fluid, and this gives an index of 3 for the normal. When the barrier is disturbed in such a fashion as to admit more bromides into the cerebrospinal fluid, one gets a permeability index that is lower than 3, and when the reverse is true, the permeability index is higher than 3. In the communication referred to, we reported, among others, results of such examinations in patients with paresis, and we found that in all cases of paresis in which treatment was not given, the permeability is increased and the index is generally below 3. Our experience with malarial treatment at that time was not sufficient to cause us to make any conclusive statements outside of the fact that in most patients successfully treated with malaria we found a tendency of the permeability to decrease toward the normal. In the present communication we wish to report the permeability, therefore, in terms of permeability index. We consider fluctuations between 2.8 and 3.2 as within normal limits. Although these limits are not universally accepted, it is agreed generally that a permeability index below 2.8 denotes a pathologic increase in passage of substances through this barrier.

All these serologic examinations were undertaken at regular intervals as follows: (1) immediately preceding malarial treatment; (2) directly following the malarial treatment; (3) two months after the conclusion of the treatment; (4) six months after conclusion of treatment, and then at regular intervals of three months each. The tests of permeability index were started a few months after the beginning of this investigation, and therefore in some of the cases these indexes are not available for the beginning of the treatment. As already mentioned, in some of our cases the patients have had only malarial treatment, others have had no treatment following malaria, although they have received treatment before, and in a third group the course of malaria was followed by supplementary treatment, usually tryparsamide. In reporting our serologic results, according to the different clinical groups, we will keep these three types apart and designate them as such. In some of our cases in which the patients have been either discharged or transferred to other hospitals and can be followed clinically, it is difficult to have the serologic investigation followed, and the results are therefore given up to the time of discharge from this hospital.

In the following tables are given the results of these tests in the first four groups. Each chart is divided into seven parts, corresponding to the number of tests. In each part, the whole series of the particular test is reported on all patients in this group. The general information, consisting of the initials of the patient, sex, and the period of time that has elapsed since the conclusion of treatment in months, is indicated in each chart on the left of the first test (the Wassermann reaction of the blood). The patients are also indicated by numbers, and in the subsequent tests those numbers alone are given, all other data being omitted. As already mentioned, we also divided our material in each group into subgroups with regard to other treatment (outside

TABLE 1.—*Data in Group 1*

	Initials	Sex	Months after Treatment		Blood Wassermann: Before Malaria	After Malaria	2 Mo.	6 Mo.	9 Mo.	12 Mo.	15 Mo.	18 Mo.	21 Mo.	24 Mo.
1	J. A. W.	M	20		+	+	+	+	+	+	..	+	..	..
2	J. W. D.	M	13		+	+	+	—	—	..	..	..	..	..
3	L. M. W.	F	26		—	—	—	—	—	—	—	..	..	..
4	W. H. G.	M	21	No treatment other than malaria	+	+	+	+	+	..	..	..	..	..
5	A. I.	F	8		+	+	+	+	..	..	..	..	..	..
6	W. M. McI.	M	27		+	..	—	..	Doubtful	—	—	—	—	—
7	W. F. D.	M	24		+	+	..	..	..	..	..	..	..	..
8	D. A.	F	21		+	..	+	+	..	+	..	..	..	..
9	F. E. C.	M	10		+	Doubtful	—	—	—	..	..	..	..	..
10	L. StP.	F	18	Treated before malaria	+	+	..	+	+	..	+	—	+	..
11	H. E. P.	M	13		—	—	—	—	—	..	..	..	..	..
12	M. D.	F	14		+	+	+	+	+	..	..	..	..	..
13	T. L. B.	F	9	Treated before and after malaria	+	+	+	+	..	..	..	..	..	..
14	E. W. C.	F	25		+	..	Doubtful	+	..	—	—	..	—	—
					Cerebrospinal Fluid Wassermann									
1					+	+	..	+	+	+	..	..	..	..
2					+	+	..	+	Doubtful	..	..	..	..	..
3					+	+	+	+	+	+	—	Doubtful	?	—
4				No treatment other than malaria	+	+	+	+	Doubtful	..	..	..	..	..
5					+	+	+	Doubtful	..	..	..	..	..	..
6					+	+	Doubtful	+	+	—	—	—	—	—
7					+	+	+	—	..	..	..	..	..	..
8					+	+	+	+	..	+	..	..	..	..
9					+	+	+	+	—	..	..	..	..	..
10				Treated before malaria	+	+	..	+	+	..	Doubtful	+	+	..
11					Doubtful	—	—	Doubtful	?	..	..	..	..	..
12					+	+	+	+	+	..	..	..	..	..
13				Treated before and after malaria	+	+	—	—	..	..	..	..	..	..
14					+	..	Doubtful	Doubtful	—	—	—	..	—	—
					Cell Count									
1					80	8	45	?	16	10	..	?	..	..
2					20	5	0	5	6	..	..	..	..	..
3					10	5	2	3	3	2	2	4	1	2
4				No treatment other than malaria	25	4	40	50	7	..	..	..	..	..
5					78	2	2	2	..	..	..	..	..	..
6					10	3	3	2	2	1	2	2	0	1
7					150	45	4	7	..	..	..	..	..	..
8					18	5	3	2	..	4	..	..	..	..
9					75	4	1	0	2	..	..	..	..	..
10				Treated before malaria	5	2	..	2	1	..	1	2	2	..
11					3	4	5	1	1	..	..	..	..	..
12					18	15	?	60	..	..	..	..	..	..
13				Treated before and after malaria	110	10	5	1	..	..	..	..	..	..
14					3	..	4	3	1	2	1	..	4	2
					Albumin									
1					3+	3+	3+	2+	2+	2+	...	2+	...	...
2					3+	2+	?	3+	2+	...	...	...	...	...
3					2+	3+	3+	3+	3+	?	2+	2+	+	+
4				No treatment other than malaria	3+	3+	3+	3+	3+	...	...	...	...	...
5					2+	2+	+	+	...	...	...	...	...	...
6					3+	2+	2+	3+	2+	2+	3+	2+	...	2+
7					2+	2+	2+	2+	...	...	...	...	...	...

7.		2+	2+	2+	2+	...	...	...	...	...	...	

8.		3+	3+	3+	3+	...	2+	...	...	...	...	
9.		2+	2+	2+	+	2+	...	...	...	...	...	
10.	Treated before malaria	3+	3+	...	3+	2+	...	3+	3+	2+	...	
11.		2+	2+	2+	+	2+	...	...	...	...	...	
12.		3+	3+	2+	2+	...	...	...	...	...	...	
13.	Treated before and after malaria	2+	2+	2+	+	...	...	...	...	...	...	
14.		3+	...	3+	3+	...	3+	+	...	+	2+	
						Globulin						
1.		3+	2+	2+	3+	+	8*	...	8	...	...	
2.		2+	+	+	+	2+	...	...	...	...	...	
3.		+	2+	+	+	2+	+	2+	+	—	+	
4.	No treatment other than malaria	3+	2+	2+	2+	2+	...	...	...	...	...	
5.		+	8	—	—	...	...	...	...	...	...	
6.		+	+	+	+	+	+	2—	+	—	+	
7.		+	+	+	—	...	...	...	...	...	...	
8.		3+	2+	2+	2+	...	2+	...	...	...	...	
9.		8	—	8	—	8	...	...	...	...	...	
10.	Treated before malaria	3+	2+	...	+	+	...	+	2+	+	...	
11.		+	—	—	—	+	...	...	...	...	...	
12.		2+	2+	+	+	...	...	...	...	...	...	
13.	Treated before and after malaria	2+	3+	+	8	...	...	...	...	...	...	
14.		+	...	+	+	+	2+	+	...	+	—	
						Permeability Index						
1.		189	248	264	353	280	303	...	321	...	...	
2.		201	219	246	305	363	...	...	...	...	...	
3.		...	...	...	...	...	375	401	354	343	382	
4.	No treatment other than malaria	257	...	277	287	332	...	...	...	...	...	
5.		261	314	304	301	...	...	...	...	...	...	
6.		...	...	...	312	...	...	318	336	300	306	
7.		...	...	...	...	...	...	...	...	...	...	
8.		270	...	355	378	...	313	...	...	...	...	
9.		254	368	308	312	330	...	...	...	...	...	
10.	Treated before malaria	303	275	...	338	332	...	344	321	342	...	
11.		274	235	286	297	300	...	...	...	...	...	
12.		200	289	260	247	...	...	...	...	...	...	
13.	Treated before and after malaria	163	...	247	257	...	...	...	...	...	...	
14.		...	...	...	...	265	351	295	...	220	300	
						Colloidal Gold Test						
1.		5555554200	5555543200	4555543210	5554432000	2334444320	1223210000	.....	?	.....	.....	
2.		5555554200	4555554300	0111434130	1223444210	5555553220	.....	.....	.....	.....	.....	
3.		5555543110	5555542200	5555432000	5555442100	5555432000	5554441100	5555222000	2544421100	1133441100	001222100	
4.	No treatment other than malaria	4555554310	5555543100	5555543210	5555543100	1221321000	.....	.....	.....	.....	.....	
5.		4432110000	1222322000	0000121000	0000111000	.....	.....	.....	.....	.....	.....	
6.		5555541000	2555541000	2555541000	2555541000	2455542100	4013333100	5555552200	1255443300	0122332200	001222100	
7.		5555543100	3355531000	1444333000	1123330000	.....	.....	.....	.....	.....	.....	
8.		5555555310	5555543000	5555554300	5555554320	.....	2555552000	.....	.....	.....	.....	
9.		0124111000	0122142200	1333322000	0011110000	1122220000	.....	.....	.....	.....	.....	
10.	Treated before malaria	5555442000	5555541100	.....	5555554210	1223543300	.....	4555544210	5555553210	4544433000	.....	
11.		0123321000	0001211000	1223322000	0123340000	1334433100	.....	.....	.....	.....	.....	
12.		5555543210	5555554400	1225543100	5555544300	.....	.....	.....	.....	.....	.....	
13.	Treated before and after malaria	0011232200	1334433100	0012332200	0112233110	.....	.....	.....	.....	.....	.....	
14.		5555510000	.....	2555542000	2344551100	5533440000	3224331000	0122322100	.....	1112230000	.....	

* In this table and in tables 2, 3 and 4, s indicates a slight suggestion of a change.

of malaria). These, too, are indicated in the report of the Wassermann reaction of the blood.

Analysis of Results in Group 1.—As can be seen, there is a tendency to improvement in all reactions as time goes on. The first to change is apparently the cell count, although the pleocytosis does not always disappear at once. In most cases, however, that are observed for a long time this usually reaches the normal limit. A pleocytosis at any time following the treatment does not in itself, however, mean a bad prognosis. Cases 1, 4 and 12 speak against that. The colloidal gold test is one of the last to show the change, and, as seen from this group, a positive result can persist for a long time (as, for instance, in case 10) concomitantly with a good clinical result. As to the permeability index, it will be noted that all cases, especially those in which no other treatment was given, show a low index (high permeability) before treatment. This may or may not decrease more immediately after the cessation of the paroxysms, but following this and early in the disease there is a gradual increase in the index, which goes up to normal and in some cases swings over to the other extreme. Only two of the fourteen cases show a permeability index lower than normal at the end of the sixth month, but even in these the index is higher than it was before treatment. This increase in the index is also noticeable at the end of two months, although at that time the index has not yet reached the normal in most cases. After six months, the indexes are practically all normal in this group.

Analysis of Results in Group 2.—Group 2, on the whole, appears somewhat similar to the first. In all tests except the permeability index, one finds a general similarity to those in group 1, but, surprisingly, with a much more pronounced tendency to negative results. This may be explained partly by the fact that there are more patients who have had some form of therapy before the malarial, and thus the serology was influenced and rendered negative even before the malarial treatment. This apparently has not influenced the permeability index to any extent, however, as all cases except one showed a permeability index below normal (an increased permeability) before malarial treatment. The permeability indexes following malarial treatment are generally in keeping with those of table 1, but do not show quite so pronounced a tendency to return to normal. Here, as in table 1, one finds at times a decrease in the index directly after the treatment, and then an increase toward normal at the beginning of the second month. This increase, however, is not so marked, and at the end of the sixth month one finds four cases in which the index is still below normal, although higher than the original. Indexes below normal are found occasionally even at the conclusion of the observation.

Analysis of Results in Group 3.—Here, too, there is no appreciable difference in any test except the permeability index as compared with tables 1 and 2. The colloidal gold curve shows, if anything, a greater tendency to decrease. The albumin, on the other hand, is the only one to stay high. Here, too, the question of treatment before the malaria has to be considered as of influence in some cases. The permeability tends to remain stationary, although in some cases it is increased. In others, however, it fluctuates, tending to a level lower than normal.

Analysis of Results in Group 4.—In group 4, in which the disease process has apparently progressed, the results of all tests other than permeability do not materially differ from those in group 1. Even in the cases in which the patients have received no other treatment following malaria there seems to be the same tendency to an improvement in the serology (outside of the permeability index), although there is a progress of the disease clinically. Even the albumin reactions show a gradual tendency to amelioration, and even if they are still positive at the end of the observation period, they are not essentially different from the cases of group 1. The permeability index, however, shows a different picture. In all cases there is a tendency to lowering of the index, which finally reaches a definitely pathologic level. It is regrettable that we have no pretreatment indexes in most of these cases. It is of especial interest to note here that the permeability indexes show fluctuation to and from the normal limits. These were not only parallel to the fluctuations in the clinical course but, in most cases, the decrease or increase in the index preceded the corresponding change in the clinical course, thus serving as a good indication of what we might expect as results of our treatment. This correlation was of most interest and value in the cases of those patients who improved following the malarial treatment but who afterwards suffered a relapse (cases 10 to 15, inclusive.) In these there are concomitant rises and falls in the permeability index and clinical picture. A case illustrating this particularly well is the following:

REPORT OF CASE

History.—R. R. S., a man, aged 38, was admitted to the hospital on Oct. 21, 1927. His family history was not significant. His early development was normal, and he had worked up to a good position as a salesman. He was of a normal type of personality. The date of the initial infection is unknown, but for about a year and a half before admission he began to show difficulties in gait, headaches, forgetfulness, and at times excitement with a tendency to violence.

Examination.—On admission, he was confused and irrelevant; there was a pronounced memory defect; his judgment was poor, and he had no insight into his condition. There were definite auditory hallucinations and a speech defect. Physically, the outstanding manifestations were unequal pupils that did not react

TABLE 2.—*Data in Group 2*

	Initials	Sex	Months after Treatment		Before Malaria	After Malaria	2 Mo.	6 Mo.	9 Mo.	12 Mo.	15 Mo.	18 Mo.	21 Mo.	24 Mo.
Blood Wassermann														
1	H. B. C.	M	20	No treatment other than malaria	+	+	—	..	..	..	..	..	..	..
2	A. G. S.	M	10		+	Doubtful	Doubtful	Doubtful	—	—	..	..	..	..
3	J. H. G.	M	9		+	+	..	+	..	..	..	.	..	..
4	P. K.	M	12		+	+	..	+	..	..	..	..	..	..
5	L. A. B.	M	11	Treated before malaria	—	—	—	—	—	..	..	..	..	..
6	A. F.	M	24		+	Doubtful	..	—	—	Doubtful	..	..	..	..
7	H. G.	M	25		—	—	—	—	..	..	..	..	..	..
8	R. L. F.	M	20		—	—	—	—	.	..	..	..	..	..
9	A. P.	M	10		Doubtful	—	—	—	..	..	..	—	..	..
10	G. H. C.	M	26		—	..	—	—	..	..	..	.	..	..
11	T. R.	M	25		Doubtful	Doubtful	—	..	—	—	—	..	..	—
12	W. E. P.	M	19	Treated before and after malaria	+	+	—	+	..	..	..	..	—	..
13	J. H. C.	M	12		+	+	+	..	+	..	..	..	..	..
14	G. H. B.	M	21		—	..	..	..	—	—	..	..	..	..
15	G. L.	M	14		+	Doubtful	Doubtful	—	—	Doubtful	..	..	..	..
16	C. E. D.	M	26		+	+	+	+	..	?	+	..	+	..
Cerebrospinal Fluid Wassermann														
1				No treatment other than malaria	?	+	Doubtful	..	..	..	..	..	..	..
2					+	+	Doubtful	—	—	Doubtful	..	..	..	..
3					+	+	..	+	..	..	..	..	..	..
4					—	—	..	—	..	..	..	..	..	..
5				Treated before malaria	+	+	+	+	+	..	..	..	..	..
6					+	+	+	—	+	—	Doubtful	..	—	..
7					—	—	—	—	..	..	..	..	..	..
8					+	+	Doubtful	—	..	..	..	..	..	..
9					+	+	+	+	..	..	..	—	..	..
10					+	+	Doubtful	Doubtful	Doubtful	—	—	..	..	—
11					—	—	—	..	..	—	—	..	—	..
12				Treated before and after malaria	—	—	—	—	..	..	..	..	..	..
13					+	?	+	+	+	..	..	..	..	..
14					+	+	..	..	—	..	..	..	..	..
15					+	?	—	—	Doubtful	+	..	..	..	..
16					+	+	+	+	..	—	—	..	+	..
Cell Count														
1				No treatment other than malaria	45	9	4	..	..	..	..	..	..	..
2					20	4	3	1	4	2	..	..	..	..
3					20	4	..	2	..	..	..	..	..	..
4					1	3	..	0	..	..	..	..	..	..
5				Treated before malaria	40	30	4	5	1	..	..	..	..	..
6					20	3	6	4	2	2	2	2	3	..
7					3	5	2	1	..	..	..	3	..	..
8					75	16	5	3	..	..	..	..	..	..
9					20	4	5	5	..	..	..	..	..	..
10					15	8	8	5	13	2	0	.	..	1
11					5	2	4	2	1	5	1	..	6	..
12				Treated before and after malaria	1	7	2	2	..	..	..	..	..	..
13					1	6	25	1	3	..	..	.	..	..
14					2	3	..	..	2	2	..	1	..	..
15					30	2	4	2	0	2	..	..	..	..
16					20	6	6	6	..	1	?	..	2	..
Albumin														
1				No treatment other than malaria	4+	3+	2+	..	..	..	..	..	..	..
2					+	3+	3+	+	2+	2+	..	..	..	..
3					3+	?	..	3+	..	..	..	..	..	..
4					+	+	..	+	..	..	..	..	..	..
5				Treated before malaria	3+	3+	2+	2+	2+	..	..	..	..	..
6					3+	3+	3+	?	3+	2+	2+	2+	2+	..
7					+	+	+	?	..	..	..	..	..	..

1	No treatment other than malaria	3+	3+	3+	3+	2+	2+	...	...	...	...
2		3+	3+	3+	?	...	...	...	...	...	...
3		3+	?	...	3+	...	...	...	...	...	...
4		+	+	...	+	...	...	...	...	...	...
5	Treated before malaria	3+	3+	2+	2+	2+	...	...	...	...	...
6		3+	3+	3+	?	3+	2+	2+	2+	2+	...
7		+	+	+	?	...	...	...	...	...	...
8		2+	2+	?	+	...	...	...	..	...	...
9		3+	2+	2+	2+	...	...	...	...	...	...
10		3+	3+	2+	2+	+	+	+	...	...	2+
11		...	+	2+	2+	2+	+	+	...	+	...
12	Treated before and after malaria	2+	2+	+	+	..	...	...	...	...	...
13		4+	3+	3+	2+	2+	...	...	...	...	..
14		3+	3+	...	...	2+	2+	...	2+	...	...
15		3+	2+	+	2+	2+	2+	...	...	...	...
16		2+	2+	2+	2+	...	2+	2+	...	+	...
						Globulin					
1	No treatment other than malaria	3+	+	+	...	...	...	...	...	...	...
2		+	+	+	+	8	8	...	...	...	...
3		3+	+	...	+	...	..	...	...	...	...
4		—	—	...	—	...	...	...	...	...	...
5	Treated before malaria	3+	3+	2+	2+	2+	...	...	...	...	...
6		2+	2+	2+	+	2+	+	+	2+	—	...
7		—	—	—	—	...	...	...	...	...	...
8		+	+	—	—	...	...	...	8	...	...
9		+	+	—	2+	...	...	...	...	...	...
10		—	+	8	8	+	+	8	...	...	—
11		..	—	—	8	8	+	8	...	8	...
12	Treated before and after malaria	—	8	—	—	...	...	...	...	...	...
13		4+	2+	2+	+	2+	...	...	...	...	...
14		8	+	...	...	—	8	...	8	...	...
15		2+	8	8	+	+	—	...	...	...	...
16		+	+	+	8	...	+	8	...	8	...
						Permeability Index					
1	No treatment other than malaria	222	287	300	...	...	...	...	...	...	...
2		227	198	250	285	237	306	...	...	...	...
3		242	203	...	270	...	...	...	...	...	...
4		264	274	...	307	...	...	...	...	...	...
5	Treated before malaria	212	194	240	252	345	...	...	...	...	...
6		...	...	269	282	304	286	304	247	323	...
7		...	...	...	354	...	...	...	...	...	...
8		240	257	268	290	...	...	...	263	...	...
9		382	249	285	301	...	...	...	...	...	...
10		...	...	...	267	260	282	247	...	...	280
11		...	...	313	300	306	294	428	...	280	...
12	Treated before and after malaria	254	199	293	321	...	...	...	...	...	...
13		255	263	282	278	285	...	...	...	...	...
14		260	228	...	...	267	259	...	295	...	...
15		244	277	276	311	284	365	...	...	...	...
16		...	...	...	342	...	331	325	...	300	...
						Colloidal Gold Test					
1	No treatment other than malaria	5555553300	3555554100	5555442000	.....	.....	.....	.....	.....	.....	.....
2		4444322000	3555544310	5554340000	0023341000	4544330000	5554330000	.....	.....	.....	.....
3		5555554410	1244433000	.....	5553331000	.....	.....	.....	.....	.....	.....
4		0001110000	0001110000	.....	0001000000	.....	.....	.....	.....	.....	.....
5	Treated before malaria	1255544100	1232332200	4555551000	1233555410	0122210000	.....	.....	.....	.....	.....
6		5555533000	5555542200	5555542100	5554410000	2555443210	3444333000	3553320000	1112221100	2232122000	.....
7		0011100000	1112100000	1112200000	0001110000	.....	.....	.....	.....	.....	.....
8		1112443100	0013331000	0011100000	1201222000	.....	.....	.....	0001110000	.....	.....
9		0012321000	0012444210	0012211000	3445454100	.....	.....	.....	.....	.....	.....
10		4455121000	1334433100	1124410000	0012321000	0012331000	1122233000	0000011100	.....	0001110000	.....
11		2343210000	1444311000	1233211000	0123331000	0012210000	0122220000	0011200000	.....	0111000000	.....
12	Treated before and after malaria	0123320000	0001224000	0012220000	0012221000	.....	.....	.....	.....	.....	.....
13		5555553100	5555554410	5555533200	1112332000	5555544200	.....	.....	.....	.....	.....
14		0012100000	0112211000	.....	.....	0001221000	1122321000	.....	0113331000	.....	.....
15		5555441000	5555441000	0123432200	2544452200	0222111000	1112220000	.....	.....	.....	.....
16		5554410000	1124330000	1244441000	3344410000	.....	1233310000	0112211000	.....	0144441000	.....

TABLE 3.—*Data in Group 3*

Initials	Sex	Months after Treatment		Before Malaria	After Malaria	2 Mo.	6 Mo.	9 Mo.	12 Mo.	15 Mo.	18 Mo.	21 Mo.	24 Mo.
Blood Wassermann													
1 J. P. H.....	M	23	Treated before malaria	—	—	—	..	—	—	—	..	—	..
2 A. L. S.....	M	11	Treated before and after malaria	+	+	+	+	+	..	..	..	..	..
3. G. R.....	M	25		Doubtful	..	—	—	—	—	—	..	—	—
4 J. L.....	M	25		+	—	+	—	—	Doubtful	—	..	—	—
Cerebrospinal Fluid Wassermann													
1.....			Treated before malaria	—	—	—	—	—	—	—	..	—	..
2.....			Treated before and after malaria	+	+	+	+	+	..	..	..	..	..
3.....				—	Doubtful	—	Doubtful	—	—	—	..	—	Doubtful
4.....				Doubtful	—	Doubtful	—	—	—	—	..	—	—
Cell Count													
1.....			Treated before malaria	2	3	2	2	1	1	1	..	1	..
2.....			Treated before and after malaria	32	5	12	2	2	..	..	..	..	..
3.....				7	8	4	4	1	1	..	..	2	5
4.....				5	2	3	3	1	1	3	..	2	2
Albumin													
1.....			Treated before malaria	2+	2+	2+	+	+	+	2+	...	2+	...
2.....			Treated before and after malaria	3+	2—	?	+	2+	...	...	...	...	...
3.....				2+	2+	2+	2+	?	2+	...	...	2+	2+
4.....				2+	2+	2+	2+	?	2+	2+	...	2+	2+
Globulin													
1.....			Treated before malaria	—	—	—	—	—	—	—	..	—	..
2.....			Treated before and after malaria	+	+	..	+	+	..	..	..	..	..
3.....				+	+	+	+	+	+	..	..	+	—
4.....				s	s	s	+	s	s	s	..	+	0
Permeability Index													
1.....			Treated before malaria	...	...	...	302	380	296	341	...	247	...
2.....			Treated before and after malaria	285	294	356	294	294	...	...	...	...	...
3.....				...	...	318	300	330	310	323	...	328	...
4.....				...	...	225	244	288	266	287	...	310	298
Colloidal Gold Test													
1.....			Treated before malaria	1122300000	1123310000	0001110000	0000100000	0012110000	0000110100	0001110000	.....	0011000000	.....
2.....			Treated before and after malaria	5555543200	4444443000	1223441000	0012221000	5433310000	.....	.....	.....	.....	.....
3.....				1123310000	1122220000	2234430000	0012222100	0001222000	0001221100	0144341300	.....	0012233300	0111221000
4.....				3444410000	1334431000	0023332100	0012321000	0000220000	0012221000	0122210000	.....	0001110000	0012310000

to light or in accommodation, tremor of the facial muscles and of the tongue, and absent knee and ankle jerks. The serology at that time was: positive Wassermann reaction of the blood and cerebrospinal fluid; 18 cells; albumin, +++; globulin, +++; colloidal gold, 5555555520, and permeability index, 2.60.

Treatment and Course.—He was inoculated with malaria on Nov. 1, 1927, and had ten chills. The serologic state immediately after the cessation of the treatment was: positive Wassermann reaction of the blood and cerebrospinal fluid; 10 cells; albumin, +++; globulin, +; colloidal gold, 5555555200, and the permeability index, 2.85.

Following this he showed clinical improvement. His speech became normal, memory was improved and he was well oriented, but physically he showed no change. Two months after cessation of the treatment, the serologic examination showed: positive Wassermann reaction of both the blood and the spinal fluid; 4 cells; albumin, ++; globulin, ++; colloidal gold, 00112444200, and a permeability index of 3.22. Clinically, he had improved definitely and was allowed to go home on a visit.

When he came for the examination at the end of six months, the permeability index was 2.91; two months later, on Aug. 16, 1928, he was returned to the hospital with the statement that he was not getting along well, that he had become irritable and had periods of excitement. At this time the Wassermann reactions of the blood and cerebrospinal fluid were positive. In the cerebrospinal fluid there were: 35 cells; albumin, +++; globulin, ++; colloidal gold, 0012442100, and the permeability index was 2.35.

He was reinoculated with malaria and following this showed a second clinical improvement. On conclusion of this treatment, the serology was: a positive Wassermann reaction of the blood and cerebrospinal fluid; 7 cells; albumin, +++; globulin, ++; colloidal gold, 1133444410, and a permeability index of 3.02. Another examination one month later, however, showed: a positive Wassermann reaction of the blood and cerebrospinal fluid; 4 cells; albumin, ++; globulin, +; colloidal gold, 1334444000, and a permeability index of 2.71. The clinical improvement, too, was of short duration and at the end of two months after the cessation of the second course he was showing the same mental picture as he did at the time of the first admission. At this time the serologic reactions were: positive Wassermann reactions of the blood and the cerebrospinal fluid; 4 cells; albumin, ++; globulin, +; colloidal gold, 1211100000, and a permeability index of 2.41.

The permeability index, therefore, is the only one of the tests that has fluctuated in correlation with the clinical picture. The cell count showed an increase after the first exacerbation but was normal after the final return of symptoms. The same relationship between the fluctuations in the clinical picture and the behavior of the serologic tests can be followed through in the other five cases (10 to 14, inclusive) of group 4 in which there have been relapses. In other words, in all these the striking correlation of permeability indexes and clinical course is contrasted by the lack of consistent parallelism between the latter and all other tests. Furthermore, the change in permeability practically always preceded that in the clinical picture.

COMMENT

The analyses of the serologic observations reported show clearly that there is a distinct difference between the behavior of the per-

TABLE 4.—*Data in Group 4*

	Initials	Sex	Months after Treatment		Before Malaria	After Malaria	2 Mo.	6 Mo.	9 Mo.	12 Mo.	15 Mo.	18 Mo.	21 Mo.	24 Mo.
					Blood Wassermann									
1	H. J.	M	18		+	+	+	+	+	..	+	..	..	..
2	C. K.	M	22		+	—	Doubtful	..	..	..	..	..	..	..
3	F. E. J.	M	12	Treated before malaria	—	+	+	+	+	..	..	..	..	..
4	A. G.	M	..		+	+	..	..	..	..	..	..	..	..
5	C. M.	M	..		+	+	+	..	..	..	..	..	..	..
6	J. E. B.	M	20		+	+	+	+	..	Doubtful	..	Doubtful	..	..
7	G. B. G.	M	23		—	—	..	—	—	—	—	..	—	..
8	V. C.	M	22		—	..	—	—	—	—	..	—	..	..
9	C. Mil.	M	23	Treated before and after malaria	—	—	—	—	..	—	..	—	..	..
10	R. A. C.	M	26		+	..	+	+	..	Doubtful	—	—	..	—
11	G. B.	M	25		+	..	+	+	+	Doubtful	..	Doubtful	+	..
12	V. Cour.	M	25		—	Doubtful	—	..	—	—	—	—	..	—
13	M. F.	F	..		+	..	..	+	..	..	+	..	..	..
14	J. E. S.	M	27	No treatment other than malaria	+	..	..	Doubtful	..	—	..	—	—	..
15	R. R. S.	M	14		+	+	+	+	+	+	..	..	..	..
					Cerebrospinal Fluid Wassermann									
1					+	+	+	+	+	..	+	..	..	..
2					—	..	—	..	..	..	..	..	..	..
3				Treated before malaria	+	+	+	+	—	..	..	..	..	..
4					+	+	..	..	..	..	..	..	..	..
5					+	+	—	..	..	..	..	..	..	..
6					+	—	Doubtful	Doubtful	—	—	..	—	..	..
7					+	+	+	Doubtful	—	—	—	..	—	..
8					+	+	+	+	+	+	..	+	..	..
9				Treated before and after malaria	+	+	—	—	+	..	..	—	..	..
10					+	+	+	+	+	+	+	+	..	+
11					+	+	+	+	+	..	..	+	+	..
12					+	..	Doubtful	..	Doubtful	—	..	Doubtful	..	—
13					+	+	..	+	..	..	+	..	..	..
14				No treatment other than malaria	+	+	..	+	+	+	..	+	+	..
15					+	+	+	+	+	+	..	..	..	..
					Cell Count									
1					2	1	2	2	2	2	4	..	..	..
2					6	4	3	..	..	..	..	..	..	..
3				Treated before malaria	10	10	5	0	7	..	..	..	..	..
4					30	3	..	..	..	..	..	..	..	..
5					7	4	2	..	..	..	..	..	..	..
6					5	2	3	3	2	2	..	0	..	..
7					4	2	3	2	6	2	1	..	1	..
8					3	2	4	2	3	2	..	2	..	..
9				Treated before and after malaria	10	7	5	5	3	..	..	1	..	..
10					10	5	3	5	2	1	2	2	..	2
11					5	3	10	4	0	5	..	2	5	..
12					6	2	5	..	3	1	?	3	..	3
13					4	3	..	6	..	..	50	..	..	..
14				No treatment other than malaria	6	3	..	8	2	3	..	3	2	..
15					18	10	4	10	35	4	..	..	..	..
					Albumin									
1					3+	?	+	3+	2+	+	2+	...	...	...
2					2+	2+	2+	...	...	...	...	...	...	...
3				Treated before malaria	2+	3+	3+	2+	2+	...	...	...	...	...
4					2+	2+	...	...	...	...	...	...	...	...
5					2+	2+	2+	...	...	...	...	...	...	...
6					3+	3+	3+	3+	2+	2+	...	2+	...	...

6	Treated before and after malaria	3+	3+	3+	3+	2+	2+	...	2+	...	...
7		2+	2+	2+	+	+	+	—	...	2+	...
8		3+	2+	3+	2+	2+	2+	...	2+	...	...
9		2+	2+	2+	+	3+	+	...	+	...	
10		3+	2+	2+	2+	2+	8	2+	2+	...	+
11		3+	2+	2+	2+	?	2+	...	+	+	...
12		3+	3+	3+	...	?	2+	+	2+	...	2+
13		3+	2+	...	2+	...	...	3+	...	...	...
14	No treatment other than malaria	3+	...	...	2+	2+	2+	...	+	2+	...
15		3+	3+	2+	—	3+	2+	...	...	...	...
						Globulin					
1	Treated before malaria	+	+	+	+	8	—	+	..	..	..
2		—	—	8	..	..	..	..	..	..	..
3		3+	3+	2+	2+	+	..	..	..	..	..
4		+	+	..	..	..	..	..	..	..	..
5		+	+	+	..	..	..	..	..	..	..
6	Treated before and after malaria	+	8	8	8	—	—	—	..	+	..
7		+	+	+	—	—	8	—	...	+	..
8		+	+	2+	2+	2+	2+	..	2+	..	..
9		+	+	—	—	2+	—	..	8	..	..
10		+	+	+	8	8	—	8	—	..	+
11		+	+	+	8	8	+	..	—	—	..
12		+	+	+	..	+	+	+	—	..	8
13		+	8	..	8	..	..	3+	..	..	..
14	No treatment other than malaria	+	..	..	+	8	8	..	—	—	..
15		3+	+	2+	+	2+	+	..	..	..	..
						Permeability Index					
1	Treated before malaria	290	213	277	262	291	285	259	...	...	...
2		...	...	276	...	...	...	...	...	...	...
3		316	182	299	232	198	...	...	...	...	...
4		282	242	...	...	...	...	...	...	...	...
5		...	...	...	...	...	...	...	...	...	...
6	Treated before and after malaria	224	...	292	259	230	246	...	246	...	...
7		...	...	294	261	270	299	275	...	277	...
8		...	...	297	299	316	272	...	259	...	...
9		...	...	281	257	258	258	...	244	...	...
10		...	...	...	...	313	325	304	312	...	248
11		...	...	...	348	338	369	...	360	259	...
12		...	...	...	...	313	268	283	281	...	257
13		...	...	...	...	...	...	225	...	...	...
14	No treatment other than malaria	...	...	...	...	377	359	...	291	208	...
15		260	285	322	291	235	271	...	...	...	...
						Colloidal Gold Test					
1	Treated before malaria	2224422000	1123331000	0124330000	4444432000	1333332000	1223332000	3444443000	.....	.....	.....
2		0001110000	0001110000	0012210000	.....	.....	.....	.....	.....	.....	.....
3		5555555430	5555555330	4555564410	2554433300	5555554100	.....	.....	.....	.....	.....
4		2234431000	0133320000	.....	.....	.....	.....	.....	.....	.....	.....
5		5565552200	5555433100	5554440000	.....	.....	.....	.....	.....	.....	.....
6	Treated before and after malaria	0002221000	0001221000	0012331000	0012221000	0001200000	0012311000	.....	0001110000	.....	.....
7		5555542100	5555451000	1244440000	0001110000	1122530000	0012223000	1122321000	.....	1223343000	.....
8		5553443100	1112443000	2434442200	0001121000	0012220000	0012233100	.....	0000222300	.....	.....
9		1445441000	0012321000	0122210000	0011100000	1112442200	0011200000	.....	0000000000	.....	.....
10		5565541000	2223330000	1124442000	1124431000	0012332000	0012142100	0012210000	0011222100	.....	0000210000
11		5555554100	2112220000	1114430000	0123321000	.....	0122211000	.....	0011221000	0011321000	.....
12		5544210000	2443310000	1234441000	.....	0011210000	1122331000	0433430000	0123210000	.....	.....
13		5554410000	2234311110	.....	0123220000	.....	.....	3555555430	.....	.....	.....
14	No treatment other than malaria	5554110000	5511000000	.....	1134430000	1214441000	1122100000	.....	0012220000	0111100000	.....
15		5555555520	5555555200	0112444200	4555544410	0012442100	1334444000	.....	.....	.....	.....

meability index and that of the other six serologic reactions that we examined. In regard to the latter, we agree with Ferraro and Fong that serologic improvement is a general, even if not invariable, result of malarial treatment of paresis. This, however, occurs without any practically applicable relationship to the clinical course. Our experiences, furthermore, with the cell counts (especially in group 1) and the albumin content (especially in group 4) show that even these two tests are not reliable as an indication of either good or poor prognosis. The cell count does decrease in practically all cases; but we found pleocytosis in the early months after treatment in our cases in which improvement occurred, and, what is more important, practically all our cases with poor results showed a decrease to normal. There seems to be no correlation even in degree of serologic and clinical improvements, as can be seen from a comparison of group 1 and group 2, which would even indicate the opposite. Furthermore, we wish to draw attention to the fact that other methods of treatment may bring about practically the same degree of serologic improvement, whereas the clinical results are undoubtedly not as beneficial as those following malaria treatment. This we have noted from the serologic study of those cases reported here of patients who have had prolonged treatment previous to malaria inoculation. Results of a somewhat similar type have been reported by Walther and Abelin,[7] who studied a series of cases in which different methods of treatment (mercury, silver-arsphenamine, malaria, etc.) were used and also in the cases in which tryparsamide was given, as reported by Solomon and Viets.[8] In discussing the paper by Solomon and Viets, Gordon also pointed out that the serologic improvement in his experience was not correlated with the clinical course. Even if such lack of correlation were not always the case, however, and in some groups some of these reactions would improve with clinical improvement, it is doubtful whether they could be utilized in determining the prognosis. As Ferraro and Fong have emphasized and as we found in our cases, the serologic reactions, with the exception of the cell count, improved rather slowly, and it was only after a long period following the treatment that one could notice the change. This was the reason why the early experiences with serologic reactions in malarial treatment of paresis were so discouraging. Almost invariably the clinical improvement preceded the serologic changes, and from that point of view such changes would be more of academic than of practical importance.

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7. Walther, F., and Abelin, S.: Ueber Blut und Liquorbefunde bei unbehandelter und behandelter progressive Paralyse, Arch. f. Psychiat. **78**:281, 1926.

8. Solomon, H. C., and Viets, H. R.: Tryparsamide Treatment of Neurosyphilis, J. A. M. A. **85**:329 (Aug.) 1925.

It is quite different, however, with the permeability tests. The first mention of the fact that the passage of substances from the blood into the cerebrospinal fluid decreases following malarial treatment was found by Walther and Abelin [7] in their experiences with the hemolysin reaction. Results pointing in this direction were also mentioned by us in our first report on experiences with the Walter bromide method.[6] In both of these, however, definite correlations have not as yet been established. Recently, a paper by Kral [9] reported a series of cases wherein the permeability for bromides was shown to be decreased in cases in which malarial treatment was employed with favorable clinical results. These tests, however, were carried out only once after the treatment. From our charts, one can see clearly that such a correlation is not only present but is reliably consistent, and can be followed through a series of examinations. (1) Practically all cases of paresis in which treatment has not been given show a low permeability index (increased permeability). This may or may not suffer a further decrease directly after treatment, but then there is a gradual increase in the index and a return to normal or even high permeability index in cases that show a good clinical result, and no change or even decrease in index in cases with a poor clinical outcome. (2) What is still more important, these changes in the permeability index occur early and generally precede the changes in the clinical course. This was especially strikingly demonstrated in the cases that showed relapses after short periods of improvement. The practical value of such an indicator is self-evident, and in our clinical work we have been able to make good use of it. In a number of cases, we have undertaken reinoculations with malaria as soon as the permeability index began to decrease (cases 1, 4 and 14 in group 1; 11 and 16 in group 2; 9, 10, 11, 14 and 15 in group 4; and 1 and 3 in group 3). These were not always successful. At times, the inoculation was not followed by the development of the fever. In such cases we have used antisyphilitic drugs, at first tryparsamide, but recently we have also used bismuth with good results.

From the point of view of final prognosis, the permeability index cannot be considered reliable; i. e., in our experience we have not been able to tell whether a change in permeability to normal meant that the patient was going to remain well and that there would be no relapse. The patients, therefore, have to be kept under constant observation and have serologic examinations made at regular intervals. Under such conditions, it has proved an excellent guide as to the probable immediate status of the process. This condition led us to believe that the permeability changes were probably closely connected

9. Kral, A.: Untersuchungen ueber das Verhalten der Blutliquor Schranke während der Malariabehandlung der progressiven Paralyse, Ztschr. f. d. ges. Neurol. u. Psychiat. **117**:315, 1928.

with some components of the histologic process and opened up an avenue of approach for a search of these components.

HISTOLOGIC OBSERVATIONS

In a recent paper, one of us,[10] on the basis of a study of forty-eight cases of paretic patients who died at different stages following malarial treatment, has described changes that seemed most likely to result from treatment with malaria. With these data, a reconstruction of the probable sequence of events in such cases was made as follows: The earliest reaction is an exacerbation of changes usually found in paresis. This exacerbation seems to begin during the treatment and increases in intensity for from three to six weeks after the termination of the fever. The changes occurring during this period were intense and were characterized by the fact that in addition to the well known infiltration of the meninges and perivascular spaces in the cortex, there were numerous macrophages in the meninges, strong proliferation of the glia (both microglia and macroglia), and the iron reaction in the walls of the blood vessels, as well as in the microglia, was highly intensified. This short period is followed by one during which a gradual diminution in the intensity of the infiltrative and proliferative reactions occurs and is accompanied by a corresponding improvement in the arrangement of the cortical layers and nerve cells. Concomitant with this, there is a diminution of iron pigment in the blood vessel walls and in the microglia. Conditions of this type were found in thirteen of nineteen cases terminating between six weeks and two and a half years following the treatment. In the other six cases, a reaction which could be looked on as a new flare-up of the inflammatory processes occurred, differing from the usual type of paresis in that the picture was characterized by scattered inflammatory foci in which there was a predominance of mesodermal proliferation even to the formation of miliary gummas. In five of these six cases, numerous spirochetes were found in the sections, whereas in the other thirteen cases the spirochete was found in one case only. This fact made it justifiable to assume that the malarial treatment conditioned a disappearance or attenuation of the spirochetes, which in some cases, however, may again proliferate and, regaining their virulence, cause a new flare-up of inflammation.

Of the different components of the picture complex, the one that was of greatest interest from our point of view as expressed in the introduction was the behavior of the iron pigment. As already described, the increase in inflammatory reaction immediately after the treatment was usually accompanied by a transitory increase of iron

10. Wilson, R. B.: Histopathological Changes Following Malarial Treatment of General Paralysis, Brain **51**:440, 1928.

pigment in the blood vessel walls and in the microglia. Along with this, there was an increased proliferation of the microglia. Both of these gradually receded as later stages following the treatment were examined, and a certain degree of correlation between the iron content and microglial proliferation, on the one hand, and the inflammatory process, on the other, could be found, although it was not always so in individual cases. The behavior of the iron contained in the blood vessel wall especially suggested an interesting possibility. It has been previously pointed out by observers in this field (Spatz and Metz,[11] von Zalka and von Lehoczky [12]) that the iron in the blood vessel wall in paresis is of hematogenous origin, and that it gains access through the walls of the blood vessel and then to the microglia as a result of the increased passage of substances through the blood vessel walls themselves. This suggested the possibility that this phenomenon is closely related to that underlying the changes in the bromide permeability. This was rendered so much the more probable as the initial increase in the iron pigment would correspond with some of our own observations, and especially with those of Krall, of an increased bromide permeability immediately after treatment. What is still more important, however, is that this pigment decreased after the initial exacerbation. We thought that the injury to the mesodermal elements increased the permeability for substances from the blood into the spinal fluid, and thus iron content in the blood might gain access into the brain tissues. This would mean that the iron found in the blood vessel wall would represent one stage of the migration from the blood outward, and that another stage was represented by the taking up of this iron by the microglia. We realize at the outset that the conditions governing the permeability for iron may be altogether different from those governing the permeability for bromides, and also that the permeability for either the bromides alone or both bromides and iron may be wholly or partly independent of the condition of the blood vessel wall. Nevertheless, the behavior of the permeability index as found in the different stages of paresis and the proportions of iron found in such cases made it at least probable that some correlation may exist between the two, and our histologic observations were primarily concerned with this factor.

For this purpose, five cases in which the permeability for bromides had been determined before death were studied histologically. Unfortunately, the period of time that had elapsed between the permeability determinations and death was not quite the same in all cases, and

11. Metz and Spatz: Ztschr. f. d. ges. Neurol. u. Psychiat. **89**:138, 1924.

12. Von Zalka, E., and von Lehoczky, T.: Zur Frage der Eisenreaktion im Gehirn, Arch. f. Psychiat. **85**:220, 1928.

this influenced the observations to some extent. The clinical and pathologic observations were as follows:

REPORT OF CASES STUDIED HISTOLOGICALLY

CASE 1.—*Clinical History.*—A. S., a white man, aged 56, was admitted to the Foxborough State Hospital on Aug. 10, 1928, with a history of failing memory and delusions of persecution of one year's duration. He appeared depressed and confused. Orientation and insight were poor. Psychometric examination revealed an intelligence quotient of 0.36. There was slurring of speech. The pupils were myotic and reacted sluggishly to light, but well in accommodation. Appreciation of pin prick was diminished on the right side of the face. The radial periosteal reflexes and the achilles and knee jerks were not obtained. The other reflexes were active and equal, and no abnormal reflexes were found. On Aug. 30, 1928, the Wassermann reaction of the blood was positive. The Wassermann reaction of the cerebrospinal fluid was positive. The fluid showed: 40 cells; albumin, +++; globulin, ++; colloidal gold, 5555444100. The permeability index was 1.78. On September 22, he was inoculated with malaria tertiana. He had four paroxysms, but his general condition became poor and quinine was administered. Notwithstanding this, he sank rapidly into coma and died on Oct. 1, 1928.

Histologic Examination.—There was a moderate thickening of the meninges which were strongly infiltrated with lymphocytes and plasma cells. In addition, there were numerous macrophages. There was considerable confusion in the cortical layers though not to the degree that the various areas could not be recognized. Many of the ganglion cells were displaced from their usual vertical position. All cortical blood vessels, large and small, were ringed with lymphocytes and plasma cells, the latter predominating in quantity. The mesodermal proliferation, however, was not strong. Both microglia and macroglia showed an intense proliferation. The microglia nuclei were mostly long and rod-shaped, with protoplasmic processes extending from both ends; these showed numerous shorter branches. The macroglia nuclei were large and poor in chromatin. Their protoplasmic processes were thickened to such an extent that many areas appeared to consist almost entirely of a dense network of macroglia protoplasmic branches. In the walls of the blood vessels there was a considerable amount of granular iron pigment, whereas the microglia contained less iron. The average quantity of iron, as taken from the different sections studied, was as follows: iron in blood vessel walls 3, in the microglia 2.[13]

Comment.—The patient in case 1 died during the malarial treatment. The permeability shortly before death was markedly increased. Histologically, the picture corresponds to the one that was most usually seen during the initial exacerbation, as recorded in a previous study. The iron content in the blood vessel walls is high, and somewhat lower in the microglia.

CASE 2.—*Clinical History.*—J. R., a white man, aged 54, admitted to the hospital on Oct. 22, 1928, about two years before began to have attacks which came on suddenly and were characterized by loss of speech, numbness of the hands, and

13. In order to facilitate comparison of the iron found in the different cases, we have designated the quantities in a similar fashion as in the previously mentioned paper as 1, 2, 3, 4, and decimal fractions between these.

unsteadiness of the legs. In November, 1927, he had a short period of confusion, disorientation and restlessness. On the day before admission, he had a similar attack during which he became assaultive. When examined on admission he was restless, excited, and at times resistive. He was disoriented and euphoric. Physically, there were speech defect, tremor of the outstretched hands, and unequal and irregular pupils, which reacted sluggishly to light. The tongue deviated to the right; the tendon reflexes were exaggerated, and on the left side there was a positive Babinski sign. The Wassermann reaction of the blood was positive. The cerebrospinal fluid showed: a positive Wassermann reaction; 4 cells; albumin, +++; globulin, ++; colloidal gold test, 5555555410, and a permeability index of 1.85. The patient grew gradually worse and on November 11, developed bronchopneumonia and died three days later.

Histologic Examination.—The meninges were thickened and infiltrated with lymphocytes and plasma cells; only a few macrophages were found. The arrangement of the cortical layer and of the nerve cells was little disturbed, although there were numerous cell-free areas. There was a fairly intense and diffuse infiltration of the cerebral vessels with lymphocytes and plasma cells, somewhat less, however, than in the preceding case. The glia was proliferated, but to a degree disproportionately smaller than the infiltration. The iron content in the blood vessel walls was 2.9, and in the microglia 2.7.

CASE 3.—*Clinical History.*—F. C., a white man, aged 44, admitted to the hospital on March 30, 1927, for the past year had shown forgetfulness, had expressed paranoid ideas and at times had been confused. In the fall of 1926, he began to complain of dizzy spells, and was unable to work. In the hospital he exhibited emotional instability, defective memory and disorientation. The pupils were unequal and reacted poorly to light. There was a tremor of the tongue, face and hands, and the tendon reflexes were overactive. The Wassermann reaction of the blood was positive. In the cerebrospinal fluid, the Wassermann reaction was positive; there were 55 cells; albumin, +++; globulin, ++; colloidal gold curve, 4555554200. The permeability index was 2.16. The patient remained confused, with transitory delirious episodes. Auditory hallucinations were elicited. He became gradually weaker, deteriorated both mentally and physically, and died on June 19, 1927.

Histologic Examination.—The histologic picture was essentially the same as in case 2. The iron content in the blood vessel walls was 3.8, and in the microglia 2.4.

Comment.—In comparing cases 2 and 3 (in both of which treatment was not given) with case 1 (in which treatment was given), one finds little to substantiate the assertion of the previous report that the malarial treatment causes an increase in the inflammatory reaction. It may be remembered that the earlier report was based on averages from a much larger series, and observations in those cases can scarcely invalidate those of a larger series. Moreover, it was pointed out that the glial reaction following malarial treatment is one of the outstanding features of the period of exacerbation. A comparison of case 1 with cases 2 and 3 may well illustrate the point; in the untreated patients, although the infiltration is strong, the proliferation of the glia is disproportionately small in degree.

Case 4.—*Clinical History.*—M. F., a white woman, aged 46, admitted to the hospital on Jan. 30, 1925, for one year previously had been forgetful, suspicious of people, irritable and untidy, and had expressed grandiose ideas. On admission she was overactive, euphoric and expansive. Memory was defective, and insight and judgment were poor. Physically, the pupils were small and reacted sluggishly to light; the tendon reflexes were diminished. The Wassermann reaction of the blood was positive; the cerebrospinal fluid showed: a positive Wassermann reaction; 95 cells; globulin, ++, and the colloidal gold curve was 5554321000. The permeability index was not determined. From February, 1925, to October, 1926, the patient received thirty-seven injections of sulpharsphenamine and twenty-one injections of mercury. During this time she showed slight clinical improvement and a decrease of the cell count to 4. On Oct. 24, 1926, she was inoculated with tertian malaria and had eleven paroxysms. On Nov. 3, 1926, the cerebrospinal fluid showed: a positive Wassermann reaction; albumin, ++; globulin, +; colloidal gold test, 2234311100. She improved rapidly and was allowed to go home on a visit of three weeks on Dec. 23, 1926. She returned to the hospital and from January to May, 1927, received eleven injections of tryparsamide. The clinical improvement continued, and she was discharged on May 18, 1927, when the cerebrospinal fluid showed: a positive Wassermann reaction; 6 cells; albumin, ++; globulin, +++; colloidal gold curve, 0123220000. On Feb. 19, 1928, the patient was returned to the hospital, having been found by the police wandering about the streets in a confused state. She was disoriented and, at times, assaultive. The Wassermann reaction of the blood was positive. The cerebrospinal fluid on March 1 showed: a positive Wassermann reaction; 50 cells; albumin, +++; globulin, +++; colloidal gold curve, 3555555430. The permeability index was 2.25. She continued to be restless and untidy, and had to be fed by tube. She died on April 6, 1928.

Histologic Examination.—The infiltration of the cortical vessels was practically as intense and diffuse as it was in case 1. In addition, many of the larger vessels showed thick rings of infiltration, composed mostly of lymphocytes. There was a corresponding strong proliferation of the microglia. The iron content, however, was not as high, being 1.2 in the blood vessels and 0.6 in the microglia.

Comment.—The patient in case 4 showed a relapse following an apparently good clinical remission. The histologic picture showed a new and intense inflammatory reaction. The permeability was not quite as high as in cases 1 and 2, and the iron content of both blood vessel walls and microglia was correspondingly lower.

Case 5.—*Clinical History.*—A. G., a white man, aged 43, was admitted to the hospital on July 10, 1925, three months after it had been noticed that his speech was defective; he was confused, made irrelevant remarks, and was becoming careless in his dress. A few days later, while in New York, he was taken to the Bellevue Hospital, presumably because he had been hit by an automobile. There he was found to be disoriented, euphoric and grandiose. Physically, he showed a scanning speech, tremor of the face and tongue, and irregular pupils which did not react to light. On admission to the Foxborough Hospital, the patient was euphoric; he had no insight, and his judgment was poor. He showed scanning speech, unequal pupils, reacting sluggishly to light, nystagmus on lateral and vertical fixation, impaired coordination, spastic gait and bilateral ankle clonus. In the cerebrospinal fluid, there were: 35 cells; albumin, +; globulin, +++; colloidal gold curve, 5555441100; the permeability index was not determined.

From July, 1925, to January, 1927, he received thirty-five injections of sulpharsphenamine, and from January, 1927, to May, 1927, eleven injections of tryparsamide. His condition remained essentially unchanged. On Jan. 20, 1927, the cerebrospinal fluid showed: 30 cells; albumin, +++; globulin, +; colloidal gold curve, 2234431000, and a permeability index of 2.82. On May 2, 1927, he was inoculated with malaria and had seven paroxysms. On May 26, 1927, the cerebrospinal fluid showed: 3 cells; albumin, ++; globulin, +; colloidal gold curve, 0133320000, and a permeability index of 2.42. The patient became gradually weaker and died on June 22, 1927.

Histologic Examination.—The meninges were diffusely but mildly infiltrated, the majority of the cells being lymphocytes with only occasional plasma cells. A few macrophages were scattered about, and there were numerous polymorphonuclear leukocytes. In the cortical perivascular spaces were found a few lymphocytes, and only an occasional plasma cell. The glia, too, was only slightly proliferated. The iron content in the walls of the blood vessels was 1.0, and in the microglia 1.6.

Comment.—The patient in case 5 apparently did not react clinically to malarial treatment. Histologically, too, there were no signs of such

TABLE. 5—*Comparison of Iron in Blood Vessels and in Microglia*

	Permeability Index	Blood Vessel Iron	Microglia Iron
Case 1	1.78	3.0	2.0
Case 2	1.85	2.9	2.7
Case 3	2.16	3.8	2.4
Case 4	2.25	1.2	0.6
Case 5	2.42	1.0	1.6

a reaction. In none of the sixteen cases (terminating within six weeks after the end of treatment) of the report quoted were such mild reactions found. The permeability is somewhat high, and there is a corresponding amount of iron in the blood vessel wall.

COMMENT

A comparison of the quantities of iron found in the blood vessel walls and microglia in the cases presented points to the existence of a parallelism between the amount of iron in the blood vessel wall and the permeability indexes which, even if it is not absolute in individual cases, follows the general rule that when the permeability index is low the amount of iron is high. This comparison is shown in table 5.

The greatest discrepancy between the permeability index and the iron content of the blood vessel is seen in case 3. One must remember, however, that the permeability index in this case was determined fully two months before death, whereas, in none of the other four cases did this period exceed a month. We know from previous experience that in a rapidly progressing mesodermal process the permeability index continues to drop, and it is highly probable that had we made another determination at a later date we would have found it so. This

parallelism, however, seems to be restricted to the iron in the blood vessels, the amount of iron in the microglia evidently being independent of it. The question also arose as to whether all conditions of the central nervous system that are accompanied by a rise in the permeability (low indexes) would show the same parallelism. For this purpose four patients suffering from diseases other than paresis, who died shortly after the determination of permeability, were examined histologically with especial reference to iron content (table 6).

Cases 2, 3 and 4 with highly increased permeabilities showed practically no iron. In cases 3 and 4, in which some iron was found, the quantity was so small that it was negligible even in comparison with the amount found in case 5 of the paretic group. This observation is also supported by the report of Zalka and Lehoczky, who in a series of 100 patients, some of whom were suffering from diseases of the central nervous system other than paresis, found no iron in the cortical blood vessels. Twelve of their patients had arteriosclerosis, five of whom had signs of arteriosclerosis of the cerebral vessels, a condition

TABLE 6.—*Iron Content in Four Patients Who Died from Diseases Other Than Paresis*

		Permeability Index	Iron Content
(1) E. R.	Dementia praecox with tuberculosis.....	2.87	0
(2) E. B.	Myocarditis with psychosis.....	2.10	0
(3) W. C.	Senile dementia with arteriosclerosis.....	1.74	Rare granules
(4) M. C.	Psychosis with cerebral arteriosclerosis....	2.20	Occasional granules

which, as we have brought out in a previous communication, is practically always accompanied by increased permeability. This would point to the probability that whatever mechanisms may be responsible for the increase in the permeability, they are not always the same in different diseases and for different substances (a point which was brought out by the results of experiments by Stern and Rapoport [14]).

Our observations in these cases have also brought up a number of interesting factors in connection with problems discussed in the first communication on permeability determinations. Of especial interest is one of these concerned with the possible mechanism underlying the permeability for bromides. We mentioned there that, if we should accept the theory of those observers who look on the production of the cerebrospinal fluid as purely a process of dialysis, we will have to search for the cause of the inequality of the amounts of bromides in the blood and cerebrospinal fluid, not in the blood vessel wall but in the blood itself. If this were so, disturbances in this proportion too will have to depend on changes in the blood itself. We have already

14. Stern, L., and Rapoport, J.: La résistance de la barrière hématoencéphalique au passage des colloides, Compt. rend. Soc. de biol. **96**:1149, 1927.

questioned the validity of such a theory in a communication dealing with the pathogenesis of paresis,[15] in which we found a definite relation between the increase in the permeability for bromides and injury to the cortical capillaries. In our present histologic material we have studied the iron content of the vessel walls in different parts of the brain, but have found that there were some areas in which such iron was not found. A consistent study of the choroid plexus, for instance, showed that the walls contained no iron. Lehoczky, too, found this to be the case. This would speak definitely for the fact that the permeability, for iron at any rate, is dependent on the condition of the blood vessel wall.

We do not consider it necessary to review the evidence in favor of the view that the iron pigment in the walls of the blood vessels in paresis is of hematogenous origin and that its presence there is brought about by an increased permeability. The works of Metz and Spatz, as well as the papers by Lehoczky[16] and Zalka and Lehoczsky, cover this in detail. Our own observations of the existence of a relationship between the permeability for bromides and the amount of iron pigment in vessel walls substantiates this view. Lehoczky[16] expressed the opinion that malarial treatment does not influence the iron content of the blood vessels or microglia. He does not, however, mention whether the cases he has studied have shown any change in clinical course, or what the clinical picture before death was like. This is important in view of the fact that one could certainly not expect any change in the iron content in cases that did not show any clinical improvement.

Our histologic material is not sufficient nor altogether ideally suitable for final conclusions. More material should be investigated from this point of view, and preferably cases in which the interval between the permeability determination and histologic examination is more or less uniform and of a shorter duration. If the relationship between the iron content of the vessel wall and the permeability index as found in our cases could be further substantiated, the value of the latter would be established not only as a practical prognostic criterion but also as indicative of what is actually taking place in the central nervous system.

CONCLUSIONS

In a study of fifty-four cases of neurosyphilis in which the patients were treated with tertian malaria, serial examinations of the blood and

15. Malamud, William, and Lowenburg, K.: The Rôle Played by the Cerebral Capillaries in the Pathogenesis of General Paralysis, J. Nerv. & Ment. Dis. **69:** 121, 1929.

16. Von Lehoczky, T.: Zur Frage der Eisenreaktion im Gehirn, Arch. f. Psychiat. **85:**229, 1928.

cerebrospinal fluid at regular intervals in the course of prolonged clinical observation showed that there exists a definite correlation between the behavior of the index of permeability for bromides and the clinical course. It was found that: (1) in practically all cases of paresis in which treatment is not given, there is an increased permeability for bromides (index below 2.80); (2) in cases in which malaria treatment was given there may or may not be a still further increase of permeability immediately after the cessation of the fever; (3) following this, however, in cases that subsequently show improvement, there is a gradual fall in permeability (rise of index to normal limits of 2.8 to 3.2), whereas, (4) in cases which do not show improvement or in which the condition becomes worse there is no such decrease and there may be an even greater increase of permeability (index below 2.8).

These fluctuations in the permeability index, furthermore, generally preceded the corresponding changes in the clinical picture. They could thus be utilized as guides in prognosis and indicators of further treatment. Contrary to this, the changes in the other reactions (Wassermann reactions of the blood and the cerebrospinal fluid, cell count, albumin, globulin and colloidal gold test) were not consistently related to the clinical picture. There was a tendency in all these to improvement, but this was exhibited in all cases (those which showed clinical improvement as well as those which showed further progress of the disease); besides that, it manifested itself some time after treatment. For both these reasons, therefore, these tests could not be regarded as reliable indicators of the probable prognosis.

Histologic studies of cases of paresis in persons whose permeability index was determined shortly before death showed that there was a definite relation between the index and the amount of iron in the walls of the cerebral blood vessels.

The determination of the permeability for bromides from the blood into the cerebrospinal fluid, because of its value as an indicator of clinical prognosis, on the one hand, and of the condition of the blood vessels, on the other, may be regarded as a link between the clinical and the histologic pictures.

THE RÔLE OF INFECTION IN THE ETIOLOGY OF TICS*

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Until recently, the problem of tic has been considered settled. Following the lines laid down by Charcot and Brissaud, Meige and Feindel, in 1905, published the results of their comprehensive studies under the title "Tics and their Treatment." According to them, the tic is of psychogenic origin. The fundamental basis on which it develops is a psychopathic personality. The essential element in that personality is volitional weakness. What begins as an expression movement, or a defense movement, or an irritation movement, by frequent repetition becomes automatized. Then, because of the volitional weakness, the automatized movement gets out of control and becomes exaggerated and deformed, a caricature of its former self.

The views expressed by Meige and Feindel were generally accepted for a number of years. They were in line with the prevailing views regarding the etiology of the group of disorders which are now known as the motility disorders. With the exception of chorea, practically this entire group was classed with the neuroses. In the last few years, facts have been accumulating which have taught that the motility disorders belong not to the neuroses, but to organic neurology. This has been accepted for most of the group, but one has been rather slow in accepting it for the tics. It is of interest that the most recent comprehensive study of tics, that of Wilder and Silbermann, still attempts to explain the condition on a psychogenic basis and uses a modified psychoanalysis as the basic method of treatment. They recognize clearly the organic nature of the tic movement as it occurs, for instance, in epidemic encephalitis; but they fail to carry the analogy over to the group of disorders which are commonly understood as tics. Failure to recognize the tic as an organic disease is largely due to the lack of a definite, clear etiology. The purpose of this paper is the presentation of new evidence bearing on this point.

Of the evidence already at hand, there is the relationship of tic and chorea. While the movements in the two diseases differ, one can always pick out isolated movements in chorea identical with those of

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tic, and conversely in cases of tic some movements identical with chorea. It is true that chorea minor, like rheumatic fever, is a self-limited disease, running a definite course, and as such differs from tic. There are cases of chorea, however, in which recurrences come so close together that the chorea minor goes over into a chorea intermittens, and even into a chorea permanens, in which the analogy to tic is very close. But most important is the fact, pointed out by Meige and Feindel and reemphasized recently in an excellent and comprehensive study by Irwin Strauss, that typical tic movements may develop as residual manifestations of a typical chorea minor.

Another factor suggesting the organic etiology of tics is the relation to torticollis. Many cases of tic show movements of the head identical with those of torticollis. As a matter of fact, torticollis is often classed with the tic group. Since Cassirer has pointed out that torticollis may be merely one part of a dystonia musculorum deformans, at least some cases of torticollis have been accepted as organic. Cassirer himself did not accept them all but believed that the identical picture might occur on a purely psychogenic basis. I believe that further observations will bring an ever-increasing number of these cases into the organic field. Rosenow's experimental work with torticollis has been suggestive.

The strongest evidence in favor of the organic origin of the tic was offered by the epidemic of lethargic encephalitis. Here for the first time one saw the different types of hyperkinesias, myoclonus, torsion, tremor and tic, occur singly and together in cases of known and accepted organic etiology. In observing tics today, one is constantly reminded of analogies to epidemic encephalitis. This is true not only of the simpler tic movements, but also of the more complex. In cases of tic one sees definite compulsive eye movements of short duration, which strongly suggest abortive types of oculogyric crises; and one sees respiratory irregularities which are related to the respiratory anomalies of the postlethargic states.

Led by these considerations, and especially by the analogies existing between chorea minor and tic, I have attempted the study of a series of cases of tic from the standpoint of the search for foci of infection and the removal of infection when found. I am reporting here the three cases which have been longest under observation.

REPORT OF CASES

CASE 1.—*Clinical History.*—P. S., a boy, aged 14, was the first child in the family and was born by a normal delivery. Development was normal. Physically, he was strong and healthy; mentally, he was intelligent and alert. He was sensitive and shy, and emotionally somewhat unstable, with a tendency to brief depressive periods. There was always some motor hyperactivity. He was definitely a nervous type.

He was well until the age of 6, when he developed a severe sinus infection which lasted three months. During this time he became run down physically, and grew pale and anemic. Toward the end of this period, during an attack of chickenpox, a blinking tic developed. From this time on he was definitely a tiqueur. At first there were free intervals, sometimes weeks long; but the tic showed a progressive tendency, and the attacks became more frequent, more severe and more extensive. Between the ages of 8 and 9, in addition to a variety of other tics, such as blinking, twitching of the face, lateral and vertical eye movements, clearing the throat and sniffing, he developed torticollis movements and even an occasional torsion movement. Everything he did was rapid and jerky. There was a constant "Rededrang." He was constantly under tension, and there was marked motor restlessness.

After the tenth year, the physical condition began to improve. He became stronger and more robust. The tics improved somewhat. From the ages of 12 to 14, there was further improvement. Blinking was reduced, torticollis movements definitely improved, and the other movements were proportionately improved. But the state of tension and the general motor restlessness were always present, and there would be spells when all the tics would return in exaggerated form.

It has been stated that the beginning of the tic followed a prolonged sinus infection. For the next few years, colds and sinus infections, particularly antrum infections, were frequent and protracted. It was noted that there was a definite relationship between these infections and exacerbations of the tic movements. After the tenth year, the infections became less frequent, and at this time the general physical condition began to improve and the tics subsided somewhat. It was felt that there was a definite causal connection. However, another factor had to be considered. Beginning at the eighth year, training movements were started. These included relaxing exercises and controlled movements of the type usually used in these cases. They were carried out for twenty-five minutes, twice a day, over a period of years. There is no doubt that they helped, and that they must be considered in the evaluation of the case.

In July, 1928, the boy developed the first severe cold he had had for a long time. It lasted for several weeks. With this came a marked and definite flare-up of the tics. For a few days they were almost violent. Examination at this time by Dr. Kistner showed a subacute flare-up of the old antrum infection.

When I reviewed the case with Dr. Kistner at this time, the following facts stood out: 1. The first appearance of the tics followed a prolonged sinus infection. 2. Recurrent sinus infections were accompanied by exacerbations of the tic. 3. Gradual lessening in the frequency and intensity of the infections was accompanied by an improvement in the general physical condition and a lessening of the tics. 4. A final flare-up of the infection was again accompanied by a recrudescence of the tics.

Operation.—The connection between sinus infection and the tic was too definite to disregard. When one considered the number of reinfections over a number of years and the long periods during which conservative treatment had been tried, it was evident that there was no chance of cleaning up the sinuses permanently except by operation. All the sinuses (the frontal sinuses excepted) had been involved, but Dr. Kistner decided to do only a radical operation on both antrums. This was chosen for two reasons: 1. The antrum had always shown more infection than the other sinuses. 2. It is not uncommon, when a relatively mild sphenoid and ethmoid infection accompanies an antrum infection, to find that the sphenoid and ethmoid sinuses will clear up spontaneously after the antrums have been radically operated on. The operation was done on Sept. 12, 1928.

Course.—This paper was written more than eight months after the operation. No one who knew the patient before and since could doubt the result. Previous to operation there was always the tension with the readiness to jump or turn or answer to any stimulus; there was the continuous restlessness, and there were tics of varying intensity. Since operation, the tics are reduced to minimum, an occasional blink or a twitch; only a little motor restlessness is present; the tension, one of the most disturbing of all symptoms, is practically gone. To the casual observer now, the patient is a normal, but rather nervous, boy.

A summary of Dr. Kistner's record of the patient from 1920 to 1929 is appended:

Nov. 11, 1920: Frequent colds. Diagnosis: acute sinusitis.

March 24 ,1921: Left chronic otitis media. Acute sinusitis.

Jan. 8, 1922: Roentgen examination of sinuses shows: left antrum less well illuminated than right; ethmoids hazy and poorly defined; frontal cells absent.

May 16, 1922: Acute rhinitis. Catarrhal otitis media. Under treatment for from three to four weeks—aspiration and douching.

Nov. 16, 1923: Acute rhinitis—duration several weeks. Roentgen examination of sinuses showed: left antrum opaque, margins hazy; right antrum apparently clear; ethmoids distinctly hazy and show little structure. Definite increase in density. Sphenoid hazy. Frontal cells absent.

Sept. 2, 1924: Acute sinusitis. Roentgen examination showed: right antrum ++; left antrum ++++. Treatment consisting of washing the antrums was given for about three months.

Dec. 11, 1924: Left antrum; while well outlined, shows distinct haziness of outline. Right antrum apparently clear. Ethmoids show haziness and moderate density on both sides. Sphenoids show haziness of G line. Illumination not bad. Frontals absent.

Feb. 21, 1925: Acute rhinitis, two weeks' duration. Antrums fairly well illuminated; margins somewhat blurred. Ethmoids hazy and show increased density on both sides. Sphenoid poorly illuminated. G line hazy. Frontals absent.

Aug. 8, 1928: Complaint: Cold of two or three weeks' duration. Roentgen examination showed right antrum smoky; borders fuzzy. Left antrum shows slight increased density; borders fuzzy. Ethmoids blurred. Sphenoids show increased density. Borders thickened. Frontal cells absent. Proof puncture showed definite evidence of subacute infection in both antrums. The sinusitis cleared slowly.

Sept. 12, 1928: Double radical antrum operation. General polypoid thickening throughout both antrums, more pronounced on the left side. Cultures from material obtained at operation showed: from the right swab, a growth of alpha hemolytic greenish short chained streptococci; from the tissues, *Staphylococcus albus;* from the left swab, a growth of *Staphylococcus albus, Micrococcus catarrhalis* and alpha hemolytic greenish streptococcus; from the left tissue, *Micrococcus catarrhalis* and alpha hemolytic greenish streptococcus.

Case 2.—*Clinical History.*—H. S., a boy, aged 11, was seen on Oct. 13, 1928. The history obtained from the father was that the patient was an only child, born at full term by instrumental delivery. There was no birth trauma. The birth weight was 7½ pounds (3,401.9 Gm.). There were feeding difficulties at 5 months, and an acute illness at 14 months. At this time the father and mother were estranged, and from then until the age of 5 the child was the object of continuous wrangling between the parents. According to the father, this was a severe ordeal for the child. The tonsils and adenoids were removed when he was 5. He started school at 6; the teacher considered him nervous and arranged his work accordingly.

For two years he did well. Then at the age of 8 he began to be lax in his studies and showed an inability to apply himself and to concentrate. He just "got by" each term. He became increasingly nervous, and a tic developed.

After a vacation he was better, but when he returned to school many new tics developed: blinking, jerking shoulders, looking suddenly back and peeking furtively into corners. He managed to finish the school year and made his grade. En route to the mountains for a vacation, he had a spell of intense nervousness; for a time muscular control seemed completely gone. A second episode of this type occurred soon after, and he was brought to Doernbecher Hospital in August, 1928. In ten days he became much more quiet and was discharged.

When he started to school in the fall, his condition became so bad that he was returned to the Doernbecher Hospital. On the train he became uncontrollable; there was jerking, twisting and writhing of the whole body; the breath came in gasps; he was unable to talk, and he could produce only staccato shrieks.

In giving the history, the father could not set any definite time at which the twitchings began. It came on in the eighth year, apparently unrelated to any acute illness. After the onset the course was definitely progressive, though with remissions.

Examination.—The patient was under observation in the hospital for some time, and showed a good deal of variation in behavior. There were times when he was fairly quiet, but these were the exception. For the most part he was exceedingly restless and was continuously in movement. When he sat reading, he twitched constantly; there were jerking movements of the arms and legs, and turning movements of the head and eyes. In moving about the ward he walked quickly and jerkily; he turned toward any slight stimulus of sight or sound, with impulsive suddenness. He was noisy, talkative and overloud in speech. He was forever interrupting others and must have his say. The voice was suddenly raised and lowered, and some words were uttered explosively. Everything he did was unsteady, jerky and irregular.

The boy had a pleasing personality, and was cheerful, friendly and cooperative. He was well liked in the ward. Occasionally, however, when the motor restlessness became more intense he made a nuisance of himself, tearing up books and pictures, throwing things around, and showing a definite tendency to destructiveness.

Neurologic examination gave essentially negative results. There was a slight muscular hypotonia. A general physical examination gave negative results except for a diffuse redness of the pharynx. The tonsils had been removed.

The sinuses were examined by Drs. Kistner and Bailey. When the nose was douched, nothing was obtained. Following proof puncture of the right antrum, a large globule of mucopus was blown from the nose. Shreds of the same type of pus were found exuding from beneath the right middle turbinate. Roentgen examination showed the right antrum to be dense, 4+; the margins were thickened. The left antrum was dense, 2+; its margins also were thickened. The frontal cells were well illuminated. The right ethmoid was somewhat blurred. The left ethmoid was well illuminated. The sphenoids were well illuminated.

The examination was rechecked, and the conclusion was reached that while the examination of the sinuses did not give definitely positive results, there was sufficient evidence of chronic bilateral disease of the antrums to justify exploration with a view to a double radical antrum operation if the observations should indicate it.

Operation.—The exploration was done on Nov. 14, 1928 by Dr. F. B. Kistner. Definite pathologic change was found on both sides. There was hyperplasia of the

lining membranes with cystic abscesses. Radical operation was done on both antrums. The observations were as follows: The right side showed moderate thickening and edema throughout. One large cystic abscess and two smaller ones were found in the lining. There was no free pus. The left side showed thickening and edema of the alveolar recess. The remainder of the lining showed little change.

Pathologic Report (Drs. Benson and Menne).—Cultures of the left antrum contained *Staphylococcus albus* and *Micrococcus catarrhalis*. Cultures of the right antrum contained alpha hemolytic greenish streptococci. From 4 to 5 cc. of broth culture from the right antrum was injected into a rabbit. No nervous symptoms developed. Autopsy after four days showed pus in the knee joint of the left hind leg, but cultures were sterile. The brain appeared normal; cultures were negative. Cultures of the heart blood were negative. No other pathologic change was found.

Course.—Nov. 26, 1928: The operative recovery was good. After the operation, there was a definite change in the condition. The boy was quieter, more relaxed and less tense. The suddenness and jerkiness of movement and the explosiveness of speech were strikingly modified. The "Rededrang" was gone. The tics were reduced to a minimum. He had changed from a high strung, hyperactive and overreacting boy to a quiet, contained, and almost normal one.

March 26, 1929: During the last four months there have been two or three periods of motor restlessness with a return of the old tics, in much subdued form. These spells usually come when he attempts to return to school. One of them was quite severe, and lasted two or three weeks. This subsided. At the time this paper was written he was quiet, and had been attending school for several weeks without any difficulty. He still has occasional tics and twitches, and is still a little overactive at times, but the approach to normality is close.

Case 3.—*Clinical History.*—E. R., a boy, aged 14, complained of jerking of the head and arms, and of inability to progress in school. The family history was not significant. The patient was the fifth child and was born at term without the use of instruments. He walked at 1½ years of age and talked at about 2 years. He had measles at 2 years and had had a few head colds. He started school at 7, and passed the first grade, but failed in the second and again in the fourth grade. The year before the present illness, he passed into the sixth grade, but he did not get along well. Up to the time of the present illness, he had been normal as far as physical activities were concerned; he was fond of athletic sports, and did well in them.

The present illness began six months before this paper was written. There was no history of any acute illness preceding it. The onset was acute; it "came all at once." He developed abnormal movements which consisted of such things as: "stretching the face"; turning up the eyes; throwing out the right arm; kicking up the right leg in running; blinking; twitching the head, and making guttural noises. At first one of these movements would come, remain for a time, and then disappear. Its place would then be taken by another. Things got gradually worse until three months before presentation, when the condition was stationary.

Examination.—Nov. 16, 1928: The patient was undersized and undernourished. The striking manifestations were the hyperkinesias. They consisted of blinking movements; facial tics of one or both sides; turning the head and eyes; jerking the head down and back; sniffing, grunting and mildly explosive noises. All these movements were quick, sudden and jerky. There were also other types of movements, such as sucking the lips and throwing the leg in walking.

The boy was friendly and cooperative. He seemed rather indifferent to his ailment—certainly he was not troubled by it. In cases 1 and 2, the basic state was one of tension, and the restlessness and jumpiness and even the abnormal movements seemed to be an expression of that tension. Here there was no tension; the basic state was one of quiet; the tics seemed superimposed, as something foreign, and the patient seemed to consider them as such and to disregard them.

Aside from the presence of the tics, neurologic examination showed nothing abnormal.

A general physical examination showed enlarged tonsils and adenoids, a congested pharynx, cervical adenopathy and a slight secondary anemia.

Mental Rating: The chronologic age was 14 years and 5 months and the mental age, 11 years and 2 months. The intelligence quotient was 77. The case belongs in the upper borderline group.

Examination of the nose and throat by Drs. Kistner and Bailey revealed the fact that the patient had two or three colds a year, each lasting two or three days. He had not had sore throats. Pus was present on the floor and in the right middle meatus as well as in the left middle meatus. The tonsils were large and slightly congested. Examination of the nasopharynx showed pus streaked over the adenoids. Roentgen examination of the sinuses revealed: right antrum dense, 2+; margins thickened; left antrum, no increased density; borders blurred. The frontal cells were dense, 1+; their structure was blurred. The right sphenoid was dense, 1+, and the margins hazy. The left sphenoid was dense, 1+, and the margins thick.

On proof puncture, the material washed from the antrums showed gross pus on both sides.

The observations at this examination were: infected tonsils and adenoids, and bilateral purulent antrums. It was decided to remove the tonsils and adenoids first and to watch the results. The operation was done on Nov. 30, 1928.

Course.—There was no improvement following this operation. Therefore, six weeks later, a bilateral radical antrum operation was done. The operative report was: "Chronic antral disease; definite thickening with polypoid edema and fibrosis on both sides. It looks as though pus were present in the other sinuses, particularly the left (probably the left ethmoid)." Nothing was done to the sphenoids and ethmoids at this time.

Following the operation, the patient showed definite improvement. There was considerable reduction in the frequency and intensity of the tics, but the improvement did not satisfy expectations. As it was known that pus was still present in the other sinuses, it was requested that they, too, be operated on. On Jan. 27, 1929, six weeks following the antrum operation, Dr. Kistner did a bilateral intranasal ethmosphenoidectomy. There was definite thickening of the lining of the cells on both sides, more pronounced on the left.

Since this operation, the patient has been showing slow but steady progress. All that is left of the mass of tics which existed before is an occasional twisting of the head and turning of the eyes. The contrast with the preoperative state is striking and gratifying.

COMMENT

The three cases are all typical of tic. Two had lasted for several years, one for several months. In all three there was definite evidence of focal infection. Two patients had chronically infected antrums; one had infected tonsils, adenoids, antrums, ethmoids and sphenoids.

Radical removal of the foci of infection in all three cases resulted in definite and striking improvement. In one case the tonsils and adenoids were removed first, without effect on the tics. Subsequent operation on the antrums, ethmoids and sphenoids resulted in marked improvement. Up to the present, every patient with tic whom I have examined, with one exception, has shown definite evidence of sinus infection. The one exception, the case of P. M., is not reported here. His tic is relatively mild. The only foci of infection found were tonsils and adenoids. Studies of the sinuses were inconclusive. The tonsils and adenoids were removed, and there was some improvement. When the patient was last seen the tic had not entirely disappeared; if the course is unfavorable exploration of the antrums will be considered.

The constancy with which sinus infection, and especially antrum infection, has been found in three cases and the satisfactory results obtained from radical treatment of these foci have led to the conclusion that: 1. Tic is an infectious disease in the same sense that chorea is an infectious disease. 2. The commonest site of the infection is the sinuses, particularly the antrums.

In none of the cases reported has there been a complete return to normality. All three patients show occasional grimaces, occasional turning of the head and eyes and some unnecessary activity. But this is expected. If, as I believe, these are cases of toxic encephalitis, it is not surprising that absorption over months and years has caused enough permanent damage to nerve cells to leave permanent residual symptoms. What has impressed me is not these residua, but the fact that in spite of long duration of the disease, and in spite of the intensity of the symptoms, the clearing up of the focal infection has still been able to produce such a striking amelioration, amounting as far as the usefulness of the patient is concerned, to a complete cure.

In looking over the three case histories, one finds the common etiologic factors to which the tics are attributed, but a different factor in each case: in case 1, a nervous constitution; in case 2, conflicts in the home which upset the child emotionally; in case 3, a subnormal mentality. These factors were individual. But the factor common to all three was infection.

As one considers the usual course in a case of tic, one finds it compatible with an infectious etiology. The mild cases clear up spontaneously. The moderately severe ones show long periods of exacerbation and periods of partial or complete remission. The most severe cases have a chronic progressive course, as in the maladie de la Gilles de la Tourette. The course in any given case will be determined by the severity of the infection. Spontaneous healing of the infectious focus—and this must happen often—leads to spontaneous cure. If the healing is temporary and reinfections occur, there will be remissions

and exacerbations. If the infection is severe and persistent, then the progressive tics will be found. I have not yet had an opportunity to study a case of maladie des tics, but the difference is one of degree and not of kind, and one would expect the same etiologic factors to be operative.

It is obvious that every sinus infection does not produce a tic any more than every tonsil infection produces nephritis, an arthritis or an endocarditis. There must be a definite specificity of the infection for certain tissues in tics, just as there is in other focal infections with which one is familiar.

The observations in the group of cases reported, and in other cases under observation justify the conclusions that have been drawn. I do not assume that every tic movement represents a focal infection, nor even that every case must be organic. It is entirely possible that a somewhat similar picture may develop on the basis of imitation. It is highly probable that cases can develop on the basis of developmental anomalies of the brain. It is certain that tic movements can occur on the basis of epidemic encephalitis, as residual symptoms of chorea minor and as a result of cerebral accidents, such as hemorrhage. But I am convinced that the overwhelming bulk of what are ordinarily classed as "tics" are the result of a toxic encephalitis due to absorption from an extracerebral focus of infection, and that the adequate treatment in these cases depends on early recognition and proper handling of the infection.

NARCOLEPSY (GÉLINEAU'S SYNDROME) AND OTHER VARIETIES OF MORBID SOMNOLENCE*

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CONTENTS

Morbid somnolence may be defined as the untimely occurrence of periods of more or less natural sleep. In recent years, considerable attention has been paid to a special variety of morbid somnolence, a variety characterized by the presence of two, usually independent, symptoms: (1) attacks of sleep and (2) weakness of the muscular apparatus, usually of the legs, coming on suddenly while the patient is in the midst of an emotional display, usually laughter. (The second symptom was named "cataplexy" by Henneberg, and will be so designated in this paper.) Gélineau, in 1880, first recognized this combination of morbid somnolence and cataplexy as a new syndrome, which he designated narcolepsy.

The purposes of this paper are: (1) to propose a classification of cases of morbid somnolence, (2) to enumerate the cases of Gélineau's syndrome already reported and to discuss them briefly and (3) to report a number of cases of Gélineau's syndrome and of other varieties of morbid somnolence that have come under my observation.

CLASSIFICATION OF THE REPORTED CASES OF MORBID SOMNOLENCE

The cases of morbid somnolence that have been reported fall into two large groups, those without and those with cataplexy.

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GROUP 1.—*Morbid Somnolence Without Cataplexy.*—Group 1 may be further subdivided into: (*a*) Cases in which, aside from morbid somnolence, there are no symptoms or signs of disease. The second case of Weech belongs in this group.

(*b*) Cases presenting symptoms of gross organic disease of the brain, such as tumor, cerebral arteriosclerosis, multiple sclerosis, paresis and epidemic encephalitis. For example, Redlich mentioned a patient with a hypophyseal tumor who had attacks of sleep. McKendree and Feinier discussed a number of cases of tumor of the brain in which somnolence was a prominent symptom. Chartier had a patient in whom it was associated with multiple sclerosis. The somnolence accompanying the acute stage of epidemic encephalitis is well known. Kraus, Perrier and others have reported cases in which attacks of sleep began to occur after the subsidence of the acute stage of encephalitis. Goldflam's sixth patient had miliary tuberculosis of the brain.

(*c*) Cases showing manifestations of metabolic and endocrine disorder. Such are the cases of obesity reported by Caton, Sainton, Williams and Goldflam (cases 3 and 4), and the case of pregnancy complicated by attacks of sleep reported by Nevermann. Legrand du Saulle commented on the occurrence of somnolence as a symptom of diabetes. Brailovsky cited the case reported by Donskow of a woman who, after an artificial menopause, began to suffer from somnolence which improved with ovarian therapy. Thomson reported the case of a child, aged 5, in whom sleep attacks were associated with evidence of liver disorder.

(*d*) Cases presenting symptoms of epilepsy, hysteria or other psychoses. Examples are Goldflam's case 5 (epilepsy), Solomon's (hysteria) and Geipel's (traumatic psychosis). Here, for want of a better place, one may put the case, reported by Siebert, of a girl aged 12, who had attacks of sleep for five weeks immediately after an attack of grip. For the next ten months, until the writing of the report, the child remained well. Siebert assumed that the attacks were hysterical.

GROUP 2.—*Morbid Somnolence with Cataplexy.*—In group 2 are the cases in which, in addition to attacks of sleep, a sudden muscular weakness accompanies an emotional display, forming the syndrome which Gélineau called narcolepsy.

The use of the word narcolepsy has resulted in a good deal of confusion in the literature:

1. The word does not describe the disease. Narcolepsy suggests a state of profound unconsciousness, whereas most of the patients with Gélineau's syndrome are easily roused from the sleep attack. Singer and Wenderowič therefore argue justly that "hypnolepsy" would be a better term. (Incidentally, Foot, in 1886, suggested the term hypno-

lepsy for a patient who had attacks of sleep but no cataplexy.) On the other hand, neither narcolepsy nor hypnolepsy includes any reference to the cataplectic symptom found in the syndrome described by Gélineau.

2. A number of writers apply the term narcolepsy to cases that do not present the two cardinal symptoms of Gélineau's syndrome. For example, Redlich, even though he championed the more restricted use of the term, included in his group of "true narcolepsy" the cases of Ballet, MacCormack, Dercum and Pitres and Brandeis, despite the fact that in none of these cases is there any mention of muscular weakness on emotional display. Redlich's own case 3 is subject to the same criticism. Kinnier Wilson, on the other hand, did not favor restricting the term narcolepsy to cases presenting the two cardinal symptoms. In his list of forty-three cases, including his own cases and those found in the literature, he included such cases as that of Gowers, and Spiller's first case, the patients in both of which suffered from sleep attacks but not from cataplexy. Since Wilson uses "narcolepsy" so inclusively, one may justifiably wonder why he does not also include in his list the cases just cited of Ballet, MacCormack, Dercum and Pitres and Brandeis, together with scores of other similar cases mentioned in the literature both before and since Gélineau's publication.

3. Another source of confusion lies in the expression "symptomatic narcolepsy," proposed by Redlich and others, as a designation for those cases in which sleep attacks occur as a symptom of some other disease, cataplexy being absent. To call such cases symptomatic narcolepsy and at the same time to restrict the term "true narcolepsy" to cases showing cataplexy is inconsistent.

4. Confusion resulted from the practice in the last decade of applying the term narcolepsy to the cases of "frequent slight attacks of childhood," first described by Friedmann. In this disease, the patient suffers from frequent seizures which resemble the epileptic petit mal. The disease has been regarded as different from epilepsy because: (1) the seizures disappear at puberty; (2) their frequency is uninfluenced by anti-epileptic drugs, and (3) there are never any convulsions. Friedmann first described this disease in 1906 under the title "Non-Epileptic Absences or Brief Narcoleptic Attacks." In 1912, he no longer referred to them as narcoleptic. In 1915, he reconsidered the question and again claimed a relationship between the disease and Gélineau's narcolepsy, and he referred to the seizures as "narcoleptic absences." Nearly every one, however, now agrees that the two clinical pictures are probably unrelated, and the disorder described by Friedmann is now generally referred to as pyknolepsy, a term proposed by Stöcker.

For the sake of clearness, I shall restrict the term narcolepsy or Gélineau's syndrome to those cases that present both sleep attacks and cataplexy. Cases showing attacks of sleep only will not be so designated. It is possible that some of the latter cases will some day be found to be identical in nature with Gélineau's syndrome, but at present the evidence for such identity is slight. Patients with attacks of sleep are common, whereas patients with attacks of sleep and cataplexy are relatively rare, and it would seem wise, for the present, to regard the latter as a separate group.

Since the term narcolepsy is often used rather loosely, confusion might be avoided by designating those cases presenting both attacks of sleep and cataplexy as Gélineau's syndrome.

REPORTED CASES OF GÉLINEAU'S SYNDROME

Previous Compilations of Cases.—Gélineau, in 1881, published a monograph entitled "De la narcolepsie," in which, according to Dufossé and Samain, he collected reports of fourteen cases of morbid somnolence. Unfortunately, this volume is not obtainable at the Surgeon-General's Library, Washington.

Redlich's article of 1925 contains the first attempt to collect all the reported cases of Gélineau's syndrome. This article is based on his four published cases, his seven unpublished cases and twenty-four cases from the literature. As I have previously indicated, one of his own cases and four of the twenty-four are not cases of Gélineau's syndrome. Furthermore, Redlich failed to include four typical cases:

1. A case presented by Gélineau before the Société d'Hypnologie on March 20, 1894, and cited by Samain. A man, aged 36, began at the age of 20 to have attacks of sleep and at 30 to experience sudden weakness on laughing out loud, on hearing a joke or on witnessing a gay scene. If, on such occasions, he was standing he had to sit down; if he was seated his head dropped forward.

2. The case of Camp, that of a man who began at 40 to have attacks of sleep and typical cataplectic attacks.

3. The case of Burr, that of a man, aged 40, who had had sleep attacks since the age of 24 and who complained that "if anything should come up that would cause me to laugh real hard, I seem to wrinkle right down, losing my strength until I catch myself." The patient's wife reported: "When he is pleased over anything, I could lead him around and throw him down. He seems to lose his strength altogether."

4. The case of Bolten, that of a girl who, according to Janzen, had sleep symptoms and cataplexy.

Many cases have been reported since Redlich's article was published. In a recent article, Adie did not pretend to include all the reported cases. His study is based on his own cases and on thirteen cases selected arbitrarily from the literature.

Kinnier Wilson recently collected thirty-nine cases from the literature and added four of his own. As previously indicated, several cases cited by him do not belong to the narrower category of Gélineau's syndrome as here used. Also, Wilson has overlooked the following cases of Gélineau's syndrome reported by: Gélineau (1894), cited by Samain; Bolten (1917); Burr (1918); Heveroch (1924); Möllenhoff (1925); Weech (1926); Strauss (1926), and Moschner (1926).

Author's List of Typical Cases Found in the Literature.—In the literature that is available to me, I find reports of sixty-six typical cases of Gélineau's syndrome. These were reported by the following authors in the chronologic order given:

1877. Westphal
1880. Gélineau
1894. Gélineau, cited by Samain, 1894
1902. Löwenfeld, Guleke
1907. Camp
1913. Stöcker (case 4), reported again in 1928 by Rosenthal
1916. Henneberg, K. Mendel, Jolly
1917. Bolten, Redlich (cases 1, 2 and 4), Singer
1918. Burr, Noack, Stiefler
1921. Somer (two cases)
1922. Kahler
1924. Goldflam (case 1), B. Fischer, Stiefler, Heveroch, Missriegler
1925. Curschmann and Prange (case 2), Matzdorff, Hilpert, Möllenhoff, Wenderowič (two cases)
1926. Spiller (cases 2 and 3), Adie (five cases), Weech (case 1), Janzen (case 2), Strauss, Stiefler (case 2), Moschner, reported again in 1928 by Rosenthal
1927. Mankowsky, Jacobssohn, Thiele (two cases), Lafora (cases 1 and 2), Strauss (two cases), E. Miller, Gillespie, Münzer
1928. Wilson (four cases), Rosenthal (cases 3 and 6), Gruszecka (two cases), Sperling and Wimmer, Brock
1929. Fulton and Bailey

Five additional cases are reported in this paper.

Missriegler's case is included in the series in spite of the fact that the report is ambiguous with regard to the presence of cataplexy. In the clinical history given by the author, no mention is made of cataplexy. Toward the end of the article, however, the author stated that "cataplectic inhibitions occurred at various times" (p. 259) and "motor weakness, coming on in attacks . . . occurred in my patient, always parallel with the sleep attacks" (p. 269). Specific details are lacking.

OBSERVATIONS IN GÉLINEAU'S SYNDROME

It is not my purpose to analyze and classify the various symptoms and physical observations of patients suffering from Gélineau's syndrome. This has been done by Redlich (1925), Adie and Wilson. I wish, however, to call attention to several points.

Incidence as Regards Age at Onset and Sex.—The sixty-six cases of Gélineau's syndrome were distributed according to sex and age at the time of onset as shown in the accompanying table. For two of the males and three of the females, the age at onset was not given. The four cases in which the onset occurred prior to the age of 10 are those of Stiefler (1918), Weech, Jacobssohn and E. Miller.

In my own five cases, respectively, the sex of the patient and the age at onset were as follows: male, 14; male, 18; male, 17; female, 13; female, 14.

Symptoms.—The typical case of Gélineau's syndrome presents two symptoms:

1. Attacks of sleep, which may occur at any time, even when the patient is walking or riding a bicycle. Gélineau's first patient had as many as 200 attacks a day. The attacks last from several minutes to a half hour or more. Usually the patient is easily roused during the attack, but sometimes, as in Kahler's case, he is roused only with great difficulty.

Distribution of the Sixty-Six Recorded Cases of Gélineau's Syndrome with Respect to Sex and Age at Time of Onset

Sex	Below 10	10–19	20–29	30–39	40–49	50 and Over
Male	1	23	16	11	2	0
Female	3	4	1	0	0	0

2. Cataplectic attacks. These consist of a sudden relaxation of parts of the muscular apparatus, usually the legs, occurring while the patient is displaying emotion. The patient may even fall. There is no disturbance of consciousness. In the large majority of the reported cases, the cataplectic weakness manifested itself on hearty laughter and during unpleasant emotions. In five of the cases (those of K. Mendel, Stiefler, 1918; Kahler, case 2 of Wenderowič, and Strauss), the weakness occurred during anger and not during laughter.

In most of the cases, the two cardinal symptoms, attacks of sleep and cataplectic attacks, exist apparently as independent phenomena. In a few of the cases, on the other hand, a relation exists between the two symptoms, so that an emotional stimulus may be followed by an attack of sleep. Such was the case in Gélineau's patient (1880), who fell asleep after the joy of getting a good hand at cards. Subsequently, even a less keen emotion was sufficient to induce sleep. Of Gélineau's second patient (1894), it was reported that "gay and humorous scenes paralyze him and put him to sleep after several moments." Kahler's patient, when she became excited or angry, suddenly became powerless, collapsed and fell into a deep sleep lasting for five or ten minutes. A

similar relation was noted in the cases of Westphal, Goldflam, B. Fischer, Adie (case 2), Weech and E. Miller.

Association with Other Diseases.—Signs suggestive of endocrine disorder are frequent, as was first emphasized by Redlich. Enlargement of the thyroid gland was observed by Strauss (1926) and Redlich (cases 2 and 4); obesity, by Stiefler (1924) and Fulton and Bailey; a small sella turcica, by Redlich (case 4), Adie (case 2) and Fulton and Bailey; posterior clinoids bent backward, by Jolly and B. Fischer; lymphocytosis of more than 40 per cent by Stiefler (1918 and 1924), Redlich (case 2), Moschner and Münzer. Jolly's patient had diminished sexual libido. Möllenhoff's patient, a boy, aged 17, had evidence of gonadal hypofunction; erections and ejaculations had not yet begun to occur. Kahler's patient, a woman, aged 21, had an infantile habitus, scanty axillary hair, a slight struma and a small sella. Rosenthal's third patient, a man, aged 21, had a sister with exophthalmic goiter, a mother with a slight exophthalmos, and maternal grandparents and two paternal aunts who had psychoses. The patient himself had slight exophthalmos, a scanty beard and a basal metabolic rate of minus 16 per cent. The cataplectic attacks of Gruszecka's second patient were more frequent during the menses, pregnancy and lactation. The basal metabolic rate was low in the cases of Moschner and of Sperling and Wimmer. Brock's patient had a calcified pineal gland.

None of the patients had syphilis. The patient in Rosenthal's sixth case had a "suspicious" Wassermann reaction of the blood.

Exceedingly significant is the development of Gélineau's syndrome in patients with a history of epidemic encephalitis. Ten such cases have been recorded: Stiefler's case 2 (1924); Wenderowič' case 2 (1925); Stiefler's case 3, reported as case 2 in his paper of 1926; Mankowsky's (1926); Adie's case 5 (1927); Strauss' two cases, 1927; Münzer's (1927); Wilson's case 4 (1928), and Gruszecka's case 1 (1928). In two of these cases, typical signs of parkinsonism developed (Wenderowič and Stiefler, 1926); Mankowsky's case showed mild parkinsonian features.

The question of encephalitis arises in Missriegler's case.

> The patient had an attack of "grip with cerebral symptoms, fever which remained at 39 C. for weeks, unbearable headaches, diplopia at times, transitory inability to speak and paralytic phenomena in the face. For several weeks he scarcely slept. Then the sleep attacks began." The neurologic examination showed irregular twitchings over the right side of the face, an irregular tremor of the outstretched fingers, and retropulsion both on standing and on walking.

Somer's first patient had 13 lymphocytes per cubic millimeter in the spinal fluid, and B. Fischer's patient had 11 cells, but in neither was there any other evidence of encephalitis.

Jacobssohn's unique patient developed multiple sclerosis.

A woman, aged 36, had had attacks of sleep since the age of 6 and cataplectic attacks on laughing since the age of 9. Until five years before, the cataplectic attacks had occurred only rarely, but throughout her life she had been unable to laugh aloud. Whenever she laughed, it seemed "as if the laughing muscles were paralyzed." In the past five years, she had had typical signs of multiple sclerosis: spasticity and weakness of the legs, bilateral pes equinovarus, hyperreflexia, bilateral Babinski sign and ataxia. With the development of multiple sclerosis, the cataplectic attacks became more frequent, until they occurred almost daily. During these attacks, the spastic legs became atonic for a few seconds. The development of multiple sclerosis was accompanied by no change in the attacks of sleep. In the past five years, she had shown pathologic laughing, a symptom encountered in no other patient with Gélineau's syndrome. During a remission in the multiple sclerosis, the cataplectic attacks became less frequent again, occurring once in two weeks. The author's report was written during this remission.

In three cases, the symptoms of Gélineau's syndrome were complicated by psychotic or epileptic features. Noack's patient, on awakening from an attack of sleep, had twitchings and paroxysms of rage. B. Fischer's patient had auditory and visual hallucinations before the attack of sleep and during the light part of the sleep; after strong emotion, he had generalized convulsions. E. Miller's patient was a schizophrenic, with recurrent catatonic stupors.

Brock's case is unique in that the patient, on awakening from a sleep attack, suffered often from "utter inability to move."

". . . Powerless at first, he begins to 'wiggle his fingers,' then his hands, and then his feet; next he moves a foot and a leg; then he stiffens his abdominal wall; finally summoning all his strength, he manages to sit up and almost always shakes his head about as if to 'clear it.' . . . It takes him from five to ten minutes to get up when so seized."

These attacks are identical with those which Rosenthal noted in his nonnarcoleptic case 7, that of a woman who would awaken from a sound sleep to find herself for the moment utterly unable to move or speak, although fully conscious. (Further details of this case are given later.) Rosenthal spoke of this phenomenon as "verzögertes psychomotorisches Erwachen" and called the attacks "Wachanfälle." Years ago, Weir Mitchell described this phenomenon, calling it "nocturnal paresis or paralysis."

One is struck by the resemblance between Wachanfälle and cataplectic attacks; in both there is inability to move, without loss of consciousness. It is also noteworthy that in Brock's case the Wachanfälle occurred only after attacks of pathologic sleep and never after normal nocturnal sleep. The disease mechanism, whatever it is, is evidently responsible for this curious type of attack.

During his Wachanfälle, Brock's patient often saw and heard, by hallucination, his brother, sister or mother, and, once, an "adult male friend, who seemed to exert a strange, evil hypnotic influence over him." Brock could find no evidence of a psychosis and regarded the patient (a man, aged 22) as possessing "a rather flighty superficial type of make-up leaning toward the cyclothymic."

Etiology.—The conditions necessary for the appearance of Gélineau's syndrome are, despite much speculation, still unknown. No case has as yet come to necropsy.

Many theories regarding the pathogenesis of this disorder have been reviewed by Redlich, Adie, Wilson and others.

Course.—In the vast majority of cases, the symptoms are more or less stationary. Goldflam first saw his patient in 1893 and reported the case in 1924; at this time, the patient was still affected with the malady. In only one case have the symptoms disappeared for any length of time: Stöcker's patient, in a communication in 1927, reported that he had had no further attacks since 1913, when he left the clinic (Rosenthal).

Treatment.—Until recently, there was no basis for optimism in the treatment of patients with Gélineau's syndrome. Various remedies had proved unavailing. In 1925, however, Matzdorff reported that his patient had fewer attacks of sleep after he began to receive thyroid extract. There was no improvement in the cataplexy. Likewise, Weech's patient had fewer attacks of sleep after taking thyroid extract. In Wenderowič' first patient, the administration of thyroid extract resulted in a complete disappearance of the cataplectic symptoms; the attacks of sleep became fewer, but did not clear up completely. Incidentally, Dercum, in discussing Weisenburg's case, stated as his conviction that "even cases which apparently are purely psychic might also be benefited by thyroid extract. . . . It is a cerebral stimulant." Dercum was referring to cases of morbid somnolence without coexistent hypothyroidism.

Missriegler treated his patient by psychoanalysis, with excellent results. When last seen, the patient had been free of symptoms for a year. Henneberg's comment on this case ("the prompt success of analytic treatment speaks against genuine narcolepsy") seems to me inappropriate in view of the fact that in no other case of "genuine narcolepsy" has the patient been treated by psychoanalysis, and there is consequently no basis for comparison.

Löwenfeld reported that in his patient the attacks of sleep became fewer after treatment which included hypnotic suggestion.

ATYPICAL CASES

The following cases do not show the classic association of attacks of sleep and attacks of cataplexy, and in the present ignorance of the

etiology of Gélineau's syndrome it would be best to regard them as atypical cases, in certain respects allied to the syndrome. In each case, the reasons for considering it atypical are given.

1. F. Fischer's (1878) case, that of a patient who had attacks of sleep and attacks of a feeling of fatigue in the knees, the latter, however, having no relation to emotion.

2. Camuset's (1880) case, in which emotion sometimes provoked sleep but never weakness of the limbs.

3. Dufossé's (1888) three cases (nos. 2, 5 and 9) to which the remark on Camuset's case also applies.

4. Quensel's (1925) case, that of a boy, aged 10, who suffered from attacks of muscular weakness in which he was unable to stand or sit. He was able to move his legs only feebly, though he moved his arms and head freely. He was fully conscious and answered questions. There was no incontinence. The pupils reacted normally. The attacks lasted for fifteen minutes. They came on without provocation, but sometimes they occurred after exertion or excitement. There was no disturbance of sleep. Quensel considered the attacks similar to the cataplectic attacks seen in Gélineau's syndrome.

5. Symonds' (1926) case, that of a man, aged 20, who had an illness in 1919 in which he suffered from drowsiness, diplopia and delirium. After this illness, he noticed that whenever he laughed or was excited he fell down. When the author saw him (April, 1925), the patient complained of insomnia. He could not fall asleep until the early hours of the morning, and at 9 a. m., when he arose, he felt so sleepy that he had to go back to bed. The case is atypical in that there were no attacks of sleep, but merely an anomaly of the sleep curve. (The patient eventually developed symptoms of parkinsonism.)

While cases of attacks of sleep without cataplexy are common, cases of cataplexy without attacks of sleep are rare. Wilson was unable to find such a case in the literature. Since then, four such cases have appeared:

1. De Villaverde's case, that of a boy, aged 10, who on hearty laughter, fell suddenly to the ground, the attacks lasting for two minutes, during which there was no disturbance of consciousness.

2. Nesbitt's case, that of a woman, aged 29, who began, at 23, to "flop in a heap" on the appearance of any strong emotion, especially laughter.

3. Wilson's case 5, that of a man, aged 32, who had attacks of pallor and muscular weakness on excitement.

4. Rosenthal's case 7 (previously alluded to), an extraordinary case of a woman, born in 1899, who had an attack of acute epidemic encephalitis in 1922, after which she suffered from cataplectic attacks, which cleared up completely in 1925. During two pregnancies (September, 1923, to June, 1924, and September, 1925, to June, 1926) she complained of frequent Wachanfälle. At the end of each pregnancy these curious attacks ceased.

One might also include in this group the case of Symonds, already referred to.

Though Gillespie entitled his report "A Case of Cataplexy," his patient presented a fairly well marked degree of morbid somnolence. This case is therefore classified here as Gélineau's syndrome.

Allied to cataplexy is the condition which Oppenheim, in 1902, named "Lachschlag." In this type of disorder, the patient falls, on laughing, and for several seconds is completely unconscious. The last point differentiates it from narcoleptic cataplexy, in which the patient is fully conscious during the toneless state. So far only one case of true Lachschlag has been recorded—Oppenheim's case 2, that of a man, aged 45, who, once at 32 and twice at 45, fell to the ground, completely unconscious, on hearty laughter. The attacks lasted for a few seconds.

The literature contains reports of three other cases in which differentiation between Lachschlag and cataplexy is difficult:

1. Oppenheim's case 1, that of a girl, aged 18, who had had attacks of falling, on laughter, since the age of 17. It was impossible to determine whether she lost consciousness during these attacks. (Incidentally, it is noteworthy that the relatives regarded her as otherwise well, "only she is *lazy and sleepy*, but they cannot designate these qualities as pathological." [The italics are mine.] Was this a "larval" case of Gélineau's syndrome?)

2. A case described briefly by Wilson, that of a man, aged 42, who complained that "when he laughed too loud he tumbled down." The patient stated that he would occasionally "go right off in the attacks" (an expression suggesting loss of consciousness), but Wilson was not certain whether or not this was actually the case.

3. Rothfeld's case, that of a man, aged 39, who began at 32 to have attacks of great weakness on hearty laughter. During these attacks, which lasted for one or two seconds, he would fall to the ground. Usually consciousness was retained, but occasionally the attack was accompanied by loss of consciousness lasting for a moment.

Perhaps the most interesting of the atypical cases are two which have attracted surprisingly little attention.

1. The case, reported by Freund, of a woman, aged 56, who had attacks of sleep and pathologic laughing. She frequently burst out laughing when she talked and was apt to do so when she was under some emotion, as, for example, when she was condoling people over the loss of a relative. She never experienced muscular weakness on laughing.

In view of the comparatively frequent association of attacks of sleep with cataplexy, this isolated instance of the association of attacks of sleep with pathologic laughing possesses the greatest interest.

2. The case, reported by Weisenburg, of a woman, aged 32, who had been having attacks of sleep for sixteen years. She improved under a course of treatment consisting of "static electricity, suggestive treatment, and change in her daily routine." Weisenburg noted that "for many years she has never been able to laugh audibly. She is usually of a cheerful, optimistic disposition, and while she would smile she would not laugh. This has been improved very much and she is now able to laugh like any other person." The reports make no mention of muscular weakness occurring during laughter.

Here, as in Freund's case, one is impressed by the association of attacks of sleep with an anomaly of the laughing function other than cataplexy.

REPORT OF FIVE PERSONALLY OBSERVED CASES OF GÉLINEAU'S SYNDROME

The following five cases of Gélineau's syndrome were studied recently. All, except case 5, came under my personal observation.

CASE 1.—*Male; at 13, slight tuberculous (?) infiltration of the left apex; nine months later, onset of attacks of sleep and cataplectic attacks, together with a change in disposition; puberty somewhat retarded; asthenic; poor circulatory response to changes in posture; orthostatic albuminuria; eosinophilia; maternal aunt had attacks of anxiety; paternal first cousin was a schizophrenic.*

History.—A. C. S., an office boy, aged 15, was admitted to the Henry Phipps Psychiatric Clinic on May 18, 1926, complaining that "when I sit still or when I am quiet I fall asleep, and when I laugh I have a weakness in my knees, my knees sink down." The patient had always been frail. At the age of 8 (1918) he had "influenza"; he was ill for three weeks, with weakness, headache, vomiting and slight fever (100 F.); there was no diplopia and no disturbance of consciousness. In September, 1924, he began to cough and was sent to a sanatorium for tuberculosis. Here the physical examination showed slight changes in the breath sounds at both apexes, and the roentgenogram showed slight infiltration of the left apex. The temperature was practically normal. The sputum never contained tubercle bacilli. He gained 17 pounds (7.7 Kg.) and was discharged on June 22, 1925, in good condition.

Immediately on his return from the sanatorium, his family noticed a change in his disposition. Whereas he had always been good-natured, he was now irritable, cross, argumentative and seldom in good humor.

One week after his return from the sanatorium, when the patient was about to laugh, he found he was able only to smile and sank to the floor. He was conscious and able to talk and to move his arms and legs. He had not hurt himself in falling. He was able to get up unaided. After this, he was never able to laugh audibly, although his sense of humor was normal. When he began to laugh, his mouth did not spread as far as it ought; he felt a "catching sensation" (or inability to get his breath) in his throat, and his eyes blinked. The facial expression on such an occasion was rather grotesque, so that other children said that he was having one of his "goofy spells." About a dozen times at the onset of the malady, he actually fell to the floor on attempting to laugh. After this, his knees would sink, but he was able to "catch" himself without falling. He was at all times able to smile naturally. Only a stimulus that provoked hearty laughter led to the cataplectic attack. Anger and fear did not bring on such an attack. The cataplectic attack was never accompanied by loss of sphincter control.

On the day after the first cataplectic attack, the patient felt drowsy in the evening and went to sleep. During the next two weeks, he felt drowsy every evening. Then (July, 1925) he began to feel drowsy during the day. After this, he had attacks of sleep nearly every day. They came on when he was quiet (sitting still or reading) and never when he was walking, running, swimming or playing ball. When the attacks came, he sometimes "fought them off," but usually he was unable to do so. When he succumbed, he slept for from several minutes to half an hour. While asleep, he was not annoyed by flies on his skin. He could

always be roused easily. During the attack, he did not dream and did not hear what went on about him. His nocturnal sleep had always been good. He had had no convulsions.

Emotional reactions never gave rise to attacks of sleep.

The pubic hairs began to appear about February, 1926. The patient had no erections and no emissions and displayed no interest in the opposite sex.

The patient said that he drank about a gallon (4 liters) of water daily. The patient's relatives, however, had never observed this. There was no polyphagia.

The patient was the second of five children, the other four being in good health. His mother was excitable. A sister of the mother had delirium at one time, probably due to drugs; she also had attacks of anxiety. A paternal first cousin had schizophrenia.

On admission to the Phipps Clinic, the patient behaved as any normal boy would. He was alert and bright, and took an interest in all the examinations. He had excellent insight.

Physical Examination.—The patient's height was 66 inches (167.6 cm.); his weight was 110 pounds (49.9 Kg.). The patient was slender. There was some suggestion of an eunuchoid habitus, the distance from the top of the head to the symphysis pubis being 31½ inches (80.01 cm.), and from the symphysis to the soles of the feet 35 inches (88.90 cm.). The scapulae were scaphoid. There was a marked lumbar lordosis. The head was well formed. The thyroid was not palpable. The testes were infantile. The penis was fairly well developed. Hirci and crines were developing. The hands were cool and moist. There was hyperhidrosis of the axillae. The neurologic examination revealed no significant changes. The heart was of normal size; the heart sounds were normal; there were no murmurs. The circulatory efficiency in response to postural changes was poor. When the patient changed from the recumbent to the upright posture, the pulse rate rose from 69 to 114. The blood pressure was: systolic 114; diastolic 52 (recumbent posture). The lungs and abdomen were normal.

Laboratory Examination.—The urine was normal except for orthostatic albuminuria. The hemoglobin content was 85 per cent; the red blood cells numbered 4,000,000; the white blood cells, 4,800 (polymorphonuclear neutrophils, 57 per cent; eosinophils, 10 per cent; lymphocytes, 23 per cent; large mononuclears and transitionals, 10 per cent). The Wassermann reaction of the blood was negative. The spinal fluid, which was colorless and of normal pressure, showed 3 cells; the globulin (Pandy) was negative; the Wassermann reaction and the result of the colloidal mastic test were negative. The blood sugar, nonprotein nitrogen, uric acid, creatinine, chlorides, free calcium, total calcium and carbon dioxide combining power were normal. The sugar tolerance test gave a normal result. The basal metabolic rate was minus 11 per cent. Roentgenographic studies showed the lungs to be clear and the sella turcica normal. The stools were normal. The Ewald test meal gave a normal result.

Course.—The patient remained in the Phipps Clinic for three weeks, during which he had, on an average, one attack of sleep a day. The attacks resembled normal sleep and he was easily awakened. The attacks came on only when the ward was quiet. Between attacks, he was bright, alert and pleasant. At night he slept soundly. He was never observed laughing heartily. The polydypsia, of which he had spoken, was not observed in the clinic.

On his discharge from the clinic, the patient was given a job in one of the laboratories of the clinic, his duties being to assist in the laboratory and to feed and take care of the animals. After he became acclimated, he appeared rather

indolent and slow. He had no mechanical ingenuity. He frequently slept at his work. At times, he slept so soundly that he would awaken only when one called him in a loud tone. On numerous occasions when the laboratory comedian caused the other workers to laugh, the patient appeared to begin to laugh, then appeared to suppress the laugh and reached for a table or chair to steady himself. He never mentioned his malady and apparently was sensitive about it.

Examination in December, 1926, showed well developed external genitalia. The patient's symptoms were unchanged.

Comment.—In this case, one notes especially the change in personality at the onset of symptoms, the history of tuberculosis, the perhaps somewhat retarded puberty, the orthostatic albuminuria and the eosinophilia. The differential blood count was repeated twice, the eosinophil cells amounting each time to slightly more than 7 per cent. Eosinophilia has been noted in none of the cases hitherto published.

CASE 2.—*Male; late puberty; July, 1918, "Spanish influenza"; December, 1918, onset of attacks of sleep while awaiting demobilization from the Air Service; at termination of attack of sleep, he invariably lost a few drops of urine; May, 1919, onset of cataplexy—relaxation of jaw, neck, shoulders and arms on laughing, and relaxation of jaw on leaving warm bath and during orgasm; vagotonia.*

History.—G. C. H., a man, aged 26, married, a time-keeper at a hotel, was admitted to the Phipps Clinic on Jan. 6, 1927, complaining of attacks of irresistible drowsiness since December, 1918, and weakness on laughing since May, 1919. His previous health had always been good. He was robust and athletically inclined. A number of things in his history suggested a mild endocrine disorder. Until May, 1918, he had been short for his age, but from May to December, 1918, while in the air service, he grew 1½ inches (3.8 cm.), accurately measured. He had always looked young for his years; when he was 18, people usually took him to be 16. He did not remember the age at which puberty occurred, but it occurred late. Sexual libido and potentia were normal. He was married in July, 1924. His wife was five months pregnant at the time of his admission to the clinic.

He was always bright and lively. His mother, however, regarded him as rather absentminded. He had many nightmares until the age of 12 or 13, walked in his sleep until 10 and wet the bed until 12. He had lived the greater part of his life in London. He was precocious at school, having reached the lower fifth form (equivalent to graduation from an American high school) at the age of 15.

In July, 1918, while the patient was recovering in an army hospital from an operation for ingrowing toenail, the place was visited by an epidemic of what was called "Spanish influenza." He was taken ill with chills, malaise and weakness; his temperature was "fairly high". The next day his temperature was over 104 F. (40 C.), and he felt prostrated and apathetic and had pains in the joints and back. The next day he felt well again; his temperature was normal, and he was allowed to be up. During this brief illness, he had been neither drowsy nor wakeful and had had no diplopia, nausea or vomiting.

Sleep Attacks.—The symptoms of Gélineau's syndrome developed as follows: When the armistice was signed, the patient was in a school of aeronautics. Following the armistice, he continued to attend the lectures, but the thrill of war was gone and he and the other cadets found the lectures tedious. Early in December, 1918, he began to sleep during many of the lectures. He ascribed this to boredom. After demobilization (Dec. 22, 1918), he spent two months at home looking for

work. During this period, he napped for three hours every afternoon and slept eight or nine hours at night. In February, 1919, he secured a job in an aeroplane factory. There he fell asleep at work, and for the first time realized that the somnolence was pathologic. After this, he had attacks of sleep nearly every day. They lasted from a few moments to several hours. The more bored he was, the more apt he was to fall asleep. In the street car, in church and while reading he almost invariably fell asleep. He often slept over his work. Less frequently, he fell asleep under more unusual circumstances. He sometimes fell asleep while walking, and at such times he usually walked on until he bumped into someone; he walked unsteadily, so that people thought he was intoxicated, but he never fell. He even crossed crowded thoroughfares while asleep. He fell asleep while driving an automobile. Once, while riding a bicycle, he fell asleep and almost ran into an omnibus, but was awakened in time by the cries of pedestrians. On two occasions, he fell asleep in the dentist's chair, while the dentist was actually drilling his teeth.

During the attack, the patient appeared to be in a natural sleep; sometimes he snored and usually he dreamed. He was easily awakened. When he slept while at work in the factory, the noise of the factory failed to disturb him; but if someone then called him, he would awaken. It never required more than two or three good shakes to awaken him.

The sleep attack was irresistible. Sometimes, on awakening, the patient felt alert; at other times, he felt a little "confused" for a few moments. Invariably, when he awakened, he was aware of a "depressed, heavy feeling" in the precordium, lasting for a quarter of an hour. Invariably, also, at the moment he awakened, a few drops of urine escaped, and he experienced an urgent desire to urinate. There had never been any disturbance of the anal sphincter. On a few occasions, his tongue felt sore when he awoke, but it was never bloody.

He injured himself only once when he fell asleep while leaning against a radiator; he was awakened by a painful burn on his shoulder. He had never had convulsions, nor had he frothed at the mouth.

The patient had been an exceptionally sound sleeper all his life. He had had nocturia all his life (once or twice a night). Before 1919, he used to get up in the morning feeling refreshed; after 1919, he had been feeling tired in the morning. He usually felt more alert in the evenings than in the mornings and the afternoons.

Attacks of Cataplexy.—Cataplectic symptoms first appeared in May, 1919. When the patient laughed, he sometimes experienced a "relaxation" of the muscles of the jaw, neck, arms and shoulders, so that the head dropped forward, the jaws dropped, the chest caved in and the arms dropped. If he was holding anything, he usually lowered it an inch or two, and if it was fluid he spilled some of it. There had never been any relaxation of the muscles of the legs. The cataplectic phenomenon did not occur every time he laughed; sometimes for several weeks the act of laughing was unaccompanied by cataplexy.

Anger had never resulted in the cataplectic phenomenon. The patient had had no opportunity to observe the effect of great fear or grief.

The patient had the habit of taking a hot bath in the evening. Occasionally, when he began to get out of the warm comfortable tub, his jaw relaxed for a few seconds.

Occasionally, the jaw relaxed for a few seconds at the moment of orgasm, during both coitus and masturbation.

The cataplectic phenomenon was never followed by an attack of sleep.

The patient's symptoms were more severe during the first six months of 1919 than at any time since. The two months after his marriage (July, 1924), constituted his best period. In September, 1924, his symptoms became more severe again.

In the summer, 1926, the patient consulted a physician, who prescribed phenobarbital, ½ grain (0.03 Gm.) twice a day. The patient took this medicine for six months, but noticed no improvement.

In the Phipps Clinic, the patient behaved normally except for the sypmtoms to be described.

Physical Examination.—The patient's height was 65 inches (165.1 cm.); his weight, 167 pounds (75.7 Kg.). His general appearance was that of a short, stocky, muscular person. The skin was dry and rough over the elbows. The thyroid isthmus was palpable. The external genitalia were well developed. The distribution of hair was of the male type; the distribution of fat was normal. There was no enlargement of the lymph glands. There was an anomaly in the formation of the lower jaw, so that the right lower first bicuspid tooth was directed medially instead of upward. There was evidence of moderate vagotonia—the pupils were small (usually under 2 mm. in diameter), the pulse rate was slow (from 52 to 76), and the temperature was usually below 98 F. The pupils reacted normally. There was a slight ptosis of the right lid (probably congenital). There was a slight tendency to lateral nystagmus. The biceps, triceps and periosteal radial reflexes were more active on the left. The neurologic examination otherwise gave negative results. The Babinski sign was absent. The heart, lungs and abdomen were normal. The blood pressure was 118 systolic, and 74 diastolic (recumbent posture).

Laboratory Examination.—The urine was normal except for the occasional presence of a small quantity of albumin. The hemoglobin content was 96 per cent (Sahli). The red cell, white cell and differential counts were normal. The Wassermann reaction of the blood was negative. The spinal fluid was colorless and of low pressure; it showed 1 cell; the globulin test (Pandy) was negative; the Wassermann reaction was negative; the colloidal gold and colloidal mastic curves were negative; the spinal fluid sugar amounted to 64 mg. per hundred cubic centimeters. The blood sugar was 95 mg. per hundred cubic centimeters; the nonprotein nitrogen, on three occasions was 46, 50 and 43 mg.; the uric acid was from 4.3 to 5.5 mg.; creatinine, from 1.5 to 2.5 mg.; the chlorides and the carbon dioxide combining power were normal. The sugar tolerance test gave a normal result. The basal metabolic rate was minus 1 per cent. A roentgenogram of the head showed the skull and the sella turcica to be normal. The stool was normal. Caloric vestibular tests gave normal results.

Course.—The patient remained under observation for six weeks. He slept from two to four times a day. During two of the attacks, it was observed that the pupils were constricted and that they dilated a little at the moment when the patient became awake. He was always easily aroused. On several occasions, while laughing, the patient felt his jaw drop a little and his head nod an inch or so. No one else noticed this. On at least one occasion, he was seen laughing uproariously with no cataplexy.

He showed no polydypsia, the fluid intake varying from 1,100 to 1,900 cc. daily.

In the absence of indications for any other drug, it was decided to try scopolamine for two reasons: 1. It was hoped the hyoscine would at least reduce the vagotonic symptoms. 2. It was considered possible, though only remotely so, that the illness of July, 1918, was an atypical attack of encephalitis of which

the subsequent symptoms were the sequelae. Scopolamine hydrobromide was started on Jan. 28, 1927, and the doses were gradually increased, so that by February 1, he was receiving 1/25 grain (2.5 mg.) daily. The only effect was that the pupils enlarged to a moderate size and the mouth became dry. The pulse rate was only slightly increased, varying from 60 to 80. There was no abatement in the symptoms.

On Feb. 19, 1927, the patient returned to his home city. According to a report from his physician (Dr. L. H. Bartemeier), on July 11, 1927, the continued administration of scopolamine resulted in no improvement, in spite of the fact that most of the time the patient received 1/16 grain (4 mg.) daily.

Comment.—The noteworthy features in this case are: the moderate vagotonia (interesting in view of the fact that normal sleep is itself a vagotonic state); the delayed puberty; the invariable loss of a few drops of urine on awakening from an attack of sleep; the relaxation of the jaw occasionally on orgasm; the fact that only the upper part of the body, and not the legs, became weak when the patient laughed, and the fact that cataplexy did not manifest itself every time he laughed.

Vagotonic signs have been recorded both for cases of Gélineau's syndrome and for cases of attacks of sleep without cataplexy. The patient reported by Sperling and Wimmer had a high grade vagotonia, as shown by his response to subcutaneous injections of epinephrine. He had increased salivation, and his basal metabolic rate was minus 14.

Chavigny reported the case of a young soldier who had sleep attacks, but no cataplexy, and whose pulse rate was always between 40 and 50. Fellows, in 1890, had a patient who suffered from morbid somnolence whom he treated with belladonna. The result was not stated. Vigdorchik reported the case of a woman, aged 32, who had suffered from sleepiness since the death of her husband two years before. She had slight exophthalmos. The author stated that, suspecting "anemia of the brain," he had prescribed atropine (about 0.0006 Gm. daily). After the patient had been taking the medicine for two weeks, her symptoms cleared up. She took the medicine another two weeks and reported three months later that she had remained free from symptoms.

Case 3.—Male; "influenza" at 13 and at 16; sleep attacks began at 17; cataplectic attacks at 19; moderate hypothyroidism.

History.—A. R., a man, aged 20, a student, was admitted to the Phipps Clinic on June 18, 1927, complaining of "going to sleep". "And I have a kind of giving 'way at times, mostly when I think of something funny or when I laugh."

The patient had been delivered by forceps. Mumps (complicated by bilateral orchitis) occurred at 11. In 1919, at the age of 13, he had "influenza." He was in bed for one week, with moderate fever, malaise, slight headache and backache, but no diplopia, and neither insomnia nor somnolence. In 1922, he again had "influenza." This time he was in bed for two weeks, with fever and sore throat, but without diplopia or any disturbance of sleep. Puberty occurred at 13. As far back as the patient could remember, he was sensitive to cold. He always required more blankets at night, and when swimming he had to leave the pool long before the other boys. (This symptom is interesting in view of the hypothyroidism which he was found to have.) He reached his full growth at 16.

Sleep Attacks.—Attacks of sleep began to occur at the age of 17. At first, the patient fell asleep only in school, at church and while writing. About a year before admission, the symptom became more marked, so that he fell asleep under more inappropriate circumstances. On four occasions, he fell asleep while walking along a country road. Each time, he was awakened by stumbling or colliding with something. He never injured himself. Once, while driving an automobile at twenty miles an hour, he felt drowsy for about a mile and finally dozed off. A moment later, the car came to a "bank" in the road where the road turned. This immediately awakened the patient, and he kept his car on the road. Occasionally, he fell asleep while conversing with some one.

Attacks of Cataplexy.—Cataplectic symptoms first appeared one year previous to the examination. When he laughed he got "weak all over"; his knees sank, his arms dropped, if they were raised, and his head fell either forward or backward. He usually caught hold of something or managed somehow to get a better grip on himself, and never fell. This momentary weakness did not occur every time he laughed. On a few occasions, it occurred when he smiled or when he became either angry or frightened. It also occurred when there was no display of emotion proper; for example, when he thought of something funny without smiling. It also occurred if, while reading a newspaper he came to some familiar statement or if the sense of what he was reading changed suddenly. On such occasions, he lowered the paper an inch or two. Sometimes, without lowering the paper perceptibly, he felt a sense of relaxation in his arms.

Emotional display had never been followed by an attack of sleep. He had never had convulsions.

On examination, the patient proved to be exceptionally quiet, and talked and acted rather slowly. He was of normal intelligence.

Physical Examination.—The height of the patient was 67 inches (170.2 cm.); his weight was 140 pounds (63.5 Kg.). The bony framework was normal. The skin was rather thick and a little dry. The distribution of hair was of the male type. The thyroid isthmus was not palpable. The pulse rate was 64. The external genitalia were well developed. The pupils were of average size and reacted well. There was a slight tremor of the fingers. The rest of the observations made on neurologic examination were negative. The tonsils were large. Physical examination otherwise showed nothing abnormal. The blood pressure was 120 systolic and 88 diastolic.

Laboratory Examination.—The urine was normal. The daily output was never more than 1,800 cc. The hemoglobin was 100 per cent. The red blood cells numbered 5,300,000; the white cells, 4,800 (polymorphonuclear neutrophils, 54 per cent; eosinophils and basophils, 3 per cent; lymphocytes, 33 per cent; large mononuclears and transitionals, 10 per cent). The Wassermann reaction of the blood was negative. The spinal fluid was colorless; it revealed 8 cells (the cells were not examined under high power); the globulin (Pandy) test was negative; the Wassermann reaction was negative; the colloidal gold and colloidal mastic curves were negative. The blood sugar, nonprotein nitrogen, creatinine, uric acic, chlorides and total calcium were normal. The sugar tolerance test was as follows: The blood sugar was 87 mg. per hundred cubic centimeters; 100 Gm. of dextrose was administered; one-half hour later, the blood sugar was 100.5 mg. per hundred cubic centimeters; one-half hour later it was 99.5; one hour later, 95; one hour later, 80. The basal metabolic rate on one occasion was minus 34 per cent and on another minus 21 per cent. Roentgenographic studies showed the skull and the sella turcica to be normal.

Course.—In view of the definite evidence of hypothyroidism, the patient was given thyroid extract. On July 3, he was given 1 grain (0.06 Gm.) a day. On July 30, this was reduced to ½ grain a day. His weight remained practically stationary. The basal metabolic rate on July 9 was minus 16 per cent; on July 12, minus 3; on July 30, plus 6; on August 5, minus 18; on September 6, minus 20. The pulse rate was usually from 60 to 70 until July 2; from then until July 22, it was low, often as low as 52, and once as low as 48, the cause of this bradycardia being obscure; after July 22, the pulse rate varied from 60 to 90, and usually was between 60 and 80. At the time of discharge from the hospital, on Sept. 11, 1927, there was no marked abatement of the patient's symptoms.

Comment.—This case is noteworthy because of the hypothyroidism. No improvement followed the administration of small doses of thyroid extract for seven weeks. In this respect, the case differed from the cases of Matzdorff and Wenderowič, in which, although the patients showed no evidence of hypothyroidism, they improved with thyroid therapy.

Another noteworthy feature of the case is the occurrence of sudden muscular relaxation even in the absence of emotional display, a feature noted in the cases of Matzdorff and Mankowsky.

CASE 4.—*Female; puberty at 13; at this time cataplectic attacks began; attacks of sleep began at 14.*

History.—E. D., a school girl, aged 16, unmarried, was admitted to the Phipps Clinic on May 12, 1927, complaining: "I fall asleep every place; and when I laugh I fall down." The first symptom had begun a year before; the second two years before. The patient was always healthy, bright and athletically inclined. At the age of 2 weeks she had an attack of "convulsions," lasting for two minutes, about which nothing is known. There were no further convulsions. She began to menstruate at 13; the periods were always regular.

Attacks of Cataplexy.—In the summer of 1924, while carrying a cup to the table, the patient started to laugh. She felt barely able to get to the table. Several persons present said: "Watch out or you'll drop it." However, she got to the table successfully. After this, she noticed that when she laughed she felt weak in the knees. Occasionally, she fell to the floor, even when holding on to something. Further, she said: "My eyes turn to either side—usually to the left, and when I try to get them back it hurts my head. I can't see plainly, it's all mixed up. My lips quiver and I can't laugh; I can't even say anything for a couple of minutes." Once she laughed while going down the stairs and fell three or four steps, but she did not hurt herself. Others observed that she threw her head back, that her eyes rolled, that she turned her head away as if to prevent others from seeing her, and that, though she tried to talk, her speech was not understandable. After the attack, she felt weak for a few minutes and then was all right again. When she was frightened, she felt a little weak, but never fell. No symptoms followed anger.

At no time was an emotional display followed by a desire to sleep.

Attacks of Sleep.—Morbid somnolence first occurred in the spring of 1925; at this time the patient noticed that after gymnastic exercises she felt weak and drowsy. Thereafter she frequently fell asleep. She slept on the street-car and the bus, and especially at school. She sometimes fell asleep while writing and awoke

to find that she had made some illegible marks on the paper. She could not resist the drowsiness. She was always easily aroused. On awakening, her head felt hot, and sometimes her tongue felt numb.

At night, her sleep was rather poor; frequently she was disturbed by anxious dreams.

She never bit her tongue, hurt herself or lost sphincter control. On one occasion, at the table, while every one was talking, she suddenly stared into space and wore a "vacant expression" for several minutes.

The family history was unimportant, except that a paternal uncle, aged 40, had for two years been "nervous" and unable to walk. Nothing further was known about him.

On admission to the Phipps Clinic, the patient impressed one as intelligent and well behaved.

Physical Examination.—The patient's height was 66 inches (167.6 cm.); her weight was 135 pounds (61.2 Kg.). She was well developed. The hands were rather large. The skin was thin and smooth, and not dry. There was some pigmentation in the axillae, over the neck and around the waistline. The distribution of hair was normal. The right lobe of the thyroid gland was rather firm. The lid slits were wide. The pulse rate varied from 80 to 90. There was no general glandular enlargement. The neurologic examination revealed nothing significant, except a slight tremor of the tongue and fingers. The heart, lungs and abdomen were normal. The blood pressure was 102 systolic and 70 diastolic.

Laboratory Examination.—The hemoglobin content was 90 per cent. The red cell, white cell and differential counts were normal. The Wassermann reaction of the blood was negative. The spinal fluid was colorless; the pressure was low, only 5 cc. of spinal fluid being obtained on lumbar drainage; it showed 1 cell; the globulin (Pandy) test and the Wassermann reaction were negative; the colloidal gold and colloidal mastic curves were negative. The blood sugar, nonprotein nitrogen, uric acid, creatinine, chlorides and carbon dioxide combining power were normal. The sugar tolerance test was as follows: the blood sugar was 84.2 mg. per hundred cubic centimeters; 90 Gm. of dextrose was administered; one-half hour later, the blood sugar was 93.4 mg.; one-half hour later, 85; one hour later, 64. The basal metabolic rate was minus 16 per cent. A roentgenogram of the head showed the skull and the sella turcica to be normal.

Course.—The patient was in the clinic a month. She had sleep attacks from which she was always easily aroused. On laughing, she was noticeably weak in the knees, but never fell; she shut her eyes and held her chin, which had a tendency to tremble.

No treatment was administered.

CASE 5.—*Female; cataplexy began at 14; sleep attacks at 16; attacks of amnesia.*

History.—V. B., a girl, aged 19, with no occupation, was admitted to the medical service of the Johns Hopkins Hospital on Jan. 14, 1927, complaining of attacks of falling when she laughed or was frightened, attacks of drowsiness and peculiar attacks of amnesia.

The cataplectic symptoms had begun five years before. When she laughed heartily or was frightened, she got weak and dizzy and fell to the ground. These attacks were never followed by sleep.

The attacks of sleep had begun three years before. She often fell asleep at the theater or while motoring. She slept for from five to fifteen minutes and was then wide awake. The onset and the frequency of the attacks of amnesia were

unstated. She was once riding with her mother and friends in an automobile. The friends were put off at a certain street and she and her mother proceeded home. After they returned to the house, she was able to remember nothing that had happened after she had entered the car. During the attack, the mother and friends had noticed nothing unusual in her appearance or behavior.

There was no history of convulsions or encephalitis. Aside from the symptoms, she had always been healthy, lively and athletic. Menstruation began at 12 and was always regular.

Physical Examination.—The patient's height was 61.5 inches (156.27 cm.); her weight was 111 pounds (50.31 cc.). The thyroid isthmus was a little full and smooth; no nodules were felt. The eyes were a little prominent, and the lid slits rather wide. The pulse rate was 76. The distribution of hair was normal. The pupils were large and reacted normally. The examination otherwise gave negative results. The blood pressure was 110 systolic and 75 diastolic.

Laboratory Examination.—The urine was normal; the daily output varied from 1,000 to 2,300 cc. The hemoglobin content was 86 per cent. The red cell, white cell and differential counts were normal. The Wassermann reaction of the blood was negative. The spinal fluid (Dr. S. E. Jelliffe, New York City) showed 4 cells; the Wassermann reaction was negative; the colloidal gold curve was negative; the spinal fluid sugar amounted to 69 mg. per hundred cubic centimeters. The blood sugar was 78 mg. per hundred cubic centimeters; the uric acid, 4.2 mg. The sugar tolerance test was as follows: the blood sugar was 92 mg. per hundred cubic centimeters; 100 Gm. of dextrose was administered; one-half hour later the blood age of 2 weeks she had had an attack of "convulsions," lasting for two mintues, about which nothing is known. There were no further convulsoins. She began to menstruate at 13; the periods were always regular.

Ophthalmologic Examination.—Moderate compound hyperopic astigmatism was observed.

Course.—While under observation, the patient at no time had fever. She had two or three attacks of sleep daily.

Comment.—The unusual feature in this case was the occurrence of the attacks of amnesia.

Dr. Llewellys F. Barker and Dr. Smith Ely Jelliffe permitted me to report this case.

REPORT OF THREE CASES OF MORBID SOMNOLENCE WITHOUT CATAPLEXY

The following cases present morbid somnolence without cataplexy, and are therefore not classified as cases of Gélineau's syndrome.

CASE 6.—*Male; at 9, began to be obese and to have attacks of sleep; puberty at 14; deficient sex drive; obesity; hypertrichosis; basal metabolic rate minus 13 and minus 21; indefinite response to thyroid extract.*

History.—G. L. S., a man, aged 20, single, an unskilled laborer, was admitted to the Phipps Clinic on June 21, 1926, complaining that for the past eight years he had had attacks of sleep. According to his parents, the attacks began at the age of 9, when he began to be obese. Following the onset, the patient never had a day free from attacks. Usually, he had two or three a day. They lasted from a few minutes to a half hour. The sleep resembled normal sleep; he was easily aroused and sometimes he dreamed. Sometimes, in a light doze, he heard what

went on about him. The attacks were more apt to occur when he was quiet or when he was seated. They also occurred while he was standing, but only once did an attack occur while he was walking. He could not resist the attack. On Sundays, when he was at home all day, he slept most of the time. His nocturnal sleep was not sound. He talked and laughed in his sleep. Almost every night, he awakened at midnight and lay awake for two hours (no nocturia). On arising in the morning, he felt fresh for about an hour and then usually began to feel tired and dull. He tired quickly on physical exertion.

There were no muscular symptoms on laughing or on any other emotional display. Emotion never led to an attack of sleep.

He was always quiet and good-humored. Puberty occurred at 14. He had normal erections, but was apparently deficient in sex drive. He did not care particularly for the society of girls and had no desire for coitus.

Mental State.—On admission to the clinic, the patient impressed one as quiet and pleasant, rather slow in thinking, but of average intelligence. He answered readily all the questions in the 14 year group of the Binet-Simon test.

Physical Examination.—The patient's height was 66½ inches (168.87 cm.); his weight was 175 pounds (79.4 Kg.). He appeared obese. The fat was universally distributed, with a rather large abdominal panniculus. The bony framework was heavy; the hips were not wide; the arms were rather short. There was marked hypertrichosis; the beard was well developed, and there was a heavy growth of hair over the chest, abdomen, legs and arms. The skin was soft and moist. The thyroid gland was not enlarged. The penis was rather small; the testes were normal. The voice was a little high-pitched. The neurologic examination gave negative results. The heart, lungs and abdomen were normal. The pulse rate varied from 60 to 100, but usually was from 60 to 70. The blood pressure was 124 systolic and 80 diastolic.

Laboratory Examination.—The urine was normal, except for an occasional trace of albumin; the daily output varied from 1,100 to 1,500 cc. The hemoglobin content was 95 per cent. The red cell, white cell and differential counts were normal. The Wassermann reaction of the blood was negative. The spinal fluid was not examined. The blood sugar, nonprotein nitrogen, uric acid, creatinine, chlorides, free calcium, total calcium and carbon dioxide combining power were normal. The sugar tolerance test was as follows: the blood sugar was 90 mg. per hundred cubic centimeters; 140 Gm. of dextrose was administered; one-half hour later, the blood sugar was 207 mg.; one-half hour later, 183, and one hour later, 107. The basal metabolic rate, estimated twice, was minus 13 and minus 21 per cent. Roentgenographic studies showed the sella turcica to be normal; there was no evidence of any mediastinal mass.

Course.—During his stay of three weeks in the Phipps Clinic, he had several attacks of sleep every day. He slept quietly, except for occasional solitary jerky movements of one arm or the other or of the whole body. He was always easily aroused. His nocturnal sleep varied in duration from five to nine hours a night.

In view of the subnormal basal metabolic rate, the patient was given thyroid extract, from July 8, when 4 grains (0.26 Gm.) a day was given, to July 22, when the amount was reduced to 2 grains (0.13 Gm.) a day. On Aug. 1, 1926, he reported that he felt more energetic, but noted no substantial decrease in the number of attacks of sleep. He added, however, that during the two weeks when he took 4 grains of thyroid extract a day, he slept less than subsequently when he took only 2 grains a day.

CASE 7.—*Male; recurrent periods of sleepiness, irritability, restlessness and polyphagia, lasting from one to six weeks, followed (in one observed instance) by almost complete amnesia for events during the attack; between attacks, pleasant disposition, alert; first attack occurred at 16, three days after the termination of a four-day illness characterized by sore throat, feverishness, severe occipital headache, restlessness and one instance of nocturnal visual hallucination (hypnagogic? delirioid?); spinal fluid four days after the third attack contained 12 cells.*

History.—D. C., a school boy, aged 16, was admitted to the Phipps Clinic on July 10, 1925, with the complaint that he was in his third attack of somnolence. Until April, 1925, he had always been well. He was cheerful, bright at school, lively, athletic and robust. In 1924, he had an attack of tonsillitis. In March, 1925, he had an attack of mumps; there were no unusual symptoms, and he was well in two weeks.

On April 24, 1925, he participated in an athletic meet in the rain. On April 27, he complained of sore throat and felt feverish. A physician diagnosed the condition as tonsillitis. On April 29, he complained of severe occipital headache and tossed in his bed until midnight. On April 30, he said that the night before, while trying to fall asleep, he had seen "glass bottles" floating around the room (he had not appeared to be disoriented). On April 30, he fell asleep without difficulty. On May 1, he felt well enough to attend a banquet in the evening. He returned and was taciturn, which was unusual for him. On May 2, he continued taciturn. On May 3, at 4 p. m., he fell asleep and slept all the afternoon, evening and night. On May 4, he was drowsy most of the day. He continued to be drowsy for the next eight days. He went to school, but fell asleep at his desk. He was dull and had little to say, in marked contrast with his usual behavior. He said: "I must have sleeping sickness," and later: "I wish I knew what was the matter with me. I'm not sick a bit, but I just can't recall things." On May 13, the picture changed: (1) he became restless, walking constantly up and down the yard; (2) the drowsiness was considerably better, though he still slept off and on, by day, and straight through the night; (3) he showed marked polyphagia, eating many things between meals. On May 18, all the symptoms vanished, and for the next two weeks he was well.

The second attack began on June 1, and followed the pattern of the first. From June 1 to June 3 the patient was drowsy, and from June 4 to June 8 he was less drowsy, but very restless and hungry all the time. On June 9, he was well again and remained well until July 3. During this period, he said that just before the second attack began, on June 1, he had felt a premonition that an attack was coming. "I just felt consciousness leaving me."

On July 3, the third attack began. The patient was drowsy until July 6; then the drowsiness diminished, and he became restless. This time he showed great irritability, which he had not shown in the first two attacks. He swore freely. He insisted on having some ice cream and said: "I'm going to holler till I get it." He walked unsteadily, but did not fall. He again was excessively hungry.

Mental State on Admission.—The patient was admitted to the clinic on July 10, 1926. He was drowsy and fell asleep frequently while conversing with the physician. On July 11, the attack came to an end. Thereafter he behaved like a normal, lively boy. In retrospect, he said that during the attack he had felt sleepy.

Physical Examination.—The patient was well developed; his height was 65 inches (165.1 cm.); his weight was 121 pounds (54.9 Kg); his temperature was 98 F. (36.7 C.). There was no evidence of an endocrinopathy. The physical examination showed nothing significant.

Laboratory Examination.—The urine was normal. The Wassermann reaction of the blood was negative. The spinal fluid (withdrawn July 15) revealed 12 cells; the globulin (Pandy) test was negative; the Wassermann reaction was negative, and the result of the colloidal mastic test was negative.

Course.—The patient was discharged on August 6. He remained well until Dec. 6, 1926; then a fourth attack occurred. On December 3, he played "soccer" in the rain. On December 6, he fell asleep after supper and slept all evening and all night. On December 7, he went to school. His classmates noticed that he was "not himself." After supper, he again fell asleep. On December 8, 9 and 10, he stayed at home. He ate huge meals and slept the rest of the time. On December 11, he was readmitted to the Phipps Clinic.

Mental State on Readmission.—On readmission, he was drowsy and slept whenever he was alone. When disturbed, in order that the physician might examine him, he became restless, tossed about in bed and smoked one cigaret after another. He said: "I've been sick again, I guess. . . . I can't seem to grasp things—just a daze." He was oriented for place and person. He thought the date was December 3. He remembered only poorly the events preceding admission.

Physical Examination on Readmission.—The patient weighed 131 pounds (59.4 Kg.). The temperature, by rectum, was 98.4 F. (36.9 C.). The pupils were slightly oval and equal, and reacted normally. There was no ptosis. Extra-ocular movements were normal. There was a coarse tremor of the tongue and a slight fine tremor of the fingers. The tonsils were large, but not acutely inflamed. The glands at the angle of the jaw on each side were enlarged and moderately tender. The physical examination otherwise gave negative results.

Laboratory Examination on Readmission.—The urine was normal. The hemoglobin content was 90 per cent. The red blood cells numbered 5,200,000; the white cells, 9,400 (polymorphonuclear neutrophils, 53 per cent; eosinophils, 1 per cent; lymphocytes, 37 per cent; large mononuclears and transitionals, 9 per cent). The spinal fluid (withdrawn December 12) was colorless; the pressure seemed increased; it showed 1 cell; the globulin (Pandy) test was negative; the spinal fluid sugar amounted to 58 mg. per hundred cubic centimeters. The blood sugar was 98 mg. per hundred cubic centimeters.

Further Course.—For more than three weeks in the clinic, the patient showed excessive drowsiness and irritability. He slept much by day and averaged eight hours of sound sleep at night. He was irritable and profane. He expectorated on the floor, which he never did when he was well. On Jan. 1, 1926, he said: "I feel I'm beginning to come out of it. Yesterday I began to wonder where I was. I knew where I was, but it seemed just like a dream." He was still irritable, however. On January 3, for the first time, he had difficulty in falling asleep at night. He lay awake reading until 2 a. m. on January 4. He then slept six hours. When he awakened it was apparent that an extraordinary change had taken place. He was bright, pleasant and happy again. "I feel like I woke up again." He had almost complete amnesia for the events that had occurred during the attack. He said, "At the beginning of the attack, I felt it coming on me—you kind of feel drowsy and hardly know what's going on around you." He began to realize the situation on the evening of January 3. "I was all right then. I lay awake a long time because I realized all this trouble I had. I realized I'd had another spell and it worried me." During the rest of his stay in the clinic, he behaved normally.

Throughout the attack he had had no fever. The rectal temperature was never above 99 F. (37.3 C.). There had been no polyuria. The appetite had been irregular.

At no time had he ever had cataplectic symptoms or convulsions.

The patient was discharged on Feb. 8, 1926. He made up the lost time at school and was graduated from high school in June, 1926. In July, 1926, he had a fifth attack, lasting six weeks; in January, 1927, a sixth attack, lasting three weeks, and in July, 1927, a seventh attack, lasting six weeks. In none of these attacks was he in the hospital.

Comment.—In this case there were recurrent attacks in which the patient showed somnolence, irritability and polyphagia. The first attack occurred three days after the termination of a four days' illness in which he had had fever, sore throat, occipital headache and, one night, difficulty in getting to sleep and visions of glass bottles floating around the room. The attacks lasted from one to six weeks. Four days after the third attack, the spinal fluid contained twelve cells. The condition was probably an encephalitic process, but the evidence is not conclusive.

The occurrence of periods of drowsiness lasting for from one to six weeks, separated by periods in which the patient was normal, reminds one of Stöcker's second case, that of a man who, from the ages of 19 to 22, had periods of deep sleep lasting at first for several days and later for from two to three weeks. During these periods, he had to be awakened for meals; he awakened spontaneously to obey nature's calls. Between these periods of sleep he was normal. Between the ages of 22 and 26, when the report was published, he had no periods of abnormal sleep.

CASE 8.—*Female; sleepy since infancy; worse at 8; at 9, her mental age (Binet-Simon) was 7; undersized.*

History.—J. N., a girl, aged 10, was examined in the dispensary of the Phipps Clinic on July 7, 1927. The complaint was backwardness and lifelong sleepiness. She had walked at the age of 18 months and talked at 2 years. She was always quiet. She had little to say and rarely played with other children. She was unintelligent. She started to school at 7 and, at the end of three years, at the time of presentation, was still in the first grade. The only febrile illness she ever had had was scarlet fever, at the age of 7. She had always been small for her age.

Since infancy, the patient had always been sleepy. When young, she often sat down during the day and slept. Shaking often failed to rouse her, and her family had to pour cold water on her head. At night she slept soundly. When awakened in the morning, she was often "dazed" and not fully awake for ten or fifteen minutes.

Since March, 1925, the sleepiness had been more marked, as indicated by two facts: she often slept while standing, which she never had done before, and at night she snored the moment her head hit the pillow, whereas before 1925 she never had fallen asleep as readily as this. Recently, one morning, when not aroused, she slept until 3 p. m., when she was awakened.

The patient never was observed to laugh heartily. She was never angry; she rarely wept. No emotional display ever resulted in muscular weakness. She had never had convulsions or bitten her tongue. She was a chronic bedwetter and voided during diurnal as well as nocturnal sleep. The appetite had always been poor. There was no polydipsia.

Mental State.—On examination, the patient was quiet and said little. She cooperated well. She did not fall asleep. A Binet-Simon test, done in November, 1926, when she was 9 years and 4 months old, gave a mental age of 7.

Physical Examination.—The patient was very small, her height being 50½ inches (128.27 cm.), and her weight 57 pounds (25.9 Kg.). The neurologic examination showed nothing abnormal, except that the knee jerks were present only on reinforcement, and the ankle jerks were absent even on reinforcement. The rest of the observations made in the physical examination were negative, except that of large tonsils. The basal metabolic rate was minus 5 per cent. A roentgenogram of the head showed the sella to be normal.

Comment.—This case of lifelong somnolence brings to mind Janzen's third case, that of a man, aged 25, who had suffered from somnolence since the age of 5. Janzen designated the latter case "constitutional narcolepsy."

In addition to the cases here reported, I have observed morbid somnolence occurring in association with hysterical symptoms in two cases, with paresis in one case, with a traumatic psychosis in one case, and with a chronic mild depression in one case.

In the first seven cases presented in this paper, the patients were examined by Dr. Curt P. Richter, in the Psychobiological Laboratory of the Henry Phipps Psychiatric Clinic, with especial reference to the electric resistance of the skin as measured by the string galvanometer. The results of these examinations were recently reported by Dr. Richter.

SUMMARY

Gélineau's syndrome is a condition marked by the presence of morbid somnolence and cataplexy (relaxation of the muscular apparatus during emotional display). This condition has usually been referred to as narcolepsy, but the term narcolepsy has also been applied to other conditions. The term Gélineau's syndrome is less ambiguous, and hence preferable. Morbid somnolence, unaccompanied by cataplexy, occurs in a variety of physical and mental disorders, especially in organic disease of the brain and in endocrine disorders.

Sixty-six cases of Gélineau's syndrome have been found in the literature, and five more cases are reported in this paper. In none of the five cases was there evidence that the patient had had encephalitis. In only one case (case 2) was this considered even a remote possibility. In case 3 there was evidence of hypothyroidism. Three cases of morbid somnolence without cataplexy are here reported. One occurred in a young man with obesity and mild hypothyroidism, one in a young man with a history suggestive of encephalitis, and one in a girl aged 10 who was mentally retarded and "constitutionally" somnolent.

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THE RELATIVITY OF PSYCHOGENIC AND OF CONSTITUTIONAL FACTORS*

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Whatever divergencies of view there may be among different schools of psychopathology, there is agreement concerning the fact that in the development of psychopathologic conditions both the constitutional make-up of the person and his past and present experiences take a part. The most ardent advocates of hereditary influences have to admit that the situational circumstances of a person's life cannot be left out in the scientific analysis of any clinical picture. On the other hand, those who see in immediate situational and more deep-lying psychogenic factors the mainspring of psychopathic states have to rely on constitutional peculiarities to explain the special type or form which the reaction takes. The difficulty and the point of disagreement lie in the more precise evaluation of these factors.

It is exceedingly difficult to arrive at general guiding-lines which would allow the estimation of the relative value to be attributed to these two factors in the individual case. Psychoanalytic investigations have contributed a great deal to the understanding of why it is that a critical experience precipitates a pathologic reaction. But it seems likely that psychologic analysis alone may often mislead by overemphasizing an apparent motivation of episodic or periodic psychopathic upheavals.

Undoubtedly, there enters into the psychiatric evaluation of the pathogenic significance of any given situation the common sense human attitude of the observer. He judges whether or not the given situation is adequate to cause an upheaval. Theoretically, these evaluations are not on a clear scientific basis. Jaspers attempted to make a sharp distinction between "causal" and "understandable" connections in the chain of events of psychopathologic behavior. This distinction is not the solution of the problem, but merely the expression of a practical modus vivendi in psychopathology. Psychoanalysis would deny *a priori* the face value of a critical experience, emphasizing the intimate dynamic relationship of any affective experience with the attitudes and constellations of childhood. But even there it would seem that the common sense judgment as to the value to be attributed to any early experience enters, if only through a back door. It is astonishing how small is the store of definite knowledge concerning normal individual reactions

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* From the Johns Hopkins Medical School.

to experiences which provoke strong emotions. Little is concretely known about the course which such affective reactions take and how long they continue to be active. Apparently, every affective reaction, sthenic or asthenic, in a sensitive person or an indolent person, takes a certain course of time to work itself out until a relative equilibrium is reestablished. It is usually assumed that in a brief reaction precipitated by an emotional experience, the significance of the situational factors is paramount. In a chronic reaction, on the other hand, there is good reason to postulate a predominant significance of constitutional factors.

It is the purpose of this study to present the history of a patient which affords unusual opportunity for a clinical experiment concerning the evaluation of situational and constitutional factors. It shows how the reaction to a seemingly adequate pathogenic situation can really be correctly evaluated only in the light of deep-rooted constitutional peculiarities.

REPORT OF CASE

A successful business man, happily married and the father of three children, began, at the age of 48, to send many lengthy letters to friends and acquaintances. In them he complained, with many details, that an injustice had been done to him by a large university in connection with the granting of a degree. He was intensely preoccupied with this subject, and for a time devoted all his attention to it. The following circumstances had led up to this behavior, which, for him, was unusual.

He had been an excellent student in college and had graduated at the age of 23. At that time his general health was not good. He was suspected of having tuberculosis and was advised by his physician to do outdoor work. He was anxious to get a Master of Arts degree. On leaving the university, he called on the president for a list of books, the reading of which would be considered by the university as entitling him to the advanced degree he wanted. He obtained this detailed list of books for reading and home study.

Later, he went into business and was successful. He married at the age of 27. As his time permitted, he read carefully and conscientiously all the books which had been listed for him when he left college. After twenty-five years, having completed his reading, he wrote to the university and applied for his degree. He received a notice to the effect that the degree could not be granted to him. He was informed that shortly after he had left the university, the faculty had discontinued the practice of granting advanced degrees without resident study, and no degrees had been granted in this way for more than twenty years prior to his application.

He became much aroused at this disappointment. He wrote long aggressive letters to the president of his old university and to alumni and trustees. He entered on an extremely lengthy and voluminous correspondence with presidents of other universities, several lawyers and many of his acquaintances. Finally, he went so far as to threaten the university with legal proceedings to compel the granting of the degree. After several weeks, his activities simmered down and finally he dropped the matter entirely.

Comment.—If one attempts to interpret the patient's reaction to his disappointment, there is little doubt that the situation in which he found himself through the refusal of the degree was adequate to cause a strong emotional upheaval. Otherwise, the patient lived at the time in happy circumstances. He had always been an active, energetic man of "kindly and gentle" disposition. He was not given to suspicion, but is said to have "thought well of every one," a trait which made him an "easy mark" for various "get-rich-quick" schemes. His persistence is shown by the fact that he continued to work toward the goal of a degree for so many years. This followed the line of his tastes, for he was much interested in literature and occasionally wrote poetry and little essays. In his family history, it is of significance that a sister suffered in advanced age from a depression with delusions of jealousy from which she recovered. A brother was of "exceedingly positive mental make-up." Of his childhood, little is known except that he was not robust and started school at the age of 9.

As for the psychiatric interpretation of the condition following the refusal of the degree, it may be designated as a sthenic-expansive reaction to an intense disappointment. It is the situation which seems to deserve the emphasis in any characterization of the condition. In view of the course taken by the patient's reaction, its short duration and the final emotional adjustment to the new status, and with the, humanly speaking, adequate provocation taken into account, the whole reaction may be regarded as a situation-determined emotional upheaval.

A study of the further course of his life affords an interesting experimental opportunity to determine the relative significance of constitutional and situational factors in his case.

As he grew older, it was noticed that he became a trifle conceited. At the age of 59 he retired from business. About half a year later, he was much upset by a lingering illness of his wife, and became somewhat irritable and quick-tempered. For several years before retiring from business he had had some worries over business affairs and also over his son's escapades at school. Following his retirement, he spent practically all of his time with his wife. At the age of 60, a little less than two years after his retirement from business, he developed an acute psychotic condition. He accused his wife of being after his money and sending spies after him. He accused the employees at a hotel of being in a plot against him, and was talkative and overactive day and night. He showed a grandiose attitude. Within a few days, he had to be taken to a sanitarium. Later, he was admitted to the Phipps Clinic, where he showed marked excitement of manic type. At times a paranoid trend was uppermost, when he accused the physicians of trying to kill him, and was combative. More frequently there was outspoken elation, with pseudowitticisms, puns, jokes, grandiose statements and flight of ideas. A great deal of the content of his talk was of a sexual nature. The patient was later admitted successively to two private institutions. His manic excitement continued for a period of five years. He recovered and remained well for two years, then suffered a second attack of manic excitement with symptoms similar to those of the first attack. It was especially noted that he showed no signs of deterioration.

The patient's psychosis must be regarded as a prolonged manic excitement. The paranoid trend, which at times was apparent, may be considered as an integral part of the condition, occurring in a setting of overactivity and expansiveness. As a study of prolonged manic conditions [1] shows, a constitutional factor must be regarded at the present stage of knowledge as being of primary importance in these conditions. The situational factors in this case, namely, retirement from business, illness of his wife and other worries, must be considered as of definitely secondary importance in the development of the chronic excitement.

What light does the study of this later psychosis throw on the interpretation of the disappointment reaction of the patient following the refusal of his degree? It would be too formal and unjustified reasoning to regard the patient's behavior on this first occasion definitely as a previous abortive manic attack. On the other hand, it is apparent that his behavior on this first occasion resembles, in a marked degree, the later psychosis, in much attenuated form and with much briefer course. In view of the fact that there seems to be a definite biometric correlation between the chronicity of manic attacks and their incidence in mature and advanced age,[1] it seems significant that the chronic excitement of the patient began eleven years after his first upheaval, at the age of 60.

In the light of the later psychosis it would seem, therefore, that the evaluation of the importance of the situational factor in the first upheaval needs qualification. All human behavior has constitutional and situational determining factors. It seemed, at first, that the refusal of the degree constituted a pathogenic situation calling forth a response which, at least in its intensity and general form, was commensurate with the provoking conditions. In some degree, the form of the reaction appeared, of course, constitutionally determined. But even allowing for this pathoplastic constitutional background, the reaction appeared predominantly conditioned by the situation. It seemed to be an affective response which could be considered as essentially human rather than as due to especially significant individual constitutional tendencies. However, if the later psychosis is taken into consideration, it appears that the constitutional factor will deserve much more emphasis in the first upheaval, not only in a general way, as a predisposing background, but in a specific qualitative sense. It becomes evident that the refusal of the degree released a potentially serious constitutional tendency. Clinically, this tendency may be designated as a disposition to

1. Wertham, F. I.: A Group of Benign Chronic Psychoses: Prolonged Manic Excitements, Am. J. Psychiat. **9**:17 (July) 1929.

manic developments with closely related affective paranoid trends. Apart from the close resemblance of the two reactions, the tendency to recurrence of affective psychoses, evidenced in this case by the occurrence of another attack after the recovery from the chronic attack, plays a part in these considerations.

From a forensic-psychiatric point of view, this attempt at a more precise and concrete evaluation of situational factors is of especial significance. The whole circumstances of the first upheaval with the potentiality of paranoid misinterpretation and of less bridled overactivity may be regarded as not unusual soil from which criminal acts might develop. In any unusual behavior which on the surface seems essentially a natural response to a provoking situation, the legal mind is particularly averse to considering the existence of pathologic elements. Examples such as this case show the futility of dogmatic formulations. From the point of view of the scientific investigation of crime, as opposed to the exigencies of current legal practice, there can be little doubt that if the patient had committed a criminal act during his first upheaval, it would not have been entirely due to environmental factors. It would have been not a universal human reaction which was responsible, but a specific individual constitutional tendency which is open to clinical psychiatric investigation.

The relativity of the significance of constitutional and situational factors in individual cases becomes evident from the study of cases such as the one described. There is no great contrast between conditions predominantly due to situational influences and those predominantly due to constitutional factors. In order to arrive at precise formulations which will put these factors in their proper perspective, it is necessary to view a whole life history, not only tracing it back to childhood, but also following it through the significant manifestations of age. In the present stage of psychiatric research, there is often, unfortunately, a large division between constitutional and heredobiologic investigation on the one hand, and psychodynamic and environmental analyses on the other. This division is also reflected in dogmatic clinical and forensic-psychiatric opinions. It would seem that the more definitely constitutionally determined psychopathologic conditions deserve systematic study of psychogenic and situational factors. In the same way it would seem desirable that just in those cases in which psychogenic features are especially well demonstrated, methods of constitutional and biologic study be applied. These consist not only of anthropologic studies and investigations of heredity, but also of the working out of constitutional trends of personality based on clinical evaluation of whole life histories.

SUMMARY

In a patient who suffered from a minor psychopathic episode which seemed to be primarily situation-determined, a later psychosis revealed a specific, clinically definable constitutional factor; namely, a disposition to manic excitement with closely allied paranoid trends. This case affords an unusually clear clinical experiment, showing that a specific constitutional background can be elucidated not only by studies of heredity, or anthropologic or psychologic types, but also from the clinical evaluation of later psychopathic conditions. It shows that seemingly situation-determined reactions may be understood only if specific constitutional tendencies are fully realized. Since proper evaluation of these tendencies is frequently possible only in the light of later personality developments, the far-reaching relativity of psychogenic and constitutional conditioning seems to deserve more attention in the interpretation of emotional upheavals which on the surface seem easily explainable.

CAPILLARY FORMS IN RELATION TO CERTAIN PROBLEMS IN DEVELOPMENT*

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It is obvious that there is an increasing tendency to look on the living organism as a series of processes rather than as a structure. But in a final analysis, function must be correlated with structure, and if both together can be conceived of as dynamic a more fundamental understanding may be arrived at. The advantage, for example, of studying constitution from a developmental point of view rather than as a finished pattern is obvious. That there are demonstrable correlations between structure and the kind of process is thought by many persons familiar with the work on the psychoses.

This study does not pretend to cope with any of the larger problems. It is merely a study from the developmental standpoint of one minor factor, the form of the capillaries, in relation to constitutional and secondary hypophrenias, which may serve as a possible aid in the early detection of a constitutional defect and in the differential diagnosis of these two kinds of defects. It may give a suggestion of the potentiality for development or the degree of retardation in a given constitution, and suggest the need for treatment when it is possible.

Lombard,[1] in 1912, showed that by placing a drop of oil on the skin and by directing illumination from the side and above, the arterioles, small veins and capillaries could be seen through a microscope. This method was utilized by Weiss [2] and Ottfried Müller,[3] who made a large number of observations, especially on patients with various diseases, and who developed theories on the relation between the kind of capillary and the constitution, as well as on the fundamental relations between the organs and systems arising out of the different germ layers.

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* Submitted for publication, April 24, 1929.

* From the Clinical Department, Letchworth Village.

* Read at the Annual Meeting of the American Orthopsychiatric Association, New York, Feb. 22, 1929.

1. Lombard, W. P.: The Blood Pressure in the Arterioles and Small Veins of the Human Skin, Am. J. Physiol. **29**:335, 1911.

2. Weiss: Beobachtung und mikrophotographische Darstellung der Hautkapillaren am lebenden Menschen, Deutsches Arch. f. klin. Med. **119**:1, 1916.

3. Müller, O.: Die Kapillären der menschlichen Körperoberfläsche in Gesunden und Kranken, Stuttgart, Ferdinand Enke, 1922.

The work with children [4] was done first, for the most part, by Walter Jaensch,[5] Wittneben [6] and Hoepfner.[7]

TECHNIC

The technic is essentially that described originally by Lombard. The skin around the base of the nail may be cleaned with oil and dried. A drop of oil is then put on and the skin is examined with a magnification of 40. Cedar or mineral oil is satisfactory. If the examination is made near a window on a bright day, no artificial light is necessary; otherwise, an ordinary 40 watt, frosted bulb with a shade is serviceable, and should be placed just in front of and slightly above the stage of the microscope.

DEVELOPMENT OF CAPILLARIES

It is generally conceded that the capillaries of the skin at the nail bed present a constant picture. Crawford [8] stated that they are as characteristic in a given person as his face, and concluded from moving picture studies that they remain essentially the same from day to day and from minute to minute. They do not appear and disappear as they are reported to in other parts of the skin. It is a rule for them all to be open.[9]

The skin capillaries have their origin in the arterioles of the subpapillary arterial network which lies near the junction of the middle and outer thirds of the cutis.[10] These arterioles, according to Krogh,[11] do not anastomose normally. A twig is sent up to form a capillary loop which approaches the corium, there being one hairpin-like loop in each scallop of the corium (chart 1, *A*1). This is the normal adult appearance from which there are many deviations, a fact that has given rise to several theories of their significance, the most reasonable of

4. Jaensch, W.: Ztschr. f. Kinderforsch. **22**:4, 1926.

5. Jaensch (footnote 4). Jaensch, W., and Wittneben, W.: Archkapillären, endokrines System und Schwachsinn, Sitzungsbericht des zweite Deutschen Kongress für Heilpädagogik, Berlin, Julius Springer, 1925.

6. Wittneben, W.: Kasuistik und Therapie archikapillärer Zustandsbilder bei Jugendlichen, Ztschr. f. Kinderforsch. **22**:4 (July) 1926.

7. Hoepfner, T.: Die Structurbilder der menschlichen Nagelfalzkapillären und ihre Bedetung in zusammen mit Schilddrüsenveränderung sowie gewissen Schwachsinns und Neurosenformen. Veröffentlichungen aus dem Gebiete der Medizinalverwaltung, Berlin, Richard Schoetz, 1928.

8. Crawford, J. H.: Human Capillaries: Observations on the Capillary Circulation in Normal Subjects, J. Clin. Investigation **2**:351 (April) 1926.

9. Lewis, T.: The Blood Vessels of the Human Skin and Their Responses, London, Shaw & Sons, 1927.

10. Spalteholtz, W.: Blutgefässe der Haut- und Geschlechtskrankheiten, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1927, vol. 1.

11. Krogh, A.: Anatomy and Physiology of the Capillaries, New Haven, Yale University Press, 1927.

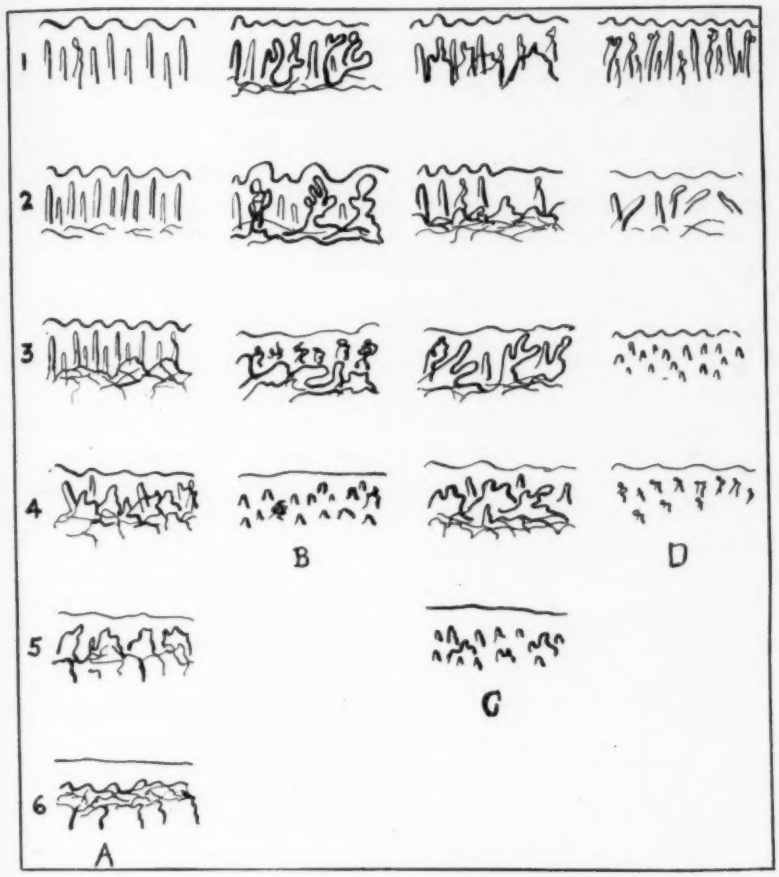

Chart 1.—Normal and pathologic development of capillaries. Column *A* shows the normal development: (6) primitive network observed shortly after birth; (5) widespread, saddle-shaped loops seen at 4 or 5 weeks of age; (4) appearance of a few hairpin-like loops with differentiation in caliber and the beginning of the scalloped form of the corium; (3 and 2) disappearance of the wide loops and development of the corium, and (1) adult form attained at from approximately 6 months to 6 years of age. Column *B* shows the abnormal development from the primitive form shown in A6, retaining some of the characteristics such as the flat corium, the network and especially the equally wide caliber of both limbs of the loops. Column *C* shows the incomplete development from the intermediate state (*A* 4 and 5), showing the more or less flat corium, network and wide loops and caliber. Column *D* shows the variations from the ideal normal development showing in irregularities of arrangement (*D* 2), corkscrew forms (*D* 1) and hypoplastic forms (*D* 3 and 4). The two latter types are also shown in groups *B* and *C* which in addition show some of the foregoing characteristics.

which is derived from a study of the development of the capillaries in normal infants and in children deviating from the normal in physical or mental development or in both.

At about the ninth day after birth, when the first observations were made, there are no capillary loops but a network supplied by arterioles from the deeper network (chart 1, *A* 6). Usually at 4 or 5 weeks of age, widely spread loops, often notched like a saddle, appear (*A* 5). Gradually they develop into the slender, hairpin-like capillary forms which do not appear in any number until from the fourth to the sixth month (*A* 3 and 4). At the same time, the horizontal network of fine blood vessels decreases in size and complexity, and the corium, which might be represented by a straight line at birth, becomes waving. When definite loops appear, it gradually assumes the typical scalloped appearance (*A* 1 and 2). The infants considered as normal were of good inheritance and normal birth and development; they were well nourished and gave negative Wassermann reactions.[12] Development of the capillaries in some was complete at 6 months of age, although in others it might not be entirely adult until 6 years. Less generally well developed children showed a slower capillary growth. Delayed capillary development in apparently normal children was especially noted in regions where goiter was endemic, and in these districts some apparently normal children never attained the ideal normal adult capillary forms. This will be discussed later.

Studies have been carried on by several investigators with large numbers of normal, retarded and feebleminded children in various regions. As a result of these studies a normal and pathologic morphogenetic scheme of development has been worked out by Hoepfner, following the original scheme of W. Jaensch, after a study of more than 3,000 cases.[13] When general development is markedly defective the capillaries may never lose the main characteristics of the primitive network form (chart 1, *A* 6 and *B*), although there may be more or less vertical hairpin forms; in addition, the horizontal vessels will be present, and both limbs of the capillary loops will have a wide caliber. The corium will be flat, except over the normal capillaries where it may be scalloped (*B* 1, 2 and 3). The size may be normal or hypoplastic (*B* 4). These are known as "Archikorrecturformen," i. e., forms which have retained certain primitive attributes relating them genetically and structurally with the primordial form, but which as a result of corrective tendencies have become approximately normal forms.

The next step in normal growth is represented by the intermediate or mesocapillaries (chart 1, *A* 5). Again in abnormal development,

12. Jaensch and Lowenthal, quoted by Wittneben (footnote 6).

13. Jaensch, W.: Die Varienten der Kapillärschichten, in Jaensch, Wittneben, Hoepfner and Leopold: Kapillarbuch, Halle, Carl Marhold, now in press.

remains of the characteristic structure of this stage may be found, i.e., relatively large caliber, broad, more or less saddle-shaped loops with undifferentiated venous and arteriole branches and the corium more or less flat (*C* 1, 2, 3 and 4). These same characteristics may be present in hypoplastic forms (*C* 5).

In a similar way there may be deviations from the ideal normal or neocapillaries (*A* 1) which, however, do not show any of the characteristics of the two groups mentioned. The deviations are in size (*D* 3 and 4) and in irregularities of arrangement (*D* 2) or in the appearance of corkscrew forms (*D* 1). All combinations may be found, and much more elaborate schemes have been worked out, but this simple one serves the needs of this work.

It has been found by others that there is a correlation between these poorly developed capillaries and a general defect in the development of what has been called the psychophysical personality. This was first observed in cretins, but is no less true in other types of defectives. Weygandt,[14] in 1912, described like deviations in capillaries of the brain cortex in similar cases.

Wittneben[6] found primitive capillaries in 10 per cent of 500 children showing all degrees of feeblemindedness and in 3 per cent, who were among the retarded, in an institution for supposedly normal children in the same town. These and other results are not entirely comparable with the work quoted later, as somewhat different criteria were used and all grades of feeblemindedness were considered as a unit.

Kahle,[15] in a study of 507 feebleminded persons, found that 17 per cent had markedly primitive forms, 53.4 per cent had deviations from normal forms and 29.6 per cent closely approached the normal. No specific statement as to the degree of feeblemindedness is made, except that about 80 per cent were in the special kindergarten and other special classes. Delbruch,[16] working in the same district, was unable to substantiate these observations, since he found the pathologic types in as many normal and superior children as in those in special classes; no mention is made, however, of the physical condition.

Recent work in Germany[17] showed that 67 per cent of 123 children in a school for normal children had normal or relatively normal capillaries of average size, whereas of 113 in an ungraded school only 29 per cent were of this type. Practically the same figures were also found by Spatz and Wandowsky[18] in the same district.

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14. Weygandt, Vogt: Weygandtschen Ztschr., 1912, Bayon.
15. Kahle, H. K.: Arch. f. Psychiat. **81**:629 (Sept.) 1927.
16. Delbruch, H.: Arch. f. Psychiat. **81**:606 (Sept.) 1927.
17. Langood: Preuss. Landesgesundheitstrat **26**, XI, 1928.
18. Spatz and Wandowsky: Preuss. Landesgesundheitstrat **26**, XI, 1928.

It is possible that marked contradictions might be due to differences in classification of intelligence; they might also have a relation to general physical development which had not been taken into account, as it is well known that with low intelligence there is more or less proportionately defective physical development. Rosenblüth [19] and others have found that the greater the degree of defectiveness, the less the child approaches normal growth.

In those children in whom endocrine treatment was indicated and given, further development of the capillaries commensurate with other changes was said to have taken place.[6]

STUDIES AND RESULTS

In the work at Letchworth Village, 679 feebleminded patients were studied. In 82 the symptoms were definitely of secondary origin; they were due to trauma, encephalitis, meningitis, etc., in children of good inheritance with previously normal development. These children might be considered a control group. In addition, 88 physically normal children in a private school who were between 3 and 6½ years of age, with intelligence quotients over 110, were studied. Each child had been given a Terman and physical examination in addition to the capillary study. The constitutional hypophrenias were divided into three groups according to the intelligence quotient: (1) an intelligence quotient from 0 to 30, which includes the idiot and low grade imbeciles and in which physical maldevelopment is most marked; (2) an intelligence quotient from 31 to 50, in which the physical development approaches normal and the patient is somewhat trainable, and (3) an intelligence quotient from 51 to 80, in which few stigmas and approximately normal growth are usual.

It was found (chart 2) that 43.1 per cent in the lowest grade had capillaries with primitive characteristics, 14.2 per cent with intermediate, 19.44 per cent with relatively normal, and 4.8 per cent with "ideally" normal forms. In the next two groups with an intelligence quotient from 31 to 50 and from 51 to 80, the results were practically the same, showing a marked decrease of maldevelopment and an increase in the tendency toward normal development. There were 143 patients with an intelligence quotient from 30 to 50, and 271 in the group with an intelligence quotient of from 51 to 80; 14 per cent and 10.7 per cent, respectively, were of the primitive type; 10.5 and 13.3 per cent were intermediate; 60.2 and 61.3 per cent were relatively and hypoplastically normal, and 15.3 and 14.8 per cent "ideally" normal.

On the other hand, the 82 patients with symptoms of secondary origin showed none of the primitive forms. Thirteen and five-tenths

19. Rosenblüth: Langen und Massenwachstum schwachsinniger Kinder, Ztschr. f. Kinderh. **46**:548, 1928.

per cent were derived from the intermediate stages. Five of 11 patients, however, developed the condition before 1 year of age and all developed it before 5 years. Thirty and five-tenths per cent were approximately normal, and 46.3 per cent "ideally" so.

These results check with the observations on superior children of known good physical development. No primitive forms were found. As these children were between 3 and 6½ years of age, it was considered normal when there were slight evidences of a network remaining (present in one-third between 4 and 6½ years of age, and in one-half between 3 and 4 years) and no signs of underdevelopment. Over one-half (52.2 per cent) were ideally normal and 38.6 per cent approximately so; 9 per cent had intermediate forms.

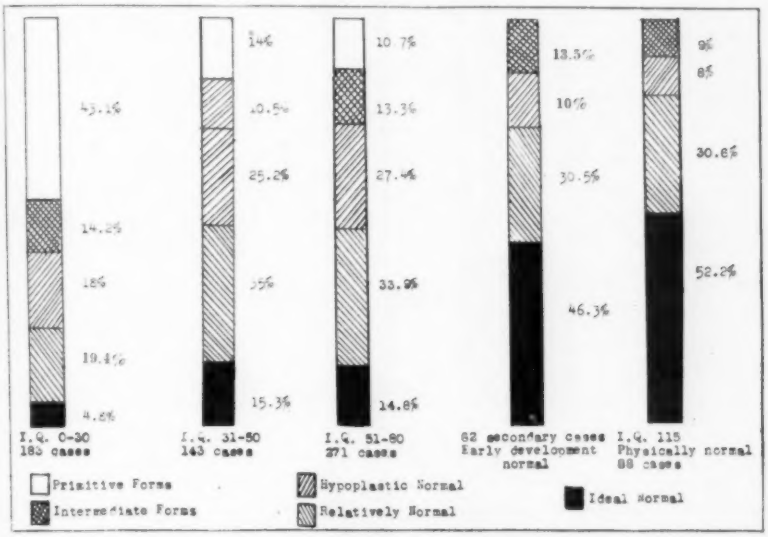

Chart 2.—The abnormal deviations from the primitive form were common in the idiot and low grade imbecile group, relatively uncommon in the higher grade imbecile and moron group and absent in children with a normal early development. The entirely normal capillaries are found in approximately 50 per cent in the latter group, 15 per cent in the middle group and 5 per cent in the lowest group. A study of this 5 per cent is shown in chart 3.

An effort was then made to find if there was any marked characteristic of those low grade patients who had normal capillaries. It was found that 21 of the 150 patients were physically normal, and of these only 2 showed primitive and 1 an intermediate form, while 12 were relatively normal, and 5 ideally so (chart 3). On the other hand, the 34 Mongols showed practically the same percentage distribution as that of other low grade imbeciles and idiots.

In a study of 33 cases of Little's disease, the criteria being those of diplegia and mental defect occurring before myelinization is complete, the picture was similar to that of other congenital idiots though somewhat exaggerated, there being even more (72.5 per cent) primitive and intermediate forms.

The question then arose as to the characteristics of the 100 patients (24 per cent) in the groups with an intelligence quotient above 30

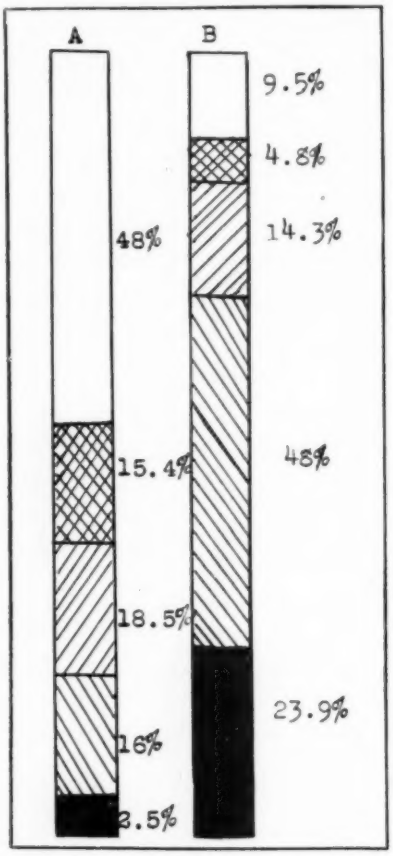

Chart 3.—*A* represents 162 patients with an intelligence quotient of from 0 to 30 who were physically abnormal, and *B*, twenty-one of the same mental level who were physically normal. There is apparently a significantly smaller percentage of primitive capillaries found even in these patients with extreme constitutional mental defect who were, however, physically normal, in contrast to the observations in those with marked physical defect associated with idiocy.

showing markedly defective capillaries. It is well known that adults of superior intelligence may have primitive capillaries, and it was thought that a study of these cases might give some understanding of this problem. It was possible to get from the physicians in charge of the

patients an estimate of the emotional status in 70 unselected cases of this group and in 74 unselected cases showing normal capillaries. They were also studied from the standpoint of endocrine and neurologic observations with the results shown in tables 1 and 2. In brief, of those persons in whom the capillaries were normal, 81 per cent had no physical or emotional abnormality as far as was determined. None were psychotic; 11 per cent were unstable, and 6.8 per cent had endocrine disorders. In those with abnormally developed capillaries, only 24 per cent had no discoverable physical or mental abnormality. Thirty-three per cent showed an endocrine dystrophy, 10 per cent were psychotic and 18 per cent unstable.

TABLE 1.—*The Relation Between Capillary Development and Physical or Psychiatric Abnormalities*

Capillaries	Total No. Cases	Marked Physical or Psychiatric Abnormalities	
		No. Cases	Per Cent
Primitive	70	53	76
Normal	74	14	19

TABLE 2.—*Distribution of Cases with Primitive and Normal Capillaries*

Condition	Primitive Capillaries, No. Cases	Normal Capillaries, No. Cases
Endocrine dyserasias	24	5
Microcephalic	3	0
Neurologic conditions	2	0
Epilepsy	1	0
Excitable	3	1
Unstable	13	8
Psychotic	7	0
No pathologic condition	17	60

It was found[7] in regions in which endemic goiter is present that development of capillaries even in children apparently normal is in many cases slower than in other regions, and that there is a higher percentage of hypoplastic forms, although no primitive forms are found in normal children.

COMMENT

It seems possible from this and similar studies that another relation between physical and mental development may be demonstrated. In those cases in which there is a marked constitutional inhibition of mental or emotional maturity, this same inhibition may operate to retard the development of the vascular system as observed in the capillaries. The possibility is suggested that by early examination of the capillaries a developmental anomaly may be uncovered which may show itself then or later as an endocrine, emotional or intellectual defect, or all three. In East Prussia this idea is being tested by including with

the physical a capillary examination of all children entering school, and the two together are made a basis for further study and treatment if indicated.[20]

It is also possible that an examination of the capillaries may help to establish a diagnosis when the early history is inadequate to distinguish between a constitutional etiologic and an environmental (disease or injury) factor.

SUMMARY

1. By means of a simple technic the development of the capillaries in the nailbed may be observed.

2. In constitutional idiots and low grade imbeciles, in whom there is a high proportion of physical abnormality (88 per cent in the present series), there was a marked tendency for a retarded or abnormal development of the capillaries; this was much less marked in the few cases which showed an approximately normal physical development.

3. In the moron and high grade imbecile group there were relatively few primitive forms. In the cases in which these forms were found there was a significantly greater proportion of emotional, endocrine and neurologic disorders.

4. No primitive forms were found in cases of secondary origin and in stable children with normal physical and mental development. The percentage of normal forms was high.

5. Suggestions were made for the possible applications to diagnosis.

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20. Personal communication.

TUMORS OF THE BRAIN AMONG FILIPINOS*

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It is a truism that many things are likely to escape one's observation unless one is particularly minded toward them. This is true of the present subject. The object of this brief survey is to point out what might be expected locally in the field of tumors of the brain or intracranial tumors, in general, and to cast a retrospective glance into the omissions and commissions of the past in the diagnosis of these conditions.

Of 13,168 autopsies performed at the city morgue from Aug. 1, 1907, to Dec. 31, 1927, I have selected only those in which an examination of the brain was performed; of these there were 4,602; 3,081 were males and 1,521 females. Exclusion was made, however, of cases in which either the brains were reported to be lacerated or in a state of decomposition (except when the tumors were definitely recognized [two cases] or the anatomic diagnosis of tumor was made with a question mark). This was done in order to do away with as much source of error as possible.

Metastatic tumors (10 cases) were likewise excluded as of no particular therapeutic interest for the time being, as were also cystic conditions or mere enlargements of the choroid plexuses, pituitary and pineal, and so-called syphilomas and tuberculomas. Inclusion of these would no doubt raise the incidence of tumors of the brain among Filipinos, to say nothing of systematic examination of the brain in all cases coming to autopsy.

This paper deals, therefore, only with tumors primary in the brain or in the meninges, of which there were 16 in 3,081 males, or 0.52 per cent, and 12 in 1,521 females, or 0.79 per cent, making a total of 28, or a combined incidence of 0.61 per cent of primary tumors of the brain among Filipinos. This compares with the figure of Bollinger, in Munich, of over 1 per cent and with that of Hale White, which is nearly 2 per cent. According to Da Costa, from whom these figures are quoted, "No region of the body is so liable to tumors as the brain."

CHARACTERISTICS OF THE CONDITION

Age.—Tumors were somewhat more frequent between the ages of 16 and 25 among females, and between the ages of 31 and 35 among

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males. The extremes of age were 8 months and 80 years; in both cases, the tumor was in the pituitary.

Sex.—Tumors were somewhat more frequent among women—0.79 per cent against 0.52 per cent among men.

Social Condition.—All patients in this survey were free patients.

Location of the Tumor.—Four, or 14 per cent, of the tumors were located about the temporal lobe; three, or 11 per cent, about the parietal; and three, or 11 per cent, either in the basal ganglia or around the optic chiasm; there was one case each in the frontal lobe and in the corpus callosum, and one of multiple gliomas. Four, or 14 per cent, started in the dura and eventually involved the brain in two. Altogether, therefore, seventeen, or 61 per cent, of the tumors were supratentorial, almost two thirds of which were on the left side; two, or 7 per cent, were located in the cerebellum, and nine, or 32 per cent, were in the pituitary.

Histologic Examination.—In eight, or 29 per cent, of the cases either histologic examination was not made or the type was not determined. In eight of the remaining 20, or 40 per cent, the tumors were diagnosed as endotheliomas; in six, or 30 per cent, as gliomas, and in 2, or 10 per cent, as gliomas; the rest were stated merely to be cystic tumors (pituitary). With the exception of a doubtful gliosarcoma, no primary malignant tumor was found.

Symptoms.—In only twenty-three, or 82 per cent, of the patients were clinical histories given; three of the remaining five had been brought in dead (one of them drowned), presumably having died suddenly. In five of those with histories there was no evidence of intracranial tumor or of any intracranial lesion, each patient having died of some acute condition, such as internal hemorrhage, internal injuries, uremia, acute dysentery (all pituitary) and acute peritonitis (supratentorial), in all of whom the discovery of the presence of tumor was accidental and unexpected. Two of these patients were particularly free from symptoms of tumor, since they died of complications following operations for routine conditions. Three gave only such symptoms as (1) unconsciousness and convulsions (pituitary); (2) headache, lockjaw, inability to sit or walk, vomiting and delirium (frontal and right parietal lobes); (3) anorexia, insanity and unconsciousness (left temporal lobe).

In eighteen, or 82 per cent, of those with histories the onset was gradual, the chief symptoms being headache, loss of appetite, hyperesthesia and rigidity of the neck, heaviness of the head, numbness and pain in the right half of the body, febrile catarrh, difficulty in walking and dimness of vision. The mode of onset was indefinite and unknown in the rest. In four, or 18 per cent, it was sudden, but differed in its nature as a rule. One started as sudden paralysis and fever during the

course of an apparently normal puerperium; two as sudden unconsciousness, one of the patients dying within five days of the onset; while the first symptom of any intracranial disease in the fourth case was a complaint of rigidity and marked tenderness in the neck and stiffness of the jaw early in the fourth week of a lobar pneumonia, the patient dying within twelve hours after the onset of the complaint.

Headache was present in twelve of twenty-two patients, in 75 per cent of whom it was the first or one of the earliest complaints made; it was constant in four. Its location, however, when specified, did not tally with that of the tumors. It was not noted in five patients, in four of them for obvious reasons—insanity, sudden unconsciousness and infancy. The significance of headache, however, is of difficult interpretation in these cases, as with the exception of three there was some other accompanying pathologic change in the brain or meninges or elsewhere—chiefly congestive, inflammatory or hemorrhagic—which of itself may cause headache.

Vomiting occurred in eight cases and is of greater significance, as there was nothing else to explain its occurrence. It was accompanied by nausea in one instance.

Examination of the fundi was unfortunately performed in only five patients, but was positive bilaterally for choked disk in all except one, in whom it could not be determined on account of existing opacities in both lenses. The cases were one each of multiple gliomas with hemorrhage, glioma of the base of the temporal lobe, cyst of the cerebellum, 8 cm. long, and a tumor of the parietal lobe, 2.5 cm. in diameter; the last case also showed a beginning optic atrophy on both sides. In one of two cases the predominance of the choked disk on the left side was to be explained by the location of the tumor in the left temporal lobe. As to other ocular changes, there was pupillary dilatation with sluggishness to light, internal strabismus and loss of vision in four cases; pupillary dilatation with exophthalmos and nystagmus in one case; dilatation with nystagmus in another, and dilatation only in two cases. Sluggishness to light with strabismus and vertical nystagmus and sluggishness only were found in one case each. Double dim vision with lateral nystagmus, and strabismus only, were likewise found once each. On the other hand, the pupils were contracted in one case (temporal lobe) and normal in another (basal ganglia), but vertical and lateral nystagmus was present in a third (pituitary).

Unconsciousness, stupor, dulness, dysmnesia or delirium was found at some time or other in fourteen cases, and it is interesting to learn in this connection that death was more or less sudden in four of seven patients in whom unconsciousness was not noted. One must therefore conceive of tumors causing such a sudden intense pressure as to depress and paralyze vital centers all at once.

The corneal and palpebral reflexes were either sluggish or lost in two cases (multiple gliomas, and base of left temporal lobe). The knee reflexes were exaggerated in six patients (right corpus callosum, temporal lobe, parietal lobe and pituitary twice) in whom there was nothing to explain them except in a case of cerebellar tumor. In addition, there were slight Babinski signs in a tumor of the basal ganglia; Romberg sign and ankle clonus in a cerebellar tumor, 8 cm. long, and a Kernig sign in a pituitary tumor 8.3 by 6.5 cm. The reflexes were apparently normal in a case of pituitary tumor, and sluggish or absent in a case of multiple gliomas and in tumors of the temporal lobe, frontal lobe, basal ganglia and postcentral gyrus; there was no other pathologic process to account for the condition of the reflexes. There were also ankle clonus and a Babinski sign on the left side in a case of multiple gliomas, and Kernig, Chvostek, Oppenheim and Brudzinski signs in a tumor of the basitemporal lobe and Kernig, Babinski, Oppenheim, Brudzinski and Gordon signs in a tumor of the base around the optic chiasm.

Restlessness was noted four times.

There were local or general hyperesthesia and also rigidity of the neck in four patients, while rigidity of the neck alone was recorded three times.

Stiffness of the jaw was noted in five cases, in one of which the tumor was in the pituitary.

Convulsions were not particularly described, but were seen in six cases.

Paralysis, either of the facial muscles or of the extremities, was noted in nine patients, but in all but one (base, left temporal lobe), in which it seemed doubtful, the site of the paralysis tallied with the location of the growths.

Fifteen patients had fever; in all but three it could be explained also by an accompanying pathologic condition, namely, tumor in the corpus callosum and parietal and temporal lobes.

The pulse generally was in accord with the temperature. In three cases, all afebrile, it was characteristically slow (60) and weak toward the later stage; in one of these cases the tumor was in the frontal lobe and in two it was in the pituitary.

Blood pressure was recorded in six patients, but is of interest only in the case of a tumor of the temporal lobe in which the systolic pressure was only 90 in the presence of an accompanying chronic interstitial nephritis and general arteriosclerosis in a woman, aged 60; in a case of tumor of the frontal lobe in which it was only 80 systolic and 45 diastolic, in a woman, aged 45; and, by way of contrast, in a case of tumor of the basal ganglia in which the initial blood pressure was 180 systolic and 90 diastolic, and an average of 125 systolic and 68 diastolic. The patient in this case was a man, aged 36.

Blood counts, more or less complete, were made in eight patients and showed a leukocytosis in five, the highest count being 21,000. It was difficult to explain in all but two cases.

A Wassermann reaction of the blood was performed only twice; the results in both instances were negative.

A lumbar puncture was performed in eight patients; only in five was there any reference to pressure, which was found increased in three, with tumors in the temporal lobe, basal ganglia and base of the brain around the optic chiasm, respectively. Pressure was normal in the case of a tumor of the parietal lobe and in one of the pituitary, in both of which the cerebrospinal fluid was also positive for protein but negative for sugar. Of the six instances in which reference was made to the cerebrospinal fluid at autopsy, four were reported to have an increased amount, only one of which was among those that had showed increased spinal pressure during life, a cerebellar tumor, the others being cases of tumor of the basihemisphere, cerebellum and pituitary, respectively.

A Wassermann reaction of the cerebrospinal fluid was taken twice; it was negative both times.

None of the three cases with reported changes in the sella turcica had tumors in the pituitary; one was a tumor at the base, one in the left temporal lobe, which was reported to be enlarged, and one in the cerebellum and left parietal lobe, with destructive changes, according to the x-rays, none of which were mentioned in the autopsy protocols.

Loss of weight was noted in seven patients.

Clinical Diagnosis.—The presence of tumor was diagnosed or suspected premortem in four, or 18 per cent, of the patients who had clinical histories. The diagnosis, however, or the symptoms, pointed to some cerebral or meningeal involvement in eighteen, or 64 per cent, to wit: tuberculous meningitis, cerebral hemorrhage, cerebral embolism, meningism, and meningo-encephalitis and even uremia in two cases. The condition in two of the unrecognized cases was thought to be malaria, presumably of the cerebral type, a fact which is not to be wondered at in view of the prevalence of malaria in the country.

No modern exploratory methods of diagnosis have been used in any of these cases, such as ventriculopneumography or other forms of cerebral visualization or even the systematic taking of x-ray pictures of the skull. This was an error of omission which, while it may account for the small proportion of cases in which the condition was accurately diagnosed, serves to illustrate what I have previously mentioned, namely, absence of mindedness toward a particular thing, in this case, intracranial tumors.

From an analysis of the symptoms, the presence of congestive, inflammatory or hemorrhagic intracranial conditions and, in the majority

of cases, the length of the patients' stay at the hospital, together with the spectacular and dramatic atmosphere which usually surrounds these cases, the failure to recognize or single out the presence of tumors was fairly excusable.

Duration of Illness.—The average duration of illness was short, being less than two months, while the extremes were one-half day (pituitary) and three years (basal ganglia), respectively.

The onset of compression, judged from the sudden unconsciousness (coma), was sudden as a rule.

Manner of Death or Onset of Agonal Period.—Death was sudden in five, or 38 per cent, of the cases of supratentorial tumors, and in two, or 30 per cent, of pituitary tumors. It should be remembered in this connection that in four of five patients who died without any symptoms referable to intracranial tumor the growths were in the pituitary. The only patient with a cerebellar tumor with history died suddenly within twenty-four hours of the patient's complaining of pain, rigidity and tenderness at the nape, the only symptoms that ever suggested intracranial trouble. Three other patients may be presumed to have died a sudden death.

Comparison Between Tumor Groups.—As there was only one case of cerebellar (infratentorial) tumor, no attempt was made to compare its clinical picture with that of any group; but an attempt was made to compare the picture of supratentorial and pituitary growths.

In cases of supratentorial tumor, headache and vomiting were more frequent in basal and frontal tumors; pupillary dilatation and other ocular changes in parietal tumors; restlessness, hyperesthesia and rigidity of the neck in temporal tumors; convulsions in basal tumors and paralysis in temporal and basal tumors. Not much can be said of the psychic features in the two cases of frontal tumors in which the histories were known.

As between supratentorial and pituitary tumors, among cases of the former the general age incidence was slightly lower (32 against 35); the incidence among men was twice as great as among women; headache was twice as frequent; vomiting was somewhat less common; there were more eye changes, restlessness, hyperesthesia, convulsions and paralysis; the proportion of patients with sluggish or abolished knee reflexes was greater; among them there was apparently a greater number of sudden deaths in those who showed indications of intracranial lesions. It must be emphasized, however, that in four of five patients who died of some acute condition without any evidence of intracranial tumor or intracranial lesion, the tumors involved the pituitary, and their discovery was accidental in the course of a routine postmortem examination. It is an

open question whether the more or less sudden demise was due to the acute condition or to the presence of the unsuspected growth, or whether the tumor was only contributory.

Unusual Features.—It is further noteworthy in connection with pituitary tumors in this survey that their behavior was a departure from the usual behavior in this type of tumor. There was no specification as to the location of the growth, whether it was anterior or posterior or both; but, with the exception of two, none of the seven pituitary cases with histories, or of all nine of them, suggested any functional disturbance of the organ (insufficiency) of either the anterior or the posterior lobe causing hyperfunction or hyperpituitarism (acromegaly) or hypofunction or hypopituitarism (adiposity, genital infantilism, amenorrhea), unless the explanation lies in the presence of some undiscovered accessory pituitary gland. The two exceptions were a tumor, measuring 3.5 by 2.5 by 2 cm., in a married woman, aged 25, who showed an infantile uterus even though she had been pregnant once, while the other was a large cystic tumor, measuring 11 by 9 by 8 cm., in a man, aged 35, who showed a rather puzzling composite picture of hyperplasia of the penis and testis on the one hand and deficiency of beard and axillary and pubic hair on the other.

Another interesting case in which the pituitary may have been indirectly at fault is that of a patient in whom the symptoms of tumor, as indicated by headache and aphasia, came on during the course of a low grade fever and cough of about six months' duration, preceded by amenorrhea of three months' duration, and in whom a tumor was found at the base of the brain around the optic chiasm, which caused pressure atrophy of the pituitary. Was the amenorrhea due to, or independent of, the pressure on the pituitary? If it was secondary to pituitary atrophy, it is hard to understand why the patient should show hyperplasia of the uterus (and of the heart) at the same time.

Other Unusual Features.—A woman with an endothelioma of the left temporal lobe had a coarse manlike voice.

Sialorrhea, impaired hearing and, in the earlier stages of the condition, a voracious appetite and later constipation were conspicuous features in a case of glioma of the base of the left temporal lobe.

A cystic tumor of the left basal ganglia was accompanied by fits of laughter during the last month of an illness of three years' duration.

One case of polycystic tumor of the pituitary, 6 by 4.5 cm., in the only infant in the series was also interesting in that the child had what to all intents and purposes appeared to be an acute pulmonary infection which had been dragging on for two months. Yet at autopsy nothing was found but a follicular pharyngitis, to which the symptoms were completely out of proportion.

COMMENT

In the majority of instances the symptoms were noted well enough, but their meaning was not correctly interpreted. This was an error of commission. The fact, however, that the examinations have been incomplete in some cases or that in four of them no description of anything to indicate intracranial lesions was given did not necessarily mean lack of diagnostic care and ability, but rather that the tumors were not voluminous enough to produce symptoms, that there was sufficient compensatory adjustment of circulation, or that the tumors were so situated as not to produce any symptoms.

It is of obvious interest to know that a cystic tumor in the pituitary can grow to be as large as 8 by 5 by 4 cm. and yet produce no apparent symptoms. Some support for this statement may be found in the fact that in four of the five patients who died without symptoms of an intracranial lesion the tumors were in the pituitary.

There is the possibility that pressure symptoms are not directly due to pressure from the tumor but to hydrocephalus resulting from blocking of the cerebrospinal passageways. There were three such cases (glioma of the base of the left temporal lobe, cyst of the cerebellum, 8 cm. long, and tumor of the base around the optic chiasm). There does not seem to be any relation between the intensity of the symptoms as judged from the duration of the illness and the size of the tumors, although such a relation is suggested between the occurrence of sudden death and size of the tumor. On the other hand, the location as a whole does seem to affect the presence of symptoms, as with the exception of one (endothelioma of the parietal dura 2.5 cm. in diameter), all fourteen supratentorial tumors gave symptoms pointing at least to some intracranial trouble, which is in contrast with the frequently silent behavior of pituitary tumors.

Attention must be called to the fact that, with the exception of a doubtful gliosarcoma, not a single malignant tumor was encountered in this series. In intracranial tumors, however, mere benignancy is of less prognostic significance than in tumors elsewhere on account of pressure being the prevailing element in their pathogenesis.

SUMMARY

1. The incidence of primary intracranial tumors among Filipinos is not less than 0.61 per cent. A systematic examination of the brain in all cases coming to autopsy will no doubt raise this proportion.

2. Primary intracranial tumors are more frequent during the second and third decades among females, and during the fourth decade among males.

3. Tumors are more frequent among females (0.79 per cent) than among males (0.52 per cent), for which no explanation is offered.

4. Sixty-one per cent of the tumors found were supratentorial; 32 per cent pituitary, and 7 per cent infratentorial (cerebellar).

5. Forty-seven per cent of the tumors were gliomas and 33 per cent endotheliomas. With the exception of a doubtful gliosarcoma, no primary malignant tumor was found.

6. Twenty-three per cent of patients for whom histories were available showed no evidence of intracranial tumor and died of acute conditions, the discovery of the tumors being accidental and unexpected.

HERPES OPHTHALMICUS FEBRILIS WITH DENDRITIC KERATITIS COMPLICATING THERAPEUTIC MALARIA *

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AND
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Horner,[1] in 1871, described a herpetic corneal disease occurring simultaneously with herpes nasalis in persons suffering from catarrhal disease of the respiratory organs. He called it herpes corneae febrilis. The case we are reporting is of a similar nature except that it occurred during the course of therapeutic malaria in a person suffering from cerebral syphilis. The cornea of the left eye was involved in a herpetic eruption which soon progressed to a definite dendritic type of keratitis. Simultaneously, there was a herpes outlining the distribution of the infratrochlear nerve on the left side of the nose. Thus, the nerve involvement was confined to the long ciliary and infratrochlear branches of the nasociliary nerve. Consequently, for the purpose of correlating the complete clinical picture, the febrile origin and the known pathology of trigeminal herpes, we used the term herpes ophthalmicus febrilis in describing our case.

The case is of interest (1) because it represents an unusual complication of therapeutic malaria; (2) because of the morbidity of the complicating process, and (3) because of the problems arising in continued treatment in such a case.

REPORT OF CASE

E. P., a man, aged 37, was transferred from the Colorado General Hospital, service of Dr. W. C. Finnoff, for malaria therapy. He entered the Colorado Psychopathic Hospital on July 7, 1928, presenting the typical physical and serologic evidences of cerebral syphilis. There was complaint of visual defect of several years' standing, and perimetric examination revealed a left superior quadrantic homonomous anopia (fig. 1). He was inoculated with tertian malaria and developed the typical tertian pattern, having six paroxysms during which a maximum temperature of 105.8 F. was reached. After the fourth paroxysm, he complained of a foreign body in the left eye. Examination revealed none, but there was circumcorneal injection with lacrimation and photophobia. Definite pain of a

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* From the University of Colorado Psychopathic Hospital.

* Read by title at the Fifty-Fifth Annual Meeting of the American Neurological Association, Atlantic City, N. J., May 29, 1929.

1. Horner: Klin. Monatsbl. f. Augenh., 1871, p. 325.

sharp lancinating character followed, and the symptoms became markedly worse with each subsequent chill. On the third day, several small vesicles appeared over the temporal margin of the cornea, and the pain was severe. A pressure bandage was applied, and the malaria was terminated. On the sixth day, herpetic vesicles appeared following the distribution of the infratrochlear nerve (fig. 2). Other vesicles appeared on the cornea to coalesce with those present, and within a short time there was evident ulceration of the branching or dendritic type (fig. 3). After the administration of quinine, the nasal herpes dried up rapidly, but the ulceration of the cornea persisted. Instillations of atropine sulphate solution were exhibited and within two weeks the process seemed in abeyance, but there was still considerable clouding of the cornea. Following discharge from the hospital, the patient was given quartz light therapy to the eye and was placed on weekly injections of neoarsphenamine. It was found, however, that there were frequent recrudescences of the keratitis. These persisted after the quartz light

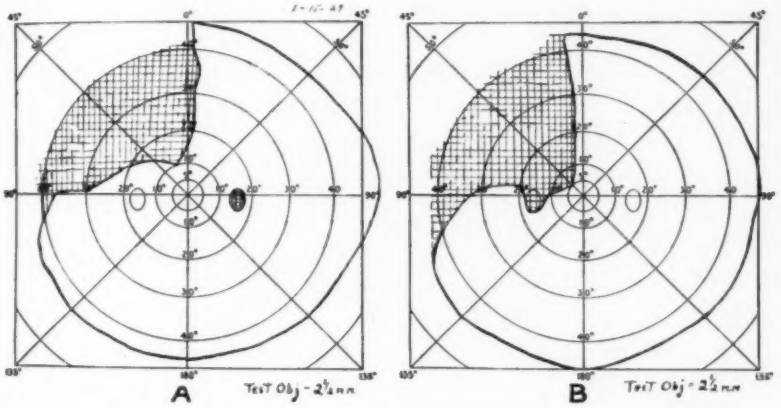

Fig. 1.—Left superior quadrantic homonomous anopia. *A*, right eye (vision = 6/5); *B*, left eye (vision = 6/20).

therapy had been discontinued. The patient associated them, first, with the intravenous medication and, second, with periods of constipation. The latter were effectually treated but with no improvement, and it was finally determined to our satisfaction that the neoarsphenamine was definitely associated with these recrudescences. Its administration was stopped, and though too short a time has elapsed to make definite assertions, there have been no other flare-ups of the keratitis and the corneal clouding has markedly diminished. At present there is still sufficient opacity of the temporal portion of the cornea to obstruct vision partially (fig. 4). There are no changes in the visual fields, and the quadrantic defect first noted continues.

COMMENTS

When Horner,[1] in 1871, described the corneal disease which he named herpes corneae febrilis, he made a theoretical correlation between this disease and herpes febrilis labialis. Since then, his ideas have been

largely substantiated by the work of Gruter,[2] who proved experimentally the transmissibility of herpes febrilis and the relationship of various forms of simple herpes to one another. Luger and Lauda [3] demonstrated the transmissibility of herpetic keratitis from man to rabbit, and Fuchs and Lauda [4] were able to produce an artificial dendritic keratitis in the human being with material from herpes simplex of the skin. Levaditi,[5] Doerr,[6] Doerr and Vochting [7] and Loewenstein [8] have associated corneal herpes with encephalitis in rabbits and have been able to start with material from herpes simplex of the skin, inoculate the corneas of rabbits and produce not only herpes corneae but a form of encephalitis as well. The Levaditi school, from their researches, have formulated a doctrine of tropism of the herpetic virus, recognizing dermatropic and neurotropic strains thereof; and this conception has been extended to explain the vagaries of *Spirochaeta pallida* also.

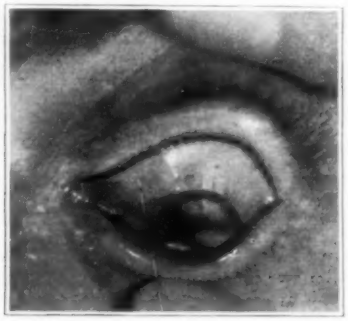

Fig. 2.—Distribution of herpes on the cornea and following the infratrochlear nerve.

2. Gruter: Experimentelle und klinische Untersuchungen über den sogenannten Herpes Corneae, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **42**:1662, 1920; Neue Untersuchungen über den Herpes Corneae, ibid. **43**:227, 1922; Zur Aetiologie der Impetigo contagiosa und anderer herpetischer Erkrankungen, ibid. **44**:220, 1924; Das Rezidiv bei den herpetischen Erkrankungen der Hornhaut, ibid. **45**:104, 1925.

3. Luger and Lauda: Zur Kenntnis der Uebertragbarkeit der Keratitis herpetica des Menschen auf die Kaninchenkornea, Wien. klin. Wchnschr. **34**:132, 1921.

4. Fuchs and Lauda: Zur Aetiologie der Keratitis dendritica, Ztschr. f. Augenh. **49**:9, 1922-1923.

5. Levaditi: Ectodermoses neurotropes, Paris, Masson et Cie, 1922, p. 269.

6. Doerr: Ergebnisse der neueren experimentellen Forschungen über die Aetiologie des Herpes simplex und des Zoster, Zentralbl. f. d. ges. Ophthal. **14**: 705, 1925; **15**:313, 1925.

7. Doerr and Vochting: Études sur le virus de l'herpes fébrile, Rev. gén. d'ophth. **34**:281, 409, 1915.

8. Loewenstein: Neuere Ergebnisse der Herpesforschung, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **42**:167, 1920.

In 1880, Charles J. Kipp,[9] of Newark, N. J., first described a keratitis occurring in malarial fever and noted the peculiar branching nature of the ulceration and the tendency to recurrence. Hotz [10] of Chicago, in 1881, Van Millingen [11] of Constantinople, in 1888 and later Ellett,[12] McConnel and McGill [13] and others have described similar conditions occurring in malaria. Kipp originally considered this type of keratitis as being due solely to malaria, and Hotz [10] thought it pathognomonic for malaria. Somewhat later, in 1889, Kipp [14] changed his views on observing the dendritic keratitis in otherwise normal persons. In 1884, Hansen-Grut [15] described a branching keratitis complicating pneumonia, and in 1885, Emmert [16] found a similar condition occurring in the tuberculous. He called it keratitis dendritica, a name by which it is known at present. In later years, it has been found in influenza by Von Reuss,[17] Denti [18] and Pretori;[19] in sinusitis by Birkhouser;[20] in pneumonia by

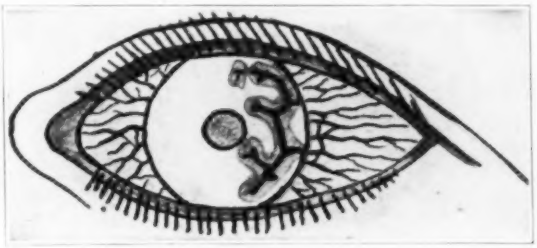

Fig. 3.—Schematic representation of the dendritic ulcer.

9. Kipp, C. J.: On Keratitis from Malaria Fever, Tr. Am. Ophth. Soc. **3**:91, 1880-1884.

10. Hotz: Malarial Keratitis, Chicago M. J. & Examiner, December, 1881.

11. Van Millingen: Hirschberg's Centralbl. f. Augenh., January, 1888; Rapport sur certaines affections oculaires consécutives à la fièvre dengue, Gaz. med. d'Orient, Constant. **32**:131, 1889-1890.

12. Ellett, E. C.: Dendritic Keratitis of Malarial Origin, J. A. M. A. **46**: 1984 (June 30) 1906.

13. McConnel and McGill: A Note on Corneal Ulcer in Malaria, J. Trop. Med. **29**:3, 1926.

14. Kipp: Further Observations on Malarial Keratitis, Tr. Am. Ophth. Soc. **5**:331, 1888-1890.

15. Hansen-Grut: Tr. International Med. Congress, Copenhagen, 1884.

16. Emmert: Hirschberg's Centralbl. f. Augenh., October, 1885.

17. Von Reuss: Zur Kenntniss der Keratitis dendritica, Arch. f. Ophth. **78**: 300, 1911.

18. Denti: Postinfluenzal Dendritic Keratitis, Ospedale maggiore **10**:183, 1922.

19. Pretori: Zur Keratitis dendritica superficialis, Arch. f. Augenh. **57**:197, 1907.

20. Birkhouser: Keratitis dendritica nach Trauma bei Kieferhöhlenempyem, Klin. Monatsbl. f. Augenh. **50**:92, 1912.

Von Reuss;[17] in dengue fever by Van Millingen;[11] in gonorrheal ophthalmia by Allport;[21] and in otherwise normal persons by Kipp,[14] Allport,[21] Theobald[22] and others.

Calhoun,[23] de Lapersonne[24] and Luna[25] have described dendritic keratitis following injections of T. A. B. vaccine into syphilitic patients. Luna's case occurred simultaneously with a nasolabial herpes in a person with an induced fever of 39.6 C. (103.2 F.). He thought that the condition was caused by a toxic neuritis of the trigeminal nerve, the keratitis being due, specifically, to a neuritis of the terminations of the ciliary nerves. This case is similar to ours, and Luna's conception of the etiology seems adequate. Theobald[22] would localize the pathologic changes in the ciliary ganglion, but obviously this conception is too limited for cases in which nasal herpes accompanies the keratitis.

Von Barensprung,[26] in 1861, definitely localized the pathologic change of herpes zoster ophthalmicus in the posterior root of the gasserian

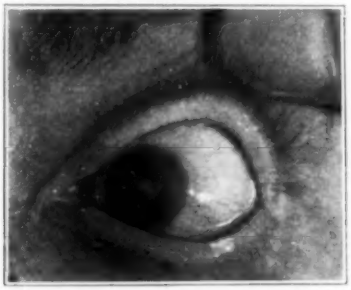

Fig. 4.—Corneal opacity at the present time.

ganglion. Head and Campbell,[27] in 1900, established the correctness of this conception beyond all doubt. Peripheral neuritis of various branches of the ophthalmic nerve has also been observed by Pitres and Vaillard[28] in 1883, and by Curschman and Eisenholer[29] in 1884.

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21. Allport: Keratitis Dendritica, J. Ophth. **13**:209, 1896.

22. Theobald: Herpes Corneae Febrilis, New York M. J. **104**:247, 1916.

23. Calhoun: Antityphoid Inoculations and Ocular Lesions, Ophth. Rec. **26**: 553, 1917.

24. De Lapersonne: Vaccinations anti-typhoidiques et lésions oculaires entrainant la cécité, Arch. de méd. et pharm. mil. **67**:236, 1917.

25. Luna: Ocular Lesions Consecutive to Injections of Anti-Typhoid Vaccine, Keratitis Herpetica Febrilis, Am. J. Ophth. **2**:488, 1919.

26. Von Barensprung: Charité-Ann., 1861, vol. 9, part 2, vol. 11.

27. Head and Campbell: The Pathology of Herpes Zoster, Brain **23**:353, 1900.

28. Pitres and Vaillard: Contributions à l'étude des névrites périphériques non traumatiques, Arch. de neurol. **5**:191, 1883.

29. Curschman and Eisenholer: Zur Pathologie und pathologischen Anatomie der Neuritis und des Herpes Zoster, Deutsches Arch. f. klin. Med. **34**:409, 1884.

Dubler,[30] also in 1884, concluded that the condition was primarily a perineuritis and that the changes in the ganglion were secondary. Marinesco and Dragonesco [31] and Goodpasture [32] have shown that the herpetic virus passes from the periphery to the central nervous system by the nerves and that any nerve, motor, sensory or sympathetic, may transmit it. When the cornea is selected for primary infection the path is by way of the first or ophthalmic division of the fifth nerve, and the primary focus is in the bulbospinal root.

That there are differences between herpes zoster and herpes simplex is not to be doubted; but that they are definitely related clinically and pathologically seems equally obvious. Gruter [2] and Goodpasture and Teague [33] have demonstrated experimentally that the chief difference is one of virulence rather than of kind. On the other hand, Paton [34] and others have failed in attempts to produce corneal infection with material from zoster vesicles, and it is well known that whereas zoster confers immunity simplex fails to do so. From a clinical standpoint, the classic description of herpes zoster ophthalmicus as given by Jonathan Hutchinson [35] in 1866 fits the essential features of our case well. In fact it would seem impossible to differentiate clinically our case from the so-called symptomatic zoster.

It is of interest also that the keratitis in our case has persisted with frequent recrudescences characterized by sharp pain, photophobia, lacrimation and circumcorneal injection. Quartz light therapy had no effect on the condition, and attention was directed to a chronic constipation. It was also suspected that the weekly injection of neoarsphenamine had something to do with the situation, since the recrudescences seemed to occur most frequently within from eight to twenty-four hours following

30. Dubler: Ueber Neuritis bei Herpes Zoster, Virchows Arch. f. path. Anat. **96**:195, 1884.

31. Marinesco and Dragonesco: Pathogenesis and Pathologic Physiology of Zona, Rev. neurol. **30**:30, 1923; Experimental Research on Neurotropism of Herpes Virus, Ann. de l'inst. Pasteur **37**:753, 1923.

32. Goodpasture: Intranuclear Inclusions in Experimental Herpetic Lesions of Rabbits, Am. J. Path. **1**:1, 1925; Axis Cylinders of Peripheral Nerves as Portals of Entry to Central Nervous System for Virus of Herpes Simplex in Experimentally Infected Rabbits, ibid. **1**:11, 1925; Pathways of Infection of Central Nervous System in Herpetic Encephalitis of Rabbits Contracted by Contact, ibid. **1**:29, 1925.

33. Goodpasture, E. W., and Teague, O.: Experimental Herpes Zoster, J. A. M. A. **81**:377 (Aug. 4) 1923; Experimental Production of Herpetic Lesions in Organs and Tissues of the Rabbit, J. M. Research **44**:121, 1923; Transmission of the Virus of Herpes Febrilis Along Nerves in Experimentally Infected Rabbits, ibid. **44**:139, 1925; Experimental Herpes Zoster, ibid. **44**:185, 1925.

34. Paton: The Trigeminal and Its Ocular Lesions, Brit. J. Ophth. **10**:305, 1926.

35. Hutchinson: Roy. London Ophth. Hosp. Rep. **5**:191, 1866; **6**:48, 1866.

injection, although this was not constant. The constipation was treated successfully, but the keratitic flare-ups continued. Then neoarsphenamine was withdrawn, with immediate cessation of the flare-ups. Clinically, it would seem that there was a definite relationship between the administration of arsenic and the recrudescences of the keratitis. Since these have disappeared the corneal clouding has also diminished and the function of the eye improved. The fact, however, that there were no other signs of arsenical intoxication during the period of its administration throws some doubt on our deductions. On the other hand, we find that herpes simplex has been known to occur in arsenical poisoning (Paton [34]), and it is of rather common occurrence following alcoholic debauches. Our patient, of his own accord, purposefully refrained from liquor of all kinds because he found that indulgence caused painful flare-ups in the affected eye.

Dendritic keratitis is known to be very resistant to treatment, with a tendency toward recurrence. It would seem that in our case these recurrences were associated with toxic factors, the removal of which served materially to aid in ultimate recovery.

SUMMARY AND CONCLUSIONS

A case of herpes ophthalmicus febrilis with dendritic keratitis complicating malarial therapy has been presented. That this complication is unusual is disclosed by the fact that there are no other reports in the literature of its occurrence during therapeutic malaria. The condition assumes importance in that (1) it is an indication for immediate termination of the malaria; (2) it may result in permanent visual defect, and (3) relapses may occur following postmalarial arsenical therapy.

Clinical Notes

DELAYED TRAUMATIC SERRATUS PARALYSIS*

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The deformity of scapula alata or winged scapula caused by paralysis of the posterior thoracic nerve, resulting from trauma, presents a problem of particular interest to men engaged in practice that necessitates the assignment of the rôle of trauma in the production of paralyses alleged to be produced by mechanical injury. The treatment of this condition also deserves particular attention.

Isolated traumatic paralysis of the serratus is generally stated to be much more frequent in men than in women; according to Berger, it is eight times as common in the former, and according to Gowers, it is six times as frequent. These investigators tabulated the reports of about thirty cases from the literature. It occurs most frequently in laborers, in muscular persons during the active period of life, between 25 and 45 years of age. The reason that it is most frequent during this period, and that a predilection is shown for the male sex, is probably that exposure and injury are more frequent during the active period of life in persons doing heavy work. For the same reason, the right side is much more frequently affected than the left, and in a great majority of cases the condition is unilateral. In bilateral cases, the nerves of the two sides are not ordinarily injured simultaneously but at successive times. Gowers mentioned the case of a man who became affected with serratus paralysis on the right side from carrying heavy beams on one shoulder, after which he carried them on the opposite shoulder and the left serratus also became paralyzed.

Injury to the posterior thoracic nerve in the part which traverses the scalenus medius muscle, seems to be the commonest cause of traumatic paralysis, whether this is due to direct trauma of the nerve in this region or to tension put on the nerve by powerful action of the scalene muscle.

Among such traumatic causes are strain from prolonged and excessive use of this muscle in such occupations as those of carrying of heavy weights or constant carrying of children in arms, prolonged use of heavy hammers, continued elevation of the arms in a vertical position as in painting ceilings or hanging on a cross bar, as also in so-called "strain" from overlifting, blows and falls on the shoulder and back (Keene), hyperextension of the arms, and puncture and gunshot wounds injuring the posterior thoracic nerve.

The signs and symptoms of serratus paralysis are characteristic. One of the symptoms described by the older writers as characteristic is the inability to raise the arm through the upper quadrant of motion. The classic explanation was that the elevation of the arm from the horizontal to the perpendicular is accomplished by the action of the serratus magnus alone in rotating the scapula. That the function of elevating the arm past the right angle is aided by the deltoid and the trapezius, especially the middle portion of the latter, the supraspinatus and infra-

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* Read at a meeting of the Chicago Neurological Society, Nov. 15, 1928.

spinatus and the levator anguli scapulae is now, however, generally admitted. It has also been demonstrated that the middle portion of the trapezius and also to a lesser degree the upper portion, develop in strength and compensatory action in case of paralysis of the serratus.

Cases of this compensatory action have been described by Baumler, Eulenberg, Bruns, Bernhardt, Oppenheim, Owen and others. Steinhauser (1900) in a collection of fifty-seven cases of serratus paralysis from all causes in the German Army records, found elevation of the arm beyond a right angle present in 95 per cent, but gave as his opinion that complete elevation of 180 degrees is probably not present unless the upper part of the serratus is still functioning. The difficulty in testing this part of the muscle electrically for paralysis makes his contention hard to disprove.

It may be difficult to determine the cause of the serratus paralysis. Before arriving at a diagnosis of traumatic paralysis, the possibility of affection of the nerve by a distant pyogenic infection, syphilis, compression of the nerve roots by osteo-arthritic deposits in the intervertebral foramens and acute anterior poliomyelitis must be excluded. The presence of progressive muscular atrophy can sometimes be ascertained only by several examinations over a considerable period of time.

DELAYED TRAUMATIC PARALYSIS

Bunts and several others among older writers have described delayed traumatic serratus paralysis. In these reports the paralysis became evident so long after the alleged trauma, and the description of the trauma was so vague that, in the light of present knowledge, it seems to me likely to have been of toxic or osteo-arthritic origin. The allegation of a traumatic etiologic factor in almost every case of paralysis and deformity is a common experience in the practice of all physicians engaged in the surgical treatment of patients with conditions due to trauma. The hope of obtaining compensation for an industrial accident may have become an important factor in the traumatic history given by the patient since the adoption of legislation providing for compensation for industrial accidents.

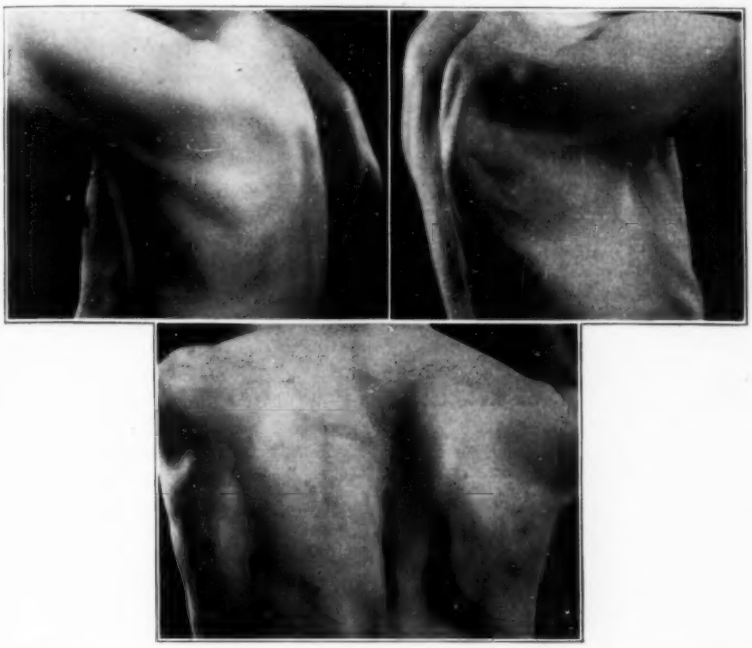
My attention was particularly attracted to the possibility of delayed traumatic paralysis of the serratus as a result of seeing the patients in two cases. I see no more difficulty in accepting the occurrence of a paralysis of the serratus which becomes manifest several days after a mechanical trauma over the scalene muscles as being due to trauma than in accepting as such the classic, slowly appearing paralysis of the musculospiral nerve following the extravasation of blood and the tissue reaction in fracture of the shaft of the humerus.

REPORT OF CASES

Case 1.—A. W., a man, aged 43, who said that he had not had any previous accident or serious illness, on Sept. 8, 1927, was driving an automobile which rolled into a ditch. He sustained a slight cerebral concussion and multiple trivial lacerations and contusions about the extremities. His right shoulder struck against a fence post. On examination the next day, with the patient stripped, an area of discoloration, about 4 inches in diameter, accompanied by apparent increase in consistency of the muscles beneath, was found over the middle of the upper border of the right trapezius. On careful examination of the back while the patient was resting and while the arms were in motion nothing of note was observed.

The wounds were dressed in the office daily. Four days after the first examination, the patient complained of inability to use the right arm fully, and on careful examination of the back, the lower angle of the right scapula was noted to be slightly more prominent than the left when the man was standing with the

arms hanging at the sides. The vertebral border of the right scapula was inclined from above downward and slightly outward. The inferior angle was a little farther from the midline than the one on the opposite side. The lower border of the greater rhomboid muscle could be distinctly seen running from the inferior angle of the scapula upward and inward to its vertebral attachment. When the arms were extended and held out in front, in a horizontal position, the scapula projected from the chest in the manner which is so characteristic of serratus paralysis—there had been no such projection of the scapula at the first examination. The backward displacement of the scapula became much more prominent when the patient attempted to push forward against resistance. The vertebral border remained close to and parallel to the spinal column during this movement,



Views taken several weeks after treatment was begun, showing compensatory muscular development.

and the absence of the serratus digitations in the right as compared with the left axilla was striking. When the arms were held laterally at right angles to the trunk, the scapula, especially the superior angle, approached nearly to the midline. The patient was able to abduct the arm only at right angles with the body.

After three months of massage of the muscles of the shoulder girdle and treatment by resisted motion, he was able to elevate the arm to a position about 45 degrees above the horizontal, although at this time the serratus muscle still failed to react to faradism, and on forward motion of the arms the same amount of winging of the scapula occurred as when the deformity first appeared. This seems to me to prove that a compensatory development in the strength of the uninjured muscles of the shoulder girdle is responsible for the increase in the power of abduction after three months of treatment, and I suspect that the argument regard-

ing whether any part of the serratus must be intact to permit complete abduction of the arm arose because early observers saw the patient at different periods in the development of compensatory strength of muscles other than the serratus.

CASE 2.—A well muscled man, aged 30, while doing heavy labor on a construction job, fell on March 9, 1922, so that the region of the middle of the upper border of the trapezius struck against a 2 by 4 timber in a building under construction; the whole weight of the man's body was exerted against this timber. When examined in the office immediately thereafter, a laceration of the skin of the shoulder was dressed. There was considerable suggillation of blood under the skin of the shoulder, and increased resistance on palpation could be felt in the muscles at the base of the right side of the neck. No signs of serratus paralysis were found at the first examination.

About two weeks later, the typical deformity began to appear. At first there was some deformity, consisting of slight elevation of the scapula and slight winging, even when the man stood in the resting position. Within two weeks, this became more marked, as in the other case, but within six weeks no deformity was seen in the resting position.

COMMENT

In regard to the treatment for these conditions, the second patient was treated only with massage; complete function of the serratus returned in about twelve months. The patient in case 1 was treated by immobilization in a position of rest for the serratus, which is, according to the work of W. C. Mackenzie, in a monograph on "The Action of Muscles," support of the arm in a sling, the elbow being brought sharply across the chest with the forearm flexed and supinated. Slow, surging sinusoidal current was applied so that only three complete phases of current stimulated the muscle daily. The photographs taken two months after the injury, show plainly the hypertrophy of the trapezius and rhomboid. Complete function returned in ten months.

It is my opinion that with the advent of present elaborate physical therapy equipment a good many patients with various isolated muscle paralysis are overtreated, and it has been my clinical experience that when prolonged electrical stimulation and massage are applied to a paralyzed muscle there is great danger of fatiguing the muscle, which is deprived of its nerve supply. The compensatory action of other muscles of the shoulder girdle is aided considerably by massage and resisted exercises.

SPECIAL ARTICLE

FRIGIDITY IN WOMAN

A REVIEW OF THE BOOK BY WILHELM STEKEL *

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WASHINGTON, D. C.

It is characteristic of psychic disorders that their onset is often so insidious and symptom manifestations so subtle as to escape completely the attention of both the patient and the physician. It is not surprising, therefore, that the subject of female frigidity, so universally common, should have so far escaped the better insight of the medical profession, and that it should have fallen to the lot of the psychotherapist to illumine this dark corner of human behavior. Toward the solution of this and related problems, the pioneer psychoanalyst, Dr. Wilhelm Stekel, devotes two large volumes which contain material and discussion of about ninety cases, some analyzed with great clarity and detail.

The work treats of the psychic disorders of woman's love life which manifest themselves as sexual frigidity (dyspareunia, anesthesia).[1] The account, like that of impotence in man, is difficult for the reason that psychic and physical disorders influence one another and may be found either isolated or combined, so that the clinical picture of the resulting morbid condition becomes obscured, its psychogenesis is masked and the disorder is mistaken for something organic.

Barring those occasional instances of some actual pathologic process present (inflammations of the vagina or vulva, pelvic and perineal conditions, fistulas, etc.), which cause local pain and thus make the patient avoid coitus, in the majority of cases painful coitus is observed in the absence of any physical condition that could explain it. As

* Submitted for publication, July 15, 1929.

* Stekel, Wilhelm: Frigidity in Woman in Relation to Her Love Life. Authorized English version by James T. Van Tesslaar, New York, Boni & Liveright, 1926, vol. 1, pp. 296; vol. 2, pp. 307.

1. Some, as Rohleder, draw a sharp distinction between the terms defining anesthesia as the absence of sexual desire, frigidity as weakened or diminished sexual desire and dyspareunia as the absence of pleasurable sensations while the sexual desire proper is still preserved. Hühner defines frigidity as lack of sexual desire in the female and makes it synonymous with sexual anesthesia, while he defines dyspareunia as that condition in women in which coitus is accompanied by more or less intense pain. These definitions are arbitrary and the conditions merge into one another, being merely different aspects of the same situation. For this reason one may regard all these terms as synonymous.

further illustrating the functional psychic nature of the reaction, a large number of cases are encountered which, in the absence of pain or physical basis, show either diminution or lack of desire (libido), or with the desire present, absence of pleasurable sensation during the act (forepleasure or voluptas) or gratification at the end of the act (orgasm).

It has been assumed since the time of Kisch that a disturbance anywhere along the reflex sexual arc may cause frigidity which may therefore be due to failure of peripheral stimulation, inhibitions from the cerebral cortex or failure of excitability of the genitospinal center. It has further been submitted that the failure of peripheral stimulation may be due to congenital sensory deficiency of the peripheral nerves, just as there may be an inherent deficiency of the excitability of the reflex spinal center. The explanation is vague, is not supported by actual demonstrations that the sensory deficiency is congenital or even can be congenital and thus begs the question by assuming that which it is supposed to prove; furthermore, many women of this type do not show local or general defects of development. It is also a frequent observation that the very woman who for a period showed "congenital sensory deficiency" of the peripheral nerves has spontaneously recovered from or been cured of it. At the same time, it is becoming increasingly clear that inhibitions from the cerebral cortex can produce peripheral anesthesia as well as lower the excitability of the reflex spinal center, through influences that are purely psychic in nature.

Although the condition of frigidity is widespread, it is seldom complained of on its own account but comes occasionally to the attention of the gynecologist when the patient seeks relief from sterility. It may be noted, however, that although a certain number of sterile women are frigid, there does not appear to be any direct relation between the two phenomena.

FRIGIDITY VERSUS IMPOTENCE [2]

A superficial survey is enough to prove that these disorders have something to do with human evolution and growth, and that they are traceable to the influence brought about by the refinement in culture. Every age has its love problems. And modern love relations are much more complicated than they were ever before. It is a sad but true reflection that a great number of men belonging to the higher cultural levels are relatively impotent, and a large number of women belonging to the same class are sexually frigid. Who can describe the vast number of tragedies behind it?

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2. For a critical discussion of the problem of male impotence see Karpman, B.: Impotence in the Male: A Review of the Book by Wilhelm Stekel, Arch. Neurol. & Psychiat. **21**:924 (April) 1929.

The psychosexual problems that confront the modern woman are in a large sense different from those of man and these differences are based on both biologic and psychologic grounds. The erotic tragedies are more frequent and express themselves differently in men than in women because of much stronger development in them of impulses and a greater exposure to temptations. To man, love is an episode, existing as it were side by side with other interests, and his erotic life shows a distinct rhythm, which does not appear so obviously in the case of women.

Man's reaction to his love inadequacy is usually that of humiliation. The frigid woman, on the other hand, may even be proud of her coldness and the reaction be elevated to the level of virtue. Suicide in men often has for its background the problem of impotence, whereas in women, so far as one knows, suicide is never caused by frigidity.

Furthermore, in any sort of sex relations, a woman, if she wishes, can assert her physical superiority and express her inner "no" by hindering coitus through vaginism. Nor is frigidity as a weakness humiliating to a woman. A frigid woman can hide or feign orgasm; she can gratify man in spite of her frigidity, at least partly so, and can make detumescence possible for him. In man, however, although his impotence is also expressive of an inner "no," this inner negation brings on a feeling of depreciation, because sexual potency presupposes a strong erection and the failure of erection implies weakness which cannot be hidden.

PSYCHIC TRAUMA AND FRIGIDITY

Partial Love Requisites.—Preliminary to the discussion of frigidity proper, Stekel first takes up in detail the problem of love requisites. Each individual carries within him certain specific determinants which must first be fulfilled before he can fall in love. These love requisites may vary greatly and pertain to some bodily or psychic peculiarity. It is usually called fetishism and has been designated by Hirschfield as "partial attraction." Genuine fetishism, however, has reference to those instances which through symbolization dispense with the whole sexual objective.

No part of the body is without some possible significance in the love choice of mankind and may become either a partial love attraction or a fetish; again it may exert an antithetic or a repelling effect. As instances of the many partial love attractions one may note: the hair—its richness, shade, color, etc.; the nose—size, shape, its function as an organ of smell; the eyes—color, form, luster, gaze, their sexual symbolization; the ear, mouth, lips, tongue; the voice—its sound, timber, vigor, etc.; stature, size, gait, etc.

Infantile Fixations.—Partial love attractions are traceable to the fixation of infantile impressions. Indeed, all of a person's likes and

dislikes in matters of love owe their origin to the fixation of the earliest impressions of childhood. These infantile feeling predispositions persevere in adult life in greater or lesser degree in spite of all repressions, and when unrestrained often lead to serious conflicts which may manifest themselves as disorders of love life. The cleft which divides the infantile and the adult love requirements corresponds to the permanent feeling-tension which exists between man's primordial and his cultural trends. For this reason disorders of love life are truly social diseases. Human beings turn sick when they find no way of harmonizing the requirements of culture with their physical needs or instinctive cravings.

In the writings of many analysts, the neurotic person is described as one of extraordinary sexuality and with polymorph perverse tendencies. Closer scrutiny, however, reveals that this apparent plethora of potentialities covers but a limited monopoly of pleasure seeking. Because of early fixations, the neurotic person's range of pleasurable possibilities is rendered extremely narrow. This is remarkably well illustrated in fetishism. Only the healthy-minded person is capable of enjoying the whole range of his sexuality. Neurosis and paraphilia not only limit the individual to certain specific love requisites, but induce a still further narrowing of the range of love possibilities, by limiting the number of possible objectives to a specific type of objective. The anesthetic woman, the impotent man, the supposedly perverse persons who appeal to us so imploringly—all these patients suffer because they insist with a certain infantile stubbornness on the fulfilment of their specific love determinants. They have not freed themselves from their infantile objectives and fantasies; nor do they want to recognize the fact that these fancies have no existence in reality.

From its birth, the child's first objectives are the nursery persons and members of the family. The various stimuli during nursing care—feeding, washing and cleaning of the child, not to speak of the tenderness and kisses showered on it—tend to produce a certain erotic atmosphere which may have most deleterious consequences for later life. As a result of extreme tenderness, the children become fixated on their parents and emotionally cannot get away from them. Thus the daughter may not be able to wean herself away from the father, which may lead to frigidity; or she may be fixated on her mother and also be anesthetic, the nucleus of the disorder being formed by an unsatisfied homosexual attachment. Fixations on the brother, sister, uncle or grandparents also play an important part.

Incest.—Of the infantile fixations, incest plays a large rôle in the development of love life. Stekel looks on incest as only another aspect of narcissism, for in the love of the family the neurotic loves only himself, the various members of the family being but mirrors in which he sees himself reflected.

Many of the peculiarities of one's behavior are traced to the incest motive. There are men who are potent only with women of the lower classes—servants, cooks, et al.—and there are women who form attachments to drivers, waiters, et al. These are immediate surrogates of the parents (servants), and the reaction may also be due to a hidden dread of respectability reminding one of the picture of one of the parents. Or the choice of love objectives from the lower ranks may express a scorn or revenge on parents because of their alleged lack of sufficient affection.

An instance of but slightly disguised parental love pattern is seen in gerontophilia—love of elderly persons. The resemblance may be very marked or ever so slight, but in any instance is made to yield the resonance of infantile affects. There are women who will yield only to men much older than themselves, but will remain anesthetic in such relations on account of their inner inhibitions. For as each man stands for the father, every attempt at intercourse is an act of incest.

Other infantile determinants often come into play—the image of the sister or brother, or of all brothers and sisters. These love choices are not so simple; they may become interwoven, producing sometimes a confusing array of details which can be cleared up only in the course of a lengthy analysis.

The love some girls have for their father may even go to the extreme of open jealousy and frank struggle to win the parent's affection and the use of all means to separate him from his wife. On such a separation the girl usually takes the place of the mother. Such a girl will never marry as long as the father lives, and should she on his death marry, she is likely to prove anesthetic. The strangest thing in the whole situation is that it is not only the mild, delicate father who chains the child to himself; it is also true of the strict, the hard-hearted, the morbid and the unmercifully cruel father. There is a longing to kiss the hand even while it punishes. Here and there one encounters instances of girls who fell in love only to break down in a severe depression when the marriage was to be consummated.

Weaning from Incest.—Love is always incestuous at first. Its subsequent development proceeds in the direction of weaning the individual from the family so that eventually he is able to choose as his love objective some person outside of the immediate family circle. The period when the emotional detachment sets in varies. Usually, the weaning from the family bonds occurs during puberty, but some persons do not attain this emotional freedom from old infantilisms until their third or fourth decade of life.

In contrast to the normal, the neurotic person usually fails to attain this self-reliance. In the course of this developmental processes of his love life, he remains emotionally anchored to his family at some point or other.

It is at this juncture that one obtains significant glimpses into the disorders of love life. Many persons, in fact, are apparently incapable of loving. Closer scrutiny reveals that such persons are fixated on their parents, without being aware of it.

Adult Trauma.—There is no doubt, then, that earliest experiences give permanent form and lasting character to the love life. Many abnormal reactions in later life owe their pathologic character to the traumatic nature of early experiences. Nevertheless, says Stekel, too much stress cannot be laid on the significance of such experiences, for there are many children who have experienced a plethora of so-called traumas without developing any neurotic disturbances as a consequence. And even more baffling is the fact that one meets persons with very severe neurosis who have experienced no such trauma. Explaining the situation by taking recourse to the formulation of the "constitutional component" theory, according to which the traumatic experience acts as a shock only in persons constitutionally predisposed to neurosis, hardly solves the problem; indeed, the whole psychogenic conception of neuroses is shattered with this conception if it is true. Granting an existing somatic cause, one is still under obligation to search minutely for the influence of psychic factors on one's behavior.

There are also the sexual traumas of adults. There are obviously many persons who get along with a labile mental equilibrium, at least apparently; however, the first real difficulty they encounter throws them down and they cannot recover. Serious psychoses, no less than neuroses, may come on as the result of severe sexual trauma. There are persons who have borne very well their infantile traumas only to go to pieces on encountering their first sexual experience at puberty and early adolescence. As a matter of fact, human beings are often ill, not so much on account of what they have gone through, as over what they have failed to experience.

An infection acquired at the onset of one's sexual life is capable of affecting his whole sexual life in a decided manner. One person so burdened turns ascetic, another reverts to infantilism, a third inverts his personality, a fourth hides his personality under various fetishisms, etc.

With some girls the loss of virginity is a psychic trauma of the first order and may even precipitate a serious psychosis. The consciousness of a moral transgression and fear that this may mean forfeiture of marriage and normal sexual life enter here as components. In other cases, yielding to the practice of some perversive activity may act as a sexual trauma. There are further traumas which do not involve a breach of ordinary inhibitions, but which nevertheless may lead to serious consequences. Such a shock is the wedding night, especially if the woman goes through the experience without experiencing orgasm. Sometimes psychoses actually break out during the wedding night.

THE PSYCHIC BARRIERS IN FRIGIDITY

The Struggle of the Sexes.—Stekel lays considerable stress on that aspect of the relation between the sexes which is spoken of as the struggle of the sexes and which is responsible for the many barriers which the frigid woman or the impotent man may erect against her or his love partner. The relations between the sexes, even at best, have never, from times immemorial, run a smooth course, the existing antagonisms being expressed as the "struggle between the sexes." This has become particularly accentuated in modern times, now that the woman has gained a certain freedom and independence. Often she insists on more than equality; she wants to dominate the situation, and this is sometimes reflected in the relation between the sexes. While it is normal for man to be aggressive and for the woman submissive, tendencies of the opposite sex are sometimes observed in each, so that the woman may seek domination rather than submission; thus there is the phenomenon of masculinization of the woman and the feminization of the man.

Sexual anesthesia is an important weapon in the sex struggle. The woman who responds cannot remain cold and calculating; frigidity gives her certain advantages. To be roused by the partner often means to acknowledge oneself conquered. There are therefore successive lovers, each leaving the woman unsatisfied, and hence unconquered. Every yielding means defeat and humiliation, and the desire to dominate may loom up in the woman's mind as more important than gratification.

The marked development of spiritual love in the course of cultural progress serves only to bridge over the differences between the sexes and to create a common neutral realm. But as the power of love grows, the antagonistic forces which make for separation become the more violent. The complete satisfaction observed among lovers is the result of many compromises, each yielding to the other at least a portion of his personality. When the lover comes to feel that his personality is almost destroyed or is in imminent danger of becoming completely absorbed by the other, a dangerous reaction to love may set in. First, there may be just indifference or depreciation of the partner. Next, differences, heretofore absent or at least undiscerned, suddenly arise and become more and more acute; these come to be reflected in the sexual relations with depressions, disorders of sensations, decreased potency, weakening or disappearance of the orgasm, until one day the catastrophe breaks out and hatred shows itself in all its pristine rawness. Then the lovers return to their families, securing separation, etc. It is not, of course, an open and frank fight, but one that is carried out beneath the level of conscious thought. Every lover is often surprised by dreams which portray the beloved as dead or dying—an inverted

wish. Usually, the hidden death wish is associated with the hypocritical thought that one could not survive such a disaster, that one would rather follow the beloved to an untimely grave. This, of course, does not contradict the occasional instances of certain love ties that are so strong that only death can sever them. And there are also lovers, the saddest of the lot, whose differences are irreconcilable. Some people instinctively avoid love, for fear of losing their freedom and independence.

The means which lovers use to free themselves from the bondage of marriage are various. One is the return of the lover to his family, which is a critical turn in one's love; for true love and family are incompatible. Another critical circumstance arises when either husband or wife becomes strongly attached to a homosexual friend. This may shatter one's great love and may cause its hatred to extend over the whole of the opposite sex, and thus accentuate one's homosexual tendencies or even lead to the establishment of a homosexual relationship. Other means of escape are regressions to infantile sources of gratification, a surprisingly new love affair which only proves too soon to have been an "imaginary" love affair, the main purpose of which was to free the person concerned from the fetters of his old love.

Sexuality and Erotism.—In human love relations there have always existed two more or less distinct components, more clearly differentiated in modern times. There is, on the one hand, the purely sexual, grossly physical aspect, having more to do with the elemental instinctive cravings (the spinal cord reaction), and on the other hand, the erotic, spiritual, refining aspects having to do with the idealized side of love (the cerebral reaction). True love consists in the fusion of both aspects on one love object, and where that fails, i.e., where physical love goes to one love object while spiritual love goes to another love object, then appear some pathologic manifestations of love life.

It is often impossible to trace a dividing line between physical and psychic objects of love, for most of the manifestations of love are mixed; in the love requisites of psychic order, one finds a number of bodily qualities which have the power of rousing certain mental states. The splitting of love into its spiritual and physical constituents, the separation of tenderness from sensuousness (Freud), leads to the strangest manifestations in the mental life of mankind.

The problem of satisfactory adjustment between sexes is not always or merely a question of potency. There are women who are frigid with every potent man because they have been antagonized, while these same women may achieve full orgasm with men of weak potency if they love the man.

There are many normal women whose unfortunate experience it is always to have met men who see in them nothing but an instrument to satisfy their physical passion. Yearning as these women do for a

love that will provide them an outlet for both the physical and the psychic, they get only the crude emphasis on the physical, for which reason they remain completely anesthetic; yearning but unsatisfied, these women are constantly in search of new adventures, each one of which leaves them cold until they meet a man who will satisfy both needs, the physical and the spiritual; whereon the anesthesia will disappear, thus affording additional evidence of the facultative nature of the reaction.

In the more advanced cases, one finds neurotic women who have split their love life into two distinct components: the physical and the psychic, maintaining relations with both types of men. For the physical partner she will choose a physically appealing but commonplace and perhaps uneducated man with whom relations are established on a purely sexual basis. There may be an orgy lasting several weeks, following which disgust overtakes her; or the man may become obnoxious to her immediately after the act. The second "sweetheart" is valued for his cultural achievements and the spiritual friendship he may offer. The relation is in the main platonic. Physical relations may or may not be indulged in, but in any case they are deprecated and appear in the background. With both men she remains anesthetic. She is anesthetic with the "physical" sweetheart, because she regards orgasm in the embrace of a common man a shame and defeat of her overstressed feeling of personality; she is anesthetic with the "spiritual" sweetheart because with him she wants a soul bond to heighten her feeling of personality. There has thus failed to take place a more perfect synthesis of sensuousness and tenderness in sex.

In many cases the flight from love expresses the obstinate struggle for spiritual values staked against physical values. There are women who are unable to find the physical and spiritual love. They perceive their sexuality as a fall, a defeat, as a blot on their womanliness and withhold from the beloved man precisely what they freely grant a stranger. Before their beloved they want to maintain the proud pose of being interested only in spiritual love; with them they cannot be roused physically. The spiritual aspect neutralizes the possibility of orgasm.

Emotional Barriers.—The conditions which influence the development of frigidity belong in general to two main categories: (1) those that have to do more or less directly with the immediate circumstances in the relations between the couple—the emotional barriers; (2) those which are rooted deeper and pertain to regressive fixations—the paraphiliac barriers. While these factors often overlap and influence each other, in clinical experience they are frequently distinct.

Very often, and without direct reference to any regressive fixations, one observes the presence of certain emotional barriers, such as hurt pride, hatred and jealousy, which interfere with the full expression of

the orgasm. The history of many anesthetic women shows that at one time they were fully responsive. Then some humiliating situation occurs—as for instance the man leaving the woman to marry a rich girl, etc.—and as a reaction to the betrayal the woman becomes a man-hater, the mind being filled with thoughts of revenge, with anesthesia as an expedient. Should there now be an opportunity for marriage, such a woman would rather choose among suitors an unloved one than one whom she loves, because with the unloved man she can remain in control of the situation and use him as a means with which she can avenge herself on all men.

A couple may enter into clandestine relations and the woman may be fully responsive at first, but if she has had inner hopes of marriage while the man does not give any evidence of any desire for marriage, the woman may develop anesthesia as a reaction to a feeling of insult.

When marriage is sometimes forced, if the woman senses the man's resistance (hesitancy, etc.), she will respond in marital relations with anesthesia. If a man has humiliated his wife on the bridal night, she will respond to his approach with anesthesia and may readily give herself to another man, perhaps even an enemy of her husband, just to avenge herself.

If the man manifests the desire to rule, vaginism may set in during the early months of marriage as the first symptomatic expression of the woman's stubbornness, while the husband may be consumed with desire. Anesthesia follows, along with other objections which the woman finds in her husband—his breath, odor, etc. She then bestows on her lover what she denies her husband.

A woman may respond with anesthesia to her husband's or lover's unfaithfulness; there may be pain.

A prolific source of anesthesia is found in those cases in which the woman yields to the man mainly for material considerations and in the face of obvious lack of love. The inner resistance then may manifest itself through anesthesia, as well as such other symptoms as nausea, anxiety and vaginism. Here belong also cases of women who presumably are in love with their man, although unconsciously their love is elsewhere.

Often the anesthesia is due in the main to tactlessness on the part of the man. Again, a humiliating requirement on the part of the man (i. e., anal intercourse—ostensibly to prevent conception, but really a transparent mask for homosexuality) will sometimes antagonize the woman greatly, and she may become so anesthetic that she may not even respond to the man's kisses.

In all, then, if for some reason or other a feeling of animosity is created, the ill-feeling, often unconscious, may be strong enough to act as a barrier so that the orgasm is regarded by the woman as defeat

and its onset is avoided. Hence many women refuse to be roused; if roused, they refuse to acknowledge their feeling and even deceive themselves on that point. All harlots are relatively anesthetic, because they feel that the men are ashamed of them, but they will respond to shown attention; indeed, they are capable of responding fully in the arms of their lovers. The anesthetic woman either disregards her orgasm or manages to subdivide and distribute the orgasm among numerous partial forepleasures or postorgastic thrills. The failure of orgasm is placed at the door of the man, and this in itself is already a victory for the woman. By thus constantly denying the orgasm, the husband is continually goaded to renewed efforts in an endeavor to bring to her an orgasm in spite of her apparent frigidity. This is the reason why women of this type sometimes choose innumerable lovers, each one of whom supposedly leaves them "unaroused." They deceive themselves only by assuming their orgasm to be a forepleasure, constantly imagining that there is more yet to come.

As an instance of frigidity that forms a transition between this and the graver types and the etiology of which goes beyond the immediate emotional relations, there is the case of a woman who remains frigid because the man failed to discover her particular sexual habits—the physical determinants, the erogenous zone, the various positions during intercourse. One knows cases in which refined skill in the ars amandi successfully annulled woman's frigidity. Havelock Ellis believes that the fact that a woman remains cool in the embrace of a man, or even in the embrace of several men successively, does not prove that she is not capable of strong sexual feeling. Often the anesthesia is obviously of a facultative type, the orgasm setting in only when the adequate form of gratification is met.

The Paraphiliac Barriers.—The next group is that in which the anesthesia, along with immediate situational determinants, appears to be also conditioned on more infantile situations. The simplest cases of this type are those in which some anxious expectation or morbid fear prevents the occurrence of the orgasm. There are women who, either for fear of losing their independence with reference to the male sex, or becoming slaves to their own passions once they let themselves go, either suppress orgasm or else refuse to acknowledge its occurrence to themselves. Some women are afraid of their own passions. The various homosexual barriers, to be spoken of, enter here as factors.

In the second volume of the series (Homosexualität and Onanie) Stekel has discussed in detail the significance of homosexuality in the relation between the sexes, and in particular its various expedients (masturbation, incest, etc.), as well as the masks and disguises under which it appears. He believes that bisexuality is a large factor in female frigidity. There is no doubt that strongly emphasized bisexuality

interferes with the normal course of one's love life. If a woman is homosexual, though unaware of it, and unconsciously longs primarily for a woman as her sexual objective, no man will be able to satisfy her unless it be a man possessing pronounced female characteristics, or in a setting in which she can enact the masculine rôle.

Many of these homosexual women, unwilling to acknowledge the fact of their homosexuality, turn the more eagerly to the male, but fail to find gratification there. There are women who in their flight from homosexuality develop the strangest behavior reactions and most remarkable physical disorders.

Equally, masturbation contributes its share as a factor in frigidity. There are women who do not achieve a satisfactory orgasm in normal intercourse unless it is accompanied or followed by masturbation practices which are the more preferred. There are also women to whom the practice yields the only sexual satisfaction they can attain, and while they may yield to sexual intercourse, the latter leaves them completely cold. They may thus go on for years, simulating the good and dutiful wife without showing any nervous symptoms of any description. Then perchance they may learn of the "terrific" consequences of the "vice"—spinal trouble, feeblemindedness, premature senility, loss of memory, emaciation, etc. Promptly the practice is stopped, whereupon there follows a condition which develops when masturbation is thus abruptly given up. The patient becomes depressed, suicidal and shows other neurotic reactions, which, wrongly of course, lead the family physician to draw the favorite old conclusion that the neurosis is due to masturbation. As a matter of fact, such patients are entirely well as long as they indulge in masturbation; it is abstinence that makes them ill. Obligatory and compulsive masturbation is always accompanied by fantasies, heterosexual, homosexual, masochistic, etc. Therefore, if the clinical history of an anesthetic woman discloses an addiction to the masturbation habit, one is not justified to infer that her sexual disorder is necessarily due to the habit.

Incest is sometimes found as an etiologic factor in frigidity. As incestuous wishes are shared by all children, the fact that the neurotic has not rid himself of such wishes shows that he is still a child. One meets such child-wives frequently in life. They do not necessarily belong to an infantile type so far as their physical structure is concerned. There are child-wives who would be taken for enchanting, full-grown women by any one who is not closely acquainted with them. But they live only in their childhood, they have children's habits and would like to remain forever the pampered child.

Women suffering from this emotional slavery never get freed of their family bonds. Virtually such a woman never belongs to her husband or children, does not tolerate any conflict between her new

family and the old household, and when such arises she promptly exhibits the "familitis" neurosis, fleeing into neurotic invalidism as a refuge which is at the same time a punishment to which she subjects her husband for his encroachment on her family feelings. Gradually, the difficulty involves her love life; by degrees she turns into a completely anesthetic state, thus expressing the inner negation.

The simplest as well as the most common form of anesthesia is the fixation of the woman or the girl on the father. These cases present themselves under strangely hidden forms and are sometimes not easily detected. With these women permissible gratification fails to bring on the orgasm; but masturbation is indulged in and preferred because the accompanying fantasies reproduce infantile situations that are charged with strong emotions.

The attachment between female members of the family may grow into open homosexuality, with competitive auto-erotic practices as expedients. On marriage such women find themselves anesthetic because their love is elsewhere than on the immediate object.

Anesthetic women possess certain erogenous zones which on stimulation are capable of inducing the first orgasm. After, the stimulation is transferred to the "normal" sexual zone and provokes the orgasm in the course of "normal" sexual relations; but in most instances the capacity for attaining orgasm remains linked to the presexual erogenous zone. The mammae are an important erogenous zone. It is true also of numerous extragenital body regions: arms, hands, feet, eyes, the nose, the ears, etc. The various love determinants, spoken of already, come into play. A certain physical fitness of sexual parts proper seems also requisite. However, when the psychic attraction between a loving couple is what it should be, the anatomic disproportions are easily overlooked.

To the simplest form of frigidity in women belong those cases in which some particular erogenous zone assumes "sexual primacy." Gradually the libido spreads from the clitoris and from other erogenous zones to the genital zone proper, the introitus vaginae. Among masturbating women this transferrence of the libido cannot take place, their excitability being more or less permanently fixed around the clitoris. But not on the clitoris alone. It would be one-sided to consider only the clitoris and disregard all the other erogenous zones.

Some women have as a particular love requisite, sado-masochistic wishes which shame prevents them from fulfilling. They usually show other neurotic or hysterical reactions and give a history of early abnormalities. Unable to fulfil their wishes, they live in the fantasies of them. Others prefer the practice of cunnilingus (with a man or woman as the case may be), masturbation, solitary or mutual (with a woman), vaginal and anal intercourse, and other homosexual and paraphiliac

reactions, in an active or passive rôle. Heterosexuality may be completely absent. All sorts of combinations are possible.

Strongly accentuated, although hidden, sado-masochistic trends may sometimes be a love requisite; failure to realize it may be responsible for anesthesia. Some women experience their strongest orgasm under great stress, fear or force; the fantasy of doing something under compulsion releases in them the highest orgasm. Such women will often masturbate with the fantasy of being assaulted.

As an expression of sado-masochistic trends one finds women who by preference choose the weak and helpless male on whom they shower a great deal of tenderness. The attachment of such a woman to those dependent on her creates situations which enable her to enjoy unconsciously the feeling of superiority and the majestic sense of "mothering" some one. The craving for power leads these women to hate the stronger element of the opposite sex. Again, the struggle against infantile love inclinations sometimes generates ascetic tendencies; as may be seen in the affection which sometimes binds the woman to an impotent man.

FRIGIDITY IN RELATION TO OTHER NEUROSES

As one surveys the type of woman who suffers from a more or less permanent state of frigidity, the observation is made that in the majority of cases one is dealing with hysterical persons who often present other psychopathologic reactions and in whom frigidity appears as but one of the many aspects of the morbid picture.

Anesthetic women sometimes exhibit marked anxiety attacks, and their anxiety appears to be related to the anesthesia. To the woman who cannot find happiness in her husband (or lover), two roads are open; she may remain faithful or else establish extramarital relations. When the woman remains faithful, anxiety may develop from deprivation and as a reaction to wish fulfilling but socially prohibited cravings to be satisfied in clandestine relations. For the moral trend of neurotic persons is very strong. When, as is often the case, extramarital relations are established, the patients expiate their transgressions with added anguish, and they can never attain an orgasm as long as their conscience does not permit it. It also illustrates the significance of the feeling of guilt when it becomes associated with gratification. All human beings are creatures of compromise. Guilt and gratification, punishment and rewards are fused in one act.

This is the reason why these patients are afraid of enjoying themselves. They feel that being a sinner they must suffer punishment. They think that the safest course for them is to pose as ill. They are thus afraid to get well for fear that they will be punished at once.

One of the most common forms of anxiety is the fear of infection, which may interfere decidedly with the orgasm. However, behind this more or less alleged motivation, there may sometimes be found deeper moral inhibitions interfering with the dangerous wishes; for the same anesthesia may remain even when the chances for infection are minimal, whereon some other explanation is offered.

Another factor in the production of anxiety is the expression of a death wish which may have been realized. This brings in its wake a guilty conscience which exacts a vow of abstinence that acts both as a punishment and as an atonement. Or the reverse may happen—the person may fail to take a vow of chastity to avert a certain situation or calamity, and if it should happen that the situation takes place, a belated oath is taken as a self-inflicted punishment. There are many women who deliberately deny themselves gratification in order to expiate their alleged sinful thought feelings. The anxiety the patient experiences in any sexual relation is thus an expression of a troubled conscience. Indeed any form of anxiety may disturb one's libido and prevent the onset of the orgasm.

Sometimes anesthesia is self-imposed in order to obtain a higher psychic goal, or it may be a self-inflicted punishment for past transgressions in accordance with the automatically operating law of lex talionis, the law of retribution.

Not infrequently one observes cases in which a formal vow was responsible for the sexual anesthesia. The failure of desire in such cases proves it to be a hysterical conversion, the moral imperative expressing the interdiction: "I must not allow myself to feel desire."

DISORDERS OF THE ORGASM

Frigidity, a Relative Concept.—In normal women orgasm is a matter of slow development. It seldom occurs during the first coitus, and very few women find first intercourse a pleasurable experience. Many complain of pain; in any case, it is disappointing. However, within a few weeks or months of apparent sexual anesthesia, there comes about a sudden change (often brought about by a particular variation of the usual position during coitus, as for instance *a posteriori*, or some chance discovery of a particular love requisite) and the woman experiences for the first time an orgasm, which on the first occasion may be profound and last for several hours.

It is characteristic of the frigid women that they state they heard about sexual intercourse inducing immense gratification, which they cannot understand, for not only do they feel frigid, but further they complain of pain, indisposition and nausea. Nor are they able to give an adequate account of their mental content during the act of intercourse. Some, even though they go through the act in a highly excited

state, claim to have "felt nothing"—a sort of dissociation followed by complete amnesia. When they come to themselves expecting the onset of the orgasm, it is already over.

The anesthesia of many women is merely superficial. In reality no woman is absolutely anesthetic. The history of many anesthetic women will often disclose that at one time, especially during courtship, they were very responsive. The anesthetic woman is often the woman who has not discovered the form of sexual gratification which alone can be adequate in her case. Very often the anesthesia seems to appertain merely to the act of sexual intercourse proper and does not include other manifestations of love life (anaesthesia vaginalis versus anaesthesia sexualis). In many cases it is found that the anesthesia is self-induced; for one reason or another, the patient does not want to feel. Just as men are capable of deflecting their orgasm, so the woman's sexual anesthesia is likewise the act of will, brought about in a similar way.

Types of Disorders.—Viewed with reference to libido, the forepleasure and the orgasm, one may differentiate the following three types of frigid women: 1. The sexual desire is strong, the forepleasure is keen, but the orgasm is nearly or completely lacking. Occasionally, and then only after considerable effort, an orgasm may be attained. Many women acknowledge a vague and rather diffuse pleasurable feeling. In numerous instances the orgasm becomes split up, as it were, and fuses with the forepleasure, so that the sexual anesthesia is not genuine. 2. The sexual desire is weak and the feeling may be entirely absent. The orgasm is either weak or rarely experienced even with utmost effort. 3. There is no craving or forepleasure, not so much as the rudimentary stages of the orgasm.

Attainment of Orgasm.—The means of attaining orgasm are numerous and varied. Not infrequently during intercourse other thoughts intrude into the woman's mind, decidedly interfering with the act. Some women bring on the orgasm with the aid of certain fantasies, as picturing men capable of releasing their orgasm. Or (which also obtains in men) they will fantasy a situation which forces them to hurry through the act either because others are present, likely to come into the room or are being suspected—a particular love requisite often conditioned on a fixation on a specific love episode (as early witnessing of bedroom scenes). Here anxiety is utilized as an aid to sexual stimulation. Others have, as a specific love requisite, the wish of being surprised in the act, and only the fantasy of it brings on orgasm. Most of these fantasies also serve the purpose of covering the woman's pronounced tendency to pluralism. Many of these fantasies stage veritable orgies. A third and a fourth and fifth person participate in the act.

Some women have recourse to fantasies of violation (rape, seduction, etc.). A seduction fantasy may be conditioned on an early experience but in later life the patient, while going through the episode over and over again and reliving it, as it were, her fantasy will carry the incident to its logical culmination and thus achieve an orgasm. Others are capable of attaining orgasm only under the strain of anxiety or fear in connection with the forbidden, as in the instance of clandestine relations when the woman is likely to find herself more responsive in the embrace of a lover whom she may not like as well as her husband. The freedom from restraint and feeling of shame in a clandestine relation permits the practice of various paraphilias (perversions) which the woman would not dare to allow her husband for fear of losing his respect. Constant preoccupation with the anal-erotic and related fantasies is sometimes a barrier which prevents the adequate realization of the orgasm.

THE PHYSICAL ASPECTS OF FRIGIDITY

The condition of frigidity has sometimes, although not at all universally or even commonly, an organic foundation. There are women with primary and secondary sexual parts in a pronounced infantile state. When one is dealing with instances of psychosexual infantilism one has, on the physical side, arrested development of the genitalia. The uterus is small, narrow and elastic like that of a child. The breasts are barely indicated, the hips are narrow transversely, the limbs are gracile, the face childish, the posture decidedly infantile. Women of this type are usually sterile; their menstruation is scant, the period of flow brief —the child-wife (Wittels). On the psychic side (not necessarily in the same person), one has also infantilistic reactions which may be extreme—"the eternal suckling."

Of the physical concomitants of frigidity, which are functional rather than organic in character, one may first note the phenomenon of vaginism. Vaginism manifests itself on every attempt at intercourse by such a spasm of thigh muscles as to render intercourse impossible. Physical examination is precluded. Salves (cocaine, etc.) are useless. Electric treatments are often impossible of application, as on the introduction of the electrode the abductors of both thighs stiffen, thus really deserving the old designation as tutores virginitatis—guardians of virginity. With the establishment of better relations between the couple, the muscular contractions gradually disappear. When intercourse is forced in spite of vaginism, the act may be followed by pain in the back, etc. Vaginism, however, may occur not only as a part of anesthesia, but as an expression of heightened sexual excitement.

Next one observes that among neurotic persons unpleasure or pain may displace gratification proper. The history of the development of

dyspareunia will often reveal the case of a woman who on marriage failed to find the great passions for which she yearned. For a while, sexual intercourse may go on tolerably well as it may be a necessity to her; but it will be noted that the response is not a particularly vigorous one. Then an attempt is made to abort the onset of orgasm by preoccupying the mind with pictures and settings not concerning the present situation. Then the patient begins to perceive discomfort and local pains instead of orgasm, which pains increase as time goes by.

Hysterical women utilize the bipolar characters of pleasure in order to protect themselves against feeling gratification during sexual intercourse, the woman foregoing the orgasm and resorting to the device of masking the gratification by transposing it into the opposite—pain. The gratification involved in achieving sexual satisfaction involves the risk of defeat. The close proximity of pleasure and unpleasure permits a conversion of pleasure into unpleasure, or orgasm into pain.

Pain during intercourse often serves to cover one's inner resistance to one's self, and may be used as a pretext for dodging the marital obligation of coitus. Occasionally, the pain is perceived only after the sexual act. There are women who on the day following intercourse get attacks of migraine or other customary pain in the back, or an attack of appendicitis, or morbid lassitude. For it is a frequent observation that whenever intercourse takes place without corresponding orgasm, it is followed by neurotic (often anxiety) symptoms. Yet the same woman, in the embrace of another man, may find full gratification; which proves only that the fervent statement of the anesthetic woman that she loves her husband is at least an unconscious deception.

Although appearing physically well, these patients have a large number of symptoms to complain of. Along with general instability and all sorts of fears, depressions, suicidal ideas and the like, there are noted abdominal and rectal pains, a part of the neurosis in general. Many of these women are of a strongly libidinous nature. They will express themselves as saying that the acme of a sexual embrace is their notion of supreme thrill; to fuse pleasure and pain and fall asleep forever in the act, this is the climax.

Yet in spite of the woman's tremendous libido, during which she will pinch and bite her husband, she may experience such sharp pain in the vaginal entrance that she will cry out or even faint from pain. Some will say that they cry out because the pains almost take their breath away. Others have stated that they will experience, from the first, sharp pains during intercourse, often persisting for two or three days. Later, pains of a sharp convulsive character set in toward the end of the sexual act. The pain during sexual intercourse has also been described as of a boring, penetrating character. It is localized either externally at the introitus vaginae or in the "small of the back"; sometimes the pain

radiates toward the rectum and is described as unbearable, or there may be particularly sharp pains in the back radiating to the lower intestines. The gynecologist then, wholly ignorant of the basic pathology of the condition, will advise moderation, whereon the patient will promptly arrange for long periods of abstinence.

Intercourse may be accompanied not only by pain, but by disgust, nausea and at times vomiting, which naturally prevents intercourse. Often there are encountered states of dizziness as well as such typically hysterical conditions as globus hystericus. Dizziness is a symptom which appears whenever unwelcome thoughts are trying to break into consciousness. This is the reason why many neurotic persons complain of this symptom manifesting itself on arising in the morning. The nocturnal dream endeavors to break into consciousness where it is unwelcome. Dizziness develops, and the dream is "forgotten."

It will also be observed that during the day these women are given to a great deal of day dreaming, and are irritable and depressed. They retain no awareness of their day dreaming, because this is promptly repressed. The irritability and depression make them unapproachable, and thus serve the function of protecting the day dreaming from foreign intrusion and insuring undisturbed activity of the fantasying.

Globus hystericus may be an expression of a fellatio fantasy (Sadger); again it may be an expression of psychosexual infantilism. These patients frequently have the sensation of holding something soft and big in their mouth, an echo of the sucking period. The patients will also show a spasmodic swallowing movement of the throat muscles.

In one recorded case the patient suffered from a profuse vaginal catarrh which baffled the gynecologists and apparently was of purely psychic origin. She also manifested strange hysterical symptoms. Suddenly her mouth turns as cold as "ice," or during intercourse her husband feels the vagina turn cold, while the usual signs of sexual excitement (constrictor cunei contractions) remain absent.

There are some who show digestive disorders conditioned on anal erotic situations. In one recorded case, the patient showed an interest in the anal zone. Here constipation was most obstinate and was due to a spasm of the sphincter ani. The spasm seemed a protection against anal erotic activities. She could move her bowels only after masturbating; then the spasm of the sphincter was relieved, and the tight anal foramen released. The analogy of this to vaginism is striking.

In the same patient much was observed to suggest that her mouth was a strong erogenous zone—her craving for sweets, her habit of sucking. She suffered from food nausea, her disgust for food being a protection against perversive oral cravings. She indulged in fellatio fantasies, but was afraid to yield to its practice. She was troubled with

the globus hystericus, related to her fellatio fantasies (she wants to "swallow down" but cannot).

Another patient evidenced on the physical side a number of significant symptoms. She suffered from hysterical deglutition. She went through and repeated again the motions of swallowing. Frequently this symptom merged into globus hystericus. The globus hystericus is associated with pains and stiffness in the back of the neck; it gives her the sensation of holding something soft and big which she could neither swallow nor spit out. She does not shrink from fellatio, although she does not like the act and it fails to rouse her libido. While she suffers from globus hystericus she has to take something in her mouth; she sucks her fingers or her hand. Her throat carries out reflex swallowing motions which are identical with the sucking motions of an infant. She is a typical illustration of the "eternal suckling." The pain in the back of the neck suggests the child leaning its head back while sucking at the mother's breast. Her attacks of dizziness are mostly a means of protecting herself against temptation and sins, an expression of an uneasy conscience as well as the means that enables her to act the rôle of an invalid.

Striking are the cases in which the patient is sensitive throughout the body, but the vagina and the introitus vaginae remain anesthetic. One encounters cases in which formerly sexual intercourse caused the patient pain in the genital region. Then the vagina became anesthetic so that the patient became insensitive to pain—a complete hysterical sexual anesthesia.

PROGNOSIS AND TREATMENT

The majority of cases of frigidity have as their etiologic background some psychic difficulty or a series of psychic difficulties on the unraveling of which the success of therapy depends. The latter in its turn depends on the type of case and the manner of approach. Cases conditioned on some emotional barrier, as described, do not as a rule present great difficulty, and sometimes a few talks with the patient and perhaps her husband may clear up the entire situation. In those cases—not at all infrequent—in which the woman's frigidity is secondary to the husband's impotence, the therapeutic efforts must of course be directed toward that source. The other group of cases which are conditioned on more profound personality difficulties and deep-rooted fixations naturally do not offer as good prognosis and require deep therapy that will often tax the skill and patience of the best therapeutic efforts.

It is the universal experience of those who deal with frigidity that medicinal, surgical or mechanical measures usually fail of their purpose. Their application is made more on faith and hope than on an honest understanding of the underlying pathology. One will not appreciate

the meaning of the failure of such remedial measures until one realizes that these patients are hysterical, and that the physical symptoms are only hysterical conversion reactions. In these emotionally dependent persons, the stubborn wish and persistent indulgence in dull fantasying count for more than the hard reality, an adjustment to which society demands. All these neurotic persons are clever play actors; psychobiologically children, they parade before one as adults. The repertoire of their complaints is rich in variety and the somatopsychic guises, of which frigidity is but one aspect, will often baffle the keenest diagnostic skill. The condition is much more widespread than is ordinarily appreciated. These patients fill the offices of the specialists in gastroenterology, endocrinology, surgery, neurology, gynecology, etc.—usually for some other complaints—and defy the best efforts that the therapeutics of these offer, because these are beside the true cause. Many human tragedies are hidden behind frigidity. The tendency to depression is frequent, and will account for much of these patients' unhappiness and occasional suicides.

It is safe to say that if every patient, whether he comes to the office of the neurologist or of the surgeon, were given the benefit of a thorough psychiatric history, many of the pitfalls of modern medicine could well be avoided and the patients spared the heavy emotional cost that prolonged neurotic illness entails.

Finally, one must not fail to note the large social significance of these neuroses. The growing dread of marriage, frigidity, dread of childbirth and breast feeding, the small family system and the divorce problem are but a few of the symptomatic social manifestations of the woman's rebellion in her struggle for equal rights. As envisaged by Stekel, the eternal clash of sexes is even more far-reaching than the struggle between the nations, for by lowering the birth rate it endangers the whole nation. Thus the prevention of dyspareunia is chiefly a social and educational problem.

The Cecil, 1026 Fifteenth Street, N. W.

News and Comment

ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASE

The tenth annual meeting of the Association for Research in Nervous and Mental Disease will be held at the Hotel Commodore, Forty-Second Street and Lexington Avenue, New York City, on Friday and Saturday, Dec. 27 and 28, 1929. The meeting will consist as usual of morning and afternoon sessions. The morning sessions will commence at 9:15 a. m., and the afternoon sessions at 2:15 p. m.

The meeting in 1929 will be subdivided into two sessions, at which different topics will be presented. On Friday, December 27, a continuation program on the convulsive state, originally taken up in 1922, will be presented. On Saturday, December 28, a continuation program on the subject of schizophrenia will be placed before the association. Dr. Stanley Cobb of Boston has acted as the chairman of the program committee for the session on the convulsive state, and Dr. George H. Kirby of New York City for the session on schizophrenia.

The following tentative program has been arranged for the session on the convulsive state.

Morning Session, 9:15 to 1:00 o'Clock

Microscopic Changes in the Brain in Epilepsy. PROF. WALTER SPIELMEYER.

Microscopic Changes in the Brain in Experimental Anoxemia. EDWARD GILDEA and DR. STANLEY COBB.

Some Clinical Results in Patients with Convulsions. DR. CHARLES BAGLEY, JR.

Pathologic Studies of the Arachnoid Villi and Pacchionian Bodies in Various Types of the Convulsive State. DR. N. W. WINKELMAN.

Interpretation of Encephalographic Observations in the Convulsive State. DR. E. P. PENDERGRASS.

The Results Obtained by Dehydrating Epileptics. DR. TEMPLE FAY.

The Effect of Induced Changes in the State of Hydration on the Occurrence of Convulsions in Epileptic Children. DR. IRVINE MCQUARRIE.

The Experimental Production of Convulsions by the Combined Action of Two or More Chemical Substances. DR. MICHAEL OSNATO, F. H. PIKE and DR. JOHN NOTKIN.

Repeated Experimental Convulsive Seizures in Rabbits. Dr. L. M. DAVIDOFF and NICHOLAS KOPELOFF.

The Problem of Localization in Experimentally Induced Convulsions. DR. C. A. ELSBERG and F. H. PIKE.

The Basilar Artery of the Epileptic. DR. WALTER FREEMAN.

Congenital Syphilis in Epileptics. DR K. A. and DR. W. C. MENNINGER.

Afternoon Session, 2:30 to 5:00 o'Clock

Report of the Association's Cooperative Neurologic Study of Noninstitutional Epileptics.

Certain Physiologic Factors in Seizures. DR. W. G. LENNOX.

The Use of Ketogenic Diet in Adult Epileptics. DR. C. J. BARBORKA.

The Effect of Cervical Sympathectomy in Epileptics. DR. C. F. MCCLINTIC.

Convulsive Attacks in Tumors of the Brain, Based on 500 C[illegible]es. A. W. PARKER.

Six Hundred Experiments on the Induction of Sleep in Epileptics. Dr. Joshua Rosett.

A Study of Neurosomatic Deterioration in Epilepsy. Dr. M. B. Hodskins and Dr. P. Yakovlev.

Intestinal Bacteria and Their Relation to Epileptic Attacks. Dr. Harold L. Higgins.

The following tentative program has been arranged for the session on schizophrenia.

The Blood-Cerebrospinal Fluid Barrier in Schizophrenia. Dr. William M. Malamud.

Study of Personality in Schizophrenia Patients. Dr. Karl M. Bowman.

Environmental Factors in Schizophrenia. Dr. C. M. Campbell.

Prognostic Implications in Early Schizophrenia. Dr. Harry S. Sullivan.

Vigilance, the Motor Pattern and Inner Meaning in the Behavior of Some Schizophrenics. Dr. Smith Ely Jelliffe.

Physical Types in Schizophrenia. Dr. Herman M. Adler and associates.

Circulatory Studies in Schizophrenia. Dr. Philip J. Trentzsch.

The Prevention of Schizophrenia. Dr. Franklin G. Ebaugh.

The Biologic Approach to Schizophrenia. Dr. Walter Freeman.

Treatment and Recoveries in Schizophrenia. Dr. Leland E. Hinsie.

The Brain Pathology of Schizophrenia. Dr. W. Spielmeyer.

Affective Reintegration in Schizophrenia. Dr. G. Zilboorg.

CORRECTION

In the article by Dr. Westburgh which appeared in the October issue (**22**:719, 1929), the double column marked "P" near the right hand side of chart 1 on page 723, should read "Nor. 2," and "Nor. 2., Normal 2nd series, (N, 7 cases)," should be substituted for "P., paretic group, (N, four cases)" in the legend.

Abstracts from Current Literature

FREUD VERSUS JUNG. H. G. BAYNES, Brit. J. M. Psychol. **8**:14, 1928.

The freudian theory belongs to a sophisticated metaphysic and is compared to the classic sophism of Achilles and the Tortoise where one is lightly led to assume the truth of a hypothesis without stopping to take the whole situation into account. The attack on the freudian "speculation" must be made at the very foundation, because if "one attempts to distinguish between the content of empirical fact in Freud's writings and the wealth of assumptions almost uncontaminated by fact, one becomes inextricably involved."

Freud discovered the mechanism of repression, and that the dreams of neurotic persons exhibited the repressed contents. He discovered that these repressed contents were connected with erotic tendencies. He discovered certain infantile sex patterns, such as the oedipus complex, and discovered the technic of free association to reveal the content of dream imagery. Such discoveries were real and epoch-making, and credit is greatly deserved, but when he assumes that the fact of universal sex repression gives him a formula to explain all human behavior and erects a "psychological system upon a one sided theory of psychic causality," then one must challenge his system. The science of psychology has been starved of sound criticism. Freud, particularly in the first decade, saw his theories contemporaneously thrust aside and himself vilified. On the other hand, real criticism has been met by the "smile of complacency," a cult phenomenon and a symptom of overcompensated doubt. Ideas and discoveries take on their proper value only through thorough-going criticism.

Freud's attitude toward repression assumes that it is a mechanism operating independently of consciousness, but at the same time speaks of it as "emanating from the conscious personality (ego) of the patient. He therefore identifies repression with consciousness and then invents a censor to guard the gates of consciousness during sleep. Repression is seen as a factor of inherited social instinct and does not proceed from the ego but is required by the needs of race-survival." It is a fundamental necessity of biologic economy and becomes morbid only when taken to excess.

Freud's attitude toward the dream is another instance in which he lost his objective judgment when he recognized the presence of sex allusion in dreams, and thus provided with a formula proceeded to apply it to the whole content of the dream. The hypothesis is true as far as it goes, but like the sophism of Achilles and the Tortoise, it does not embrace the whole of the facts, and therefore falsifies reality.

In his discovery of the method of free association which established the connection between the dream and the hidden stream of the mental life, Freud neglected to recognize the powerful influence of suggestion arising from his own presence and relation to the patient. This he failed to take into account in appraising the dream with associated material. In other situations he has emphasized the importance of the infantile character of the transference, which brings the patient profoundly under the influence of the analyst, and probably acts like a selective stain, bringing associations conforming to a formula to the foreground and keeping others back. The associations are profoundly affected by the analyst and, furthermore, the same dream under different setting will bring up entirely different sets of associations.

Jung, through his association experiments, recognized the influence of the personal equation in giving different emotional values to the same ideas. He also recognized that it was impossible to eliminate this. Freud, with his belief in the mechanistic causality of the natural sciences, felt he could obtain constant conditions for his inquiries and thus satisfy laboratory requirements. That this is not so

is apparent to all who know that in the analytic relationship the interplay between the two personalities is the essence of the entire situation.

Rickman has insisted that the freudian position is based on a rigid determinism. The doctrine of determinism is in itself an attitude and a mental product and not an a priori condition of all minds. Many minds are convinced of the reality of freedom, a thing which the determinist senses but looks on as a delusion. The science of psychology is interested in the experience itself and the basis for interpretation made. It is concerned with determinism, not as a doctrine but as an attitude. The deterministic attitude approaches the psyche from the historical point of view, assuming that if all past experiences and determinants are known the present could be fully understood. The analytic situation, in which the patient has an infantile transference to the analyst, focuses entirely on the past determinants, the relationship influencing which of these determinants are brought to the surface. It has the effect of excluding not only present but further determinants. Freud sees the libido as purpose, but nowhere does he discuss its goal nor take it beyond that of direct or vicarious gratification and assumes that because the present situation can be understood on a historical plane, the future can also be absolutely determined. "The vital importance of Jung's conceptions in handling the real problems of life is that they embrace this purposive orientation of the libido."

The whole purpose of the analytic situation is to release the individual from a neurotic past and provide a goal for the future. The physician is the way toward that goal, and the patient is dependent on him to release him from his past ramblings and give direction to the future. This would indicate that a purely deterministic or exclusively historical attitude is not scientific. It excludes present and future factors which play an important rôle in the process of living. Haldane argues against this mechanistic attitude when he says that taking a living organism out of its environment and studying it under laboratory conditions means that one is no longer dealing with the organism as a whole but only with structure. Such observations are of value but are not to be confused with total individuality.

Freud, in supporting his dogma as all-inclusive and others as unscientific, has in reality created a cult and has placed himself outside the scientific world. Science is never bigoted or partial. A psychology based on a scientific attitude must be plastic and comprehensive and not based on a formalized rule-of-thumb method.

There is a need of a fundamental restatement of psychologic conceptions and a closer cooperation between science and philosophy. Freud took over the mechanistic hypothesis current in the nineteenth century and isolated himself from a philosophic approach which Jung was able to bring, the resistance to which on the part of Freud increased his isolation. The difference between Jung and Freud is illustrated by Jung's conception of the collective unconscious which gave weight to general human experience in ages past and which recognized that the power of the parental image is not solely affected by the infantile memories of the actual parent. Freud could not accept this concept as scientific and on this and other fundamental disagreements they parted, and then Jung elaborated this concept to "embrace the whole sphere of inherited function-complexes by means of which the historical continuity of all human experience exists as ways of functioning in the individual psychology."

Freud's tendency to exclusiveness hindered him from gaining a full appreciation of the significance of dreams. A dream of a patient is cited with conclusions drawn after a deductive type of interpretation. The same dream was presented to Jung, whose concept is that the major meaning of the dream is lost when subjected to an exclusively reductive treatment. The test of a dream interpretation is whether or not the interpretation gives a truthful and balanced likeness of the patient as a whole.

Jung was the first to recognize the importance of the conscious attitude in the investigation of unconscious processes. Re-evaluating the conscious attitude exercises an important influence on dreams, and an objective study of one's own

dreams produces an alteration in the conscious attitude. Freud has been unscientific and constantly undervaluing the importance of consciousness.

Jung in his "Psychological Types" sought to analyze carefully the various types of consciousness and to show the relation between his conscious attitude or philosophic outlook and his psychologic type. Jung in his work "saw the greatness of the spirit of man and could not be satisfied with concepts that dealt solely with his elementary origin."

ALLEN, Philadelphia.

INVESTIGATIONS OF THE PHYSIOLOGY AND PHYSIOPATHOLOGY OF COORDINATION: I. THE SIGNIFICANCE OF SENSATION IN THE ACTIVITY OF AGONISTS. H. ALTENBURGER, Ztschr. f. d. ges. Neurol. u. Psychiat. **116**:471 (Oct.) 1928.

The relation of sensation to the action of agonists has been studied by many, but without much success. While others were concerned with the action of different muscles, Foerster (1902) investigated a simpler mechanism, the single muscle. Following him, Wacholder and Altenburger have shown that the problem of coordination is concerned with the single fiber bundles of a muscle. The relation of these single fiber bundles is different in various conditions of activity. Posture and motion produce fundamental differences. In the first case there is a universal independence of activity of the fiber bundles, and in the latter condition there is coordination, often to the finest details. The nature of the movement, the degree of tension, etc., are of significance for the relation of these very small coordinations. Altenburger has investigated the relationship of single muscles and their fiber bundles (agonists) in their dependence on the sensory system.

To investigate this, simultaneous movement records were made of the action currents and mechanical motion curve in the agonists. The action current was recorded by an Edelmann double string galvanometer in which platinum or gold-platinum strings were used and placed under tension so that 1 millivolt caused an excursion of 10 mm. Platinum or silver electrodes were used in leading off from the muscles. Bass and Trendelenberg, and Wacholder, have shown that in this way records of single muscle fiber bundles are possible. Movement records were made according to Wacholder's technic.

Action Currents After Posterior Root Section in Man.—These currents were investigated in a patient with an amputation neuroma of the upper arm on whom Foerster had cut the posterior roots from the second cervical to the fourth dorsal level. After the operation, there was complete anesthesia in the dermatomes affected, and passive movements were no longer perceived in the shoulder joint. While the operation was being carried out under local anesthesia, action currents were recorded from the stump. They were taken from different fiber bundles of the pars acromialis deltoidei with the electrodes 3 cm. apart. Shortly after the movement, there set in, in both fiber bundles, first small and then larger waves which continued throughout the movement. Both sets of action currents are similar in both individual and group variations. After the roots were cut, the patient was requested to make the same motion. If the action currents after dorsal root section are compared with those before, there is found a great increase in the amplitude of the string extension present in both strings. Three possibilities present themselves in explanation of this increase in amplitude: 1. A purely physical one consists in a decrease of resistance between the electrodes and hence an increase in the amplitude. Altenburger discards this possibility, because a comparison of resistance with normal persons showed no change. 2. The relationship of the fiber bundles to one another in their discharge rhythm is a synchronous one, but after section of the dorsal roots many of them are out of phase. 3. The relationship of the different fiber bundles to one another is not changed, and the increase in amplitude is caused by an increase in intensity of the individual excursions. Before deafferentation, the average frequency was 110.5, and after this it was 122.6 per second, or an increase of 10 per cent. The form of the action current was unchanged by deafferentation; it consisted of primary and secondary waves as in the normal. Observations similar to those in the deltoid were recorded in the pectoralis major.

After section of the posterior roots in man there is an excess of agonistic activity which involves the intensity as well as the frequency of the impulses. This coincides with the subjective observation: Immediately after deafferentation, the carrying out of a movement is severely disturbed. In the weeks following the operation, a compensation occurs so that action current records taken weeks after the operation show that the amplitude of the excursion or the intensity of the impulse has returned to normal, whereas the increased frequency of the rhythm remains the same as immediately after deafferentation.

Action Currents in Pathologic Lesions of the Sensory System.—Action currents were taken in tabes from the gastrocnemius and tibialis anticus. On dorsiflexion of the foot, action currents which were present in the gastrocnemius at rest cease, and there is a marked increase in amplitude in the action currents from the tibialis anticus. The action currents in the gastrocnemius do not disappear completely, but during the movement and in the subsequent posture there are fine, numerous vibrations. A similar situation is seen in the gait of tabetic as compared with that of normal persons. In the tabetic person there is a marked increase in amplitude of the action currents of the tibialis anticus and, to some extent, of the gastrocnemius. These increased amplitudes occur in groups, separated by a more or less quiescent string in the phase of extension. The antagonists showed an increased amplitude in these movements, but this is not always the case. The amplitude of the excursions in the string galvanometer is independent of the speed of the movement in cases of tabetic ataxia. There is some relation, however, in the sense that increase in the speed of the movement increases the tendency to abnormal amplitudes. In a comparison of the frequency of the electrical responses in tabetic and normal persons, it is found that in some cases there is a much greater frequency in tabes and in others the same or only a slight difference. The difference in the various cases may be explained on the basis of the varied degree of pathologic changes in different cases of tabes. The generally increased frequency in these cases is of interest, however, in demonstrating the parallelism between the electrical record in cases of transection of the roots and of pathologic involvement of the roots. The only other studies of electrical records in tabes have been made by Gregor and Schilder, and by von Weizsäcker. These investigators found an increase in amplitude and a decrease in frequency. The former, however, worked with a low tension galvanometer string, and in the latter's cases of tabes the pathologic process had spread beyond the root zone and hence they are not parallel to the cases of Altenburger.

Action Currents in Lesions of the Anterior Horn.—Action currents in cases of old poliomyelitis taken from the quadriceps femoris and biceps brachii are similar to those found in sensory lesions. There is a greatly increased amplitude in the muscles affected, with greatly increased frequency, the action currents of great amplitudes showing between them or superimposed on them smaller action currents.

Double Sensory Supply of Striated Muscle.—If one looks on the action current as the product of activity of the central nervous system, one has some clue to the relation of sensation to the action of the agonists in a movement. "The increase in activity of the agonists after section of the posterior roots is only to be understood by the fact that in the normal during muscular contraction there occurs an excitation of intramuscular sensory elements, so that afferent impulses are sent out which regulate the innervation current coming to the different central nervous stations, and so modify it, that its intensity is adequate for the movement. Every muscular act is simultaneously bound up with the occurrence of sensory inhibitory impulses, and by the inter-working of both processes there results the coordinated innervation of every single muscle. If this inhibition is lacking, there occurs an exaggeration of activity. The loss of the sensory inhibition impulse leads to an increase in intensity." The work of Foerster and of Sherrington has shown that these inhibitory impulses come from the muscle and not from the skin.

ALPERS, Philadelphia.

RELIABILITY OF INFANTILE MEMORIES. J. A. HADFIELD, Brit. J. M. Psychol. **8**:87, 1928.

Modern psychopathology generally accepts the importance of early childhood impressions as a predisposing cause of the psychoneurosis. This makes important the question concerning the amount of reliance that can be placed on the adult's retrospective account of early experiences. Objections to their reliability are: (1) it is impossible for any one to remember that far back; (2) it is inconceivable that a child could have the thoughts ascribed to him.

There is no priori reason why an adult cannot recall childhood experiences. There is no question that the very young child has a memory and recognizes such things as the mother's breast very early in life. If it has a memory, there is no reason why it cannot be reproduced. These memories are usually of two types.

There are many reported instances of a spontaneous recall of an infantile memory. An instance of a child, aged 8, recalling a fire when he was 8 months, with checks indicating the reliability of the details, is given. Then there are memories obtained by free association during an analysis and these fall into three groups: (1) memories that can be objectively verified; (2) memories that are corroborated from similar conditions observed in childhood, and (3) experiences commonly emerging in analysis which can neither be proved nor corroborated.

As an example of the first type, a case is cited of a man with bronchial asthma who, under hypnosis, recalled an experience of his mother putting something under his nose which nearly suffocated him. The mother verified the experience as occurring during her treatment for whooping cough. In this case the patient did not know that the event had actually occurred, even after he had described it.

In the second group, there are the experiences corroborated by observing similar behavior in children. All the symptoms of the psychoneuroses can be duplicated in childhood behavior of normal children. The factors leading to such behavior, however, are mostly subjective and observation of the actual causes is not an easy matter.

Can an experience happening at or before birth be recalled, is a question frequently raised. A case is described which gave an accurate description of the events and feelings surrounding the patient's birth, though she claimed no knowledge of such things from personal observation. The story was reproduced with all the emotional vividness of a relived experience. Watching a child at birth and after leads one to realize that the feelings of terror and anxiety and of strangulation might be experienced, and an adult reviving such a memory might be actually recalling such feelings.

The third group do not lend themselves to either objective verification or corroboration but are the recall of subjective impressions of an early experience. As an example, a detailed account of an early childhood experience is given, in which a revulsion to the mother's breast is described and this was associated with the administration of an enema and fondling by the father who was drunk. If such an account is taken as a recall of early impressions, then it is certain that the child's experiences are much richer and fuller than a superficial observation would suggest.

Except for the regularity of the reproduction of such experiences one would be inclined to dismiss them as the fabric of the imagination. There are two other possible considerations: (1) that they are literally true; (2) that a real experience has been interpreted and colored by later experiences. All experiences related during an analysis are not based on fact. Some are symbolic; some are composite; some are pure fantasy. The imagination and the fantasy are interesting and important. The reading into a past experience of the attitude of the present is also possible, and yet the present is colored and directed by the past and therefore bears an important relation to it.

The mind of a child consists mainly of feelings which are vague and not differentiated. When one accepts that feelings as well as thoughts can be reproduced and also accepts that the sophisticated language of the adult which is used to

describe these infantile feelings is a mixture of words describing both feelings and interpretations of them, one clears up some of the difficulty about the reliability of infantile memories.

The mind of the child consists of vague and undifferentiated feelings, some of which are later shaped into thoughts. These feelings are not different in kind from those experienced in later life. But the adult groups his feelings and differentiates between them — the child has not the capacity to do this. Children are too young to understand, but never too young to feel. The fact that the feelings are not understood but strongly felt is one explanation why some experiences have such a profound effect on the mind of the child. These are the experiences which may be recalled even though the language in which they are recalled is the product of a much later period. Archaic memories might be explained, not as an inherited endowment but as "merely the later interpretations of crude and primitive feelings closely associated with organic needs" and sensations. The organic feeling may be the only archaic factor.

Feelings can be reproduced as well as thoughts, and they can be expressed either by the primitive emotional response itself or by language. The infant has only the former and the adult in an analytic situation may find that a situation can be reproduced only through feeling it. The language used to describe it may be the true interpretation of this experience.

In translating the feeling into words there are three possibilities: (1) pure translation; (2) interpretation in the light of later experience but retaining the correct explanation of this feeling, and (3) interpretation so colored by later experience that the original feeling is invested with a wrong meaning. It is on this question of interpretation that large differences of opinion exist. The example is given of the boy who slashes off the heads of flowers, and it is then discovered that the flower represents the father. Adler and his followers would interpret this as a striving for power and the elimination of the father who thwarts this craving. Freud and his followers would place a sexual significance on this eliminating of the father as a means of satisfying desires with the mother. The previous formulation and school of thought would cause the analyst to read his own interpretation into the facts. This type of fallacy can come from both analyst and patient; from the analyst by reading in his own interpretation and from the patient ascribing to early experiences the interpretations of a later period.

The present view of freudians is to regard as of little significance the question about the reality or unreality of the experience. It is the wish for incest or the fear of castration that form the important things, and they exist apart from real experiences. Freud is quoted as saying "I should be glad to know whether the primal scene was a phantasy or a real experience. But taking similar cases into consideration, I must admit that the answer to the question is a matter of little importance." Since the whole freudian psychopathology is based on recollection, it leads one to ask what is left of the whole system when it is that of little importance whether or not these rather objective experiences are true or not.

Besides objective verification of a recalled childhood memory, there are other things that should lead one to take these memories seriously. The warmth and feeling with which an experience is recalled is different from that associated with an imaginary experience. Another factor is strong emotional tone and the effect such recall has either in producing or in abolishing symptoms. The witnessing of such recollections strongly impresses one with the reality of the experience. The final factor that is convincing is the fact that the psychoneuroses and the character traits are so fully explained by the memories brought out by free association.

All of these considerations are not to be considered as positive proofs. It is only the cumulative evidence which commands that the fact be taken seriously when associated with the factors enumerated. Absolute proof for the capacity to recall infantile memories is not possible. Evidence points to the possibility of such recall. Skepticism may be justified but "complete incredulity can only be a mark of prejudice."

ALLEN, Philadelphia.

MYOTONIA CONGENITA. MAX BIELSCHOWSKY, J. f. Psychol. u. Neurol. **38**:199, 1929.

Bielschowsky had occasion to study five cases of this disease, three of which are described in detail in this contribution. All patients presented the characteristic symmetrical paralysis and flaccidity of the extremities, especially of the proximal segments. The tendon reflexes were either abolished or diminished, and the joints were hypotonic. The presence of "flail shoulders" and inability to hold up the head were common features. Some motion was possible with the hands and feet, but the excursions were unusually feeble. The hypotonic and paretic muscles were not atrophied, nor were they doughy in consistence. Of the auxiliary respiratory muscles, the intercostals and abdominal muscles were involved; aeration of the lungs was actually carried on by the diaphragm; it is therefore not surprising that all the children afflicted with the disease succumbed readily to acute respiratory infections. The condition was congenital in four cases, and in one it manifested itself during the third month of life, although in this patient's sister, also affected with the disease, the mother noticed the condition at birth. This factor is an important diagnostic point. In these two cases, the anatomic observations were the same as in the other three cases.

There was a striking uniformity in the microscopic observations in all cases. Slight deviations from the usual features were encountered in some of the cases, but these were of no significance as far as the symptomatology and nosology of the condition are concerned. Some of these abnormalities were: variations in the persistence of the external embryonal granular layer of the cerebellar cortex, heterotopy in some of the ganglion cells, smaller groups of ganglion cells in the cord, slight anomalies of the ependyma, polygyric type of convolution with cyto-architectonic abnormalities (cases 4 and 5), etc. While these anomalies per se are not of much importance, they are significant because they indicate a tendency to a dysgenesis which also has some bearing on the characteristic pathologic features of the disease.

The typical pathologic changes of myotonia congenita are found in the ventral horns of the cord, in the striated musculature and in the intramuscular innervation apparatus. In all cases the motor ganglion cells in the ventral horns, especially in the enlargements of the cord, were markedly diminished in number; this was more noticeable in the lumbosacral than in the cervical portion of the cord. In areas in which the motor cells were destroyed, there seemed to have been a peculiar predilection for individual groups of cells; in some areas for the median and in others for the lateral groups. These sites of predilection for cellular involvement were observed not only in different cases, but also in the same case. Most previous observers have reported similar observations. The involvement of the cells did not appear to be a purely systemic one; it affected not only motor cells, but the mesial cell groups, or the cells in Clarke's column. In case 3, the entire gray substance of the cord seemed to have been reduced although, even here, the loss of the multipolar ventral horn cells was by far the most prominent observation. The dorsal cord showed the least involvement. The so-called sympathetic cell groups in the lateral horns of the cervical and dorsal cords were almost entirely intact. The motor nuclei of the pons and medulla were only moderately affected. Quantitative involvement was observed in the facial nuclei in two cases (1 and 2), and the hypoglossal nuclei in cases 4 and 5 showed striking cellular defects. Every case showed qualitative variations in the motor nuclei of the hindbrain. The absence of clinical signs of bulbar involvement in these cases was striking. The nuclei of the ocular muscles were normal in every case.

There were no evidences of vascular or inflammatory processes to account for the cellular involvement, so that there could be no question that one was not dealing with a fetal poliomyelitis or some vascular disease as the basis of the cellular changes. Nor could any conclusions be drawn as to the nature and origin of the pathologic process from a study of the glial preparations. The cellular defects were replaced by fiber-forming astrocytes. The nestlike grouping of

astrocytes was, according to Bielschowsky, evidence that a large number of ganglion cells must have fallen out at one time and had soon been replaced by astrocytes.

A study of the ganglion cell in this disease raises the question whether one is dealing with a degeneration — a shrinking process — or with a maldevelopment. The discussion of this question, Bielschowsky says, "is as old as the disease itself," and has led to various theories as to its causation and as to whether or not it was identical with the spinal amyotrophies. He himself believes that the disease is a malformation or arrest of development during which the motor innervation of the striated muscles has remained inadequate quantitatively as well as qualitatively. The anatomic substratum is one of agenesis and dysplasia of the ventral horn cells with a consequent insufficient supply of axons for the muscles supplied by these cells and a failure of maturation of the axonal terminations. Bielschowsky attaches more importance to the lesions of the axons and their endings than to those of the ganglion cells. The essence of the pathologic process, according to him, is a defective muscular neurotization. With this conception as to the pathogenesis of the disease in mind, much of the speculation as to the reason for possible recovery can be dismissed; one can readily conceive that improvement may occur because later in life either some of the contact organs within the muscles become mature or motor fibers invade the immature contractile substance of the muscles, both of which processes have a tendency to enhance voluntary innervation of the defective muscles. Bielschowsky is certain that this gradual change in muscle neurotization is the only substratum for the increasing functional capacity noted in these cases during the period of improvement.

So many writers have attempted to put myotonia congenita in the same nosologic category as the Werdnig-Hoffmann type of muscular atrophy that the author feels it incumbent on himself to emphasize the points of differentiation between these two conditions. The changes in myotonia have the earmarks of arrested development, which is complete at birth. Werdnig-Hoffmann's atrophy is due to a regressive process in previously normal spinoperipheral neurons which leads clinically as well as pathologically to typical manifestations of secondary degeneration in the musculature. A relationship between the two is conceivable, but only causopathogenically; namely, the arrested development in myotonia and the progressive cell degeneration in Werdnig-Hoffmann's atrophy might be due to similar endogenous factors. Because in some cases of myotonia the neurologic symptoms are associated with obesity, genital hypoplasia and myxedematous skin changes, some observers (Glanzman) have attempted to attribute the disease to endocrinal disturbances, particularly of the hypophysis. These observations, however, are few, and the results of anatomic investigations in these cases from the endocrinal point of view do not support such a hypothesis.

It must be mentioned, however, that the histologic differential diagnosis may occasionally present great difficulties. This is especially the case when regressive processes are found implanted on the dysgenesis and hypoplasia of the ganglion cells. Even in the cases that are the subject of this communication, the vessel walls in the region of the ventral horns and the differentiated wide muscle fibers show some evidences of a progressive degeneration, but these are too insignificant and far overshadowed by the dysgenetic phenomena. KESCHNER, New York.

SYMPTOMATIC PARKINSONISM. (POSTAPOPLECTIC PARKINSONISM.) EUGENE BRZEZICKI, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **30**:198, 1928.

The conception of cerebral circulatory disturbances has recently undergone significant changes. Pal has shown that hypertension does not depend only on arteriosclerotic changes in the blood vessels, but that there also is a hypertension attributable to functional disturbances in the musculature of the vessel walls. This functional disturbance frequently appears on a constitutional vasoneurotic basis and older physicians noticed that apoplectic attacks appear more frequently in families composed of thick-set, red faced, short necked persons than in others. Hanse offered statistics based on a study of 135 patients. He showed that the number of cases of apoplexy is greater in spring and in fall, and that most

strokes occur in the morning hours. It is surprising that the percentage of alcoholic and syphilitic cases is not more than 12.5 and of rheumatic cases, 20, while a "nervous disposition" occurs in as many as 40 per cent of the patients. Premonitory symptoms are found in 45 per cent.

Doubtless, organic and functional vasomotor disturbances can combine with each other in various ways, and vessels with demonstrable morphologic changes are predisposed to functional disturbances. Otherwise healthy young persons without hypertension, however, may show such an abnormal irritability of their vasomotor systems that slight stimulation such as mild trauma or toxemia can result in fatal hemorrhage. In such cases the vessels are healthy but the vasomotor mechanism, the vegetative system, is at fault.

The author mentions several theories of cerebral hemorrhage and then states that each theory fails to explain all cases of cerebral hemorrhage and softening. He states that it is much more probable that different explanations must be adopted for different kinds of hemorrhage but reminds one that the functional element present in every case must not be lost sight of.

Why do some parts of the central nervous system suffer more frequently than others? It is known, for example, that arteriosclerotic changes are found far more frequently in the brain than in the spinal cord. Also, hemorrhages are more frequently from the sylvian artery and its branches than from others, such as the lenticulostriate, in which thrombosis is most frequently encountered (Schwarta-Goldstein). Kodama has shown that arteriosclerosis may occur in the meninges independently of the brain stem. The basal ganglia are not all equally affected, the putamen, caudate and globus pallidus being most frequently involved in the order named. The same proportions hold also for softening. The only explanation that one can offer for this is that certain areas of the brain possess a greater vulnerability to noxae than do others.

C. Vogt and O. Vogt have called this particular vulnerability "Pathoklise," while Spatz and Mueller believe that there is a variability in iron content of tissues in various parts of the brain, i. e., a chemical difference in the various tissues. Brzezicki believes, however, that one must attribute many of these differences primarily to a functional vasospasm or a vasodilatation.

Among the numerous theories of the cause of hypertension, that of Bordley and Baker is of interest. It has been shown that decrease in the amount of blood flowing to the vasomotor center in the medulla oblongata is accompanied by a compensatory rise in blood pressure. The vessels of the medulla were examined in twenty-four arteriosclerotic patients. Fourteen of these had hypertension, and in all of them arteriosclerotic changes of the medulla were found. However, this theory can hardly explain hypertension appearing in persons with normal cerebrovascular equipment.

It is known that arteriopathies may result in softenings in the basal ganglia. Brzezicki reports four cases of hemorrhage into the corpus striatum, resulting in parkinsonism. Two of these hemorrhages were on a basis of syphilitic cerebrovascular disease, but because they had apoplectiform onsets they are regarded as cases of postapoplectic parkinsonism rather than as cases of syphilitic parkinsonism. The other two cases were on an arteriosclerotic basis. None of these cases gives a clear explanation of the cause of cerebral hemorrhage and it can be seen that a variety of changes may account for the appearance of a hemorrhage.

In none of the cases was the hemorrhage in the lenticular nucleus of any great size, so one must regard the bleeding either as hemorrhage by diapedesis, or as capillary or, at most, arteriolar bleeding.

The present work brings out some interesting facts concerning the relationship between the substantia nigra and lesions in the striatum and pallidum. The substantia nigra was involved in two of the cases here presented, and in both of these cases the outer portion of the globus pallidus also was involved. This signifies that these two areas probably have the same blood supply. It is known that the putamen, the medial part of the caudate and the lateral part of the outer division of the pallidum are supplied by the lenticulostriate artery. The

inner division of the pallidum is supplied by the anterior choroid artery. In the cases reported here, the lenticulostriate must have had some "predisposition to hemorrhage" (Blutungsbereitschaft) so that the outer portion of the pallidum was involved with the substantia nigra, while the inner division remained intact.

In all the cases of symptomatic parkinsonism, softening and cyst formation were found in the striatum. In the two cases in which the outer part of the pallidum was involved, the melanin-containing cells of the substantia nigra were degenerated on the homolateral side. In the other half of the brain, where there was no demonstrable lesion of the pallidum, the substantia nigra appeared to be intact. The degeneration of the substantia nigra on the affected side could not have been primary (such unilateral condition is unknown) so one must assume that the degeneration was axonal and secondary to the lesions in the homolateral pallidus.

The facts cited support the belief that the striatum has only an indirect connection with the substantia nigra, and that this is through the pallidus.

KAMMAN, St. Paul.

COMPARATIVE CYTO-ARCHITECTONICS OF THE AREA STRIATA. I. ALOUF, J. f. Psychol. u. Neurol. **38**:5, 1929.

The area striata presents in all mammals the following histotectonic characteristics: (*a*) The cortex is narrower and the cellular elements more or less smaller than in the adjacent cortices. (*b*) The lamina zonalis (I) is somewhat narrower than in the neighboring cortices. (*c*) The lamina corpuscularis (II) is generally inseparable from the third layer; in the cases in which it represents a separate and individual layer (man), it is narrow and consists predominatingly of very small pyramidal cells. (*d*) The lamina pyramidalis (III) is very narrow, its pyramidal cells being of the small and medium-sized varieties. (*e*) The lamina granularis (IV) is generally wide and well developed. In animals in which this lamina is poorly developed (*Erinaceus europaeus*), it is most developed in the area striata. In some animals (mouse, hedgehog, bat) the fourth layer shows generally no divisions — typus unistriaris — in others (rabbits), it is divided into two parts (IV *a* and IV *b*) — typus bistriaris — and in still others (kangaroo, cat, hog, dog, monkey, man) it is divided into three and even more layers (IV*a*, IV*b* and IV*c*) — typus tristriaris. In cases in which lamina IV is subdivided, IV*b* is always lighter in color and contains large cells (Cajal's astrocytes). In the fiber preparations lamina IV*b* corresponds to Gennari's striation. (*f*) Lamina ganglionaris (V) is considerably narrower than in the adjacent cortices and shows generally no or indistinct subdivisions. (*g*) Lamina multiformis (VI) is narrower and considerably denser than in the adjacent cortices. The cellular density of this layer is, in all mammals, very striking, and must, therefore, be considered as a characteristic feature of the area striata. (*h*) Lamina infima (VII) is narrower than the adjacent cortex and can be seen definitely separated from the white substance.

All borders of the area striata are sharply defined. Structural differences are noticeable in all layers, but are most distinct in the fourth, and especially in the sixth.

(*a*) The Width of the Area Striata: In the kangaroo this area is relatively wider than the adjoining cortex; in the hedgehog, on the other hand, there is little difference between it and the adjacent cortices. In the hog, horse, mouse, squirrel, rabbit, cat, dog, bat and half-apes the area striata is constantly narrower than the adjacent cortices. In apes and in man its narrowness is especially striking.

(*b*) The Cellular Content of the Area Striata: This is greatest in primates; next follow the rodents, cat, kangaroo, dog, hog, horse, hedgehog and bat.

(*c*) Lamina Zonalis (I): In the kangaroo and hedgehog this lamina is as wide as in the adjacent cortex, whereas in all other animals that were examined it is always narrower.

(*d*) Lamina Corpuscularis (II) Corpuscularis (II): In the kangaroo, hedgehog, bat and to a certain extent also in the hog this lamina consists of narrow, dense striae made up only of small pyramidal cells. In the horse, mouse, squirrel, rabbit, cat, dog and half-apes this lamina is not separated from lamina III; the same is true of man but here relatively more granules are found.

(*e*) Lamina Pyramidalis (III): This lamina is relatively wide only in the mouse and rabbit. Its subdivisions are generally speaking poorly marked. The kangaroo, hog and bat are the only animals in which this lamina has two subdivisions; the hedgehog has none. Among the animals having an undifferentiated lamina II and III are the horse, squirrel, rabbit and monkey. These animals show a division of this lamina into two parts, whereas the mouse, hedgehog, cat, dog, half-apes and man show no subdivisions at all.

(*f*) Lamina Granularis (IV): In the kangaroo, this lamina assumes a tristriar type. The two representatives of the *Ungulata* (hog and horse) differ from each other in this respect. Whereas in the hog this lamina is tristriar in type, it is unistriar in the horse. In the mouse it is unistriar, in the rabbit bistriar and in the squirrel propetristriar. In the hedgehog it is unistriar. The cat represents a tristriar type. The dog has a tristriar type. In the bat, with merely an indication of an area striata, there is no subdivision of the IV layer. In half-apes (*Lemur catta*) lamina IV is tristriar, whereas in monkey and in man it is markedly tristriar or, correctly speaking, multistriar. It is also noteworthy that in the *Ungulata* and *Carnivora* the nuclei are relatively large, whereas in monkey and in man they are small. In the bat and hedgehog the area striata is poorest in cells, whereas in monkey and in man it is richest.

(*g*) Lamina Ganglionaris (V): In the kangaroo this lamina is widest; in the horse and mouse it is comparatively wide, whereas in other animals it is strikingly narrow. In the mouse, hedgehog, cat, dog and bat there are no subdivisions; in the kangaroo, horse, rabbit and man this lamina has two subdivisions, and in the hog, squirrel and half-apes three. The subdivisions of lamina V are caused by the fact that the center contains magnicellular elements densely packed together, above and below which the layers are poor in cells. As in the lamina granularis the subdivisions of this lamina (V) cannot be used as a criterion as to which order the animal belongs.

(*h*) Lamina Multiformis (VI): In the kangaroo this lamina is wider, but in all other animals it is narrower than in the adjoining cortex. The cellular content of this lamina is only moderate in the kangaroo, but is extremely rich in all other animals. In man, monkey, half-apes, dog, cat, horse and hog no subdivision of this lamina can be made out, whereas in the kangaroo, mouse, squirrel, rabbit and bat two, and in the hedgehog three subdivisions can be seen. The large size of the cells in this lamina is striking in *Lemur* and in the primates. Together with the large cellular elements in IV*b* and of a few cells of lamina V, the cells in lamina VI are the largest cells in the area striata.

(*i*) Lamina Infima (VII): In this lamina comparative tectonic characteristics cannot be determined.

(*k*) Border Between Cortex and White Substance: In the kangaroo this border is not distinct; it is somewhat more distinct in the horse; in all other species on the other hand, it is unusually clearcut.

Alouf, in closing this study, points out that from the anatomic point of view it is impossible to substantiate the hypotheses offered by Bárány and by Kleist that binocular and monocular vision have any relation to the splitting or nonsplitting of lamina IV in the area striata.

KESCHNER, New York.

LOCALIZING VALUE OF OPHTHALMIC EXAMINATION IN SUPPURATIVE DISEASES OF THE BRAIN. WELLS P. EAGLETON, J. A. M. A. **92**:713 (March 2) 1929.

Some time ago, Eagleton came to appreciate the importance of repeated examinations of the visual fields in cases of suspected intracranial complications. Only patients whose mental condition allowed cooperation are reported in the paper.

There are thirteen abscesses in twelve cases, with seven recoveries. There are also two cases of bulbar cisternal meningitis and four cases of edema of the brain and localized meningitis of the middle fossa. It is Eagleton's purpose to show that: (1) edema of the brain may be diagnosed early by the presence of hemianopic indentations of the fields; (2) papilledema, by its presence or absence, may be of some localizing value in suppurative diseases of the brain; (3) localized meningitis of the bulbar cisterna presents a symptom complex consisting of both posterior and middle fossa symptoms with bitemporal hemianopic indentations, associated with a peculiar semistupor and paralysis of the sixth nerve, and (4) oculomotor or group dysfunctions may be studied through their connections with the vestibular apparatus.

There follow interesting sections on development and the relation of vision to motion and of motion to color.

The next section of the paper deals with different nervous mechanisms variously susceptible to edema and to general or local pressure. The central visual tract is especially susceptible to edema. The central vestibular mechanism is affected by an increase in the general intracranial pressure (at times long before papilledema develops). On the other hand, the oculomotor nerves are paralyzed only by direct pressure.

Swelling of the nerve head from suppuration around the optic foramen from cold in the head and consequent spheno-ethmoid disease causes neuritis, as evidenced by swelling of the nerve head, central scotoma with rapidly failing vision and pain on pressing the eyeball back into the orbit and on movement of the eyes. The author believes that in these cases opening of the sphenoid is called for even without positive roentgen observations.

In spite of the general opinion to the contrary, the presence or absence of papilledema may be of some localizing value because different types of lesions originate papilledema in different ways. In tumor, the brain is subjected to gradually increasing pressure; in vascular disease, localized areas of cerebral tissue suffer from nutritional disturbances, and in septic disease, the infected area is subjected to an invading edema. Brain abscess and septic infarcts offer acute insults to cerebral tissue. Both are largely edematous processes. The visual fields in edema of the brain caused by infection, abscess, septic infarcts and retrograde thrombophlebitis are as follows: (1) they are rapidly changing; (2) the hemianopic indentation often precedes by days or even weeks outspoken neurologic signs sufficient to localize the lesion, and (3) the field defect is not recognized by the patient. The author believes that cerebral edema involving the homolateral optic tract causes the visual defect. Cerebral edema may last for months. Convulsions, transient paralysis and hemianopia in blood stream infections may be the manifestation of a small infarct and associated cerebral edema. Eagleton emphasizes the importance of the taking of daily fields by a trained technician and states that while today the diagnosis of brain abscess is seldom made before the supervention of compression, there is a long interval in which a provisional diagnosis should be made.

There follow case reports as follows: (1) sinus thrombosis associated with localized meningitis in septic infarct, and an associated cerebral edema causing paralysis of the face and arm and complete hemianopia relieved by the removal of the focus of infection in the bone and the evacuation of cerebrospinal fluid from the cortex and base; (2) abscess of the temporosphenoidal lobe, each exacerbation of suppuration being preceded by sudden hemianopic indentations with a complicating streptococcus meningitis; (3) cerebellar abscess and sinus thrombophlebitis with hemianopic indentations of fields, and (4) sinus thrombosis followed by cerebellar abscess and purulent meningitis of the subdural space above the temtorium associated with hemianopic indentation of field from edema of the visual tract of the homolateral side. In the fourth case death occurred from meningitis. A postmortem examination was made.

Eagleton then discusses paralysis of the nerve and comments that while a suppurative lesion of the brain may cause sensory disturbances, only direct pressure

can cause motor paralysis. The third nerve is paralyzed only when an exudate of inflammation completely fills the arachnoid space in which it runs. The fourth nerve is seldom affected and when it is the sign is pathognomonic of pathology in the posterior fossa. Exudate in the cavernous sinus may involve both the third and the fourth nerves. A case of combined cerebellar abscess with paralysis of the fourth nerve and recovery from abscess of the temporosphenoidal lobe is presented.

The sixth nerve presents several susceptible points after it leaves the pons. Abductor paralysis can occur in suppurative lesions of the posterior or middle fossa or in cavernous sinus thrombophlebitis.

In localized meningitis of the bulbar cisterna one gets first middle fossa symptoms, then posterior fossa symptoms and then a bilateral hemianopic indentation of the field. If to these is added a homolateral, contralateral or bilateral abductor paralysis, exploration of both the petrous tip and the bulbar cisterna is indicated. A case of localized bulbar cisternal meningitis following caries of the apex of the petrous pyramid without labyrinthitis is reported with postmortem observation.

The oculomotor group can be tested by vestibular tests, which will frequently give evidence of abnormality in the absence of individual paralysis.

Eagleton presents eleven conclusions.

CHAMBERS, Syracuse, N. Y.

STUDIES ON SOME MODERN METHODS OF TREATMENT OF SCHIZOPHRENIA. F. VON HORANSZKY, Arch. f. Psychiat. **84**:181 (July) 1928.

The author reports experiences with three methods of treatment: 1. Continuous narcosis was employed, the method being that described by Schaefgen. The patient is given a mixture of a derivative of allyl-isopropyl barbituric acid, phenobarbital and bromides; beginning with 5 drops of somnifen, 0.05 Gm. of phenobarbital and one tablespoonful of a 10 per cent bromide solution three times a day, the doses are increased gradually until the patient receives 15 drops of somnifen and 0.1 Gm. of phenobarbital three times a day, leaving the bromide solution unchanged. The continuation of the administration of these drugs depends on the condition of the patient; in the patients treated by the author, the duration was from five to sixty-three days. When the medication could not be given by mouth because of resistiveness, the bromides had to be left out and the phenobarbital and somnifen were given by injection. Sixteen patients were treated. They represented different types of schizophrenia. Two showed a definite improvement following the cessation of the treatment (in both cases, however, the condition was episodic schizophrenia). In some of the others there was some improvement during the treatment, but a return to previous symptoms after its cessation; in some of the cases there was no improvement. In general, one can say that when there are states of excitement the treatment brings about a sedation and better contact with the patient. In pronounced hallucinosis, the hallucinations disappear during the treatment but return after its cessation. The best results were obtained with cases of a periodic nature, whereas in paranoid schizophrenia there was no influence.

2. Sixteen patients were treated with endocrine gland extracts. Among the patients selected for this purpose were ten male and six females. General hypoplasia of the sex organs, infantalism, disturbances of menstruation, etc., were regarded as indications for this type of treatment. In only one case was there definite improvement. The other patients showed either transitory improvement or no change.

3. Sixteen patients were treated with metal salts (manganese and beryllium). Here, too, the different types of schizophrenia were represented; some of the cases were of recent onset, in others the psychosis had existed for some time. The duration of the treatment was from four to ten weeks, and the patients were given from twelve to thirty injections, receiving altogether from 30 to 100 cc. of the solution. The results were as follows: there were no complete recoveries; in two cases there were good remissions; in three cases there was slight improvement; in all other cases there was no change.

In summing up the results of the three methods of treatment, the author comes to the conclusion that so far nothing definite can be said in favor of any of these methods of treatment. In his material, consisting of forty-eight cases, only two patients showed an apparently complete recovery; both of these, however, had cases of periodic schizophrenia, and had had spontaneous remissions before. In most of the other cases the improvements were transitory and not definite. None of the methods, however, has been definitely injurious, and with care, should not produce any untoward symptoms. Further attempts should be made and the material should be chosen carefully.

MALAMUD, Foxborough, Mass.

FEVER AND TACHYCARDIA IN CEREBRAL TUMORS. G. R. LAFORA, Arch. de neurobiol. **8**:223 (Sept.-Oct.) 1928.

Lafora has seen some cases of cerebral tumor with persistent tachycardia (104 to 112), and less frequently slight fever (37.5 to 38.5 C.) (99.5 to 101.3 F.). After reviewing the literature on the subject and discussing the results of physiologic experiments on the thermic centers of the brain he reports a case.

A girl, aged 13, who was without tuberculous history and in good general health up to the beginning of the symptoms of cerebral tumor, presented first muscular cramps in the right hand, followed a year and a half later by slight jacksonian convulsions of the right hand, with paresthesia when playing the piano. Symptoms later increased until the arm was involved. About this time, the patient suffered from typhoid fever from which she recovered completely. After a month, there was occasional vomiting and slight fever. At the time of examination she was emaciated, anorexic, with frequent vomiting and a little headache on some occasions, and jacksonian attacks in the right arm that sometimes were also felt in the leg of the same side, more rarely in the same side of the face. There was marked awkwardness in the use of the right hand, the patient frequently dropping objects. The temperature was from 38 to 38.5 C. (100.4 to 101.3 F.); the pulse rate, 108. The right arm and hand were usually pale and cold.

The neurologic examination disclosed paresis in the right arm and hand with hypesthesia, hypalgesia and considerable diminution of the discriminating faculty and of the articular and muscular sensibility. The tendon reflexes were somewhat diminished in the right arm and normal in both legs. Cranial percussion was painful in the left temporoparietal area. There was no edema of the papilla of the left eye, but slight venous congestion was present. There were normal pupillary reactions with slight mydriasis. No other symptoms were present. Careful analysis of the cerebrospinal fluid and blood gave negative results for tuberculosis and syphilis. Several tests for tuberculosis as well as clinical explorations were negative.

A diagnosis of cerebral tumor was made. Operation disclosed a tumor, measuring 8 cm. in diameter and 5 cm. thick, situated in the upper portion of the left parietal lobe. Part of the tumor was situated on the sensory center for the arm, and there was slight involvement of the center for the leg. The tumor was easily removed, but the patient was in such a weakened condition that she died eighteen hours after the operation. The tumor was a fibrosarcoma with numerous, irregularly distributed, softened zones surrounded by fatty cells and small caseated foci filled with cell detritus. Neither plasma nor giant cells were present. Lafora concludes that this case confirms the existence of cortical centers that produce fever through vasomotor alterations.

NONIDEZ, New York.

THE PREFRONTAL SYNDROME OF AMNESIA. G. DEMORSIER, Encéphale **24**:20 (Jan.) 1929.

This article, based on experiment and observation, concludes with the following statement: Physiologic experimentation and anatomicoclinical observations prove that the deficiency of the two prefrontal lobes produces necessarily an amnesia, an apathy and a peculiar type of irritability. In man this amnesic function is generally resident in only one of these lobes. The concord of arguments from the morpho-

logic, the physiologic and the pathologic spheres proves that the association pathways relating the prefrontal lobes to the posterior cortical areas cross in the posterior portion of the corpus callosum, where lesions effect the same troubles as do those of the prefrontals. The integrity of this pathway conditions the memory acquisitions which permit adaptation of the individual to the exterior world.

In evidence of these conclusions two detailed case histories are included. One may be briefly reviewed as typical of the clinical syndrome. A man, aged 54, became suddenly obtunded, somnolent, confused, disoriented and blind. Presently he became able to recite clearly all the details of his life prior to the onset of the illness; on the other hand, he seemed to show not the slightest knowledge or concern of what was going on about him. He was totally disoriented in time and space, although awake and not presenting evidence of confusion. If not spoken to he would lie apathetically indifferent alike to his surroundings and his malady. Occasionally, outbursts of humor would occur — now ironic, now almost virulent. On examination of the brain there was found symmetrical bilateral softening of the prefrontal lobes extending back along the course outlined, to the knee of the corpus callosum, without other pathologic changes. Such an isolated pathologic process affords a better study of the "pure" type than does a neoplasm, in which pressure at a distance is so often a factor to confuse.

There exist a certain number of cases of unilateral lesion of the prefrontal lobe without the syndrome of memory defect. On the other hand, there are clearly reported many cases of such unilateral impairment in which the syndrome is as well marked as in bilateral lesions. Statistics on these facts can be interpreted only by assuming that one frontal lobe can suffice for preservation of the mnemic function, but that individual determiners are at work in different persons, so that at one time the left may be predominant and at another the right.

There is included considerable experimental work in outline concerning the course of the pathways from the frontal lobes through the corpus callosum to the occipital region.

ANDERSON, Kansas City, Mo.

OCULAR LESIONS IN CERTAIN CASES OF TUMORS OF THE ANTERIOR FOSSA. MENINGIOMA OF THE CRIBRIFORM PLATE OF THE ETHMOID. M. J. CHAILLOUS, Ann. d'ocul. **165**:881 (Dec.) 1928.

After referring to Cushing's work on meningiomas arising from the olfactory groove, Chaillous reports two of Cushing's cases, one a meningioma of the cribriform plate with optic atrophy on the side of the lesion and papilledema on the opposite side, anosmia and mental disturbance. The patient in the second case suffered from bilateral anosmia, atrophy of both optic nerves and severe mental disturbance. In this case there was slight displacement backward of the clinoid processes.

He then gives a detailed report of the case of a patient seen by him in consultation with Vincent. There was severe headache, marked mental disturbance and retrobulbar neuritis followed by atrophy of the right and then of the left optic nerve. Anosmia developed and a meningioma finally appeared in the nasopharynx. In spite of two cranial operations and removal of part of the meningioma of the anterior cranial fossa, the patient died of meningitis. In grouping the symptoms found in cases of meningioma arising from the region of the cribriform plate, one frequently finds more or less complete atrophy of one nerve with edema of the other nerve. In a second clinical group, one may see retrobulbar neuritis with diminution of visual acuity not accompanied by objective signs. Later on, the optic nerve becomes atrophic and assumes the characteristics of a primary optic atrophy. In the last clinical type the diminution in visual acuity may be accompanied in some cases by central scotomas. The mental condition of the patient sometimes prevents a complete systematic examination of the eyes.

In the first clinical type, with atrophy of the optic nerve on one side and papilledema on the other side, diagnosis of intracranial pressure should immediately

be considered. If the ocular symptoms of retrobulbar neuritis are manifest, the etiologic diagnosis is more difficult. He suggests particular study of patients who have retrobulbar neuritis which excessively affects the eyes, particularly when the ocular troubles are accompanied by anosmia and mental disturbances. Study of the olfactory sense should not be neglected.

With these points in mind and with the aid of roentgenograms, one is usually able to make a diagnosis which permits operative intervention before the patient becomes blind or insane.

BERENS, New York.

THE CEREBROSPINAL FLUID SUGAR IN NERVOUS DISEASES AND ITS DIAGNOSTIC SIGNIFICANCE. A. E. KULKOW and M. E. TARNOPOLSKAJA, Arch. f. Psychiat. **85**:672 (Nov.) 1928.

The authors have examined the cerebrospinal sugar in a large number of cases of disease of the central nervous system, and compared it in some of these cases with the blood sugar. The method of the sugar determination was that of Hagedorn-Jensen. They took as the normal value for cerebrospinal sugar from 50 to 60 mg. per cent (average 56), and for blood sugar from 100 to 130 mg. per cent (average 115). They sum up the changes in cerebrospinal sugar in organic nervous disease as follows: (1) In the case of bacterial meningitis a diminution of the sugar occurs, this being pronounced in the epidemic, cerebrospinal and tuberculous meningitis (from 9 to 34 mg. per cent). They found that an increase of the sugar in the spinal fluid during the course of the disease can be taken as a favorable prognostic sign. The reverse, however, is not always true. (2) Tabes and paresis show a normal sugar content. (3) In cerebrospinal syphilis and congenital syphilis, a slight increase of the sugar was found; a similar increase was found in some cases of tumors of the brain. (4) The acute forms of epidemic encephalitis show an increased spinal fluid sugar, whereas the late postencephalitic parkinsonians show normal content. (5) An increased sugar was found also in the fluid of multiple sclerosis, as well as in cases of epilepsy and in some cases of polyneuritis.

There is no constant relation between the sugars of the blood and those of the cerebrospinal fluid, and although usually the normal relationship is found, the cerebrospinal fluid sugar may oscillate with simultaneous changes in the blood sugar and vice versa. An increase of pressure of the cerebrospinal fluid is not always accompanied by an increased sugar content. The introduction of some chemical substances (neoarsphenamine) into the cerebrospinal canal may cause a change in the sugar content.

MALAMUD, Foxborough, Mass.

REMARKS ON KLEPTOMANIA. FRITZ WITTELS, J. Nerv. & Ment. Dis. **69**:241 (March) 1929.

The author describes a series of cases of kleptomania in an effort to discover the underlying psychopathologic mechanisms. In most of the cases he finds a lack of an effective "super-ego" and a defiance of authority. In both men and women, stealing, he believes, satisfies sexual libido in a perverse fashion. One girl showed other compulsions as well—washing, scrubbing and scouring—which the author interprets as an overcompensation for her anal impulses.

Because of the skilfulness of the pickpocket's fingers, the author goes so far as to invest them with libido. In addition, he discovers, besides the instinctive impulse to do the forbidden thing, a masochistic pleasure in its dangerous consequences. Like mountain climbing, it is a form of sport, but besides this is the symbolic significance of the robbery, which it is the duty of the psychologist to discover. In one small boy, the motive was the longing for love and tenderness; in another case, it was revolt against paternal authority, which the analyst finds resting on a castration complex.

In the author's opinion, women are more subject to kleptomania than men, and male kleptomaniacs present signs of physical and psychic femininity. Their sex

life is rather meager, and he finds that the efficiency of the endocrine system is likely to be disturbed. The "super-ego" of the child who steals is underdeveloped, i. e., his antisocial instincts are not sufficiently repressed. Punishment gives him satisfaction, and successful stealing gives him the joy of victory. He does not want to be changed. To hold him to treatment requires a consummate art, and he is apt to escape at the moment when the physician thinks he is successful in his treatment.

The author concludes with excerpts from the memoirs of J. J. Rousseau, who derived a positive satisfaction from stealing from a woman and accusing an innocent girl whom he really loved. The constant feeling of guilt was a form of suffering that he really enjoyed, and the act of calumny became a sexual act.

HART, Greenwich, Conn.

EXPERIENCES WITH CHORDOTOMY. GILBERT HORRAX, Arch. Surg. **18**:1140 (April) 1929.

Horrax reports his experiences with eight cases of chordotomy. Of the eight patients, six were distinctly benefited by the operation and two were apparently not relieved except for a month or two. One death occurred as the result of postoperative pulmonary complication. In five patients the operations were performed because of pain due to a previous syphilitic infection. Three of the patients were relieved; in two of them the chief pain was due to some form of abdominal "crises." The two who were not relieved had the typical "lightning pains" of tabes. In one of the latter, the faulty result may be attributed to an incomplete tract section, but in the other the anterolateral columns were apparently completely incised as shown by the resultant analgesia. Of the three patients who did not have syphilis, in one the pain was due to a chronic hypertrophic spondylitis, in another to a recurrent tumor of the spinal cord, and in the third to erosion of the spine and pressure on the posterior roots by a hypernephroma. In all of these, relief was obtained by the operation.

With regard to technic, Horrax believes that chordotomy is best done with a pointed knife, carrying the point inward to a depth of 4 mm. Some of these cases were done under local anesthesia, and as a result the author believes that the most superficial fibers in the tract represent the lowest, that is, the sacral levels, and that the fibers for the segments progressively higher lie respectively deeper in the tract. This is merely a deduction from the fact that apparently the greater the depth to which the cord incision was made, the higher the level of the analgesia on the opposite side of the body became. If this is true, it probably means that the fibers of these tracts, starting from the lowest levels and working upward after crossing in the cord, take a position in the opposite anterolateral tract medial to those which have entered just previously, thus pushing these earlier entering fibers outward toward the periphery of the cord.

GRANT, Philadelphia.

THE RÔLE PLAYED BY CEREBRAL CAPILLARIES IN THE PATHOGENESIS OF GENERAL PARALYSIS. WILLIAM MALAMUD and K. LOWENBERG, J. Nerv. & Ment. Dis. **69**:121, 1929.

The therapeutic effects of malaria and other therapeutic agents in paresis have led investigators to renew the search for the pathogenesis of the disease, and to attempt to correlate histologic pictures with the clinical syndrome. Paresis is a special type of syphilis, namely, syphilis of the parenchyma of the brain, and the problem should express itself in the attempt to identify the components of the whole syphilitic process that determines the invasion of the parenchyma. Meningeal syphilitic involvement may occur commonly without any symptoms for a long period.

Six cases are presented showing varying proportions of meningeal and parenchymatous involvement. Some of the cases seemed to accentuate the close relationship between involvement of the cortical vessels and of the parenchyma. The authors think that their studies support the observations of Spalz that parenchymatous disturbances are always associated with vascular involvement in the

parenchyma, and that when the disease remains in the vessels of the meninges without affecting those of the cortex there is little or no involvement of the cortex itself.

It seems that disease of the capillary network of the parenchyma is associated with reduction in the resistance of the blood-cerebrospinal fluid barrier. Observers have found that in cases improved by malarial treatment a reduction of inflammatory process in the mesodermal elements occurs. One case presented by the authors seemed to indicate that, both qualitatively and quantitatively, the reaction of the parenchymatous tissue depends on the involvement of the capillary network; hence, it is possible that a primary disease of the meningeal capillaries may lead to meningitis, while that of the cortical capillaries may lead to paresis.

HART, Greenwich, Conn.

THE INFLUENCE OF THE PROPRIOCEPTIVE NERVE ENDINGS OF THE HIND LIMBS ON THE POSITION OF THE FORE LIMBS. J. PI-SUÑER and J. F. FULTON, Arch. de neurobiol. **8**:176 (July-Aug.) 1928.

The experiments were performed on cats. The animals were decerebrated after previous section of the left sciatic nerve and of all the branches ending on the muscles of the flexor group with the exception of the semitendinosus. The external cutaneous, internal saphenous, obturators and nerve for the sartorius were also sectioned. The abductor and adductor muscles of the limb were rendered useless through resection of their tendons and of the tensor of the femoral fascia. The same operation was carried out on the right limb, but the innervation of the adductor muscles was preserved.

The results of these experiments are summarized by the authors as follows: 1. Forced flexion of the knee against the resistance offered by contraction of the quadriceps in decerebrated cats causes extension of the fore limb of the same side and flexion of the fore limb of the opposite side, accompanied by extension of the other hind limb (Phillipson). 2. Strong traction of the isolated knee tendon or pressure on the same tendon without any tension of the muscle causes the same responses on the other hind limb. 3. A weak faradic current applied on the central stump of the sectioned sciatic nerve or one of its components also causes the same type of response. 4. Strong traction of the semitendinosus or other of the flexor muscles of the knee does not exert any influence on the position of the fore limbs and the unaffected hind limb. 5. The flexors of the knee show active tendon reflexes after the animal recovers from operative shock. 6. These observations may furnish an orientation for the interpretation of the mechanism of motor coordination in quadrupeds. 7. The pathways of the reflexes described are probably predominantly intraspinal.

NONIDEZ, New York.

OBSERVATIONS ON THE MENOPAUSE: II. THE EFFECTS OF VARIOUS OVARIAN PREPARATIONS ON SYMPTOMS OF THE MENOPAUSE AND ON BASAL METABOLISM. JOHN T. KING, JR., with the Technical Assistance of Ellen Patterson, J. A. M. A. **91**:1423 (Nov. 10) 1928.

There is need of more careful study of the effects of ovarian preparations in the menopause. No objective criteria are available, but patients under treatment and those not under treatment can be carefully followed and other substances than ovarian preparations can be tried. Symptoms of the menopause are not static, and this probably accounts for the multiplicity of favorable results. In this study forty-two patients were treated with whole dried ovary, corpus luteum by mouth or follicular extract subcutaneously. Twenty-three others were treated with bromide or phenobarbital or both and these were used as controls. Complete disappearance of symptoms under treatment was recorded as plus 100 and lesser improvement as plus less than 100 down to 0; if symptoms were made worse a scale of 0 to —100 was used. Phenobarbital gave a result of plus 33.7 per cent. Corpus luteum showed only 19.6 per cent, which is no better than one would

expect to find in the natural course of symptoms during the menopause in an untreated patient. Whole ovary was only slightly better, yielding 22 per cent.

Follicular extract, the most recent preparation, gave the least satisfactory results of all—only 9.1 per cent. There was no significant alteration in basal metabolism.

The authors conclude that corpus luteum and follicular extract are useless and that bromide or phenobarbital or a combination of the two is distinctly helpful, probably not in a specific way but rather as mild sedatives.

CHAMBERS, Syracuse, N. Y.

EXPERIMENTAL CARBON MONOXIDE POISONING. A. M. GRUENSTEIN and N. POPOWA, Arch. f. Psychiat. **85**:283 (Oct.) 1928.

Six animals — four cats, one dog and one rabbit — were subjected to the effects of carbon monoxide. The concentration varied in the different experiments from 2 per mill to 3 per cent. Some of the animals were subjected to the gas only once, and others as many as twelve times, care being taken to remove the animal from the gas as soon as it became unconscious. The animals lived from one to six months after the experiment, at the end of which time they were killed and examined histologically.

The results of the observations are summed up as follows: The effects of carbon monoxide consist of clinical and histopathologic changes. Clinically, paralyses of temporary or permanent types can be observed. Histologically, the following changes are seen: a diffuse degenerative process in the nerve cells with apparent predilection for localization in the extrapyramidal system; changes in the cells of the anterior horns of the spinal cord, and a reactive neuroglial proliferation and vascular change. In some cases isolated foci of softening and hemorrhages occur. The clinical symptoms seem to be due to diffuse, rather than localized changes, whereas some of the most marked histologic changes can occur without giving rise to clinical symptoms. As far as can be seen, these changes are practically the same as those that occur in the human being.

The authors do not agree with the view that the electivity of the reactions is due to difference in vascular supply, but lean more toward the pathoklitic theory of Vogt. Just how the carbon monoxide causes these lesions remains questionable. The authors think that the most probable mechanism is that of an immediate chemical effect of the carbon monoxide on the nerve cells.

MALAMUD, Foxborough, Mass.

LEPROSY AND PSYCHOSIS. M. PRADOS, Arch. de neurobiol. **8**:161 (July-Aug.) 1928.

A case is reported of typical tubercular leprosy in a woman, aged 33, in whom an acute psychosis suddenly developed after ten years of the disease, during which there was only a mental crisis of short duration. The beginning of the psychosis was characterized by psychomotor agitation with logorrhea, verbigeration, auditory hallucinations, a feeling of anguish and occasionally of terror, and marked depression. The motor agitation slowly disappeared, but the incoherent logorrhea persisted. The patient became highly irritable. The crying spells ceased and on some occasions seemed to have been superseded by a feeling of euphoria. Catatonic symptoms were present at this stage and the patient spent most of her time sitting on the floor with her head between her knees. This stage of the psychosis was followed by another characterized by marked eroticism and flight of ideas. Later, the patient was unable to leave the bed, lost weight rapidly and became negativistic. She fell in profound stupor, awakening occasionally, and died five months after the onset of the mental symptoms. Unfortunately, an autopsy could not be performed.

The case described is similar to a case reported by Jakob and Meggendorfer. Although it is impossible to affirm that the mental condition of the patient was directly due to the infection with Hansen's bacillus, Prados is inclined to believe,

from his own observations in other cases of leprosy and the reports in the literature, that in certain cases of this disease there is a mental syndrome in which infection with the bacillus may play an important rôle.

NONIDEZ, New York.

A CONTRIBUTION TO THE PROBLEM OF THE FUNCTION OF THE CORPUS CALLOSUM IN ANIMALS. W. SELETZKY and J. GILULA, Arch. f. Psychiat. **86**:57 (Dec.) 1928.

The authors carried out experiments on ten rabbits and six dogs. In seven rabbits the whole corpus callosum was cut through in the midline. In the other three, only the anterior portion of it was sectioned. In one dog the whole corpus callosum was cut through, and in two the anterior, and in three the posterior parts. They summarize the results as follows: (1) Section through the corpus callosum is followed by certain disturbances which are apparently due to sections of the anterior or middle parts, whereas section through the posterior portion causes no evident disturbances. (2) The symptoms conditioned by such section are as follows: (a) disturbance of sensation of all extremities, of isolated ones or of the body alone; (b) the gait becomes ataxic; (c) there are involvements of the auditory and visual sensory apparatus, and of taste and smell; (d) some of the animals show cataleptic reactions (allowing the limbs to remain in whatever positions they are placed, no matter how uncomfortable); (e) psychic disturbances: excitements, fear reactions, inhibitions, apathy, etc.; (f) sometimes the animals tend to run around in circles for some time following the operation. (3) All these disturbances disappear after a shorter or longer period of time, leaving the animals apparently without symptoms.

MALAMUD, Foxborough, Mass.

APRAXIA IN WALKING AND STATIC ATONIA. LUDO VAN BOGAERT and PAUL MARTIN, Encéphale **24**:11 (Jan.) 1929.

Two cases are presented with autopsy reports. One was a case of abscess of the left prefrontal lobe; the other of hemorrhagic glioma of the white substance of the left prefrontal lobe. In both instances there had been apparent a particular maladroitness of the inferior member, whereby there occurred confused movements of the usual alternate movements of walking in the erect position, as well as a sort of "anarchic nonuseful pawing motion." The muscular power of the separate segments of the lower limbs was relatively well conserved and the apraxia could not be accounted for by a paresis. Even when supported the patients were unable to place their limbs properly. These symptoms have been previously described by Gerstmann and Schilder and explained as dependent on a prefrontal lesion, or an extension into the anterior part of the corpus callosum. Since a bilateral motor apraxia is recognized as affecting the face, head and speech, the question is asked as to why it is more difficult to admit such a bilateral apraxia of the inferior extremities. These symptoms, amounting to astasia-abasia, when coexisting with preservation of the primary motor function, can serve to locate the lesion as prefrontal.

ANDERSON, Kansas City, Mo.

EARLY DETERIORATION IN CEREBROVASCULAR DISEASE OF COMPOUND PATHOGENESIS (SYPHILIS AND OTHER FACTORS). A. BOSTROEM, Arch. f. Psychiat. **86**:1 (Dec.) 1928.

The author discusses a series of cases of mental disorders in which either the history or the serologic examinations, or both, left no doubt as to the presence of the syphilitic infection, but in which there were also other factors that contributed to the development of the psychosis. He summarizes the syndrome as follows: The patients, mostly men, who give a history or show reactions pointing to syphilis, show their first mental abnormality at about the age of 40. The first symptom generally consists in an outbreak of excitement. During the course of the disease they develop definite organic disturbances of the emotional tone, with

excitability and at times compulsive crying spells. As time goes on, there is a development of a cloudiness of consciousness and emotional flattening with occasional outbreaks of excitement. It leads to intellectual deterioration with a special disease of the retention memory, loss of interest in the environment, untidiness and dilapidation of thought. As a rule, focal signs are rare. The serologic examinations may or may not be positive. Pathologically, one finds a disease of the cerebral vessels which is rather difficult to differentiate from arteriosclerosis, and which approaches very nearly the picture of endarteritis syphilitica.

MALAMUD, Foxborough, Mass.

MUSCLE TONE IN DECEREBRATE RIGIDITY. LOYAL DAVIS, Arch. Surg. **18**:1687 (April) 1929.

The pattern of rigidity following decerebration depends on the level of section of the brain stem and on the influence of other reflex activities. The pattern and degree of rigidity in a decerebrate animal are not changed by removal of the cerebellum. Lasting patterns of rigidity in flexion occur in decerebrate animals in which the labyrinth has been destroyed, and are unchanged by removal of the cerebellum. Coexisting normally distributed tone, patterns of rigidity in extension and flexion, crawling, climbing and springing are produced in animals in which a segment of the basilar artery has been isolated between two ligatures some distance apart, and in animals decerebrated at a relatively high level. Removal of the cerebellum does not affect the pattern or degree of these activities. The cerebellum as a whole inhibits, in a general way, the tonic labyrinthine reflexes. Removal of the cerebellum permits the regular and forceful occurrence of rhythmic reflexes in decerebrate animals. Tonic labyrinthine reflexes produce a change in the physical property of muscle which permits it to be purely mechanically stretched while other reflex adaptations occur. Muscle tone may be produced by reflexes other than stretching.

GRANT, Philadelphia.

THE TENSION OF THE EYEBALL IN INCREASED INTRACRANIAL PRESSURE. SALVATI, Ann. d'ocul. **165**:919 (Dec.) 1928.

Salvati made ten clinical observations on patients with increased intracranial pressure. He also made experimental observations on monkeys. After trephining the skull of monkey no. 1, salt solution was injected into the lateral ventricle under increased pressure. The tension of the eyeball did not increase, although papilledema developed. This experiment was repeated in a second monkey, 15 instead of 20 cc. of physiologic salt solution being injected. The tension remained at 18 mm. of mercury. After the third experiment (20 cc. injected), papilledema developed and the intra-ocular tension was 18 mm. of mercury. In experiments 4 and 5 on monkeys, 10 and 15 cc. of physiologic salt solution injected into the lateral ventricle produced papilledema, but in monkey no. 4, the ocular tension was 19 mm. of mercury, and in monkey no. 5, 20 mm. of mercury.

The author concludes that intra-ocular pressure shows no change in the course of intracranial hypertension and that there is no relation between the pressure of the cerebrospinal fluid and the intra-ocular pressure.

BERENS, New York.

PYRETOTHERAPY WITH "DMELCOS" VACCINE IN FOUR CASES OF PARESIS. J. M. VILLACIAN, Med. ibera **585**:1 (Jan.) 1929.

Since it has been stated that the beneficial effects of pyretotherapy in paresis are due to the production of fever rather than to an antagonistic action between the treponema and the injected plasmodium, Villacian has tried to produce fever in patients through injections of "Dmelcos" vaccine, used with success by Sicard, Haguenau and Wallich. Four cases are described. The pyretogenic effect of the injections was evident in every case. In two of the patients temperatures between 39 and 40 C. (102.2 and 104 F.) were produced, while in the other two the

temperature seldom exceeded 39 C. No shock phenomena, lipothymia or collapse, said to occur in patients into whom injections of paludic blood have been made, were ever observed in any of the cases described, and other undesirable effects due to the vaccine were equally absent. In two of the cases the specific treatment for syphilis was given simultaneously with the pyretotherapeutic treatment without retarding the fatal evolution of the disease. Villacian concludes from these observations that fever alone is not the cause of the remissions obtained after infection with the malarial agent.

NONIDEZ, New York.

INTRACRANIAL SARCOMATOUS TUMORS OF LEPTOMENINGEAL ORIGIN. PERCIVAL BAILEY, Arch. Surg. **18**:1359 (April) 1929.

Bailey tries to show, by the description of his series of these rare tumors, how true sarcomas of the brain may arise, and follows their structure and evolution. Although some of the tumors reported were not proved by necropsy to be primarily intracranial he has used these doubtful cases to point out the structural characteristics by which sarcomas may be distinguished from gliomas. Although all these tumors must arise from the leptomeninx or its derivatives, their microscopic structure is exceedingly diverse. The various types which they assume for the most part emphasize the kinship of the pia-arachnoid with the extraneural connective tissues. Only the occurrence of melanomas might be considered to argue in favor of a neuro-ectodermal origin of these tumors, in case the melanin-bearing cells differentiate directly from the meningoblasts. However, in the absence of any direct embryologic observations on the development of the meningeal melanophores, it is possible that they migrate into the pia-arachnoid as they are said to do into the choroidal coat of the eye.

GRANT, Philadelphia.

HYPNOTISM AND CONDITIONED REFLEXES. B. BIERMANN, J. f. Psychol. u. Neurol. **38**:265, 1929.

The problem of hypnotism in the light of conditioned reflexes raises three questions: 1. What is the physiology of the hypnotic state? 2. What does the physiologic mechanism of hypnotization (hypnosogenous factors) consist of? 3. What is the relation of hypnotism in man to that of so-called animal hypnotism? Biermann answers these three questions as follows: 1. The hypnotic state in man and in higher animals is conditioned on a partial inhibition of the cerebral cortex. The various degrees and forms of hypnosis depend on the extent and localization of the inhibitory process. 2. The physiologic mechanism of the effect of hypnosogenous factors consists of the development of the process of internal inhibition. This process may become effective through uniform feeble as well as through strong stimuli (rarely in man). Besides this the process of inhibition may be brought about by the use of conditioned inhibitory stimuli. The last factor (suggestion of sleep) is most important in man, so that hypnosis in man is a conditioned reflex partial sleep. 3. It is only by the latter (conditioned reflex partial sleep) that hypnosis in man differs from that in animals.

KESCHNER, New York.

NORMAL MENSTRUATION AND GASEOUS METABOLISM. F. G. BENEDICT and M. D. FINN, Am. J. Physiol. **86**:59 (Aug.) 1928.

A report is here presented of well controlled metabolic studies relative to the effects of menstruation, in the case of an extremely good human subject, over a period of twelve years. The authors found menstruation definitely to exert a lowering effect on metabolic rate. Thus the average heat production for thirty-two menstrual days was 7.5 per cent less than the Harris-Benedict expectancy, as compared to an average of minus 4.5 per cent for eighty-nine intermenstrual days. Likewise, for the menstrual days, there were distinctly more deviations greater than minus 10 per cent than for the nonmenstrual period, i.e., 34.3 in contrast to 7.8 per cent, respectively. These observations, it may be

mentioned, corroborate observations published by F. L. Gustafson and F. G. Benedict (The Seasonal Variation in Basal Metabolism, *Am. J. Physiol.* **86**:43, 1928) on the basis of a study of twenty Wellesley undergraduates.

RAPHAEL, Detroit.

VIDIAN NEURALGIA FROM DISEASE OF THE SPHENOIDAL SINUS: REPORT OF A CASE. HARRIS H. VAIL, Arch. Surg. **18**:1247 (April) 1929.

A case is reported with symptoms hitherto ascribed to Meckel's ganglion neuralgia, which were demonstrated to be due to vidian neuralgia, the result of an infection in an extensively pneumatized sphenoidal sinus. Stimulation of the vidian nerve in its canal by means of the injection of iodized oil 40 per cent caused an attack of pain located not only in the typical distribution of vidian neuralgia, as described by Sluder, but also in the region hitherto ascribed to the anterior or maxillary part of the syndrome, namely, pain in the eyeball, a contraction of the pupil with irregularity of its contour, reddening of the conjunctiva and pain in the upper canine, bicuspid and molar teeth. The case reported seems to show that it may be possible for the painful symptoms assigned to Meckel's ganglion neurosis to be relieved by treatment directed to the sphenoid disease, without any treatment directed to the sphenopalatine ganglion itself.

GRANT, Philadelphia.

THE BASAL METABOLISM OF MAYAS IN YUCATAN. G. D. WILLIAMS and F. G. BENEDICT, Am. J. Physiol. **85**:634 (July) 1928.

The authors found, from a careful study of twenty-three whites who had been in Yucatan less than eight months, that there occurred no appreciable effect on metabolic rate traceable to the subtropical climatic situation as such. A series of thirty-two male natives (Mayas) revealed an average metabolic rate of 5.2 per cent above the prediction standard obtaining for similar subjects of northern type. This relatively higher metabolic level is accounted for on the basis of some racial or constitutional factor, since the presumable climatic effect, if any, would have a reduction in rate rather than an increase. The authors are further led to state that metabolic rate in itself cannot be taken as an index of racial superiority or inferiority, this chiefly in view of the fact that for orientals, whose civilization is of a distinctly higher order than that of the Mayan, the metabolism level is a definitely lower one.

RAPHAEL, Detroit.

THE OCCURRENCE OF IRON-CONTAINING PIGMENT IN THE CEREBROSPINAL FLUID. ALEXANDER PILCZ and ROBERT STERN, Jahrb. f. Psychiat. u. Neurol. **46**:163, 1928.

Pilcz and Stern confirm the method of staining the cellular contents of the cerebrospinal fluid for iron as published by two American observers, Proescher and Arkush, in January, 1928 (*J. Nerv. & Ment. Dis.* **67**:21, 1928). The former were able to demonstrate iron-containing pigmented granules in most of the cases of paresis as well as in tabes, cerebrospinal syphilis, congenital syphilis and latent syphilis. These granules could also be demonstrated in cerebral arteriosclerosis and dementia praecox, occasionally also in epilepsy and alcoholism, and even in patients without organic changes in the nervous system. Although iron-containing pigment is found most frequently in the cerebrospinal fluid in syphilitic and metasyphilitic diseases of the central nervous system, its presence cannot be utilized in differential diagnosis in the same sense as the "four phase" reaction.

KESCHNER, New York.

THE SEASONAL VARIATION IN BASAL METABOLISM. F. L. GUSTAFSON and F. G. BENEDICT, Am. J. Physiol. **86**:43 (Aug.) 1928.

For the purpose of this investigation the authors utilized a series of twenty normal Wellesley undergraduates, ranging in age from 18 to 22. On this series, two careful metabolic determinations were carried out, as far as possible, once

each month for sixteen months, excluding July, August and September. From the data thus obtained, the authors conclude that there is a definite tendency to metabolic depression during the winter period with a subsequent rise during the spring. Thus, for two groups of five and eight students, studied continuously for fifteen and thirteen months, respectively, the averages for the readings in January were 194 and 198 cc. of oxygen consumed per minute and, for the June readings, 213 and 214 cc. From this study, also, it appears that the metabolic rate may be somewhat lowered during the catamenia.

RAPHAEL, Detroit.

ELASTIC TISSUE IN MENINGEAL FIBROBLASTOMAS SO-CALLED "DURAL ENDOTHELIOMAS." WILLIAM P. VAN WAGENEN, Arch. Surg. **18**:1621 (April) 1929.

The type cell of the meningeal fibroblastomas — so-called "dural endothelioma" — can apparently give rise to elastic tissue. This is taken as another bit of evidence of the potentially fibroblastic nature of these tumors. A description of a tumor is presented in which elastic tissue was found in abundance well away from and unassociated with the dura. The elastic tissue was in too great abundance to come from the stroma. The variations in size, shape and staining reactions and its close relationship to the tumor cells strongly suggest its origin from the cells. Coalescence of and condensation about fibroglia fibrils to form elastic tissue were observed. The designation of the so-called "dural endothelioma" by Penfield as meningeal fibroblastomas seems to be a most comprehensive one.

GRANT, Philadelphia.

AN ALTERNATING TYPE OF SYNKINESIA. JOSEPH RUSSETZKI, Encéphale **24**:251 (March) 1929.

The case is described in detail of a man, aged 28, who presented the not uncommon picture of left hemiparesis due to cerebral thrombosis in the capsular region. In addition, he evidenced an unusual degree of synkinesia. Lying on his back, he would show a whole series of interesting movements on being subjected to certain other voluntary movements. Forceful closing of the eyelids yielded a slight flexion of the left arm at the elbow; opening of the mouth did the same; forced inspiration or expiration produced flexion of the left arm and left toes. A well maintained contraction of the diaphragm, such as in coughing or yawning, gave marked arm flexion. All these movements were rhythmic, succeeding in a regular manner and indicating a central area under the control of volition.

ANDERSON, Kansas City, Mo.

SIGNIFICANCE OF A DILATED PUPIL ON THE HOMOLATERAL HEMIPLEGIC SIDE IN CASES OF INTRACRANIAL HEMORRHAGE FOLLOWING HEAD INJURIES: REPORT OF SEVEN CASES. CARL W. RAND, Arch. Surg. **18**:1176 (April) 1929.

Rand reports seven cases of intracranial hemorrhage in which the hemiplegia and the dilated pupil were homolateral, intracranial hemorrhage being found on the same side. He believes that hemorrhage is usually greatest on the side of the dilated pupil, but that at times apparently bitemporal decompressions are indicated in such cases, as either a dilated pupil on the homolateral side or a hemiplegia on the contralateral side are too important to disregard in looking for hemorrhage. He emphasizes the importance of avoiding the use of drugs which contract or dilate the pupils in the control of these cases. If such drugs are used this important pupillary phenomenon may be masked.

GRANT, Philadelphia.

CONSTITUTIONAL STUDIES OF STAMMERERS. L. M. TIHOMIROVA, Collected Papers. Moscow State Neuro-Psychiatric Dispensary **1**:218, 1928.

One hundred stammerers, eighty-six men and fourteen women, were studied in the Neuro-Psychiatric Dispensary. Kretschmer's system of measurements and his classification of constitutional types were used. On examination, forty

patients were found to be of asthenic type, twenty-four of the asthenic-athletic, seven of pyknic and the rest of mixed or dysplastic type. Most of the women belonged to the latter two types. The asthenic and asthenic-athletic types are the most common constitutional physical types associated with stammering. The pure pyknic types are the least frequent.

KASANIN, Boston.

THE CAPACITY OF RESISTANCE OF THE CENTRAL OPTIC PATHWAYS TO MECHANICAL PRESSURE. HIRSCH, Ztschr. f. Augenh. **57**:592, 1925; Ann. d'ocul. **165**:611 (Aug.) 1928.

Hirsch reports two cases of cyst of the hypophysis, followed by remarkable return of vision after operation. He calls attention, however, to the fact that the tracts are slightly resistant to infection.

RETINAL BLOOD PRESSURE, SITTING AND LYING. SALVATI, Ann. d'ocul. **165**: 917 (Dec.) 1928.

Salvati, after making ten observations, concludes that blood pressure in the retinal vessels is not influenced by posture, except in anemia, when, as Magitot has pointed out, the volume of blood is diminished and the local blood pressure may be elevated in the recumbent position.

BERENS, New York.

ANATOMIC CHANGES IN THE LABYRINTH SECONDARY TO CEREBELLOPONTILE AND BRAIN STEM TUMORS. S. J. CROWE, Arch. Surg. **18**:982 (April) 1929.

Crowe made careful serial sections of the auditory apparatus in a case of cerebellopontile angle tumor and in a case of tumor involving the brain stem. As a result of study of these sections, he confirms the experimental work of Wittmaack that the atrophic changes in the spiral ganglion, peripheral nerve fibers and organ of Corti in these two cases were secondary to a lesion of the auditory nerve central to the cochlear ganglion, and that this ganglion is an exception to the wallerian law of degeneration.

GRANT, Philadelphia.

THE EFFECT OF NEUROPHIL DRUGS ON THE FIDDLER CRAB, UCA PUGNAX. OSCAR W. RICHARDS, Biol. Bull. **56**:28 (Jan.) 1929.

A reversal of the usual motor responses of the legs of the male fiddler crab, *Uca pugnax*, has been observed when the animals have been in solutions of picrotin, strychnine, phenol, pilocarpine or veratrine in sea water. Camphor increases the irritability of the animals. Atropine, apomorphine, morphine, codeine, caffeine and digitonin are without effect on the crabs. The results of these experiments are discussed in relation to those made with other arthropods.

COBB, Boston.

A CASE OF OCULAR SYNDROME OF HYSTERIA. GIOVANNI LODDONI, Ann. d'ocul. **165**:357 (May) 1928.

Loddoni reports the case of a girl, aged 16, who worked in a silk factory and was struck in the eye by a piece of iron which broke off from a bobbin. Following this slight injury, there were repeated hemorrhages from this eye and two small foreign bodies were removed. She also had convulsive seizures of short duration, blepharospasm, amblyopia and amaurosis. She developed strabismus and rotary nystagmus. All the symptoms were attributed to hysteria.

BERENS, New York.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

Regular Meeting, April 2, 1929

LOUIS CASAMAJOR, M.D., *President, in the Chair*

AN UNUSUAL TYPE OF INVOLUNTARY MOVEMENT, RELATED TO THE DYSTONIA GROUP. DR. S. BROCK.

Clinical History.—E. S., a man, aged 24, an American-born Jew, was first observed from June to September, 1925, and then in November, 1928. He was abroad in the interval. In 1916, at the age of 12, the patient first noticed tremor in the fingers of the right hand, which made writing difficult. About six months later, choreiform movements appeared in the left leg. The movements of the left lower extremity lessened, but the leg was dragged and walking became difficult. Periods of decided improvement occurred. In 1922, he was still able to run and do ordinary gymnasium work; then the right lower extremity became involved so that it was "thrown outward" in walking. By 1923, locomotion was seriously disturbed. In the period from June, 1924, to March, 1925, some improvement was reported; thereafter he "relapsed." He then complained of cramps and also of tremors in the right lower extremity.

Following is the neurologic status in November, 1928. It differs so little from studies made in the summer of 1925 that the few differences will be merely alluded to.

Neurologic Examination.—The gait was bizarre, due to the occurrence of tonic spasms in the quadriceps extensors, abductors and external rotators of the thighs, more especially the right. It was at times reminiscent of the movements seen in Huntington's chorea. The right lower extremity was thrown outward and externally rotated with the knee joint stiffly extended. With the outward thrust, the toes of the right foot, especially the big one, dorsiflexed, so that the foot "landed" somewhat on the heel. In 1925, there was hyperreflexion at the right knee and hip joints, with a peculiar bowing over of the body toward that side in gait. There was a definite twist of the pelvis, with elevation of the right hip. The left lower extremity was first rotated externally and then slightly rotated internally, with a tendency toward eversion of the foot. Both knees exhibited genu recurvatum. The right upper extremity swung freely; the left less so. He walked backward much better than forward, due to the fact that the hamstring muscles which initiate backward stepping were practically uninvolved in the spasmodic contractions.

In standing, one noted spasm of both quadriceps extensors. Occasionally there was external rotation of the left thigh with projection of the glutei on the outer aspect of the left buttock. At times these tonic contractions also occurred in the muscles of the back of the thigh. The spasms occurred mainly in the quadriceps and glutei groups, in the dorsiflexors of the right toes, and to a lesser extent in those on the left. The quadriceps spasm produced bilateral genu recurvatum. Some of these spasms were painful; others were not. There was little tortipelvis and no myostatic postural fixation. Except for an infrequent slight spasm in the left triceps the trunk, neck and upper extremities were not involved.

In sitting, few involuntary movements were to be seen. The main one was a tremor-like external and internal rotation movement of the left thigh, on which was superimposed at times a larger abductor (gluteus maximus) spasm. Crossing the left knee over the right caused its disappearance, and was his position of election.

In recumbency, the same features were noted as in sitting. The left lower extremity was rotated outward. A general hypotonus was noted. When relaxed, it was possible to elicit the tonic spasm in the quadriceps and other muscles by

passive overflexion of the joints of the lower extremities. Under these circumstances, the hyperhypotonus changes seen in dystonia were elicited.

He ran better than he walked. There was, however, a peculiar overflexion of the legs on the thighs, especially on the right; both feet tended to become everted.

The fundi and the fields of vision were normal. The pupils were equal and reacted well to light and in accommodation. There was a slight, questionable ptosis of the right eyelid. There were distinct nystagmoid movements on extreme lateral gaze. At previous examinations this had amounted to actual nystagmus in lateral and vertical directions. The external ocular movements were normal. There was no strabismus or diplopia, but at times there was a slight tendency of the right eye to turn inward. The functions of the fifth nerve were normal. The corneal reflexes were active. There was facial asymmetry, but no paralysis. Hearing was normal. The movements of the palate, tongue, trapezius and sternocleidomastoid muscles were normal. Speech was unaffected.

The outstretched right hand revealed a parkinson-like tremor, which was more evident when the palms faced each other. At times a parkinsonian cupping of the right hand was apparent. The same hand showed a tendency to overpronation when the upper limbs were upstretched. There was no atrophy and no paresis, and the reflexes were normal.

The abdominal and cremasteric reflexes were lively and equal.

There was no paresis or atrophy in the lower limbs. The knee reflexes were present, but somewhat diminished. In 1925, a distinct pendular element was noted in both knee reflexes, especially the right. The ankle jerks were active and equal. There was no Babinski toe reflex.

There was no ataxia on finger-to-nose or heel-to-knee tests; and no adiadokokinesis. A slight rebound phenomenon was noted in the left upper extremity. There was no dysmetria. When the patient's eyes were closed, the outstretched right hand sank a little. There was no past-pointing.

Sensation was everywhere intact.

Mental: This patient was unusually intelligent and at the time of presentation was studying for a Ph.D. degree, having obtained an M.A. from Columbia University. He was, however, definitely euphoric, and at times loquacious.

General Examination.—Except for a depressed pigeon-breast deformity of the sternum, the results of the physical examination were entirely negative, as were also all laboratory tests.

Comment.—The case presents dystonic, involuntary movements, largely confined to the lower extremities and almost entirely elicited by other voluntary effort, such as walking. Emotion increases their intensity. I cannot account for the remarkable restriction of the dystonic movements to volitional innervations definitely subserving other purposeful activities. In a large experience with dystonia musculorum deformans, I have never encountered this phenomenon before, nor am I aware of any description of such a case. In the present stage of knowledge, it would be difficult to speculate on this point.

The parkinson-like tremors (right hand; left lower extremity) and nystagmus have been noted before in undoubted cases of dystonia musculorum deformans.

The generalized hypotonia at rest, the previously observed pendular knee reflexes and possibly the genu recurvatum speak for the involvement of cerebellar pathways. The nystagmus also points to involvement of structures other than the basal ganglia. The pathologic process underlying this condition is obscure. However, the history of remissions, the nystagmus, the evidence of involvement of the cerebellar pathway and, of special importance, the definite euphoria might well suggest an atypical multiple sclerosis.

DISCUSSION

DR. I. S. WECHSLER: I find it difficult to discuss this case. I presume one can discuss it from the point of view of the clinical manifestations, of pathology and perhaps from that of nosologic classification. To take the last first, I do not believe that one can put this patient into any definite group, nor does he belong

in the group of the primary dystonias. At best he shows dystonia of a postural nature without the spontaneous movements, and one might classify him with the myostatic variety of dystonia musculorum deformans. Apparently, the condition has remained stationary all these years; at least there have been enough remissions to keep it fairly stationary. While it is true that there is such a disease as Oppenheim's, one sees a great many cases in which the dystonia can be regarded only as a symptom, particularly as a sequel of encephalitis, and I think that one should look on most dystonias as symptoms rather than diseases. As regards pathology, multiple sclerosis has been mentioned, but clinically I see little to justify such a diagnosis. Whether the patient has a degenerative process of that disease or one of a similar nature is difficult to say. Whatever the process, it has manifested itself in a parkinsonian tremor, in a cerebellar element of movements and in dystonia. At best one can make a symptomatic diagnosis, possibly localize the multiple lesions anatomically and say that they are on the basis of some degenerative process.

DR. LOUIS CASAMAJOR: This is a most peculiar case and I think that one cannot do much more than guess at the etiologic factors concerned in it.

A CASE OF A KORSAKOFF SYNDROME. DR. IRVING SANDS.

Clinical History.—K. S., a white woman, aged 40, was admitted to the Montefiore Hospital on Dec. 28, 1928. Her family history was unimportant. She was born in Austria and came to America in 1906. She married at the age of 17, and had two healthy children. One miscarriage resulted at the end of the third month of pregnancy, due to physical overexertion. Her husband died six years after the marriage. She remarried five years later. She has had no serious illness and has led an apparently normal life. She would take a glass of beer with meals. During the past year, she has taken two or three highballs a day, but she has never been intoxicated.

The present illness dates from December, 1927, when she began to vomit in the mornings. This lasted for about three weeks. Appetite became poor and she lost considerable weight. In May, 1928, she went to the country to recuperate. She felt well until September, 1928, when she complained of numbness of the fingers and toes. The toes would turn blue. In October, 1928, she complained of pain in the legs and forearms. Then there occurred increasing numbness and stiffness in the extremities. In December, 1928, there was considerable impairment in walking and in the use of the hands, so that at the end of the month she could not use the hands nor could she walk. During the latter part of December, she became confused, showed decided impairment in cerebration and became forgetful.

Examination.—Physical examination revealed a well developed and poorly nourished woman showing dilated capillaries of the face, and flatness and impaired breathing at the left base of the chest. The liver was enlarged and was palpable at the umbilicus. There was atrophy of the muscles of all extremities, with bilateral foot drop and wrist drop. There was paresis of the extremities. All deep reflexes were absent. Tactile sensibility was lost in the hands, forearms and in the lower extremities. Hyperesthesia was found in the soles and in the palms. There was diminution of vibratory sensibility and loss of muscle joint sensibility in the limbs. There was tenderness of the muscles and nerves in the limbs, especially in the calves. The gag reflex was lost. The pupils were unequal, the right being larger than the left. There was nystagmus in the horizontal plane when looking either to the right or to the left. The urine showed albumin. A blood count showed: 3,900,000 red cells, 65 per cent hemoglobin, and 9,400 white cells, of which 78 per cent were polymorphonuclear leukocytes. The Wassermann reaction of the blood and spinal fluid was negative.

Mentally she was confused, disoriented and showed memory and retention defect. She fabricated and confabulated freely. She said "I saw you yesterday. This is Sloane Hospital. I came here to visit somebody. This is 1998. I was here yesterday, visiting."

DISCUSSION

DR. MOSES KESCHNER: There may be a question whether or not we are having prohibition in this country, but there is no question that the Korsakoff syndrome is not so common as it was prior to the enactment of the prohibition laws. It is of interest that, long before Korsakoff published the report of his cases (1887), von Hösslin described an identical syndrome following toxemia of pregnancy with or without polyneuritis. It was after this that Korsakoff described the syndrome, which he designated encephalopathia psychotica toxemica. Korsakoff, more than his followers, emphasized that polyneuritis was not an essential component of the syndrome. The Korsakoff syndrome is observed in carbon monoxide poisoning and other toxic conditions; it is common in dementia paralytica, in drug addiction, in typhus and in other organic cerebral conditions due to toxemia or infection. A point to be borne in mind is that alcoholism is not the only condition in which the syndrome is observed. Dr. Sands' patient was extremely euphoric on admission, so much so that with the irregular, somewhat sluggishly reacting pupils some observers at first thought there might be an element of syphilis in the case. Serologic examinations, however, disproved that theory. It is also of interest to note that while most patients showing a Korsakoff's syndrome due to alcohol do not improve, this patient is beginning to show evidences of improvement, at least as far as her mental condition is concerned.

DR. MICHAEL OSNATO: The typical Korsakoff's psychosis is occasionally associated with chronic pulmonary tuberculosis in the toxic exhaustive states. Typical pathology has been reported, and this should be added to the list of toxemias and chronic intoxications of which Dr. Keschner spoke.

DR. IRVING SANDS: I have seen a typical Korsakoff reaction in a young woman suffering from gonorrheal arthritis. I have not come across a similar case. The syndrome lasted six weeks and then disappeared.

AN UNUSUAL CASE OF MUSCULAR ATROPHY FOR DIAGNOSIS. DR. A. H. RUBINOWITZ (by invitation).

A girl, aged 14, was perfectly well until three years before presentation, when pain began in the right foot, and spread gradually as far as the right groin. The severest pain was in the right ankle and the right groin. The pain was described as very sharp and more or less continuous; occasionally she would limp. In 1927, a diagnosis of flatfoot was made and she wore arches, without relief. As late as 1928, at Bellevue Hospital, the results of an examination were completely negative; x-ray pictures of the bones of the lower extremities were normal and neurologic examination also gave negative results; the patient was discharged with a diagnosis of hysteria. Subsequent observations are inconsistent with this diagnosis. At the time of presentation she showed a symmetrical atrophy of the right lower extremity, and the right thigh and leg were definitely smaller than the left. In addition, neurologic examination showed a commensurate loss of motor power; especially on passive examination all the movements in the right lower extremity were definitely weaker than on the left. The reflexes were practically normal. There were no sensory disturbances. Roentgen examination of the bones gave negative results. Lumbar puncture gave negative results. Manometric studies were normal. The reaction of degeneration was tested; the muscles gave a normal response to faradism and the galvanic reaction was normal. Roentgen examination of the spine showed some arthritic changes, although with no destructive changes in the bone. There was no fibrillation.

DISCUSSION

DR. WALTER M. KRAUS: This case is easy to classify and hard to discuss. One can say definitely that it is a segmental atrophy, with a reservation about the word atrophy. Within the last two years I have been much interested in cases of this sort and reviewed the literature of cases of hemiatrophy since 1859.

My conclusion was that these cases are really allied to the dystrophies, and are due to disorder of the central beginnings of the visceral nervous system. Consequently, I believe that the term hemidystrophy should be used, though there is a question whether such a change in terminology would prove acceptable.

The number of reported cases since 1859 was eighteen, and the age of onset was between 16 and 20, except for one case which began when the patient was 4. This case is also interesting because it involves only the leg. In the reported cases the condition sometimes involved the arm and leg on the same side, and in some cases there were crossed forms of the disease, so that one cannot prophesy here which of the other limbs may be affected next.

The occurrence of pain interested me greatly, for syndromes involving the thalamus and hypothalamus have been reported with considerable frequency; bearing in mind the central origin of dystrophy, it would not be unexpected to find that pain from the thalamus is associated.

Dr. Rubinowitz's report, however, and my own examination of the patient at the hospital, did not elicit any added evidence to substantiate the central origin of this pain.

DR. HENRY A. RILEY: Were any careful studies made of the blood pressure in the lower extremities, particularly with the view of determining whether there was any difference in the blood pressure in the two legs? Also, were studies made of the surface temperature and of the sweating reactions and were any of the special drugs, such as pilocarpine, used?

DR. THOMAS K. DAVIS: Was there any evidence of sensory dissociation? I am struck by the fact that it is difficult to think of this case as fitting into any definite syndrome, but the proportional atrophy reminds me of how one sometimes sees that in syringomyelia. I wonder if syringomyelia may yet develop in this patient.

DR. BYRON STOOKEY: At Bellevue this last fall, I saw a patient who had had pain for two years in one extremity, the cause of which was undetermined in spite of numerous examinations. That patient showed symmetrical atrophy of that extremity as a result of the pain. This patient showed no real finer atrophy in one particular group of muscles, but a general symmetrical atrophy of the entire extremity, and the outstanding symptom, as I understood Dr. Rubinowitz, was pain. I am wondering if the atrophy was not secondary to the pain, that this patient has for a period of time saved this leg, and the atrophy is only secondary if the pain could be relieved. The patient I have in mind was kept in bed, and was there for about a year; finally, when nothing further could be done for him, it was decided, without knowing the cause of the pain, to make an attempt to get rid of it by cutting the anterior lateral column on that side, through a hemilaminectomy. This was done with complete relief from pain, without any impairment of motor function. I have followed that man from time to time, and the atrophy which he had has gradually disappeared, so that one extremity is practically the size of the other. I am wondering if this may not be a comparable case. The etiology of the pain I do not know.

DR. E. D. FRIEDMAN: Would it not be possible to make a biopsy on some of the muscles for the purpose of seeing whether there are any changes in the muscles themselves? All are familiar with the syndrome of polymyositis, or dermatomyositis. There are cases in the literature in which the polymyositic process was limited to one limb. I recall a reference by Wertheim-Salmonsen to localized forms of polymyositis. In view of the symptom of persistent pain, I should strongly urge a biopsy to determine the presence of a chronic exudative process in the muscles.

DR. RUBINOWITZ: In answer to Dr. Riley, we did take the blood pressure, but in the upper extremity, not the lower. There was no change in the surface temperature in the right leg as compared to that of the left. The patient, as far as sweating is concerned, did have sweating more on the right leg, but probably it involved all the extremities; even the upper perspired considerably.

The most interesting part of the case was that we found no objective sensory disturbances. This makes the diagnosis difficult. We realized that if she were later to develop sensory disturbances it would help one to understand the pathology.

Dr. Stookey mentioned the possibility of the atrophy being secondary to pain. The patient was not confined to bed very much, and there was little difficulty with motor power; we did not think that there was enough to cause as much atrophy. I have seen patients confined to bed considerably more than this one was, without causing as much atrophy as in this girl.

Dr. Friedman suggested a biopsy. In polymyositis or dermatomyositis I understand that something is usually felt locally in the skin or under the skin; not only did that not occur in this case, but the particular sort of disturbance involved symmetrically the whole limb and no other portions of the body.

In regard to the possibility that this condition is thalamic, I should have mentioned that the child, at the age of 3, had an injury to the head; she fell from a height of about half a story, and was unconscious for from fifteen to twenty minutes; she had no convulsions. Possibly at that time there was some intracranial lesion, but in the absence of any objective sensory disturbances indicative of thalamic involvement, it is difficult to place this case in that category, except on the general principle that dystrophies come with thalamic involvement. The view that possibly some dystrophies are associated with definite endocrine disturbances was suggested; we studied the case from this angle, and examined roentgenograms of the skull. They showed no involvement of the sella turcica or of the clinoid processes. Metabolic studies were normal for sugar metabolism and sugar tolerance; so I think that this case is a progressive hemiatrophy, for the time being localized in one leg. Possibly the other limb may be involved later.

HODGKIN'S DISEASE WITH DIABETES INSIPIDUS. DR. NATHANIEL E. SILVERMAN.

Clinical History.—A carpenter, aged 39, born in Hungary, had a past history without significance except for influenza in 1920, which confined him to bed for four days. The illness began in January, 1926, with a painless swelling in the right side of the neck. In December, 1927, the patient complained of a "tired feeling" in the right arm, dry cough, difficulty in swallowing and a loss of 20 pounds (9 Kg.). In addition to this, he experienced pain in the lumbar region in the morning, which wore off during the day.

Physical examination at that time revealed matted glands in the right supraclavicular region with a few soft axillary and inguinal nodes. There was mediastinal widening and bronchovesicular breathing in the upper lobes. The liver was palpable for two fingerbreadths below the costal margin. The spleen was not felt. Repeated blood counts at the time were within normal limits. Roentgen examination revealed a mass in the superior mediastinum about the size of an apple, pushing the trachea to the left. The appearance was suggestive of Hodgkin's disease or lymphosarcoma. Roentgen therapy was instituted on Jan. 11, 1928, cross-firing over the anterior and posterior mediastinum and the right supraclavicular area. Biopsy revealed typical Hodgkin's disease.

Reexamination of the chest, one month later, showed marked diminution in the size of the superior mediastinum, which appeared almost normal at the time of presentation. There was marked relief from symptoms with disappearance of the glands in the neck.

The patient felt perfectly well from February, 1928, to August, 1928, when after a head cold he began to complain of supra-orbital headaches and insomnia. Roentgen studies of the sinuses revealed cloudiness of the right antrum. At this time he complained also of slight blurring of vision.

After five roentgen treatments over the base of the skull, the headaches disappeared and the patient felt well up to about Jan. 15, 1929, when he developed excessive thirst, drank large quantities of fluids during both day and night, and was distressed by passing large amounts of pale colored urine.

Examination.—Neurologic examination gave negative results except for deep tenderness over the lumbar spine. He was again hospitalized. During his stay in the hospital, intake and output varied between 14,500 and 3,600 cc. Owing to poor cooperation from the patient these figures are probably considerably low. Roentgen studies of the skull and sella turcica gave negative results.

Reexamination of the chest showed a normal superior mediastinum. Roentgen study of the spine was normal. Urinalysis showed: specific gravity, 1.006, and negative reactions for sugar and albumin. The blood sugar was 100 mg. per hundred cubic centimeters, and the urea was 9.7 mg. Consultations concerning the nose and throat yielded negative results. Perimetric readings were normal.

Course.—Immediately following therapy with pituitary extract, the intake and output dropped to about 3,000 cc. It remained at this level, and the patient felt perfectly well.

He was discharged to the outpatient department for roentgen therapy to the pituitary region.

On discharge the patient obtained a job out of town and failed to report to the clinic. His intake and output again slowly resumed the previous high level.

Comment.—On going over the literature, I was unable to find a description of this syndrome associated with Hodgkin's disease, but I did find many cases showing involvement in other parts of the brain and spinal cord. Not only are the lymph glands and spleen involved, but also the bone, skin, gastro-intestinal tract, thyroid, heart muscle, kidneys, blood vessels, meninges and other organs. Pathologically, there is hyperplasia of the reticulo-endothelial system, and it has often been demonstrated that no tissue is exempt from invasion. A comprehensive paper on this subject was written by Dr. S. Ginsburg of the Radiotherapy Department of Montefiore Hospital, in which a series of neurologic complications in Hodgkin's disease was reported. The protean manifestations of this disease were stressed, and while, in the majority of cases, involvement of the nervous system is usually secondary to extensive disease in the lymphatic system, this is not necessarily the case, and involvement with Hodgkin's disease may arise primarily anywhere in the body. Involvement of the nervous system at times may give symptoms for long periods before that in the lymphatic system, and so give rise to great diagnostic difficulties. In these cases the ready response to irradiation not only will be a great aid in diagnosis, but will possibly prevent permanent neurologic damage.

DISCUSSION

DR. S. PHILIP GOODHART: One point which was emphasized by Dr. Ginsburg in his study from the Montefiore Hospital is important; that is, all tissues are likely to be invaded, and disease of the nervous system is not uncommon. That is one of the things that is of rather recent introduction into the literature. This case brings out a point of importance: the likelihood of the invasion of the nervous system long before there is any glandular involvement. It is important, from the standpoint of paraplegia, and those instances in which, even before there seems to be demonstrable enlargement of the glandular structures, the nervous system is invaded.

The question as to just what the lesion in this case is, so far as the brain is concerned, is speculative and interesting. It opens the question whether the tissue of the posterior part of the pituitary may be involved; whether there is an involvement of the structure itself, and a malignant cellular involvement of tissue mingled with the normal tissue of the gland, or whether there is the usual situation, that is, nodular enlargement of the base of the brain. It is possible that the last is the case, although one would wonder, if this is true, why no other structures in the areas adjacent to the pituitary are involved. One interesting feature is the visual symptoms, the nature of which was not entirely decided in this case. It is not at all improbable, however, that there is a secondary involvement of the pituitary, and perhaps an involvement of a neoplastic nature, since one knows that in cases of neoplasm of the pituitary, the development of the syndrome peculiar to that organ may be preceded for a number of years by diabetes insipidus.

ACROMEGALY, PERIPHERAL NEURITIS AND DIABETES. DR. J. H. LEINER.

Clinical History.—A man, aged 61, had a history that dated back seven years, when he noticed he became weak. The weakness was progressive, and compelled him to leave business. In 1923, he noticed that he had to obtain larger sized clothing—collar, gloves, shoes and hats. He had some trouble sexually with erection, but libido was maintained. He also noticed diminished vision in the right eye, for which he went to a clinic, where he was told he had a cerebral tumor. About this time, he began to show generalized symptoms of cerebral neoplasm. He had severe headaches in the frontal region, and marked vertigo. He also showed polyuria, polydipsia and diplopia at times, and a reversal of sleep. He slept in a chair during the day, but could not sleep at night. During the early part of 1929, he went to the hospital and presented a rather advanced type of acromegaly.

Examination.—He showed unequal pupils, the right larger than the left, and diminution in hearing. He also showed a generalized hyporeflexia; the muscles were flabby and he showed a correspondingly diminished amount of muscle power. There were no pathologic plantar phenomena. He began to have uncinate fits and would show perversions of taste and smell. The optic nerves showed no atrophy. There was some constriction of the fields of vision.

Mentally, he was unreliable and noncooperative. He showed a marked macroglossia, large spade hand and large feet. He had a basal metabolism of +13 on one occasion and +19 on another. The blood count was normal; the blood pressure showed a hypotension of 120 systolic and 80 diastolic. The x-ray plates of the skull showed a thickening of the bones of the vault; the sella was enlarged, and there was no erosion of the floor. The sinuses were greatly increased in size.

Comment.—This case is one of acromegaly due to a condition of adenoma, which is beginning to invade the base of the brain, showing temporosphenoidal involvement, with uncinate attacks. He shows, in addition, diabetes mellitus and peripheral neuritis which is secondary to the diabetes.

DSCUSSION

DR. L. DAVIDOFF: After having seen this patient, I think that no one will question the diagnosis of acromegaly. The points of especial interest are the presence of diabetes and the neuritis of which Dr. Leiner spoke. The relation of diabetes to acromegaly is not merely coincidental. Other physicians have found it many years ago, and in a series in Boston we were able to show that 25 per cent of 100 consecutive cases of acromegaly showed either a frank diabetes or a tendency toward it in the sense of showing a certain amount of glycosuria at times with an increase in the blood sugar. The difference between the diabetes in acromegalic patients and true diabetes mellitus is difficult to prove. At the same time, this patient may be an example of this very thing, the difference between this type of diabetes and the other. This man is known to have had severe diabetes mellitus clinically for at least six years. He is, as Dr. Leiner said, wholly incapable of cooperation, and never attempts to follow a diet. We had him under observation for six months in Boston and attempted to treat him dietetically, as adequately as possible, but in spite of this he always broke through his diet and ate tremendous quantities of candy and fruit. He has continued to do that from then until now. I am sure that the physicians at Montefiore will agree with me that he will eat anything and everything at any time of the day. I am sure that no ordinary diabetic person could have stood this and be alive today. Acromegalic diabetic patients occasionally die in diabetic coma; I think, also that Dr. Leiner was not correct in saying that they do not respond to doses of insulin; they do, but perhaps not as actively as ordinary diabetic patients.

Neuritis is associated with ordinary diabetes mellitus. In acromegaly, however, one frequently observes neuritis without glycosuria; another explanation is possible in these cases. For example, it is known that acromegalic persons are prone to develop hyperostoses. The lantern slides shown illustrate the skeletons of two diabetic patients; the one of a normal person, was shown for comparison. The curvatures in the spines, which one frequently sees clinically, are brought out

in these skeletons. They are not due to a general increase in the size of the bones, but to an actual hyperostosis in the individual vertebrae. Another slide was shown to illustrate two vertebrae from the largest of the three skeletons on the previous slide, and two normal vertebrae for comparison; the tremendous exostosis of the bone can be noted. In another slide were seen three of the upper thoracic vertebrae in the same case; they are fused together by the proliferative bony process, and the vertebral canal is almost completely obliterated. The cervical region in the same patient showed a similar process to a slightly less degree. The changes in these acromegalic persons may be due to hyperostoses which impinge on the peripheral nerves, and the sensory changes may be accounted for on the same ground.

Dr. Strauss has reminded me of a patient with severe diabetes whom he sent to Dr. Cushing. The urine sugar was as high as 5 or 6 per cent. On the train he had an extremely severe headache, which was suddenly relieved by a discharge of a large amount of fluid from the nose; he arrived in Boston with a striking cerebrospinal rhinorrhea. The diabetes disappeared from that time on.

Dr. Louis Casamajor: What would be the result of the administration of insulin in a case of this sort? Was insulin tried for this man? I recognize that he is extremely difficult to manage, and that the use of insulin requires the cooperation of the patient.

Dr. Michael Osnato: On what grounds was the diagnosis of tumor of the brain made in the absence of focal signs, and particularly in the absence of any evidence of involvement of the chiasm?

Dr. J. H. Leiner: I believe that I have read that Cushing, in reference to insulin therapy in pituitary diabetes, made the statement, in discussing a case, that insulin therapy is not the remedy in this condition that it is in the classic type of diabetes. In this case insulin has been tried with no improvement in regard to the sugar. I think that that corroborates Cushing's view.

This man is becoming more and more somnolent. The uncinate attacks point to an adenoma. This explains the temporosphenoidal involvement. The progressive symptoms in this case led us to believe that we were dealing with a neoplasm.

Changes in the Spinal Cord in Anemia: A Clinicomicroscopic Study. Dr. Charles Davison and Dr. Arthur Weil.

This article was published in full in the November issue of the Archives, **22**:966, 1929.

NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY

Regular Meeting, April 9, 1929

Junius W. Stephenson, M.D., *in the Chair*

Brain Abscess Following Pyemia of Long Duration. Dr. Charles Davison.

A woman, aged 48, was admitted to Montefiore Hospital on Nov. 24, 1926, with the history that eighteen years before she had had an abortion performed which was followed by pelvic peritonitis. She was in a critical condition for four weeks. After convalescence and until October 24, she suffered from multiple large abscesses in various parts of the body. In 1913, a large abscess formed in the right temple close to the eye; the abscess was incised and drained by removal of part of the temporal bone. This was followed by projectile vomiting and severe headaches over the right frontotemporal region.

She remained in this condition until June, 1925, when she was admitted to the Mount Sinai Hospital where a diagnosis of osteomyelitis of the skull was made. Trephining of the skull in the temporofrontal region revealed extensive granulation tissue and a few drops of pus between the skull and dura. The headaches and vomiting continued and pronounced exophthalmos of the right eye soon developed, with a thin discharge from the outer angle of the orbit external to the lid.

In May, 1926, she lost consciousness for a few minutes. On regaining consciousness she had severe headaches, projectile vomiting and mental confusion. On readmission to the Mount Sinai Hospital, she showed weakness of the left hand and ataxia of the left upper extremity with slightly increased deep reflexes on the same side.

A craniotomy performed over the right frontal bone revealed an extradural collection of pus. In the right frontal lobe an abscess situated rather superficially was found, and 20 cc. of pus was removed. Following the operation there developed symptoms of meningitis and a hernia cerebri with hemiplegia and deep stupor.

On admission to the Montefiore Hospital she showed numerous scars over various parts of the body (residuals of old abscesses). There was a healed depression over the frontoparietal area and another over the forehead. The right eye bulged considerably and was rotated toward the nasal side. The right pupil was contracted and slightly irregular, and reacted well to light but poorly in accommodation; the left corneal reflex was diminished; the left corner of the mouth was drooping; the tongue deviated to the left. There was a flaccid left hemiplegia, a questionable Babinski sign and diminished vibratory sense, with loss of postural sense in the big toe on the same side. There were also a left hemihypalgesia, hemihypesthesia and hypothermesthesia with astereognosis of the left hand. The patient remained in this condition till June, 1927, when she had a convulsion, the precise nature of which could not be determined. She recovered from this the next day and was apparently well till Nov. 23, 1928, when she became confused, apprehensive and incontinent and died suddenly the same night.

All laboratory examinations gave negative results.

Necropsy—The brain and pituitary were removed. The brain weighed 1,200 Gm. The dura was adherent to the skull at the right temporal region by thin fibrous adhesions, and to the brain over the frontal, precentral and central convolutions by dense fibrous tags. The right hemisphere was smaller than the left and soft in consistency, especially over the frontal, precentral, central and temporal convolutions. On the left side there was an extensive subarachnoid hemorrhage, extending from the precentral convolution to the occipital lobe and involving chiefly the superficial branches of the middle cerebral artery. Part of the clot was found at the base of the brain between the right cerebellum and occipital lobe, involving branches of the right anterior inferior cerebellar artery, and possibly some superficial branches of the right posterior cerebral artery. On sagittal section the left hemisphere showed a clot in the lateral ventricle. The right hemisphere was cut vertically in the anteroposterior direction. The abscess made its appearance at the tip of the right temporal lobe. A vertical section at this level showed a mass 2 by 2 cm., partly firm and partly soft in consistency; in some areas this mass showed signs of organization. The structures adjacent to it (external capsule, internal capsule, caudate nucleus) were either completely destroyed or distorted. Further back, the thalamus and the lenticular nuclei were compressed and their outlines were indistinct. The right lateral ventricle was somewhat enlarged.

Vertical sections at the level of the abscess, stained by the Weil method, showed complete destruction of the cortical gray matter as well as demyelinization of the white fibers of the cortex, diencephalon, internal capsule and basal ganglia. These structures were completely distorted. With the van Gieson stain no well defined capsule, but only numerous bands of connective tissue traversing the destroyed brain tissue, were seen; these bands were more marked at the outer border where

the abscess was adherent to the dura. Some of the connective tissue bands extended deep into the brain substance and midbrain structures.

Between the dense mass of connective tissue and the damaged brain tissue there was a thin inner cellular zone consisting of lymphocytes and plasma cells. The brain tissue beyond this area consisted mainly of large foci of plasma cells, lymphocytes, gitter cells and congested blood vessels with proliferative and infiltrative changes. In some areas there were distinct nonencapsulated purulent foci.

The blood vessels, both in the vicinity of the drained abscess and distant from it, showed proliferative changes, and some showed marked perivascular infiltration. The adventitia in some vessels was enormously thickened and consisted of numerous bands of connective tissue fibers with narrow interspaces packed with plasma cells, fibroblasts and gitter cells. In some of these vessels the adventitia represented an old inflammatory condition, producing a fibrous ring around them.

The ganglion cells near the inflammatory process showed various changes from mild chromatolysis and swelling to complete neuronophagia. Milder forms of similar changes were found at some distance from the damaged tissue. The subarachnoid space remote from this area was distended and infiltrated by lymphocytes and gitter cells. The vessels in these areas showed changes similar to those described.

Microscopic sections of the brain from the left hemisphere were practically normal, except that the vessels showed slight adventitial thickening and perivascular infiltration.

Summary.—The record of a patient is presented in whom pelvic peritonitis developed, followed by pyemia, immediately after an abortion. Five years later, a retro-orbital abscess formed and was drained; this was followed by attacks of headache and vomiting for thirteen years until a brain abscess was localized in the frontal lobes near the central fissure. Following drainage of this abscess, meningitis and hemiplegia with hemianesthesia developed. The meningitis cleared up, but the paralysis and sensory disturbances remained unchanged. Two years after the cessation of drainage of the abscess the patient died suddenly, the ultimate cause of death being a subarachnoid hemorrhage, which was apparently caused by the damaged vessel walls following the infection.

DISCUSSION

DR. CHARLES A. ELSBERG: Was the abscess cavity still there, and was it filled with purulent material?

DR. CHARLES DAVISON: Yes.

DR. CHARLES A. ELSBERG: In the location where the original abscess had been?

DR. CHARLES DAVISON: Yes.

THE STUDY OF NORMALS. DR. THEODORE H. WEISENBURG, Philadelphia.

A study of normal persons was begun by members of my staff about four years ago. The results so far indicate that there are just as many variations among normal as there are in pathologic subjects. Moreover, the mistakes made by normal persons are frequently similar to those of pathologic persons. The value of such work is obvious. Its chief interest lies in the fact that if the errors of normal persons are known, clinical observations in pathologic subjects will be of more value. Moreover, these studies have so far demonstrated that there is great need for revision in methods of clinical examination, for obviously if the same errors are found in normal and in pathologic cases one cannot put as much reliance on examinations as heretofore.

Aphasia.—Our first studies were directed to aphasia. This work was done by Dr. G. H. J. Pearson and Dr. Bernard J. Alpers. The tests used by Head in the determination of aphasia were tried on a series of normal persons corresponding to the type of cases used by him in his pathologic group. Our studies showed that normal persons often give responses which are exactly the same as those obtained

in cases of aphasia. Even with people of superior intelligence mistakes are fairly frequent, and they are often exactly the same as those made by patients with aphasia.

We came to the conclusion that it is impossible to separate in any way the function of speech from intelligence, as the psychic processes are too subtly interwoven to permit such dissection. Other tests give similar results.

Since this study we have been engaged in the formulation of a series of tests for aphasia based entirely on a study of normal persons. This topic is receiving the special attention of Dr. Bernard J. Alpers, who has just finished a thesis on the "Origin and Development of Speech," with special reference to psychologic mechanisms.

Sensation.—Pearson investigated vibratory sensation and found that adolescents perceived vibration best. Decade by decade there is a slight decrease in the sensibility over the lower extremities and this decrease becomes striking after the age of 50. Many of the older persons had lost vibratory sensibility entirely. A similar decrease in relation to age was found in twenty-one cases of parkinsonism and fourteen cases of hemiplegia. There was no alteration of vibratory sensibility in the upper limbs.

Norvelle C. LaMar examined about 200 normal persons for the following types of sensibility: recognition of size, recognition of shape, recognition of passive movement of the great toe and forefinger on both sides by two methods—(1) grasping the toe above and below, and (2) grasping the toe at either side; recognition of form and three dimensions; recognition of figure writing; similarity and difference in textures.

LaMar found that normal persons make as many mistakes as patients in pathologic cases, and the same kind of mistakes. Many of the mistakes were the result of a lack of attention on the part either of the patient or of the examiner. In fact, so far as the tests for sensibility are concerned, unless one is careful in the technic of the examination, especially in regard to the psychologic reactions, the results can be utterly disregarded. This same principle would apply to any tests in which the results depend on the mentation of the patient and the examiner.

Enuresis.—Forrest N. Anderson not only studied enuretics but a normal control group. This work will appear soon in monograph form. Diurnal control of the bladder is secured at an average age of 17 months, nocturnal control at an average age of 23 months, with an average duration of training time of six months. Emotional factors constitute by far the largest group of elements in the causation or at least in the continuance of enuresis. Physical factors, while of undoubted significance in a limited number of cases, probably exert their influence through suggestion and erroneous assumption of parents and others as to being causative.

Secondary Sex Characters.—This work was undertaken by Paul E. Kubitschek who studied 725 boys ranging in age from 9 to 17 years inclusive. Each examination included, in addition to the study of the secondary sex characters, approximate determination of the size of the sex glands and external genitalia and anthropometric measurements for the determination of structural type and, in 500 cases, personality studies. This was done for the purpose of finding the presence or absence of significant correlations between the state of development of secondary sex characters, sex glands, the structural or constitutional types and personalities. On the basis of development of secondary sex characters and sexual development the subject falls into three arbitrary groups: normal, retarded and precocious. These are not sharply defined, but merge from one into the other. The following points were emphasized: the development of the pubic hair, the rate of appearance and extent of other secondary body hair, changes in voice, the size of the sex glands and so on.

This paper will soon appear in monograph form.

Work in Progress.—Dr. R. W. Waggoner and Dr. W. G. Ferguson are engaged in the study of the plantar reflexes in infants up to the tenth day following birth. The study is made by means of moving pictures. The extensor and Babinski

responses tend to be greater with a minimum stimulus and when the infants are awake, while the flexor responses are more common with the greatest stimulus and when children are asleep. This has a practical application in that the nature of the plantar response in the adult will depend on the degree of stimulation. Frequently, a flexor response is noted because of the stimulation of the flexor muscles, whereas a lighter stimulation would give a typical Babinski response. Another interesting point so far demonstrated is that the extensor or Babinski response becomes fairly stable to stimuli at the third day.

Further work is planned on sensibility, vasomotor and vegetative reactions and so on.

Conclusion.—Enough work has been done to demonstrate that clinicians could with great profit study normal persons.

(This paper was published in full in *The Journal of the American Medical Association* **93**:377 [Aug. 3] 1929.)

DISCUSSION

DR. FREDERICK TILNEY: The closing sentences in Dr. Weisenburg's paper emphasize its importance. They indicate that the mistakes which are so often made in the interpretation of clinical signs are due to the fact that one is not sufficiently familiar with what actually is the normal; consequently one is often embarrassed in deciding what may be a departure therefrom.

There are features in Dr. Weisenburg's studies which interest me particularly. I could not attempt to cover the wide sweep of his investigations as a whole. But what he has to say of aphasia is most pointed. To my mind, it is most difficult to tell when a patient is actually manifesting signs of organic speech disturbance. The point is well taken, especially in borderline conditions. The tests for speech are fundamentally and primarily tests of intelligence. There are too many neural currents converging on the mechanism which produces speech to allow one to unravel this mechanism in terms of defects in the motor, the sensory, the visual, the kinesthetic or any other single element. What is actually operating behind this process, as I see it, is a complex synthesis which goes to make up the entire composite of intelligence.

In dealing with the matter of sensation, Dr. Weisenburg came into a field in which I have been interested in the past few years, especially in my studies of Helen Keller. In observing the sensory capacities of this remarkable woman I have had the opportunity to apply many of the tests of which Dr. Weisenburg has spoken, also all of Head's methods, endeavoring to employ them with care and discrimination, not only to Helen Keller, but to a group of so-called normals. My impression is exactly that of Dr. Weisenburg.

In the normal persons it is difficult to decide what is actually normal. For example, in the vibratory sense, there are great variabilities, not only in different parts of the body, but in different tissues of the body. Some subjects are much more sensitive than others in the skin for the transmission of vibratory sensibility, and some have a peculiar lack of sensitiveness, particularly in the lower extremities. I have never observed that depreciation of vibratory sense in the upper extremities.

One interesting fact in testing Helen Keller was the keenness of her stereognostic sense. The patients, or the subjects, who were used in contrast with her were all New Yorkers, but I found on giving them coins—and these are what I used extensively although not exclusively in the tests—that none of them showed any such discrepancy in coin recognition as Dr. Weisenburg found in his subjects. This may be wholly a matter of locale.

One other point that has impressed me with reference particularly to stereognosis is the fact that education so distinctly influences it. In making the tests in which weighted cubes are used, up to this time I have never found any one with less than a high school education who will call a cube anything else than a square, and often the conditioning by education has led many of the patients to call these cubes dice.

So it is obvious that anything like a real norm in the decision with reference to sensory discrimination is, indeed, difficult to find. And for that reason I would like to sound perhaps what might be called a gentle note of warning in our attempts to establish the norm. It comes from overseas, from Dr. Leyden, a physician of the London Hospital, who after paying American physicians in the *Lancet* a pleasant compliment for their vigorous pursuit of medical studies, said that it is quite the custom now for the British people to send their students and nurses here to be educated in postgraduate medicine. One of the almost invariable results of this emigration is the return of students with sheaves of standards, standards which apply to almost everything in medicine, and which, according to Leyden, are of secondary importance only to the stone tablets brought down by Moses from Mount Sinai.

He then went on to show how variable a thing temperature is, and how it may vary from individual to individual, under different circumstances, times of day, conditions of rest and digestion. Also he brought to the front a subject which has been of great importance in past years, namely, blood pressure, and showed that in all probability it is also difficult here to arrive at any definite norm. One particular case in point which he mentioned was that of an old lady who, at the age of 77, had a blood pressure of 280, lived for the next ten years maintaining that blood pressure, and died at 87 from causes entirely dissociated, as far as could be made out, from hypertension.

He also indicated the difference in the renal threshold of sugar retention and pointed to the fact that apparently there is some difference in this threshold on the two sides of the Atlantic. It is known from studies made long ago that the Babinski reflex is a variable phenomenon, that athletes after running a race have often been known to present a typical Babinski sign (quite independent of the amount of pressure or the tension of the stimulus) which, on resting, disappeared.

Thus, while it is most desirable in every way to obtain some sort of composite picture of what the normal is, it must be borne in mind that this normal is a rather figurative factor, one which will be extremely difficult to establish, and can be established only in the hands of experts such as Dr. Weisenburg is developing, and after most extensive studies and comparisons.

I sincerely hope that Dr. Weisenburg will pursue this subject which he has so brilliantly begun, with all the energy and enterprise for which he is noted.

Dr. Smith Ely Jelliffe: I feel that I should be bringing coals to Newcastle after Dr. Tilney's thorough discussion, and so I shall resist the temptation to bring up clinical observations. In so doing, I feel sure that I shall run counter to curiosity, because it is more or less universal, not only for those of us who have lived three-score, if not three-score and ten, to seek units and definite criteria. Still, with the years it seems as though most of these attempts prove to be futile. At least, Dr. Weisenburg is telling something about the futility of what is usually called "normal." It would appear to me that some of the truths that the ancients have already outlined have strangely been forgotten.

There has been and still remains a great proclivity on the part of most of us to think in terms of one's own individuality—what one is is normal; what the other fellow is is abnormal. As may be recalled by the guest from Philadelphia, so quoth the Quaker to his wife in other language. And so as one applies the so-called rules of tests one finds just exactly what Dr. Weisenburg has accentuated, the personal note, the individual psychologic bias, the effort to see only the things one wishes to see in terms of the training more or less that has been acquired, or badly achieved, and the authorities that one wishes to follow.

Let me again, for I often have done so, call attention to an ancient soliloquy. It was a soliloquy of Protagoras, the sophist, who put the attitude, it seems to me, that is being accentuated here in contrast to the attitude that, unfortunately, all have been trained in, namely, the attitude of Plato and the Socratics. Plato and the Socratics looked for absolutes in this world; they were constantly talking about goodness and truth and normal persons. They did not hunt for them, they simply stated them. Socrates, in his devious and yet quite absolute way, told

people what was "good," just as one knows that the Methodists tell what is "good," the Baptists tell what is "good" and the Catholics tell what is "good." The Russians have their normal persons and the English have their normal persons. We have learned this evening something about "normal" persons for coin discrimination in New York, as contrasted with Philadelphia; and other kinds of normal persons.

Now Protagoras, in F. C. S. Schiller's charming invention, was interested in the subject of color blindness. That subject concerns sensory perceptions. Let us see how he approached the matter. In a sense, it seems to me, he stole much of Dr. Weisenburg's thunder. This was 340 B. C. Protagoras had a dialogue with a chap who was called Morosophus, which as may be seen to be a skilful little play of words on Sophomorus, because, after all, this search for normal persons is sophomoric, with apologies to Dr. Weisenburg. Whether one tests 5,000, 50,000 or 500,000 cases, one never scratches the surface of the possibilities of the 1,400,000,000 people on this globe. One can never get near an understanding of the "variables" in this world. What one should remember is exactly what Protagoras really said, "Every tub stands on its own bottom, and no two people ever are alike."

In talking with Morosophus—and I take this out of the body of the dialogue, without giving it all, and solely from memory—he said, "Have you ever met Xanthia the son of Glaucus?"

"Oh, yes," said Morosophus, "I remember him. But he seemed to me a very ordinary fellow, and quite unfit to aid in such inquiries."

"On the contrary," said Protagoras, "to me he seemed quite wonderful, for the wretch was actually unable to distinguish red from green. He could not tell the color of grass from that of blood. You may imagine how he dressed and how his taste was derided. But it was his eye and not his taste that was in fault. I questioned him closely and saw he could not help it—he simply saw things differently. How and why, I was unable to make out."

[Protagoras, you see, was a great deal wiser than Dr. Weisenburg.]

"How and why, I was unable to determine. But it was from his case and others like it, but less startling that I learned that truth and reality are to each man what appears to him. For the differences, I am sure, exist even though they are not noticed unless they are very great and inconvenient."

"Ah," said Morosophus, "Xanthias was diseased and you would not take the opinions of a mad man about color."

To which Protagoras said, "By calling it madness or disease you do not get rid of the differences. And how would you define the essential nature of madness or disease?"

Morosophus said, "I am sure I don't know. You should ask Esculapius about that."

To which Protagoras answered, "He is one of those gods I have never yet been able to meet."

I should like to meet the god who can tell me what is normal. Nevertheless, the problem presented is full of fascinating situations. I nearly always fume at the mouth when anybody talks "normal," because there is implied a radical sophomoric attitude toward observation. Why not talk about variables. Does one mean averages, or does one mean ideals? Is the normal state a so-called ideal into which one should like to thrust everybody, for instance, Methodists, Baptists, Catholics, the short-sighted, flat sella turcicas, or what not, because they are ideal from one's own individual point of view, or average from the standpoint of fifty-one out of one hundred? One must think in terms of variables and variations. And if, as he says, there are more errors among the normal persons, or as many errors among the normal persons as there are among the pathologic persons, then what is the difference between pathologic and normal? I remember an interesting experience I once had. Full of fervor and enthusiasm, I went to the Binghamton State Hospital when I was a youngster to learn something about psychiatry. I also went with ideas about what the ears should be like, as judged

by some of Dr. Lombroso's ideas of the normal about healthy ears, the ears of degenerates, the ears of insane people, etc., up to the Museum of Natural History and to the Zoological Garden. I measured and drew the ears of primates, etc. I was going to solve the problem of the normal ear, the ear of the degenerate person, the ear of the insane person, etc. I measured 2,400 ears, and drew pictures of them, and thought I knew all about it. I was going to tell wonderful things to the world at large in New York. I came home one morning. I rode up on the Ninth Avenue Elevated Railroad from downtown to where I lived on 71st Street, and in one car I saw more "so-called" pathologic ears than I had seen in the whole 2,400 in the institution. Then and there I made up my mind that I was barking up the wrong tree. It seems to me one is barking up the wrong tree when one wishes to draw too many inferences from psychiatric tests, from chemical measurements, from all this type of measurement work. Variations well and good, but "normals" never.

DR. WALTER TIMME: I was challenged by Dr. Weisenburg to state something regarding norms in the endocrine field. One never talks of normal persons in endocrinology. One talks of the facts seen and of theories used on the facts seen. It is simply a question of relativity, if I may use that much overworked term. If one would make an analogy, let us say between a human being and a machine (the analogy is really far-fetched), it will serve perhaps for this purpose. One might say, is a Ford car normal; is a Mack truck normal? Yes, of course, for its particular type of car each is normal. It does its work. However, a Ford car and a Mack truck will not answer the same stimuli to the same degree, nor present the same reaction either as to time or as to effect from the same stimuli; yet they are both "normal." But when one of these cars through some maladjustment of itself and within itself fails to perform what is asked of it, then it becomes for itself abnormal, irregular. And so it is with human beings. What difference whether the pubic hair remains horizontal up to the age of 18 years, or becomes pyramidal in its outline at the sixth year, so long as the person's endocrine glands are working on a basis which allows him to perform with others of his kind his daily tasks and is, by reason of that, able to compete in this world of difficulties with others. So long as he does that he is a "normal" creature; although different entirely from his "normal" fellow in external appearance. So it is a matter of relativity entirely. There is no such basic thing as normality. There cannot be. If one assumes, for instance, that there are ten glands of internal secretion, the number of different normal persons having such ten active variable glands, as Dr. Jelliffe has brought out (not positive fixed constants, but variables), according to the number of permutations and combinations with the variable factors, is almost infinite. It would cover the universe. If all the beings on the universe were normal, we would still have sufficient variables in this field to allow each one of these billion and more beings a system of his own, different from every other system, and still a normal creature.

So that with this as a basis, it is difficult to say that one will start with one particular unit as a normal unit from which any departure is an abnormality. I believe that answers the question that Dr. Weisenburg put to me particularly. One other point I would like to bring out is in regard to the actual presence of the Babinski reflex in apparently normal subjects. Babinski some years ago demonstrated two apparently normal patients with positive reactions; that is, positive Babinski reactions in people absolutely normal from his own point of view. I have since seen that to my own satisfaction, so that if one is deprived of such an actual dividing line symptom between what has heretofore been considered normal and abnormal—if that is taken away—one really must be at a loss to establish anything in a category of normality.

DR. WEISENBURG: I am grateful for the discussion. It is obvious that the speakers are impressed with the variations in normal persons and are critical of our results. That is the attitude of those of us who have worked in this field. On the other hand, may I point out that one should be equally critical of the clinical results in pathologic subjects.

So far as I am concerned, this work has made me much more careful in my clinical examinations and I find that with knowledge of the type of errors that normal persons make, my clinical interpretation is better and I am likely to make fewer mistakes. Moreover, I have learned to take into consideration the patient's psychologic aspect as well as my own.

PHILADELPHIA PSYCHIATRIC SOCIETY

Regular Meeting, April 12, 1929

WILLIAM DUFFIELD ROBINSON, M.D., *Vice-President, in the Chair*

COMPARATIVE STUDY OF HEREDITY IN MANIC-DEPRESSIVE PSYCHOSIS AND INVOLUTIONAL MELANCHOLIA. DR. C. B. FARR and DR. L. H. SMITH.

The age of onset and hereditary factors were compared in two groups of women; 50 cases of each were studied and the number was subsequently—before publication—increased to 100 of each. One group consisted of manic-depressive patients, unselected, except that those with isolated attacks in the involutional period were excluded. The other group consisted of cases of typical involutional melancholia, with one or two exceptions in the involutional period; similar syndromes of senile origin, etc., were excluded. The average age of onset in the manic-depressive group was 26.1 years, with a range of from 12 to 39 years. The average age of onset in the involutional group was 49 years, with a range of from 38 to 63 years. In the manic-depressive group, a psychotic heredity was present in 38 per cent. This included parents, grandparents, uncles and aunts and siblings. In 22 per cent, there was direct parental heredity. In this group of thirty-eight patients there were fifty-two additional psychotic relatives in addition to the thirty-eight nearest of kin already noted. This suggests a heaping up of hereditary factors. In the involutional group the total psychotic heredity amounted to 16 per cent, with direct inheritance in six and added factors in six only. Other hereditary factors, such as a history of suicide, psychopathic personality, nervous disease, alcoholism, senile dementia and apoplexy, were also recorded, each one being mutually exclusive in the order named. For example, if a patient had a frank psychotic heredity he was thus classified. If there was no psychotic heredity but a history of suicide, he was recorded under this heading. Following this, psychopathic personality was noted, etc. None of these factors were common except psychopathic personality, which occupied a prominent place, especially in the manic-depressive group. Psychopathic included all sorts of temperamental and other deviations from the normal by relatives. Seventy-six per cent of the manic-depressive patients showed "tainting" with some one of these factors. Similarly, 52 per cent of those with involutional melancholias showed similar "tainting."

Involutional melancholia shows a markedly less degree of heredity "tainting" than manic-depressive insanity, the direct parental inheritance being less than 6 per cent, not far from the "normal" average. It is possible that these figures are diminished by the less complete histories in older people, and on the other hand, increased by the inclusion of a certain number of manic-depressive cases. Full statistical details were shown in tables.

INVOLUTIONAL MELANCHOLIA: ITS PROGNOSIS. DR. C. L. MCCORD.

In the 100 cases of involutional melancholia studied, the following results were obtained: In length of onset, the greatest number of patients recovered in from one to six months (sixteen); the greatest number were improved in the same time (seven); the greatest number were not improved in the same period (twelve).

In regard to the duration of the psychosis, the greatest number of cases which ended in recovery were nine — in forty-eight months.

Minimum (recovered)	3 months
Mode	48 months
Maximum	144 months

Four patients improved in from six to twelve months; three patients improved between thirty-six and forty-two months.

Menopause past	55 cases
During menopause	19 cases
Menopause not reached	9 cases
Menopause not known	17 cases
	100 cases

Number of patients married	74
Number of patients single	26

DISCUSSION

DR. EARL D. BOND: One important point is that 46 per cent have already recovered from this severe type of manic-depressive psychosis. Of the eighteen patients who are improved and the thirty-six not yet improved, certainly four are going to recover in time, so that the recovery rate in these cases can safely be put at or over 50 per cent.

DR. EDWARD A. STRECKER: The interesting point here seems to be the high recovery rate after a long duration of psychosis. This would seem to disprove the idea of Dr. Hock and Dr. McCurdy, who insisted that one of the most important features is that the recovery rate became much smaller after twelve months. Here the higher recovery rate occurred with great frequency after three years and longer.

BLOOD PRESSURE READINGS IN MANIC-DEPRESSIVE PSYCHOSIS. DR. P. SLOANE.

Blood pressure readings were taken in an unselected series of 100 patients, including thirty-one with manic cases, fifty-four with depressions, ten with involutional melancholias and five with mixed cases. The condition of the patient at the time of the reading as well as any possible complicating physical condition was noted.

The conclusions to be drawn are that there is practically no difference in blood pressures between the manic and the depressed patients. In the older age groups one naturally finds slightly higher readings in both manic and depressed patients, but these are apparently more constant in the manic cases. It was noted that in excited patients the blood pressure often fluctuated during the reading, depending on the degree of the patient's unrest. For instance, in one case an overactive patient, aged 25, gave a reading of 150 systolic and 105 diastolic, but when she became quiet the blood pressure dropped 20 or 30 points. In some of the younger age groups, readings as high as from 150 to 170 systolic were obtained, but these must be ascribed to the mental condition of the patient at the time of the reading, since physically nothing was found. I believe that I have excluded physical causes in all cases of hypertension, but even so the results would indicate that there is no sharp dividing line between the various forms of the disease, the higher readings being distributed impartially.

In ten cases of involutional melancholia the average age was 48 and the average reading 134 systolic and 85 diastolic. In only four cases was the reading below 140, the highest reading being 160.

SPECIFIC DYNAMIC ACTION OF PROTEIN. DR. C. B. FARR and DR. K. E. APPEL.

Basing our work on Bauman's review of the literature on the specific dynamic action of foodstuffs (S. D. A.) on endocrine dysfunction, we applied this method of study to psychotic cases. Briefly, it consisted in determining the basal

metabolic rate, subsequently administering a standard protein (meat veal) and reestimating the metabolic rate at thirty minute intervals for one and one-half hours. Subsequent to our study we found that Fischer, in Germany, had reported characteristic disturbances of metabolism (S. D. A.) in schizophrenia, particularly a diminution of the specific dynamic of the S. D. A. to protein, and later a more persistent depression of the basal metabolic rate itself. We studied twenty-eight cases—twenty-six of the miscellaneous psychoses and neuroses—but found no uniform results; thus, a marked diminution of the S. D. A. was noted in the five patients, one with early uncomplicated dementia praecox, the rest with toxic or nutritional factors. Other patients with dementia praecox showed normal reactions. There is an undoubted tendency for patients with dementia praecox to show evidences of endocrine dysfunction, particularly when the basal metabolic rate is diminished. In our experience, this new and more elaborate test does not yield results commensurate with the amount of time required.

A Case of Manic-Depressive Insanity with Unusual Features. Dr. Edward A. Strecker.

The chief feature of interest in this case was the association of definite and well marked blood pressure oscillations with the variation in the presenting emotional phase. The patient had circular insanity, and the mood changed abruptly about every twenty-one days. With the onset of the active stage, the blood pressure mounted 50 or more points, and with the appearance of melancholia it dropped a corresponding amount. During the period of mania, the patient's memory for the events of the psychosis was accurate, but during the depression there was an amnesia. I think that the striking aspects of the psychosis could probably be referred to a fundamental endocrine imbalance.

Manic-Depressive Psychosis: Report of a Case. Dr. D. H. Fuller and Dr. E. V. Eyman.

A manic-depressive patient, a man, began to have mild depressions at the age of 25. At 32 he had the first severe attack of excitement, requiring hospitalization. There was another at 34, and a third at 36, when he went for the first time to the Pennsylvania Hospital. He remained at home after this, going through several depressions and mild exhilarations and was again hospitalized at the age of 42. Since that time he had been in the Pennsylvania Hospital continuously, with the exception of about three months in 1914 and one or two brief visits home of a few days each. At the time of presentation he was 49; in March, 1929, he was transferred to a state hospital.

There was nothing unusual about his depression or excitements except that in 1924, following his return from a visit to his home, there were sudden and unusual changes from one extreme of the emotional field to the other, occurring almost daily. These changes were sudden, occurring in a few minutes. On one occasion, after several days of depression, while in the yard he began talking to the sun and then asked to be allowed to return to the ward. He did so, began to pray, arose from his knees and went dancing and singing down the ward, continuing from that time until midnight in an excited, euphoric, extremely manic condition. In the continuous bath he became quiet at midnight, went to bed and was depressed and quiet until the second day after, when a similar attack occurred, lasting until 1:30 at night.

He continued to slip suddenly from wild excitement to agitated depression and back again, neither condition prevailing longer than twenty-four hours and usually lasting nearer twelve.

When depressed, he was more confused than when euphoric. A nurse reported that after being in a state of depression for twenty-four hours, this patient suddenly jumped from his chair and exclaimed in a loud voice—"I must preach the word of God," and for the next twenty-four hours he was excited and active, talked loudly and was boisterous and destructive.

This type of manic-depressive psychosis, called circular insanity, was recognized and most fully described as a distinct form of mental disease before the days of Kraepelin and was differentiated from the mania and melancholia of that period. Two forms of transition from one phase to the other, sudden and gradual, are described. In the excellent description given in the Dictionary of Psychological Medicine, the sudden transition is described as follows: "It usually takes place during sleep and in a short space of one night, complete change has been brought about. The patient who went to bed as a melancholic or manic, wakes up again as a manic or melancholic. It has been observed that the change takes place at a certain hour, which is constantly the same, at one or two o'clock, a. m."

A French physician quotes an interesting case "where the first symptom of transition from one stage into the other was a dream. When the patient passed from excitement into depression, she generally had had a sad dream, but when she passed from depression into excitement the dream was gay."

"This sudden transition from one stage to another may also take place during the day time, and then as Krafft-Ebing says, in Handumdrehen (in the turn of a hand)."

Circular insanity was described as usually ending in a chronic manic or chronic melancholic condition without deterioration. The patient presented had been excited for about four years, his quieter periods being without depression but characterized by irritability, profanity, destructiveness, and a sullen mood with rapidly ensuing euphoria when aroused. The prognosis was not good. While Kraepelin has classified these so-called circular cases in the manic-depressive group, they are of such a nature as to suggest differences either in mental mechanisms or in personality makeups.

1. *Long normal interval between two psychotic attacks.*

A patient, aged 72, who had an attack of depression at 26, recovered in six months and remained free from psychotic symptoms for forty years, when he was readmitted to the hospital at 65, with another depression which persisted for seven years. The diagnosis was manic-depressive psychosis and depression. The symptoms of the two attacks were identical. There was no history of intervening manic periods. The patient was markedly successful in business ventures in the long interval between the two attacks.

2. *Typical manic-depressive picture gradually shifting to a typical schizophrenic picture.*

A patient, aged 37, had his first psychotic attack at 22, which was followed by alternating attacks of excitement and depression over a period of ten years, during which his case was repeatedly diagnosed manic-depressive, circular type. Normal periods during the ten years totaled five years—the two longest normal periods lasting a year and a half each. There were six distinct psychotic attacks during this period. The manic attacks were at first typical in every respect, as were the attacks of depression. The diagnosis of manic-depressive psychosis was repeatedly made at each of two prominent hospitals for mental diseases. Ten years after the first attack, the swings became more frequent, less typical and schizophrenic symptoms became more and more pronounced. For the past five years, the picture has been quite typically schizophrenic. A personality study revealed mixed characteristics.

Affective and Schizophrenic Features in One Case. Dr. Kenneth E. Appel.

A case showing both affective and schizophrenic features was presented, the different symptoms discussed, and a chart presented showing contrasting symtoms helpful in a differential diagnosis. The case was interesting because the older members of the staff had thought that the patient was suffering from an affective psychosis, while the younger members thought it was schizophrenic.

The patient, a woman, aged 30, whose family history was without significance, had had convulsions at 18 months. She had always been a typical extrovert.

While teaching school she became worried over a serious accident to a suitor and had to stop teaching for several months. Later, she married this man. While separated from her husband on a vacation after two and a half years of an apparently happy marriage, she broke down following the attentions of another. She became abstracted, moody and depressed. At home she was excited and overproductive—at times silly. She thought her suitor was dead. She believed she had communicated with spirits and could read the thoughts of others with her hands on the Bible. At first she was uncooperative, antagonistic, unclean, attitudinized and hallucinated and thought the physician was causing her to have unclean thoughts. She was afraid she was bewitched. In six months she improved and cooperated on the ward. As improvement appeared she showed more manic symptoms, e. g., rhyming and punning. At the time of presentation she was cooperative and went out shopping, etc., with relatives. She still showed much fantasy, was evasive and had ideas of influence and perhaps hallucinated—but apparently she will recover from this attack.

Her prepsychotic personality was extroverted. The habitus was athletic. The psychotic personality was not an exaggeration of the prepsychotic one, but a changed one. The physician had little empathy for her. Her early behavior was odd, impulsive, negativistic and showed mannerisms, grimacing and at times smiling. It was unaccounted for by an impaired sensorium. She attitudinized and dramatized with reference to hallucinations. Often she was excited and was fearful for herself. Her attention was concentrated and turned in. Judgment and reasoning showed serious defects. She expressed absurd and magical ideas. There were ideas of reference and influence. At times she had peculiar trends of thought. There was much thought blocking and evasiveness and irrelevance. At times she was perplexed, and this inhibition seemed due to something the patient could not communicate. There were hallucinations and illusions. One would think of the symptoms mentioned as being schizophrenic.

As time went on, overactivity became more prominent, with playfulness, rhyming and punning. The fear disappeared and was replaced by a feeling of well-being. The emotions and behavior became more consistent with her thoughts. Distractibility and flight of ideas appeared.

It was thought that the fact that the first psychotic manifestations were schizophrenic and thus a reversal of her personality type reactions (namely, extroverted) made the prognosis grave. Notwithstanding her improvement, the persistence of fantasying, ideas of influence and occasional hallucinations made the physician feel that the fundamental process was schizophrenic, notwithstanding many affective symptoms.

DISCUSSION

DR. CLARENCE E. PATTEN: Is this the patient who had so much infection?

DR. K. E. APPEL: Thirteen days after she came in, she had a great many boils on the arms and hands, but she was unclean and would not allow us to bathe her without force. Handling her was a problem. We thought that the boils were the result of local friction, uncleanness, etc., rather than an internal condition, especially as they were limited to the arms and hands. The blood sedimentation at the time of entry did not point to infection.

CINEMATOGRAPH OF ACUTE REACTIONS. DR. P. SLOANE and DR. L. H. SMITH.

A moving picture of several patients who were active in an acute psychosis of a manic-depressive type was shown. Although there were several similar cases, the different causative factors in each case were striking. These reactions followed childbirth, an attack of acute epidemic encephalitis, an emotional shock and the menopause.

When the patient was 16, during the World War, her parents died, and two brothers were massacred within a short time. She was raped by a soldier. At the age of 20, she married a man who apparently did not care much for her. A the age of 25, she developed a mild depression with somatic symptoms.

Her only brother, whom she tried to protect in her great love, resented her attention and left her. At the age of 27, the horrors of her former life were revived by a letter from a relative in Russia. She developed somatic complaints, became depressed, then excited, restless, elated and even ecstatic. She showed hyperproductivity with variability of mood, confusion, visual hallucinations, illusions and lack of insight.

The only etiologic factors that could be found in connection with this case were psychogenic in origin.

HEREDITY. DR. EARL D. BOND.

Two charts are presented. The first shows four generations of a family many members of which have been in the Pennsylvania Hospital. It is remarkable that the third generation of six, all of whom became adults, left only one child. The very bad strain has almost died out. The second chart shows that the coming together of two families full of manic-depressive members has produced a child with dementia praecox.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, April 18, 1929

LOYAL DAVIS, M.D., *President, in the Chair*

THE RÔLE OF THE HYPOTHALAMUS AND MESENCEPHALON IN LOCOMOTION. DR. J. C. HINSEY, DR. S. W. RANSON and DR. R. F. MCNATTIN.

This paper will be published in full in a later issue of the ARCHIVES.

DISCUSSION

DR. LEWIS J. POLLOCK: I think that this is an exceedingly important contribution; it is the first I have known in which a specific differentiation is made between the functions of locomotion and progressive movements. In all the experimental work I have seen reported, attempts have been made to locate the center for progression, and this center has been taken to mean that portion of the nervous system from which there may originate alternate movements of the fore and hind legs. From the work of Cobb, Mella and Brown in their attempts to localize the center for progression, one may infer that by this is meant a center for walking. They have stated that it is necessary, among other things, to conserve the red nucleus to preserve progression in the animal. This seems inaccurate, because it has been shown by Davis and myself in our low tie animals, in which there is anemia of the brain stem cephalad to the exit of the fifth cranial nerves, that the animal will have active running movements, so active that at times it is impossible to pose it in a position of extensor rigidity.

Similarly, in a decerebrate animal with no alternate movements of the fore and hind legs, if the labyrinth is removed that animal will become so active in running motion that at no time does it come to rest. It will exhaust itself by the running movements. Brewer has pointed out that for the elicitation of progressive movements it is necessary to have a certain optimum of tone beyond which the movements cease. His conclusion is, I think, correct, for it is noted that in the preparations that are very rigid alternate movements of the fore and hind legs are absent. On the other hand, if the labyrinth is removed, the animals may have increased flexor tone as well as extensor tone.

It is obvious from these experiments that the red nucleus cannot both conserve the righting reflex and distribute tone. If that were true, one could not obtain

decerebrate rigidity in an animal capable of walking. That emphasizes the point Davis and I have often spoken of—that the decerebrate animal does not have rigidity alone. In our animal with a double tie, we observed walking, crawling and climbing, and the moment the animal got in a position with the occiput down it would fall and remain in the typical extensor position, as in the ordinary decerebrate animal.

It is necessary to differentiate between the type of progressive movements we have reported and what Dr. Hinsey reports as locomotion, which includes not alone progressive motion, but the ability to progress in walking.

DR. J. C. HINSEY: Dr. Pollock's remarks are very apropos. This same thing impressed us, in that we feel that we should always differentiate between coordinated movements of progression and the complete act of walking. The occurrence of coordinated movements in the fore and hind legs does not necessarily constitute locomotion. Magnus and de Kleijn also have observed alternate running movements in decerebrate animals, and they found that they never occurred when the tonus of the muscles of the extremities was minimal, but only when it was from medium to maximal. We believe that the extreme hypertonicity of which Dr. Pollock .speaks would inhibit their presence.

THE QUANTITATIVE DISTRIBUTION OF THE PYRAMIDAL TRACT IN MAN. DR. A. WEIL and DR. A. LASSEK.

This article was published in full in the September issue of the ARCHIVES, **22**:495, 1929.

In addition it was stated: In a search for an explanation of the abundant supply of the upper cervical segments with pyramidal tract fibers one may point out the significance of this region for different righting and tonic reflexes. Changes in the position of the head right the rest of the body to follow the neck to the normal posture. At the same time, a change in the position of the head elicits vestibular stimuli in the muscles of the neck in order to bring the head back to the normal position. This interplay of reflexes may be best demonstrated in decerebrate animals and in man after pyramidal tract lesions. Simons demonstrated the tonic reflex of the neck on the extremities in hemiplegia producing extension of the hemiplegic arm and leg when the head was turned toward the paralyzed side and flexion when it was turned away. The same phenomenon could be demonstrated in our dogs in which the motor cortex (sigmoid gyrus) of one side had been removed. To overcome these strong stimuli, proprioceptive and vestibular, acting on the muscles of the neck by the way of the upper cervical segments, strong opposite stimulation is necessary to protect the body from an overaction of righting and tonic reflexes following slight changes of the neck under normal conditions. This rôle of "destroying stereotyped posture" (using a term of W. M. Kraus) is assumed by the pyramidal tract. Consequently, the stronger such postural stimuli acting on a given level of the spinal cord are, the stronger the pyramidal impulses which have to overcome these "stereotyping" influences have to be. The fact that the cervical segments of the spinal cord are better supplied with pyramidal tract fibers than all the other segments may be taken as a mathematical expression of this physiologic function.

DISCUSSION

DR. S. W. RANSON: I think that the observations which have been made, in man and in the dog, of the very large percentage of fibers which end in the upper cervical segments are especially significant. When one considers the relatively large volume of gray matter in these segments in proportion to the size of the roots that emerge from them and the importance of the tonic and righting reflexes that have their centers in these segments and that act on the extremities, and puts these facts together with the enormous pyramidal innervation of these segments, it raises in one's mind the question whether or not one may not have in this upper cervical portion of the cord a center for the regulation of the automatic movements of the extremities, such as are involved in walking. In the sheep, in which the legs are used only for walking, the pyramidal tract

ends in the first cervical segment. In the mole it ends in the cervical cord. A study of serial sections of the cervical cord of the mole shows that the tract diminishes rapidly, more than one half of the fibers being given off to the upper cervical segments. In the sheep, the only influence which the pyramidal tract exerts on locomotion must be through centers situated in the upper cervical portion of the cord.

Dr. Lewis J. Pollock: I might mention a rather interesting clinical case that will bear out part of Dr. Weil's interpretation of the influence of the pyramidal tract on the inhibition of neck and labyrinthine reflexes.

As I was passing through the hospital one day, a child, aged 3 months, was lying on a table in continuous motion, and I made the remark that the child was paralyzed in all extremities, to the great surprise of the intern who was with me. It was obvious, however, that the child was moving its head from side to side, with accompanying movements in all extremities, but on holding the head firmly the movements ceased in the extremities. I do not believe that in the adult the neck has the whole inhibitory influence in the neck and labyrinthine reflexes, but in the infant it must possess considerable influence.

Dr. Arthur Lassek: The preference of upper cervical and thoracic segments with supply of pyramidal tract fibers in man might be explained as related to his erect position. But in quadruped animals the same scheme of distribution of the pyramidal tract was found, as was demonstrated in the comparative measurements of new-born animals. Furthermore, we found a similar preference in the supply of the higher cervical segments in dogs, in which the motor cortex of one side had been removed. The spinal cords were examined after from two to four weeks and stained by the Marchi method.

Myostatic Contractures. Dr. S. W. Ranson.

Myostatic contracture designates a condition of permanent shortening in resting muscle which is maintained in the entire absence of nerve impulses, the muscles having acquired, usually as a result of prolonged immobility, a new and shorter than normal resting length. In its genesis it is dependent on the nervous system and it is probably an abnormal manifestation of tonic innervation. But after it is once established it is independent of the nervous system and persists under deep anesthesia and even after section of the motor nerve.

In the late stages of local tetanus myostatic contracture develops. The muscle and its constituent fibers become set at a shortened length. In response to direct stimulation it shortens less than a normal muscle and relaxes more slowly. Its elasticity is decreased and its ductility increased.

Histologic sections show that the contracture is not due to fibrosis. The contracture must be due to changes in the muscle fibers themselves. There is a blurring of the cross striations, a mottling of the fibers due to an uneven staining of the anisotropic substance, and an increased longitudinal striation due to the individual myofibrils becoming more evident. None of these changes, however, adequately accounts for the muscle becoming fixed at a new and shorter than normal resting length.

The Chemistry of Muscles in Tetanic Contracture. Dr. H. A. Davenport.

Muscles in contracture produced by tetanus toxin were analyzed for lactic acid, glycogen and acid-soluble phosphorus compounds. Local tetanus in one hind limb was produced, and the opposite flaccid limb served for control. The gastrocnemius muscles were usually used for analysis.

The lactic acid found varied from 8 to 40 mg. per cent in muscles in contracture, and from 8 to 34 mg. per cent in the flaccid controls. Variations between the two muscles from the same animal were within technical limitations, hence lactic acid has no causative relationship to the contracture.

Glycogen was markedly reduced in all stages of contracture in guinea-pigs, but only slightly reduced in rats. Rabbits sometimes had more glycogen in the tetanus than in the control gastrocnemius. Tetanus toxin caused no decrease

in the glycogen of gastrocnemii which were denervated at the same time as the toxin was injected. The effect on the glycogen content and likewise the formation of the contracture are dependent on motor innervation.

The study of phosphorus compounds included the inorganic (ortho) phosphate, phosphocreatine, pyrophosphate and total acid-soluble values. No characteristic difference in quantities of these substances was seen except that there was about 10 per cent less total in the tetanic muscles and a similar relative decrease in the three fractions. It seemed likely that the apparent decrease was due to a loss of muscle substance through replacement by fat.

DISCUSSION

DR. A. B. YUDELSON: I should like to know whether Dr. Davenport thinks that the process of autolysis that goes on in the muscle produces the change, and whether the degeneration, which is apparently the terminal change replacing the muscle cell by fat, is not the "masked" fat which is held in the cell prior to its destruction.

DR. H. A. DAVENPORT: We have no direct information from chemical data as to the location of the fat. The fat has appeared as soap, a by-product formed in the determination of glycogen, and was obviously greater in tetanus muscle than in normal, especially if the contracture had existed for two or three weeks. We do not know whether it is a true fatty degeneration or a fat infiltration.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, April 26, 1929

CLARENCE A. PATTEN, M.D., *President, in the Chair*

A CASE OF AMYOTROPHIC LATERAL SCLEROSIS IN A GIRL AGED 13. DR. HUGH M. GALBRAITH.

R. C., a girl, aged 13, came to the clinic of Dr. F. W. Sinkler, in the Orthopaedic Hospital, on Feb. 27, 1929, complaining of an expressionless face, of difficulty in talking and swallowing and of weakness in the hand. The family history showed that a sister, aged 19, was just recovering from a nervous breakdown of undetermined nature. The patient had always been a normal, healthy child until the onset of her present trouble. Late in June, 1928, according to the mother, it was noticed that the patient was growing rapidly and outgrowing her dresses. At the same time she matured quickly, and began to menstruate in July. Early in the same month, the face began to twitch markedly and gradually both hands began to behave in a similar manner. This continued until late in August, when suddenly the face became paralyzed over night. Early in September, the patient began to have difficulty in speaking, in swallowing and in controlling movements of the mouth. This condition quickly attained proportions that made it difficult for her to make herself understood, and she was able to swallow only by holding her chin up. At about the same time, the hands became very weak and dorsally flexed, making it difficult for her to use either hand. All of the paralytic symptoms reached a climax in September, and it is believed that there has been a slight improvement since. She has continued to grow rapidly, but a very poor appetite has caused her to become emaciated. There has been no disturbance of gait except a slight unsteadiness.

On examination, the patient was poorly nourished. The gait was slightly spastic, and there was a slight swaying in the Romberg position. There was bilateral paralysis in all branches of the facial nerve; the motor branch of the fifth was not involved at that time. There was little action of the muscles of the palate; the vocal cords approximated well. The tongue could not be protruded, and fibrillary tremors and marked atrophy were present. The sternocleidomastoid muscles on

both sides showed definite weakness. The intrinsic muscles of both hands showed marked atrophy producing, bilaterally, a claw hand. The muscles of the arms and shoulders appeared to be somewhat weak, but no definite atrophy could be found. The reflexes of the arm were normal, while the knee and achilles jerks were markedly exaggerated. There was true patellar clonus on the right and quickly exhaustible ankle clonus on both sides, more marked on the right. A true Babinski sign was found on the right side, a suggestive one on the left. The blood Wassermann test was negative and a lumbar puncture was not done.

This case undoubtedly is one of an amyotrophic lateral sclerosis syndrome. The interesting features are the age of the patient and the predominantly bulbar symptomatology.

DISCUSSION

DR. W. G. SPILLER: Amyotrophic lateral sclerosis at the age of this patient is extremely rare. It might be advisable to compare this case with those of the infantile form of progressive bulbar paralysis described by Fazio, Londe and others.

A CASE PRESENTING THE PICTURE OF PROGRESSIVE MUSCULAR ATROPHY DUE TO SYPHILIS. DR. LOUIS ADRIAN SCHWARTZ.

A negress, aged *32*, single, a presser, was seen in the neuropsychiatric dispensary of the Pennsylvania Hospital, service of Dr. Strecker, on March 9, 1929. She complained of "weakness of both arms from the shoulders down." She was well until fourteen months before presentation, when she noticed dull throbbing pains in the left arm. At times the pain began in the shoulder and at times in the arm. It was never excruciating, however, and was inconstant. There was no history of numbness, tingling or dysesthesia. The patient usually noticed a dull ache and weakness, especially at night, at the onset of the illness. Her left arm gradually became weaker until she had to give up work six months ago. She was walking down the street with an armful of bundles when she suddenly felt the left arm give way, and she dropped the packages. Three months ago, the right wrist became somewhat swollen. This subsided, and the patient noticed a marked weakness in the right arm. One week later, she was unable to raise either arm above the head.

The past history was uneventful, except the usual diseases of childhood. The anal and vesical sphincter control was normal as were the menses. There had been no girdle pains. The patient said that she had not had syphilis by name and symptom; also said that she had not come in contact with lead and was not addicted to the use of alcohol or drugs.

The father died about twenty-eight years ago, from an unknown cause. The mother and a sister were living and well. There was no evidence of familial organic, nervous or mental disease.

Following are the positive neurologic observations: the pupils were equal and regular, but reacted slowly to light and better in accommodation; extra-ocular movements were normal; the retinal vessels were not sclerosed; the disks were of good color and clearly outlined; the cranial nerves otherwise were normal in both motor and sensory components.

There was marked atrophy of the shoulder girdle muscles, including the deltoid, biceps and triceps, as well as the muscles of the forearms, specially the extensors of the wrist. There was some involvement of the interossei and thenar muscles, while the rounded contour of the shoulders was flattened. Slight inconstant fibrillations were noted in the right biceps region, especially, but were also found elsewhere, in both shoulder girdles but not in the hands. The patient was unable to raise her arms to the head or to level the hands to a plane at right angles to her body. There was marked weakness in all types of movement: of abduction, adduction and rotation of the shoulder joint and flexion and extension of forearm. The power of supination was decreased bilaterally. There was some weakness of the extensors of the wrist. The atrophy and weakness mainly involved the scapulo-

humeral muscles and to a lesser extent the muscles of the forearm and wrist. Myotatic irritability was increased, as shown by tapping the muscle.

The biceps reflexes bilaterally were barely elicited, while the right triceps was very weak; the left triceps jerk was absent. The radial periostal reflexes were decreased. The Hoffman sign was positive on the right. Thermal, tactile and pain sensations were intact throughout in the upper extremities. The finger-to-nose test was impossible to determine owing to the muscular weakness. In the lower extremities, the knee and achilles jerks were exaggerated. On the right side, by combining the Babinski plantar stimulation with squeezing the gastrocnemius muscle, dorsiflexion of the great toe occurred. The Babinski sign was absent on the left; confirmatory signs were absent bilaterally; there was no ankle or patellar clonus. The gait and the station were normal. There was no Romberg sign; no impairment of vibratory, thermal, tactile and pain sense or of position of the toes. The heel-to-knee test gave normal results.

Mentally, there was no change in affect, and the stream and content of thoughts were normal.

The urine, blood and hemoglobin were normal. The Wassermann and Kahn tests of the blood were strongly positive, 4 plus; with the spinal fluid the tests also were strongly positive (4 plus) in all dilutions; the colloidal gold curve was 33445510000. The spinal fluid sugar was 62 mg.; there were 12 lymphocytes per cubic millimeter. Globulin and albumen tests gave 2 plus reactions. The spinal fluid pressure was 11 mm. mercury with evidence of obstruction and the fluid was water clear. Electrical reactions indicated reaction of degeneration, especially in the scapulohumeral muscles, less marked in the muscles of the forearms and hands. There was slight response to faradic stimulation, and the formula under galvanic stimulation was altered.

The patient was admitted to the ward and received a course of mercurial inunctions and potassium iodide. Massage and physiotherapy were started. The muscles and motor points of the nerves were stimulated with electrotherapy, the sinusoidal current and faradic stimulation being used. The weakness and atrophy are gradually increasing in spite of the treatment. No bulbar symptoms or signs have developed.

This case presents a syndrome characterized by the slow wasting of the scapulohumeral muscles, including the muscles of the forearm and, to a lesser extent, the interossei and thenar muscles, with diminution of reflexes. The observations are due to degenerative changes in the anterior horn cells as a result of the syphilitic process. There is possibly a similar degeneration in the fibers of the motor nerve which spring from the ganglion cells. In addition, there is evidence of early pyramidal tract involvement.

Of interest is the history of dull aching pain in the muscles which afterward wasted. Gowers described slight, subordinate sensory symptoms confined only to dull, rheumatoid pains in this condition. The muscles are often more sensitive to pressure and extension.

Of further interest is the history of complete loss of power with absolute paralysis coming on quickly after a preceding gradual weakness. Such cases have been described by Oppenheim, Gowers and others. Weakness and wasting may come on together, but either may first attract the attention of the patient. The loss of power is usually first noticed in the shoulder and back and, in such covered parts, the wasting may become considerable before it is observed. Most of the observations in this case are those of progressive spinal atrophy.

Idiopathic muscular dystrophy presents so close a resemblance to the spinal atrophy just described that it is distinguished with some difficulty, especially in the absence of syphilis, serologically or clinically. In this case, with the positive serologic observations, the diagnosis is simplified. However, a good number of such cases show a negative clinical serology without evidence of syphilis, so that the differential diagnosis at times is difficult. The distribution, and the age of onset, usually below 20, and familial involvement, favor a diagnosis of dystrophy. Many observers treat such patients with antisyphilitic therapy, believing that

syphilis is the basic underlying factor. Two cases have been reported in the literature in which the wasting commenced during, and another directly after, an energetic mercurial course. It is possible that the disease may be a product of the syphilitic virus and not due to the organisms directly. This patient had had no antisyphilitic treatment prior to the onset of symptoms. With the anterior horn cell and pyramidal tract involved, a diagnosis of amyotrophic lateral sclerosis is justified.

Cervical pachymeningitis with considerable damage to the nerve roots may show wasting that resembles progressive muscular atrophy, but in this condition there are distinctive sensory symptoms with acute pains and usually anesthesias of irregular distribution.

The same disfinctions suffice for diagnosis in disease of nerve roots, such as are produced by tumors. Syringomyelia is ruled out by the absence of dissociated sensory disorder. A chronic disseminated myelitis may cause widespread muscular atrophy, but is distinguished by the presence of symptoms of irregular damage of the cord producing sensory disturbances.

In amyotrophic lateral sclerosis the thenar muscles and interossei are usually the first to suffer; when the disease involves the shoulder muscles the deltoid is generally first to manifest the disease. The rounded contour of the shoulder becomes changed, and the head of the humerus can be recognized beneath the acromion. It is not rare for part of the deltoid to suffer and part to escape, due to separate involvement of distinct groups of anterior horn cells. The wasting of the deltoid is soon followed by that of other muscles of the upper arm and the scapula. The levator anguli scapulae generally escapes when all the muscles about it are wasted. The electrical irritability of the wasted muscles presents changes which vary in character in different cases. When the wasting is slow, there is usually a diminution in both faradic and galvanic irritability, which fails with the muscular nutrition. When the wasting is great only a slight contraction can be obtained even with a strong current. The quality of the galvanic irritability may be normal, but sometimes anodal closure contractions occur more readily than the cathodal. In this case the tetanic contraction during the passage of the current is produced with undue readiness when compared with the closure contraction, and opening contractions are often caused by currents but little stronger than those that cause closing contraction. Mechanical irritation of muscles is considerably increased, as is shown in this case, a tap causing a local contraction of the fibers struck. Fibrillation is so frequent as to be characteristic, but it is not invariable. It sometimes may be observed in muscles that are not yet invaded by the wasting, but when it is observed atrophy usually follows. Reflex action is abolished, owing to damage of the motor part of the reflex arc. Sensory symptoms are usually slight and subordinate, and are confined to dull and rheumatoid pain. It is possible in this case that the slow degeneration has been varied by a more acute process of destruction of the anterior horn cells with a more rapid degeneration of the nerve fibers, so that the muscular tissue presents paralysis, especially in the forearm, in excess of wasting, with a galvanic irritability in excess of the faradic irritability of the nerve endings.

DISCUSSION

DR. N. W. WINKELMAN: Did the symptoms come on suddenly?

DR. L. A. SCHWARTZ: There were symptoms of weakness for about fourteen months, but when the complete atrophy and paralysis finally came on they did so suddenly. It is possible to have a gradual destruction of anterior horn cells producing progressive weakness, but with an acute exacerbation of the process the picture of sudden paralysis is produced.

DR. N. W. WINKELMAN: Then why is not this a case of thrombosis of the anterior spinal artery? The clinical picture is almost similar, and syphilis is a well known cause. It is interesting that the vessels of the cord are rarely affected by ateriosclerosis, but syphilis is well known to produce vascular disease.

DR. L. A. SCHWARTZ: I think that the patient should show evidence of more patchy lesions of the spinal cord. She has a Babinski sign on the right, but there are no pyramidal tract observations on the left side. I do not see how thrombosis could pick out the anterior horn cells bilaterally and yet permit pyramidal tract signs on the right side only and also without disturbance of other lateral column fibers such as the spinocerebellar. The history of weakness for fourteen months is against this also.

Sir William Gowers reported a series of cases in which he said the paralysis may occur very quickly—in fact after a few hours, and he thought that this is true also in syphilis and is probably vascular. This case is reported as one of syphilitic amyotrophic lateral sclerosis with the brunt of the lesion in the anterior horn cells of the lower cervical region.

MULTIPLE ABSCESSES OF THE BRAIN IN BRONCHOPULMONARY CASES. DR. ALFRED GORDON.

The literature on the subject of multiple abscesses of the brain in bronchopulmonary cases is not extensive. The examples that are recorded are sufficiently illustrative to emphasize the value of a more detailed study in pulmonary or bronchial cases of long duration. Complicating cerebral manifestations may be overlooked, especially when the original condition runs a severe course with very conspicuous symptoms.

C. G., a white man, aged 34, had suffered from bronchitis for many years, bronchiectasis was discovered later. There had been purulent expectoration in abundance. Drainage and treatment by the bronchoscopic route had been repeatedly performed. The condition, in spite of the treatment, became so grave that a radical thoracoplasty was being considered. Recently the patient had contracted an acute upper respiratory infection which was intractable. He suffered from intense headache. He began to lose weight and appetite, vomited considerably and gradually became somnolent. An examination of the pulmonary discharge revealed the presence of gram-positive cocci in chains.

At that time my examination of the patient showed congestion of both ear drums. The eye globes were prominent and bulging. The pupils were unequal and reacted poorly to light. No paralysis was present. The reflexes, tendinous and cutaneous, were all normal. A spinal puncture was performed, and 10 cc. of cloudy fluid was removed. It showed pus with 75 per cent polymorphonuclear cells and 25 per cent lymphocytes; the albumin was 2 plus, the globulin 1 plus; the colloidal gold curve was 0022210000. A direct smear showed no organisms. Urinalysis gave negative results. A blood count showed 18,000 leukocytes. The antrums and other sinuses were punctured, but no pus was obtained. The nose and throat were apparently intact. No source of infection other than the pulmonary area was detected. The expectoration became enormous. Cerebral symptoms became more and more conspicuous. The patient became stuporous and finally comatose. He died four days after this examination.

Removal of the brain alone was permitted. When the skull was opened, the entire brain was bulging. When the dura was opened, the entire surface of the brain was found covered with thin yellow pus. The veins were much dilated over the cortex and base. The apex of the right temporal lobe was unusually soft and showed a yellowish area about the size of a silver dollar. The anterior end of the left frontal lobe was also visibly softened. An anteroposterior section showed destruction of tissue with cavity formation at the apex of the left frontal lobe. In the same hemisphere, the posterior horn of the lateral ventricle was filled with pus. A section of the right temporal lobe showed another cavity surrounded by yellowish tissue. At the time of the necropsy, these cavities were filled with pus. A culture of the pus from the cavities revealed the presence of nonhemolytic streptococci.

The case is instructive from a pathologic and etiologic standpoint. Abscess of the brain has been observed in a large variety of bronchopulmonary conditions of serious nature. Among them bronchiectasis occupies the first place. With

many others, Shorstein personally collected sixty-nine cases of abscess in the brain, secondary to lung conditions, among which 55 per cent were in bronchiectasis. The remaining cases are divided in order of frequency between lung suppuration, tuberculosis, empyema and pneumonia (*Lancet* **11**:842, 1909). The reason for the greater frequency in bronchiectasis probably lies in the longer duration of this condition. My patient's condition extended over a period of many years, during which the characteristic abundant expectoration was present with great intensity. The size of the two cerebral abscesses in the present case, together with the extension of the destroyed cerebral tissue, indicate the long duration and the gradual formation of the necrosis of the nerve tissue. The patient suffered from severe headache for many months, but in view of the absence of gross objective motor and sensory phenomena, the nervous complications were overlooked, especially when the distressing bronchopulmonary condition was conspicuously predominant.

As to the mechanism of formation of abscess in the brain in these conditions, it is logical to admit either a thrombophlebitic or a metastatic origin, and finally a direct bacterial invasion. Arnold injected particles of bran into a large vein of the neck and found some of them in the opposite venous sinus. He figured out that these foreign particles were carried first into the small bronchial or pulmonary veins and then to the superior vena cava; from the latter, via the jugulars, to the cerebral sinuses and finally to individual veins in the brain (*Arch. f. path. Anat.*, 1891, p. 385). The violent and frequent coughing of my patient produced intense forcible expiratory paroxysms and venous engorgement, thus facilitating the penetration of pathologic material into the cerebral tissue.

While this contention regarding the etiology of the isolated foci of destruction in the brain seems to be correct, one cannot ignore the discovery of the identical streptococci during life in the bronchopulmonary excretions and the cerebrospinal fluid. It is therefore also possible that infection set in simultaneously in the bronchial apparatus and the cerebrum, thus producing similar destruction in both tissues. Finally, it may be admitted with equal right that the foci found in the brain may be simultaneously both vascular and bacterial in character.

The case described consequently presents two special points of practical interest, namely that cerebral manifestations of a pathologic nature may occur in bronchopulmonary condition of prolonged duration, and that the lung disorder may be so pronounced that the brain symptoms may be overlooked. Metastasis or independent invasion of the brain tissue by the same bacterial agent that produced the pulmonary condition may occur. Recovery in some such cases as the result of surgical intervention on the brain have been recorded. (H. Houlland: *Paris Chirurg.*, 1917, p. 613; S. Barling: *Lancet* **1**:121, 1925). It should be borne in mind that absence of gross symptoms of an organic involvement of the central nervous system does not always exclude invasion of brain tissue. When the motor or sensory cortex or other important cerebral centers are not involved, the respective clinical manifestations will be absent. In my case, for example, the destroyed areas were confined exclusively to the apexes of one frontal and one temporal lobe. A diagnosis of a lesion of the brain cannot be based exclusively on the presence of phenomena referable to the motor or sensory apparatus. The cerebral manufestations in this case consisted of persistent severe headache with periodic attacks of somnolence. This condition is sufficient to suggest cerebral involvement, and the suspicion should be directed to areas other than the special centers of the brain. W. P. Eagleton in his book on "Brain Abscess" collected a large number of such cases, and it is interesting to note his conclusion that the "frontal lobe is the most frequent seat of abscess." The temporal lobe, especially its apex, is another seat in the brain which may not give rise to disturbances which could arrest attention.

DISCUSSION

DR. C. K. MILLS: It is true that multiple abscesses such as Dr. Gordon has shown in this case are not infrequent. It is probable that the condition in

the lungs was due partly to involvement of a region of the brain. The important thing, I think, is the existence of multiple abscesses in a case like this.

DR. TEMPLE FAY: The question of a widespread meningitis comes up in this case, and one cannot overlook the possibility of a preceding sinus infection. Dr. Gordon mentioned abnormalities of the ear drums. In encapsulated deep-seated metastatic abscesses, one does not find organisms in smears or cultures of the spinal fluid. If an abscess is metastatic through the blood stream, it is most often deep in the substance of the brain. This frontal lobe abscess is superficial, and that suggests the possibility of a sinus origin. The majority of cases of sinusitus are associated with pulmonary infections. Occasionally, when one finds multiple metastatic abscesses, they are so scattered and deep-seated that there is little doubt concerning the source of invasion.

THE PRESENT DAY POINT OF VIEW OF THE MOTOR SYSTEM. DR. T. H. WEISENBURG.

This paper will be presented in full in a later issue of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY.

EXTENSOR REFLEXES FROM THE KNEE IN RELATION TO THE KNEE JERK AND TO REBOUND. DR. GRAYSON P. MCCOUCH and DR. BERNARD J. ALPERS.

This paper appeared in full in the October issue of the ARCHIVES, **22**:672, 1929.

DISCUSSION

DR. G. E. COGHILL: This paper is of great interest to neurophysiologists, both for the technic employed and for the highly technical questions of physiology that are involved. These features of the paper, however, I shall not try to discuss.

I am interested particularly in the conclusion that the patellar reflex is excited by a complex of proprioceptive and exteroceptive stimuli, because it seems to give a physiologic demonstration of what one sees in the history of the development of the reflexes. One sees the principle clearly demonstrated in the fish and in lower forms of animals in which the sensory neurons of the first order in the trunk region are both proprioceptive and exteroceptive. These sensory cells, instead of being arranged in spinal ganglia, occur within the spinal cord. One branch goes to the skin and one to the muscle, so that their single fibers must serve both skin-sensory and muscle-sensory functions. One gets through them, however, distinctly exteroceptive reflexes, for there is a stage in the development at which the movements are almost constantly away from the side involved. A little later in the development, this regularity of response ceases and there are about as many movements toward the side touched as away from it. But if at this stage one cuts the head off far enough down the trunk to leave a few muscle segments attached, the regularity returns, and the movements are again almost constantly away from the side touched. The conclusions certainly would be that the operation has eliminated a factor in the reactions and that this factor is proprioceptive. Dr. McCouch's central thesis is certainly supported by these facts of comparative anatomy and physiology.

Referring to the embryologic phase of the problem in man, I do not know that one can establish such clear relations, although this particular reflex as it occurs in fetal life has been studied by several investigators. At least three have reported on it: Drs. Minkowski, Bolaffio and Artom, and Krabbe. Dr. Krabbe, in 1912, reported the case of a fetus of 24 cm. total length in which he was able to excite other reflexes but not the patellar reflex. On the other hand, Drs. Bolaffio and Artom were able to demonstrate patellar reflexes in a 28 cm. fetus, but not earlier than that. They also found it in a fetus of 33 cm., but only after decerebration. In these stages a patellar reflex must be demonstrable. Dr. Minkowski goes much further back in fetal development and states that he has

seen the patellar reflexes in a fetus of 6.5 cm. total length. Of course, in a fetus of that size it is a delicate matter to localize the stimulus exactly, but Dr. Minkowski is certainly one of the most skilled observers of movement in the human fetus. Furthermore, he found another case of a 7 cm. fetus in which there was extension of the leg on the thigh as a patellar reflex. In the 7 cm. fetus this was accompanied by extension of the other leg and flexion of the elbows. It is certain, therefore, that the patellar reflex has a very early origin in fetal life. The significance of this fact, for the question under discussion, is that the reflexogenous zone for a particular reflex is relatively general and diffuse, and that the definitive localization of the appropriate point of stimulus arises by a process of concentration. For example, in early development the plantar reflex, which in adult life occurs typically to stimuli on the sole, has been elicited by Dr. Minkowski by stimuli on the dorsum of the foot as well as on the plantar surface.

There is, in this principle of progressive localization of stimuli in embryologic development, and in the phylogenetic history of the nervous system as seen in the development of the lower forms, a biologic background which would support Dr. McCouch's conclusion that there is a complex of stimuli involved in the patellar reflex, some of which are obscured by the more apparent modes of excitation and can be demonstrated only by very special study.

Book Reviews

NORMALE UND PATHOLOGISCHE ANATOMIE UND HISTOLOGIE DES GROSSHIRNS. By PROF. A. JAKOB. Volume II, first part. Price, 40 marks. Pp. 870, with 266 illustrations. Leipzig and Vienna: Franz Deuticke, 1929.

This is the second volume of Prof. Jakob's work on the histology and microscopic anatomy of the nervous system. As was the first volume, the book is based entirely on the material studied by Jakob and his many pupils.

Chapter 1, comprising twenty-five pages, takes up tuberculosis of the brain and its membranes. In it, meningeal tuberculosis and tuberculoma of the brain substance are discussed. Excellent examples are brought forth to illustrate the various phases. The exposition of this subject is fairly complete. Only one or two points might be emphasized. The author includes from the literature the cases of tuberculous meningitis in which recovery has been reported, with a discussion of each report. He quotes the case of Henkel in which death nine years after recovery from a tuberculous meningitis was studied histologically. The second point worthy of emphasis is the description and illustration of the characteristic changes in the blood vessels in tuberculosis of the brain, which Jakob calls "panarteritis tuberculosa," an excellent designation. This condition can easily be mistaken for the syphilitic form of vessel change, if one is not alert to the possibility.

It is, however, in syphilis of the nervous system that Jakob reaches his acme. This he divides into meningitis and meningo-encephalitis; syphilitic disease of the vessels, including Heubner's endarteritis; chronic syphilitic disease of the vessels; syphilitic arteriosclerosis, and the endarteritis syphilitica of Nissl and Alzheimer. Any one who has worked in Jakob's laboratory, as the reviewer has done, can appreciate the carefully selected illustrations in this part of the book.

It is of interest to know that in twelve years in Jakob's laboratory 3,100 cases have come to autopsy and of these 890 were cases of paresis. One can get an idea of the mass of this syphilitic material by comparing the type of material at the Philadelphia General Hospital, where, in nine years, over 3,000 cases have come to autopsy of which less than 100 were cases of paresis. It is only natural that with 890 cases at his disposal, Jakob should be able to write a masterly chapter on the subject of paresis. If this chapter alone were to constitute the entire monograph, it would be worth its price. One hundred and fifty pages are taken up in the discussion. He divides the material of the chapter as follows: (*a*) the usual type of paresis, to the discussion of which he gives 60 pages, (*b*) atypical paresis, divided into paresis on the basis of congenital syphilis, Lissauer's type of paresis, the combination of paresis with cerebral syphilis, the spontaneous remission, or the "healed," paresis and the form in which remission has been obtained by malaria or other forms of fever therapy. Practically no form of paresis is described in the literature of which Jakob does not have examples. Every newer method of study has been utilized in the making of this chapter.

The final chapter in the book takes up the discussion of trypanosomiasis, Chagas' disease, multiple sclerosis and diffuse sclerosis. There is only one criticism on this part of the work: in multiple sclerosis, Dawson's work is not quoted. To the reviewer's mind, this is one of the most important pieces of work on this subject in the last few years.

All in all, this is a work that is based on years of study. It forms one of the milestones in the progress of what is fast becoming one of the major interests of the clinical neurologist; for today no one can be a clinical neurologist intelligently and honestly without a good grounding in the pathology of the nervous system. So that this work is important not only for the man who is devoting his life to the advancement of the science of neurology, but for the man who is devoting himself to the diagnosis and care of patients afflicted with nervous disease.

The illustrations are excellent and clear; the book is printed on good paper. The publishers have done their part well. The book fills a gap in the neurologic literature that has existed for years, since Spielmeyer's second volume has never appeared. Professor Jakob has earned the eternal gratitude of all those whose work it is to advance neurology.

DIE PSYCHOSEN DER SCHWACHSINNIGEN. By DR. RUDOLF NEUSTADT, Asst. Physician in the Clinic and Instructor in Psychiatry and Neurology at the Medical Academy in Düsseldorf. Paper. Price, 15.60 marks. Pp. 188. Berlin: S. Karger, 1928.

The material studied comprised the histories of 380 patients selected as having psychoses from 7,000 admissions to the institution at Düsseldorf-Grafenberg between Jan. 1, 1924, and June 30, 1927. This hospital receives only adult persons and therefore, as the author points out, the results of the investigation cannot be applied statistically to cases of feeblemindedness in general. For various reasons the histories of 160 patients were excluded. Care was taken to exclude cases in which the feeblemindedness was accompanied by epilepsy or by alcoholism, morphinism, arteriosclerosis, paresis or other organic disease. The diagnosis of feeblemindedness was based on defective intelligence which was congenital or acquired in the earliest years and which was stationary. Psychopathic states were excluded and conditions were considered as psychoses only when—in accordance with the definition of Gruhle—the mental manifestations were new and foreign to the previous mental make-up of the patient.

The forms of the psychoses observed are divided into the following groups: manic states, 13; melancholic states, 13; retarded and depressive states, 28; anxious excitement, 20; confusional states, 20; hyperkinetic and catatonic states, 9; hallucinatory pictures, 22; paranoid pictures, 8; borderline states (excitement and depression), 35; hysteria (pseudodementia), 14; socially and sexually unstable, 15; differentially uncertain and undiagnosed cases, 23.

The material is admirably presented, and excellent, detailed histories are given in illustrative cases in the various groups. In the general summary, Neustadt has adopted for all the psychoses the general term introduced by his chief, Sioli, "Episodic Psychoses of the Feebleminded." He points out that these psychoses have certain elements which distinguish them from the psychoses in the non-feebleminded and suggests that they constitute a nosologic entity. He discusses in detail their relations with dementia praecox and the manic-depressive psychoses and his conclusions are particularly striking. With regard to the former he comes to the conclusion that feeblemindedness and schizophrenia are mutually exclusive (Wir haben vielmehr den Eindruck gewonnen, dass Schwachsinn und Schizophrenie sich zwangsläufig ausschliessen). "Wenn es überhaupt echte Pfropfschizophrenien gibt, so ist sicher ein sehr seltenes Vorkomnis." Neustadt considers that the many superficial similarities between feebleminded psychoses and schizophrenia are to be explained by recent studies of the schizoid reaction type. Catatonic symptoms can readily be understood as the result of brain injuries, such as occur for example in epidemic encephalitis. Likewise, manic-depressive psychoses are rare in the series of cases reported, though there is not the same antagonism here. In feeblemindedness the psychoses are overwhelmingly the result of external situations and are characteristically episodic.

The book presents an excellent piece of clinical investigation in a field that has been much neglected, and is deserving of the consideration of every psychiatrist.

DIE PSYCHOPATHISCHEN PERSÖNLICHKEITEN. By DR. KURT SCHNEIDER, A. O. Professor, Oberarzt der psychiatrischen und Nervenklinik der Universität Köln. Second edition. Paper. Pp. 87. Leipzig: Franz Deuticke, 1928.

In the second edition of this book the subject matter has been largely rewritten in the light of recent studies of character and bodily constitution. The aim is the formulation of a system of classification of psychopathic personalities which is not

merely symptomatic or founded on social behavior. The author specifically excludes from consideration abnormalities in intelligence, though recognizing the intimate relation between this and the personality. He adopts the concept of quantitative normality of Kant and regards as abnormal, deviations from an undefined average—deviations in the direction of more or less. The varieties of abnormal personality are therefore endless, and he deals only with those deviations that result in harm to the individual himself or to society. This conception necessarily includes not only the abnormalities that result in unsocial or antisocial behavior but also many of the conditions described as psychoneuroses.

The first part of the book contains an excellent discussion of the literature dealing with psychopathic personalities and outlines the various methods of subdivision into types that have been followed. These include the so-called unsystematic types of Kraepelin, Bleuler and others, but also the systematic schemes of Gruhle, Kretschmer and Ewald. For practical purposes Schneider has preferred to adopt an unsystematized scheme, in constructing which he makes full use of the characterologic studies of Kretschmer and the formula for analysis of character of Ewald. He also uses the Kretschmer scheme of body types.

The second part of the book is devoted to a description of ten types of abnormal personality with discussion of their relations to the groupings used by others, to body constitution, to the psychoses and to one another. These are not regarded as diagnoses; combinations between them are encountered. The types are: (1) hyperthymic; (2) depressive; (3) self-doubting, in which there are two subforms —the sensitive and the anankastic or obsessive; (4) fanatic; (5) limelight-seeking (geltungsüchtig); (6) affectively labile; (7) explosive; (8) unfeeling (gemütlos); (9) unstable (willenlos); (10) asthenic.

The book is thoroughly worth while and throws a great deal of light into this extremely difficult field. It is perhaps unfortunate that the author has not given some illustrative case histories and analyses, but his descriptions are excellent and his discussions helpful.

STUDIES IN PSYCHO-EXPEDITION. By PROF. F. SCHNEERSOHN. Price, $3.50. Pp. 209. New York: Nicholas L. Brown, 1929.

A more difficult book to swing into would be hard to find. It concerns the bringing of the artistic intuition of the soul into the scientific world: Its subject matter is ecstasy, passion, the unplumbed depths of human personality. Art and religion must bring their treasures to science. It would be hard, indeed, for the neurologist to get any hold of psycho-expedition if it were not for its criticisms of psychoanalysis and experimental psychology. The latter is dismissed as disregarding, essentially, everything but the normal consciousness. The former concerns itself with primitive instinctive life, shutting its eyes to everything else, and even in its own field "interprets" instead of observing.

What the book claims is that it begins the science of man in his infinite multiformity. What it emphasizes is a psychic life of three modes, "which alternately flow into each other" — the primitive, the normal conscious and the intimate. The last is the most important, and can be observed in the exceptional outbursts of emotion in the average person, in the lives of artists and saints and in the milder and more permanent moods. What the book practically offers is a method for the diagnosis and cure of neuroses, which are forced workings of the intimate urges that cannot be satisfied with one's actual way of life. The method is an "expedition" into the intimate life of the human being: by a recollection of passions and of ecstasies, by keeping a diary, by letting one's imagination run along or by getting descriptions of the patient made by himself or by some one else. The cure-expedition is undertaken by the patient, who sets a date, arranges his dress and his room "in an intimate manner, and gives himself to ideas, images, emotions and longings" which "become inwardly intertwined into a concentrically radiating consciousness" and "allow the person to face the Absolute."

Clinical illustrations are given. Probably most of the book will make little appeal in America. It is worth reading to keep oneself reminded that "there are more things in Heaven and Earth than are dreamed of in our philosophies."

INTERNATIONAL CLINICS. A Quarterly of Illustrated Clinical Lectures and Especially Prepared Original Articles on Treatment, Medicine, Surgery, Neurology, Pediatrics, Obstetrics, Gynecology, Orthopaedics, Pathology, Dermatology, Ophthalmology, Otology, Rhinology, Laryngology, Hygiene, and Other Topics of Interest to Students and Practitioners by Leading Members of the Medical Profession Throughout the World. Edited by Henry W. Cattell, A.M., M.D., Philadelphia; with the collaboration of Charles H. Mayo, M.D., Rochester, Minn.; Sir John Rose Bradford, M.D., London; Hugh S. Cumming, M.D., D.P.H., Washington, D. C.; William S. Thayer, M.D., Baltimore; John G. Clark, M.D., Philadelphia; Frank Billings, M.D., Chicago; James J. Walsh, M.D., New York; A. McPhedran, M.D., Toronto, Canada; John Foote, M.D., Washington, D. C.; Sir Humphry Rolleston, Bt., K.C.B., M.D., F.R.C.P., Glasgow, Scotland; Seale Harris, M.D., Birmingham, Ala.; Charles D. Lockwood, M.D., Pasadena, Calif.; A. H. Gordon, M.D., Montreal, Canada; T. M. Devine, B.S., Melbourne, Australia; Fielding H. Garrison, M.D., Washington, D. C.; R. Bastianelli, M.D., Rome, Italy. Volume III. Thirty-Sixth Series. Pp. 302. Philadelphia and London: J. B. Lippincott Company, 1929.

The present volume contains a number of neurologic articles, among which the most interesting are: Brain Anatomy at the University of Zürich; The Extra-Pyramidal System and Its Diseases; The Unconscious and the Instincts, and Psychoanalysis.

Like previous numbers of International Clinics, the selection of the authors and the material is up to its former standard.

A STUDY OF THE EDUCATIONAL ACHIEVEMENT OF PROBLEM CHILDREN. By RICHARD H. PAYNTER and PHYLLIS BLANCHARD. Price, $1. Pp. 72. New York: The Commonwealth Fund Division of Publications, 1929.

There has been some tendency to generalize, on the basis of individual case studies, that deviations of personality and behavior necessarily imply an impaired educational achievement. In order to investigate the reliability of this generalization, the authors studied the educational achievement and accomplishment ratios of 167 Los Angeles and 163 Philadelphia school children who had been referred to Child Guidance Clinics because they presented behavior problems. As a result of their study, they conclude tentatively that problem children show no general tendency toward low educational achievement, but that certain ones fail to rise to normal levels of achievement owing to emotional maladjustments. Although in many cases educational achievement is not being impaired to any great extent by their difficulties, these children are being ill prepared for adaptation to the economic and social demands that will be made on them in maturity.

Besides these important conclusions, the monograph contains many useful data on the maladjusted school child. One chapter, which is devoted to methods of handling statistical data regarding behavior, will prove a valuable aid in the treatment and presentation of such data.

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The star (*) preceding the page number indicates an original article in The Archives. No author entries are made except for original articles and society transactions. Book reviews, Obituaries and Society Transactions are indexed under these headings in their alphabetical order under the letters B, O, and S, respectively.

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