

all times readily available for study in the living patient. Second, from its physiologic position in the body, it must of necessity reflect in some way every variation from the normal that may take place in the organism, whether it be a general or a local variation. It is plain of what vast value it would be if we possessed definite information as to what these changes are and under what circumstances they occur. Thus far our studies of the blood have been chiefly in morphology. It is to the chemistry of the blood that we must look for the greatest aids in diagnosis in the future, but at the present, blood chemistry is practically an unexplored field. In five or six diseases, a diagnosis rests entirely on the blood examination. In every febrile disease, an examination of the blood will offer some light. In malaria, leukemia, filariasis, pernicious anemia, relapsing fever and chlorosis, exact diagnosis is impossible without a blood examination. However, the most generally valuable finding is the presence and extent of any leucocytosis. Every febrile process has a leucocytosis, with the following important exceptions: Malaria, typhoid fever, pure tubercular infections, measles, and the early stages of la grippe.

Leucocytosis is most pronounced in lobar pneumonia and suppurative diseases. Its presence or absence in a given case is of great diagnostic aid in placing a febrile disease in or out of the above list. A diagnosis of anemia should be based on the estimation of the percentage of hemoglobin and the number of red cells present. Any other method allows of a wide range of error.

We have endeavored to briefly outline a few of the more important aids in diagnosis afforded by the laboratory. No effort has been made to detail technic, as our space is too limited. In spite of the generally recognized great value of technical diagnostic aids, they are not employed by the average practitioner, and to any extent by very few indeed outside of the larger hospitals. Several factors go to account for this: First, to equip a laboratory requires considerable expense; second, many physicians have not the technical training and lack the time to acquire it; third, some because of a lack of diversity in their available material, do not have an opportunity to become proficient. In other words, the average physician is too busy, thinks it won't pay or lacks the taste or opportunity to become skilled in technic.

The solution of the problem lies in co-operation. With an expenditure of \$10 and an arrangement with a laboratory within twenty-four hours' ride, any physician can have laboratory advantages at his disposal. All that is needed is a few glass slides, some culture tubes, platinum needle, alcohol lamp, Thoma-Zeiss white cell pipette, Tallqvist hemoglobinometer and a book on technic to obtain and forward material for a leucocyte count, hemoglobin estimation, bacteriologic examination of blood, urine, exudate or whatever else may be desired. One member of the community of physicians can go to the expense of fitting a laboratory and becoming proficient in technic. His neighbors can either share the expense and go there to make their examinations, or what is better perhaps for both, have one who has the time and taste, do the work for all. Practice makes perfect, and better observations would be secured by such a plan. A mutual agreement should be made as to the sharing of expense, and by a very small personal outlay a town possessing a faculty of only six or more physicians could have a sufficiently well-equipped laboratory and afford enough work to enable one or more of their number to

become reasonably expert. We have not the time to elaborate this plan, but simply wish to state how, by means of harmony and mutual aid, it would be possible to bring the means of diagnostic precision within the reach of practically all the profession.

In many of the larger cities municipal laboratories have been established and have done much good work, especially in dealing with the epidemic and contagious diseases. Being municipal laboratories only, they reach but a small proportion of the profession. It is not unlikely, in view of the good work done by the city laboratories, that the state will eventually place at the disposal of the general profession an adequate number of well-equipped laboratories that would not only offer technical aid in general diagnosis, but would require that their services be employed in all cases that might have a bearing on the health of the general public. It must of necessity be some time before any generally efficient state aid can be expected, and meanwhile the profession must rely on their own efforts to obtain laboratory aid in diagnosis.

A CASE OF CIRCULAR INSANITY STUDIED FROM CLINICAL, DIFFERENTIAL AND FORENSIC STANDPOINTS.*

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WITH GROSS AND MICROSCOPIC ANATOMY OF BRAIN (FROM THE PATHOLOGICAL DEPARTMENT OF THE UNIVERSITY OF CHICAGO), BY THOR ROTHSTEIN, M.D., CHICAGO.

(Concluded from p. 1133.)

FIFTH MANIC-DEPRESSIVE CYCLE.

In October, 1895, a petition *de lunatico* was again filed, and at the hearing it was testified that he had shown an immoral photograph to a young girl of twelve. This hearing was interrupted by a scuffle and altercation in court between X. and a witness, and by his calling the judge a "cheap skate." He was committed to jail for contempt. Immediately after he made three applications for a habeas corpus before as many judges, two of which were denied. He was released on the third and immediately rearrested, and when later the hearing on the question of insanity came up before a jury, the latter brought in a verdict of sanity, and he was never declared insane or committed to an institution for the insane again; not because he did not continue to exhibit an unbalanced mind, but apparently because all attempts to control him were given up in despair. From this date I have no detailed knowledge of the periods or phases of exaltation and depression. They continued to recur in an irregular manner. X. had in September, 1893, been restored to his property rights and his conservator had been removed by the probate court. His property was at that time estimated to amount to \$45,000, and was eventually dissipated by him so that at his death, seven or eight years later, I understand little or nothing was left.

In November, 1898, he was arraigned before a justice on charge of assault with deadly weapon, could not secure bail and was sent to jail. Later he appeared in circuit court, poured a pitcher of ice water over his head, rubbed it with a newspaper in lieu of towel, and then asked for a *certiorari* on the justice who had committed him.

In January, 1897, he started in the superior court 16 suits for personal damages aggregating \$1,464,000. At

this time two of his children filed affidavits desiring to stay with their mother.

During the years from 1893 to 1898 much of the time he occupied his residence on one of the handsome boulevards. He would sit at his door and anyone passing by in the street was liable to be made the subject of some practical joke. He would array himself at times in kilts and in dress and conduct was absurd and eccentric in the extreme. Often nonsensical signs and notices were hung in his windows, and the interior of the house was a scene of disorder and confusion indescribable.

SURGICAL INTERFERENCE.

In 1898 he consulted me about an operation on his brain, stating that Dr. J. B. Murphy had agreed to operate if I would recommend it. His claim was that the injury to his head in 1889 was the cause of his insanity. I stated to him that I could not recommend the opera-



Fig. 4.—Deterioration. Obesity, aged about 40.

tion, because his insanity undoubtedly antedated the injury, because there were no focal symptoms, motor or sensory, pointing to any certain region of the brain as the seat of disease, nor were there local evidences of injury to the cranium sufficient to justify an operation. I informed him finally that a physician who had seen him immediately after the injury had informed me he presented no signs at the time of concussion or compression. X. stated that granting all that to be true, still he was determined to have the operation, if for no other reason than for its effect on the public. He felt he had lost to a great degree the confidence of the public, and, if he could announce that the disorder of the brain caused by the injury to the head had now been removed by operation, he would regain the standing he had lost and could again get back his formerly lucrative law practice. The

operation of trephining was done in October, 1898, I learned from the newspapers. The particulars of the operation have not, so far as I know, been reported. Evidence is lacking, however, in the subsequent history of the case to show material change in the patient's condition either for better or worse. Before submitting to the operation X. made his will—a characteristic document, in which he directs that his funeral be held in the West Side High School, of which he considered himself the "father and partial designer." He names for honorary pallbearers several of the first citizens of Chicago, beginning with Mayor Harrison. Some of these men had been his friends in years gone by. He gave his cranium to one physician, the rest of his skeleton to another and his heart to a third. He directed that models should be made of his skull and given to the medical colleges of Chicago, also to about twenty physicians in New York and Chicago; also that a death mask should be made and cast in bronze and presented to his friends, etc. This will, though eccentric, seems inconsistent with the theory of paresis having existed nine years.

ASYLUM DELIVERIES.

In the fall of 1898 and the following winter and spring the patient spent some time in Elgin, Ill., led thither, as he stated, by an "affair of the heart." (He had previously been divorced.) During this time he came in contact with patients in the state hospital who claimed they were wrongfully confined, and he secured the legal release of three such patients on habeas corpus; he also brought suit in three other cases and obtained the discharge of a girl from the State Home for Juvenile Offenders at Geneva. His "affair of the heart" did not run smoothly. A paragraph in the press in April, 1899, told of his engagement being severed at Moline, Ill. After he had lost sight of his fiancée for some time he sought her in three different states and spent "\$86 in trying to trace her by telephone." He implored her to accept him at the police station in the presence of patrolmen and reporters. She remained obdurate and later returned his presents.

In November, 1900, X. secured the release from the Hospital for the Insane at Kankakee of an undoubtedly insane patient, and began suit against the authorities for \$250,000 for illegal detention.

In February, 1901, the patient successfully defended a patient in an insanity inquest in Milwaukee. This patient, too, was palpably and undoubtedly insane.

In August, 1901, the death of X. occurred at the Cook County Hospital from typhoid fever. It was stated in the press at the time that an autopsy was held by Dr. E. P. Noel. The brain was placed in possession of Dr. Ludvig Hektoen, who gave it for examination to his associate in the laboratory of Rush Medical College, Dr. T. Rothstein, who will submit his findings at the close of this paper.

DIFFERENTIAL DIAGNOSIS.

From such knowledge as I had, I arrived at the diagnosis of circular insanity, and here offer a few words on the differential diagnosis.

In reviewing this case from the standpoint of diagnosis, it may be stated that circular insanity is unmistakably shown by the clinical history. Circular insanity is a psychosis, the essential characteristic of which is the occurrence of alternating periods of mental exaltation and depression, or manic-depressive cycles, with periods of lucidity or normal condition between. Some writers insist on a normal interval between each of the exalted

and depressed phases; some between the cycles only, and find one phase passing almost immediately into the other, but the classical *folie circulaire*, as first described by Falret and Baillarger (also called *folie a double forme* and *cyclo-thymia*), consisted of exaltation, depression and sanity in regular succession. The only question that can arise in diagnosis of this case is whether the various cycles were or could be merely phases associated with paresis, since that disease is sometimes marked with alternating or circular mental symptoms, and since the expansive or grandiose phase of X.'s psychosis was suggestive to many minds of paresis, general paralysis or paralytic dementia, as the disease is denominated by different writers, but which I will here call general paralysis.

It is, of course, possible to refine endlessly on the points of diagnosis and to emphasize the exceptions to almost any rule, but it can be truly said, if this case was one of circular insanity, it was a typical one, and if it was a case of general paralysis, it was non-typical in the extreme. The cases of general paralysis are few indeed in which complete manic-depressive cycles occur for three or four years with strict regularity to the number of four or five; in which mental power and brilliancy are retained with accurate memory for recent and remote events, dates, etc., with logical cogency; sharp and clear power of discernment and statement and of concentration of mind and sustained attention. It is of the very essence of general paralysis that a blurring, so to speak, of perceptions and conceptions is present, even in the early stages, and the will is weak, the expansive ideas or delusions in the exalted state are fatuous and inconsistent. In the depressive state, there is more confusion and stupidity and more tendency to hypochondria, less retardation, less anxiety and discomfort—all of which considerations speak for circular insanity in the case of X. The moral deterioration in his case and the change in disposition and increased irritability might be common to either condition.

If it be granted for the sake of argument that the mental symptoms shown by this case might possibly be present in a case either of general paralysis or circular insanity, we must then look to the somatic signs for settling the diagnosis, and here, again, I find, so far as my observation went, that in the case of X. the most characteristic symptoms of general paralysis was lacking—fibrillary tremors of facial muscles and tongue, speech defects, immobile pupils, implication of cranial nerves, convulsive, spasmodic or paralytic phenomena, heightened deep reflexes—all were lacking in my observation of the case of X. It may be that colleagues who examined X. at various later periods during the twelve years of his insanity saw some of these signs, but none of them were present when I examined him. Furthermore, evidence of syphilis was lacking, which is known to be present in 75 per cent. of paretics and claimed by many the invariable cause. Again, the fact of the patient living for twelve years and dying of intercurrent disease is unusual in general paralysis, though, of course, not impossible.

The symptoms speaking for general paralysis were the grandiose ideas, which, on closer view, are, in my opinion, not characteristic, the mental and moral deterioration, which are, however, common both to general paralysis and to circular insanity. The abnormality of diminished reflexes on the left side is of neutral significance. Finally, there have been cases of general paralysis with remissions, in which great brilliancy was

shown, and with a duration of ten or more years and subsidence of the symptoms, but not, so far as I know, cases in which all the symptoms were consistent with a diagnosis of circular insanity. In view of Dr. Rothstein's examination, I will not speak further of the pathologic anatomy than to say the examination of the brain fails to furnish evidence of changes such as are required to establish general paralysis.

THE ANATOMIC FINDINGS.

BY DR. THOR ROTHSTEIN.

(University of Chicago.)

The patient died of typhoid fever at the Cook County Hospital in Chicago. From the autopsy, the brain, the part of dura covering the convexity of the brain and the middle part of the frontal bone were secured and preserved in formol and Müller's fluid. On the internal surface of the frontal bone are two groups of exostoses, one on each side of the middle line, at the point where the crista frontalis meets the sulcus frontalis. The exostoses on the left side consist of five eminences spread over a surface that is 4 cm. in length and 2.5 cm. in width. They protrude about 1-1.5 mm. The exostoses on the right side are confluent, protruding at one point 3 mm. and covering a surface 4 cm. in length and 0.5-2 cm. in width. Some parts of the dura adhere around the exostoses. The dura has the appearance as if it had been adherent to the skull, then the external layers were torn off from a considerable part of it, but the internal surface of the dura is perfectly smooth and of normal luster.

The brain, with cerebellum and medulla oblongata, weighs 1,720 gm. and hence is to be classed with the heavy brains. The pia is loosened from the brain over a small surface at the frontal pole of both hemispheres, but the underlying brain tissue is intact except that in the left hemisphere there is a horizontal cut 2-3 cm. long (made at the autopsy). There is a depression on the right hemisphere close to the fissura longitudinalis at the level of the brain corresponding to the situation of the exostoses. It is hardly noticeable on the left side, but a little more marked on the right hemisphere (Fig. 16, at the frontal pole), here measuring 1.5 cm. in height and about 0.25-0.50 cm. in depth.

The veins of the pia are distended with blood, especially over the convexity, but no thickening of the pia can be found and it is easily loosened, leaving the gyri perfectly smooth. The lateral ventricles are unusually narrow.

LEFT HEMISPHERE.

Fissura sylvii (f sy, Fig. 13) is 50 mm. long. The ramus anterior horizontalis (r h, Fig. 13) is short but deep. The sulcus designated as ra (Fig. 13) cuts down to the insula and thus must be considered as ramus ascendens anterior. It is unusually long and keeps its depth till it comes close to the sulcus frontalis medius, but the anastomosis with the last named sulcus is shallow, both sulci being separated by a deep gyrus. Fissura sylvii is bifurcated at the posterior end. The ramus posterior descendens is very short. The ramus posterior ascendens passes upward and backward, joining the sulcus intermedius (i, Fig. 13).

Sulcus centralis (c, Figs. 11 and 13) commences on the medial surface of the hemisphere and runs in a nearly straight direction forward and downward. Near its lower end it sends a branch backward and downward through gyrus centralis post. to the fissura sylvii.

The sulcus cinguli (ci, Fig. 14) runs as a continuous sulcus from the genu corporis callosi to the incisura cinguli (i c, Fig. 14). In its posterior part (pars posterior—Eberstaller) it is single, but a doubling of pars intermedia and pars anterior (Eberstaller) (p a and p i, Fig. 14) exists. Orbitally from the sulcus cinguli a sulcus rostralis (s r, Fig. 14) is seen, which reaches the edge of the hemisphere anteriorly. Below a short sulcus rostralis inferior (Retzius) (s r i, Fig. 14) exists.

The sulcus frontalis superior is represented by two furrows (f s₁, f s₂, Figs. 11 and 12). The posterior furrow (f s₁) sends

out several side branches. One passes vertically and anastomoses with sulc. frontal med. (f m, Fig. 12). The anterior furrow (f s₁₁) starts half-way between the posterior furrow and fissura longitudinalis and runs parallel to this fissure, but is far from reaching margo orbitalis.

The sulcus frontalis medius (Eberstaller) (f m₁ f m₁₁, Figs. 12 and 13) is the most prominent furrow on the frontal lobe. Its posterior portion (f m₁, Figs. 12 and 13) communicates with the sulcus præcentralis inferior (pr i, Fig. 13) and thus has replaced the sulcus frontal inferior. The anterior portion (f m₁₁, Fig. 12) sends out several side branches and ends at margo orbitalis in sulcus fronto marginalis (pars anterior—Eberstaller) (f m a, Fig. 12). Side branches from sulcus frontal med. form pars media and pars lateralis (Eberstaller) of sulcus fronto marginalis (f m a₁₁ f m a₁₁₁, Fig. 12). Sulcus frontal inferior (f i, Fig. 13) is short and commences in front of ramus ascendens fissura sylvii (r a), from which it is separated only by a very narrow gyrus. After a short course forward it turns downward and ends in a short horizontal sulcus (f m a) a little in front of ramus horizontal foss sylv.

Sulcus præcentralis inferior (pr i, Fig. 13) is short, with a nearly straight vertical course. It communicates with sulcus frontal medius and from its lower part sends a short horizontal branch in gyrus front inferior (F. III). Sulcus præcentralis superior (pr s, Figs. 11 and 13) is composed of two sagittal sulci united in their middle by a vertical sulcus. The lower sagittal sulcus passes backward to the sulcus centralis, then cuts through gyrus centralis posterior and finally joins sulcus postcentralis (p o, Figs. 11 and 13). In this way both the central gyri are divided into an inferior and a superior portion.

On the mesial surface the gyrus central anterior forms the greater part of lobulus paracentralis (P A R, Fig. 14). On the upper surface it takes an unusually straight course. Gyrus frontal superior (F₁, Fig. 12) is very narrow in its anterior portion. Gyrus frontalis medius (F₁₁) is the most prominent of the frontal gyri and is divided into one superior and one inferior gyrus.

Nearly all of gyrus frontal inferior (F₁₁₁, Fig. 13) is taken up by Broca's convolution (operculum intermedium—Retzius), which is of an unusually large size and has an incisure on its surface.

LOBUS PARIETALIS, LOBUS TEMPORALIS AND LOBUS OCCIPITALIS.

Sulcus postcentralis (retrocentralis) (p o, Fig. 11) exists as a well marked sulcus. Below it starts in the fissura sylvii and goes slightly curved in the form of a retroverted S upward and backward. Not far from its upper end it joins a semicircular sulcus (p s, Fig. 11) which, with its posterior end, descends on the mesial surface of the hemisphere. This sulcus may be considered a sulcus parietalis superior (Retzius).

Laterally from parietalis superior a strongly developed sulcus interparietalis (i p, Figs. 11 and 13) is found. Posteriorly it enters the occipital lobe as a sulcus interoccipitalis (i op, Fig. 12) and ends as sulcus occipitalis transversus (o t). Anteriorly sulcus interparietalis has a vertical position, which is parallel with sulcus postcentralis and ends long before it reaches fissura sylvii. From this last named fissure a sulcus subcentralis posterior (Retzius) (s c p, Fig. 13) ascends toward sulcus interparietalis, thus completing the posterior boundary of gyrus ascendens. On the mesial surface of lobus parietalis a free sulcus subparietalis (s p, Fig. 14) exists, which anastomoses with a vertical sulcus precuneus (p c, Fig. 14).

The sulcus intermedium (i, Figs. 11 and 13) starts anteriorly from sulcus interparietalis, goes backward and downward, communicating with ramus ascendens fissura sylvii and with sulcus temporalis superior and turns finally upward, ending near the sulcus interparietalis.

The sulcus temporalis superior (t s, Figs. 11 and 13) in its posterior part, sends out one upward branch (t s₁, Fig. 14), anastomosing with the sulcus intermedium. Further back it

divides into two sulci, one (t s₁₁, Fig. 11) passing upward and ending near sulcus interparietalis; the other passes backward, joining the ascending part of the sulcus temp. medius.

The sulcus temp. medius is subdivided in three portions (t m₁ t m₁₁ t m₁₁₁, Fig. 13). The posterior portion anastomoses by a short, shallow sulcus with incisura preoccipitalis (pro, Fig. 13), thus forming a sulcus preoccipitalis (Wernicke), and communicates with sulcus temporalis superior and sulcus occipitalis lateralis superior.

The sulcus temporalis inferior (t i, Fig. 14) runs forward from incisura preoccipitalis and is represented by two separate sulci. A deep sagittal sulcus exists in gyrus fusiformis.

The sulcus occipito temporalis (fissura collateralis) (o t, Fig. 14) presents at its posterior end two transverse branches, the mesial of which reaches the occipital pole, the lateral ends in incisura preoccipitalis. At the point where it reaches the gyrus hippocampus it is joined by a sulcus lingualis (s l, Fig. 14).

Fissura parieto-occipitalis is typical, but anastomoses even deeply with fissura calcarina, which further on at the occipital pole, turns over on the lateral surface. Behind fissura parieto-occipitalis a sagittal sulcus cuneus extends (s c, Fig. 14) to the occipital pole.

On the lateral surface of the occipital lobe are found the sulcus occipitalis lateralis inferior (o l₂, Fig. 13) and the sulcus occipitalis lateralis superior (o l₁, Fig. 14), which anastomoses anteriorly with sulci temporalis superior and medius and sends one transverse branch which ends near sulcus interparietalis.

The gyrus centralis post (C P, Figs. 11 and 13) has a nearly straight course. It is very broad in its middle part and upward it passes into a short, thick gyrus parietalis superior medialis (p s, Fig. 11) (gyrus arcuatus ant.—Retz). A well-defined gyrus parietalis ascendens (P A, Fig. 11) that stretches backward to fissura parieto occipitalis is formed, giving the brain a three gyral type.

The gyrus supramarginalis (S M, Fig. 13) is divided by sulcus intermedium and not very well defined. Gyrus angularis (G A, Figs. 12 and 13) is well developed, but has taken the place of the gyrus parietalis inferior posterior. Lobus parietalis inferior as a whole is poor in gyri. Gyrus temporalis superior (T S, Fig. 13) is very narrow. Gyrus temporalis medius (T M, Fig. 13) is unusually broad and in its posterior portion perfectly insulated by sulci.

The gyrus fusiform (Fu, Fig. 14) is divided into two parts by a sagittal sulcus and is posteriorly perfectly insulated. Anteriorly it goes over in the gyrus hippocampi and the gyrus temporal inferior (T I, Fig. 13). The gyrus cinguli (C I, Fig. 14) is a well-developed gyrus. The pentagones anterior medius and posterior (mentioned by Brissaud) (P E N a. m. p., Fig. 14) are easily made out.

On the lateral surface of the occipital lobe are gyrus occipitalis medius and gyrus occipitalis inferior well marked.

On the insula and the other parts of the left hemisphere not mentioned nothing is found worth recording.

RIGHT HEMISPHERE.

Fissura sylvii (f sy, Fig. 15) is 51 mm. in length and at its posterior end is divided into two short rami posteriores (r p a, r p d). From its anterior portion one branch passes upward, which then bifurcates to form ramus anterior ascendens (ra, Fig. 15) and ramus anterior horizontalis (r h, Fig. 15), which latter anteriorly becomes sulcus fronto marginalis lateralis (Eberstaller) (f m a₁₁₁, Fig. 15). Although the two last named sulci communicate, the boundary between them is marked by a deep transverse gyrus (by x, Fig. 15). Behind ramus ascendens the sulcus diagonalis (d, Fig. 15) goes upward, sending forward a branch, which anastomoses with sulcus frontalis inferior. Sulcus diagonalis is shallow, not penetrating down to the insula, and consequently can not be considered as ramus ascendens fissura sylvii.

The sulcus centralis (c, Figs. 12 and 15) commences on the upper surface near fissura longitudinalis. It is slightly curved in its upper part, but its lower part has a straight course.

The sulcus cinguli (ci, Fig. 16) is continuous, but is double

in its pars anterior and pars intermedia (Eberstaller) (pa₁ pi, Fig. 16). Parallel to the lower part of sulcus cinguli the sulcus rostralis (ro, Fig. 16) runs forward, making an incision in the anterior edge of the hemisphere.

The sulcus præcentralis superior (prs, Fig. 11) in its upper part is about parallel with the sulcus centralis, but at the level of the middle of the hemisphere it goes backward and downward, cutting through the gyrus centralis anterior and the gyrus centralis posterior, ending in the sulcus postero-centralis (p o, Fig. 15). The lower portion of the sulcus præcentralis superior thus has the same course in both hemispheres, dividing the gyri centrales in the same way on both sides. An upper short and shallow branch goes forward from sulcus præcentralis superior, and a little laterally to this branch the sulcus frontalis superior (f₁, Fig. 11) passes forward. The latter soon divides into two sulci, neither of which reaches the orbital margin. The lateral branch of the sulcus frontalis superior (f₁₁, Fig. 11) might be considered as an upper portion of the sulcus frontalis medius. The sulcus frontalis medius (fm, Fig. 12) starts a little back of the anterior end of the sulcus frontalis superior, branches freely and ends in sulcus fronto marginalis medius (f m a₁₁, Fig. 12), (Eberstaller). The sulcus præcentralis inferior (pr i, Fig. 15) is connected with the sulcus frontalis inferior, but ends free, both in its upper and lower end, with which latter it nearly reaches fissura silvii.

The sulcus frontalis inferior (fi, Fig. 15) even on this hemisphere is short and anastomoses anteriorly with sulcus diagonalis (d). From fissura sylvii a sulcus subcentralis anterior (sb a, Fig. 15) is directed upward into gyrus centralis anterior. On the orbital surface a circular sulcus exists, from which three radiating sulci are directed forward and three backward.

The gyrus frontalis superior (F₁) is broader than on the left side, but the gyrus frontalis medial (F₁₁) is even in this hemisphere the most prominent. The gyrus frontalis inferior (F₁₁₁, Fig. 15) is small, with a small operculum intermedian. The gyrus centralis anterior (C A, Fig. 11) is, as mentioned, divided by the sulcus præcentralis superior into one superior and one inferior portion. The gyrus, which takes a nearly straight course, is narrow in its upper part, but is otherwise broad. The gyrus paracentralis is oval and anteriorly bounded by sulcus præcentralis medialis (pr m, Fig. 16).

The gyrus centralis posterior (C P, Fig. 11) is also divided by sulcus præcentralis inferior into one superior and one inferior portion. It is broad and takes a straight course.

LOBUS PARIETALIS, LOBUS TEMPORALIS, LOBUS OCCIPITALIS.

The sulcus postcentralis (retrocentralis) (p o, Figs. 11 and 15) is continuous, passes upward from fissura sylvii and ends in a sulcus that arches around incisura sulci cinguli (i c, Fig. 16). This arciform sulcus (p o s, Fig. 11) is probably best considered a part of sulcus postcentralis superior. Behind sulcus postcentralis superior a typical sulcus parietalis superior (Retzius) (p s, Fig. 11) exists, which starts on the mesial surface and then goes transversely over the superior surface, sending one branch backward. The sulcus interparietalis is represented by two sulci. The anterior (i p a, Figs. 11 and 15) has one vertical forward concave part, which forms the posterior boundary of gyrus parietalis ascendens. Between the anterior end of sulcus interparietalis and fissura sylvii is found a short compensatory (Retzius) sulcus, which arches around the end of fissura sylvii. It is identical with the sulcus that Brissaud names sulcus lobuli parietalis inferior (sp i, Fig. 15). The posterior part (i p p, Fig. 11) of sulcus interparietalis is a short angular sulcus. With a short transverse part at its anterior end it helps to form the posterior boundary of gyrus parietalis ascendens. Posteriorly it communicates with the fissura parieto-occipitalis (f p o, Fig. 11) and ends behind this fissure as sulcus interoccipitalis (i o p, Fig. 11). Sulcus subparietalis (sp, Fig. 16) is anteriorly connected with sulcus cinguli and posteriorly with sulcus præcuneus (p c, Fig. 16). The sulcus temporalis superior (t s, Fig. 16) is divided by a narrow gyrus into one anterior portion, which is double, and one posterior portion,

which extends high up in the parietal lobe and here joins the interparietal furrow.

The sulcus temporalis medius (t m, Fig. 15) is broken up into several short sulci. Its ascending part sends one anastomosis forward to sulcus temporalis superior and divides then into two branches, which both extend upward, ending (t m, Fig. 11) as deep sulci in sulcus interparietalis posterior. The sulcus temporalis inferior (t i, Fig. 16) is well developed. It communicates with incisura præoccipitalis and joins posteriorly sulcus occipito temporalis (o t, Fig. 16).

The sulcus occipito temporalis (o t, Fig. 16) extends backward to the occipital pole and is joined at its central part by a sulcus lingualis (s l, Fig. 16). A three-stellated free sulcus exists in the posterior part of gyrus lingualis (s li, Fig. 16). The incisura præoccipitalis (pr o, Fig. 15) extends upward on the lateral surface of the occipital lobe, forming a sulcus preoccipitalis. Short branches go out from it forward and backward, which together might be considered to form a sulcus occipitalis lateralis inferior (ol₃, Fig. 11). The fissura calcarina is typical.

The fissura parieto occipitalis (f p o, Figs. 11 and 16) communicates only by a shallow furrow with fissura calcarina and takes on the upper surface a very remarkable course. There it communicates with sulcus interparietalis, but continues further laterally and divides then into two branches, one going forward, the other backward. The anterior branch goes forward and downward and soon joins a longitudinal furrow which runs on the lateral surface of the lobus occipitalis. The posterior branch goes backward, but turns forward again and joins also the above-mentioned longitudinal sulcus (o l₁, Fig. 15). This longitudinal sulcus goes downward and backward and sends, near its end, a branch forward, which anastomoses with another longitudinal sulcus (o l₂, Fig. 15), which enters anteriorly the temporal lobe and reaches posteriorly the occipital pole.

The fissura parieto occipitalis keeps its depth even laterally to sulcus interparietalis and becomes more shallow first at the point of division. The here described course of fissura parieto occipitalis is certainly abnormal and very seldom seen in the human brain. It corresponds to the course the fissura parieto occipitalis usually takes in the monkey brain and to what is described in the brain of the Egyptian by Elliot Smith.¹

On the upper surface of the occipital lobe exist several small irregular sulci.

The gyrus parietalis ascendens (P A, Fig. 11) is well defined in its upper part, and forms here an irregular gyrus which can be followed to fissura longitudinalis. The posterior part of gyrus parietalis superior forms a plump polygonal gyrus. The gyrus supramarginalis (S M, Fig. 15) is beautifully formed. The gyrus angularis (G A, Fig. 15) takes its regular place, but is cut by the anastomosis between the sulcus temporalis superior and the sulcus interparietalis. The gyrus temporalis superior (T S, Fig. 15) is not as narrow as on the left side and continues upward in the gyrus supramarginalis and the gyrus angularis. The gyrus temporalis medius (T M, Fig. 15) is in its anterior part very broad, but becomes narrow in its gyrus angularis ascending part. The gyrus temporalis inferior (T I, Fig. 15) is rather narrow. The gyrus præcuneus (P C, Fig. 16) is divided by the sulcus præcuneus into one gyrus præcuneus anterior and one posterior. The cuneus (C U, Fig. 16) has one sulcus which runs parallel to the fissura calcarina.

The gyrus lingualis (L, Fig. 16) and fusiformis are well developed. The gyrus cinguli (C I, Fig. 16) is well formed and shows the three pentagones very distinctly (PENa PENm PENp, Fig. 16). About the other parts of the hemisphere nothing remarkable.

MICROSCOPIC EXAMINATION.

The pia is normal. The intima piæ runs perfectly smooth over the gyri, but most of its veins are distended with blood and in some places hemorrhages are found, both on top of

1. The Morphology of the Occipital Region of the Cerebral Hemisphere in Man and Apes. Anatomische Anzeiger, vol. xxiv.

the gyri and between them. The largest of the hemorrhages may be seen with the naked eye as punctiform. They exist also in the brain substance, usually the white, and are most numerous in the motor region, and in the frontal and temporal lobes, but may be occasionally found in the other parts of the hemisphere and in the central ganglia. They have all the character of fresh hemorrhages and must have occurred shortly before death. The blood vessels in the pia and in the brain substance are normal. Their walls are not thickened and no infiltration exists around them, but in the parts of the brain

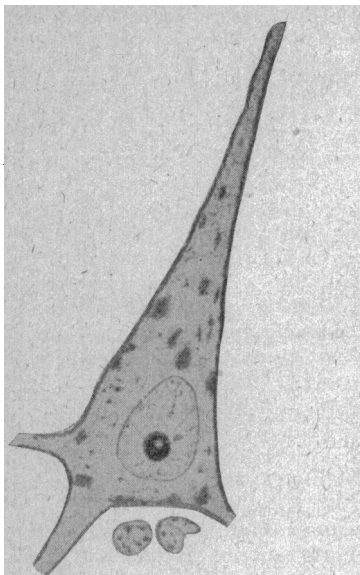


Fig. 5.—Pyramidal cell from the deeper part of cortex. (Leitz system 1/12, ocul. iii.) (Rosin.)

where the hemorrhages are numerous a slight edema is found around some of the veins. There is no measurable change in the thickness of the cortex, and estimations of the number of the ganglion cells have given the same figures as in normal brains. The giant ganglion cells (Betz) are normal, but in the large pyramidal cells and in those of medium size a slight chromatolysis was frequently met with (Fig. 5). In the layers of the medium sized and the small pyramidal cells,

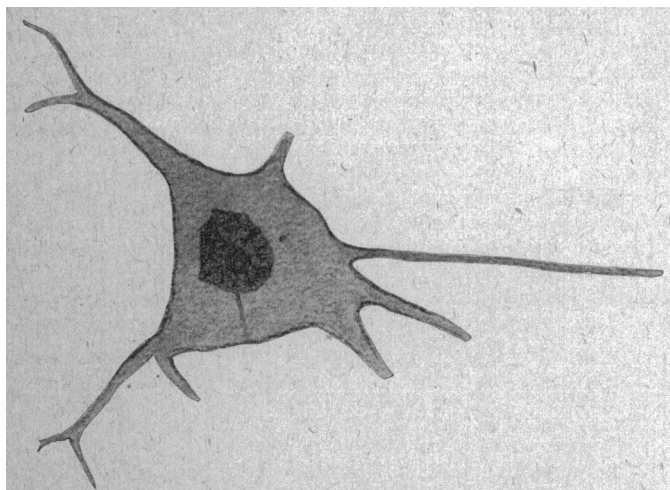


Fig. 6.—Degenerated pyramidal cell from the middle layer of cortex. (Leitz system 1/12, ocul. iii.) (Rosin.)

especially in those parts of the brain that showed numerous hemorrhages, many cells were found showing the degeneration described by Hoche (Fig. 6). The same kind of degeneration was also found in nucleus dentatus.

Notwithstanding repeated trials it has been impossible to stain the neuroglia with any of the elective methods. There seems, however, to exist a slight increase in the neuroglia nuclei in the motor region and parts of the frontal lobe, but the in-

crease is so very slight that I do not venture to decide positively if the glia is increased or not.

The number, size and arrangement of the medullated fibers correspond perfectly to the normal. In Figs. 7, 8, 9 are reproduced sections from different parts of the brain, and in Fig. 10 a section from a sane man of the same age as the patient.

Section 10 is about 5 microns thicker than the other sections and serves mostly as a control of the reproduction. The thickness of the other sections varies between 15 and 20 microns. They show the different strata and the radiating fibers without any perceptible deviation from what normally is found in the corresponding gyri. The ependyma is normal in the lateral ventricles, the aqueductus sylvii and the fourth ventricle. Especially important is that the brain tissue beneath the above mentioned depressions in the frontal lobe was found perfectly normal in all its layers.

The brain can be said to have in general broad gyri, but it does not present altogether well formed and regular gyri and sulci. Although there is nothing in the formation of the gyri and sulci that can be classed as positively pathologic, certain features are worth mentioning. Thus the right frontal lobe does not extend as far back as the left, and several of the gyri give the impression of a conformation lacking in proportion and symmetry as, for instance, Brocas gyrus, certain parts of the gyri centrales, parietalis and temporalis. Furthermore, an unusual number of deep anastomoses between the great sulci and, finally, the abnormal development of fissura parieto-occipitalis. It may be that the irregularities mentioned have not any significance, but, on the other hand, they may be signs of a faulty development. As heredity is generally considered one of the most important factors in the etiology of circular insanity, it has seemed to me important to look for formations that could possibly indicate a faulty development and to make the material available to comparison with brains from other cases of the same disease, I deemed it necessary to give a full description of the gyri and sulci with accompanying drawings. The microscopic examination has, as seen, not given any positive results, except the finding of hemorrhages, the slight chromatolysis and the Hoche's degeneration, and all these changes have their natural explanation in the typhoid fever of which the patient died. But, on the other hand, the microscopic examination has strengthened the clinical diagnosis, making it possible to exclude a dementia paralytica.

One distinct pathologic change has been found in this case and that it is the exostoses with adherent dura. Now the opinion has been expressed that exostoses could perhaps have some influence on the development of a circular insanity by the changes they cause in the cortex. In Dr. Dewey's case, however, there was not found the slightest alteration of the cortex in that part of the brain which was exposed to a supposed pressure of the exostoses, and I can not, under such circumstances, see any reason to suppose that the exostoses have had any influence on the insanity in this case.

SOME GRAPHIC OBSERVATIONS OF ANKLE CLONUS.

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PHILADELPHIA.

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The phenomenon known as ankle clonus consists in alternate extension and flexion of the foot on the leg, repeated a varying number of times, following passive flexion of the foot effected rather rapidly and forcibly and maintained somewhat firmly. It is best induced with the leg flexed slightly on the thigh and supported in the palm of one hand, although it can at times be