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ON THE DEVELOPMENT OF THE LARGE COMMISSURES OF THE TELENCEPHALON IN THE HUMAN BRAIN.

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THE brains of five human embryos, ranging from the beginning of the fourth month to the end of the eighth, were at my disposal for these researches. Two of these brains, one of the beginning and the other of the middle of the fourth month, were cut into a series of frontal sections. These sections were stained by means of hæmatoxylin and eosin. Of the three other brains only a median section was made for studying the later stages of development of the corpus callosum.

The sections I made of the brain at the beginning of the fourth month were 20 μ thick and slightly deviated from the frontal plane, so that the sections meet the hemisphere, which is on the left in the photographic reproductions, in a more posterior level than the one on the right. In this brain the anterior commissure is so far histologically differentiated that it can be recognized as a strand of a fibrous structure; among the fibres are spread the nuclei of glia cells. The diameter of the commissure amounts to 0·4 mm. The commissure lies partly in the lamina terminalis, covered from behind by the ependyma of this lamella. The more anterior part of the commissure probably exceeds the limit of the lamina terminalis. This limit cannot be traced out with certainty, because in front of the lamina, and in direct continuity with it, the mesial surfaces of the pallium are united.

The corpus callosum and the fornix are also recognizable, but the tissue composing these systems is not so far differentiated as that of the

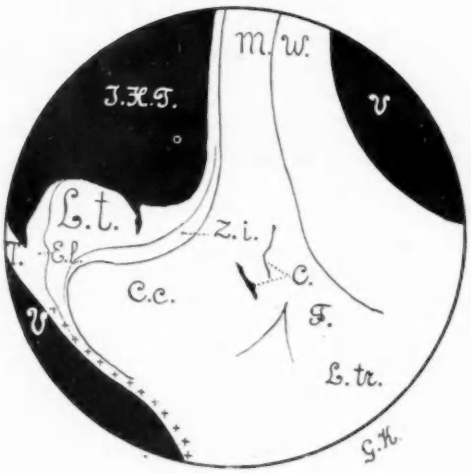
anterior commissure. Both systems are built up by a reticular tissue, the meshes of which are irregularly shaped, with a prevalence of hexagonal ones. In one direction the meshes are stretched, whilst in another direction, at right angles with the first, the diameters of the meshes are pretty nearly equal. Now the direction in which the meshes are stretched coincides with the ultimate direction of the fibres which differentiate out of this meshwork. Therefore we are able to recognize in the sections different directions, in correspondence with the final course of the nerve fibres generated by this tissue. Beyond its particular structure the reticular substance of the corpus callosum and of the fornix stains more deeply with eosin than does the surrounding tissue; moreover, it is characterized by a considerable want of nuclei; only here and there glia cells with dark stained nuclei are met with. Large capillary loops are seen in the reticular substance of the corpus callosum, penetrating from the surrounding connective tissue into the substance of the commissure.

If we compare the tissue of the commissura anterior with that of the corpus callosum, the difference is very noticeable. In the anterior commissure the transverse septa have largely disappeared and the longitudinal septa have become thicker, resulting in a more fibrous structure. Moreover, the fibres are united in bundles, which are wrapped up by glia cells.

In order to determine the place where the corpus callosum originates I reproduce two frontal sections through the lamina terminalis. The first of these sections (figs. 1 and 1A) just touches, at the left, the ependyma (E.l.) of the lamina terminalis (L.t.). At this place the lamina is continued in a tænia (T.), which goes over into the mesial wall of the pallium. What appears in the preparation as a tænia is the transverse section of an ependymal membrane. This fleece is the continuation of the lamina terminalis, which is bent in and then passes over into the mesial wall of the pallium. A little backwards this membrane is invaginated into the lateral ventricle forming the ependymal covering of the plexus choroideus of this cavity.

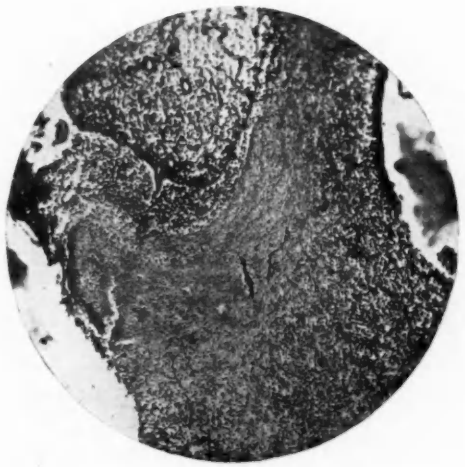
Now in the lamina terminalis, at a little distance from its superior margin, the corpus callosum (C.c.) is situated. At the left appears the lateral border of the commissure. This edge is convex and bordered by the ependymal cells of the lamina terminalis. Here the section is torn, so that at the place I indicate by crosses the connection between the lamina terminalis and the wall of the pallium is broken. Going to the right in the section, the continuation of the callosum into the mesial

wall of the pallium is visible. The superior border of the commissure, so far as it lies in the lamina terminalis, is concave and covered from



Frontal section through the lamina terminalis.
Human embryo of the beginning of the fourth month.
C., capillaries.
C.c., corpus callosum.
E.l., ependymal layer.
F., fornix.
I.H.T., interhemispherical tissue.
L.t., lamina terminalis.
L.tr., lamina trapezoidea.
M.W., mesial wall of the pallium.
T., tænia.
V., lateral ventricle.
Z.i., intermediate zone.

FIG. 1.



Frontal section through the lamina terminalis.
Human embryo of the beginning of the fourth month. Photograph Zeiss obj. B.B., enl. $\frac{4}{7}$. Fig. 1 is the outline of this photograph; the crosses in that figure indicate the place where the section is torn.

FIG. 1A.

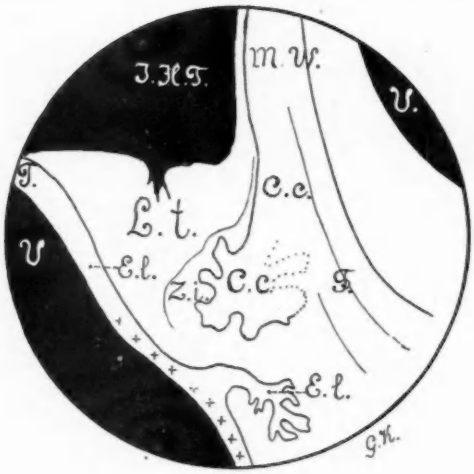
above by the tissue of this membrane. The inferior border, which is the direct continuation of the lateral border, is convex

Aside from the corpus callosum, and entirely confined to the mesial wall of the pallium, lies the fornix (F). In the mesial wall the two systems cannot be distinguished. At the place where the systems diverge the difference becomes evident, the callosum forming a dense meshwork, whilst the tissue of the fornix spreads out in passing over into the lamina trapezoidea (His). Apart from the spreading out there is also the difference in the form of the meshes, those of the fornix being more stretched.

In figs. 2 and 2A I reproduce a section through the most posterior part of the callosum (C.c.) lying in the lamina terminalis. It shows this most posterior part also at a little distance from the superior border of the lamina. The tissue in which it is imbedded (Z.i.) consists of cells, the nuclei of which are more distant and stain less intensely than those of the ependyma. A narrow band of this tissue penetrates between the commissure and the ependyma, which is situated at the left and underneath the commissure. The same fact may be observed in fig. 1, where at the left a small strip of this tissue is also interposed between the callosum and the ependymal layer of the lamina terminalis. This proves that the posterior part of the callosum, though situated in the lamina terminalis, does not touch the ependyma, but is always separated from it by a thin layer of non-ependymal cells. These cells belong to the layer which in this stage of development is described by His as the intermediate layer (*Zwischenschicht*) [8 (pp. 91, *et seq.*)] of the wall of the pallium. The distance by which the most posterior part of the callosum is separated from the posterior surface of the lamina terminalis amounts to 0·1 mm.

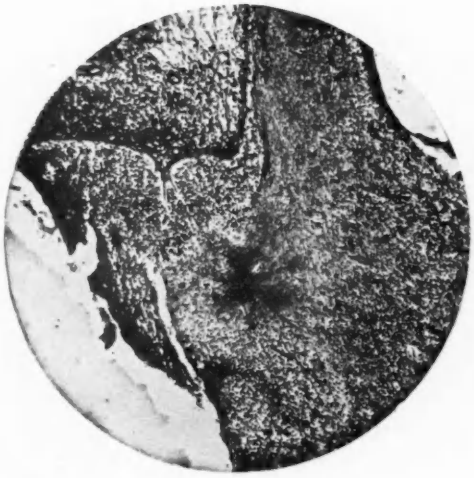
In the more frontal region of the callosum the relations are less simple, being complicated by the union of the mesial walls of the pallium. The fronto-occipital diameter of the united part of the laminæ trapezoideæ attains to 0·32 mm. Both pallia meet in the zone of union at a sharp angle, in which the interhemispherical tissue (commonly called the falx cerebri) penetrates like a wedge. This formation is built up by fusiform cells with elongated nuclei; as to its structure, it is a lacunar tissue, the lacunæ of which are filled up with blood-corpuscles. At a little distance from the wall of the pallium the lacunæ are wide, and as such easily recognizable, but approaching the wall they become smaller, and at last are not to be distinguished from the small slits always occurring in connective tissue. In these small clefts, however, I often find one or two blood-corpuscles. For this reason I believe that at least some of these clefts have a connection with the larger lacunæ. Besides

these lacunæ small blood-vessels are met with in this tissue which pass over into capillaries. Quite near the wall of the pallium the nuclei of



Frontal section through the lamina terminalis just touching the most posterior part of the corpus callosum.
Human embryo of the beginning of the fourth month.
C.c., corpus callosum.
E.l., ependymal layer.
F., fornix.
I.H.T., interhemispherical tissue.
L.t., lamina terminalis.
M.W., mesial wall of the pallium.
T., tænia.
V., lateral ventricle.
Z.i., intermediate zone.

FIG. 2.



Frontal section through the lamina terminalis just touching the most posterior part of the corpus callosum.
Human embryo of the beginning of the fourth month. Photograph Zeiss obj. B.B., enl. $\frac{4}{1}$. Fig. 2 is the outline of this photograph; the crosses in that figure indicate the place where the section is torn.

FIG. 2A.

the connective tissue are arranged with their long axes parallel with this wall. The outer surface of the pallium itself is covered by a thin layer

of glia cells with rounded nuclei. This sheath is one cell thick. Approaching the zone of union the elongated nuclei converge towards the top of the wedge so that this top is formed by two or three of these nuclei. Now these nuclei are pressed together very flat, perhaps a little longer than the nuclei of the adjacent cells, and thereabove curved, or bent like an S. At some places one of these nuclei seems to penetrate a little into the substance of the corpus callosum and is less stained than the other nuclei. I should be inclined to interpret the process I just now described as a marginal atrophy of the interhemispherical tissue.

The substance of the corpus callosum along the lateral border of this wedge-shaped interhemispherical tissue shows stretched meshes; towards the top these meshes become shorter and more hexagonal, whilst at the same time they become smaller. The mesh-work itself at this spot stains more deeply with eosin. The whole structure gives the impression as if the tissue of the callosum sharply bends round the top of the interhemispherical tissue and is compressed. I think, therefore, that the growing commissure exerts a pressure upon the interhemispherical tissue and that this is the direct cause of the atrophy of this membrane.

Now this process of atrophy must be sharply distinguished from the ingrowth of capillaries into the commissure. In the brain under description these capillaries mainly penetrate from above through the superior border of the lamina terminalis. Not only capillaries, but also a larger blood-vessel, pierces this lamina. At the border of the commissure the vessel goes over into a capillary that penetrates into the callosum (C.c., fig. 1). It follows already from the bare topographical relations that the atrophy of the interhemispherical tissue and the ingrowth of capillaries from this tissue are two distinct processes, which may take place independently of each other. In most cases, however, especially in the more advanced stages of the development, the two processes are mixed up in such a manner as to make an analysis quite impossible. From the drawings of Zuckerkandl [30] I should be inclined to conclude that in the rat this is the case from the very first. Probably the difficulty which the analysis offers in this case induced the author to describe a *Durchwachsung* of the falx cerebri by the fibres of the callosum. In my opinion this statement is erroneous, as I never found free ending fibres nor rudiments from the connective tissue other than in direct connection with the blood-vessels that pierce the lamina terminalis and penetrate into the commissure.

The fronto-occipital diameter of the callosum amounts to 0·5 mm.; about one-third of this (0·18 mm.) is situated in the lamina terminalis,

and two-thirds (0·32 mm.) transgress the limits of the lamina and lie in the zone of union. The thickness of the lamina terminalis attains to 0·28 mm. Fig. 3 represents these relations diagrammatically.

The most posterior part of the callosum passing over from the lamina terminalis into the wall of the pallium is recurved backwards. It may be followed for some distance in an occipital direction, reminding one of the final state when there is a forceps major. In the same way the more anterior part of the callosum is continued in a frontal direction. The fornix, which lies laterally of the callosum, cannot be distinguished from it in the more occipital region. There the two structures together occupy a triangular field with its top turned upwards. The impossibility of discriminating between the two structures is caused by the fact that in this region the fornix and the callosum have about the same direction, so

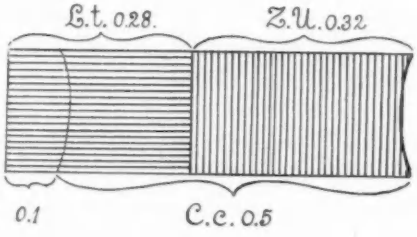


FIG. 3. Eul. $\frac{100}{1}$.
C.c., corpus callosum.
L.t., lamina terminalis.
Z.U., zone of union.

that there is no appreciable difference in the shape of the meshes. At the place where the callosum passes over into the lamina terminalis the two structures diverge, and this produces a difference in the shape of the meshes in the frontal section. The fornix spreads out and goes over into the lamina trapezoidea on its way to the hypo-thalamus. In the more frontal region the fornix is no longer present and only the callosum remains in the wall of the pallium. Callosum and fornix in the lamina terminalis, as well as in the mesial wall of the pallium, are strictly confined to the intermediate layer of this wall.

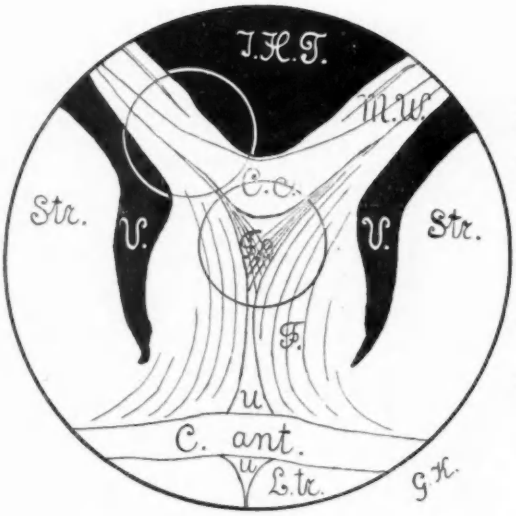
In its general features I find that the structure of the mesial wall of the pallium corresponds with the description given by His [8 (pp. 91 and 111)]. First comes an ependymal layer bordering the lateral ventricle, then a matrix (*Matrix*), next the intermediate stratum (*Zwischenschicht*) ; upon this follows the cortex layer (*Rindenschicht*), which is

followed by a marginal zone (*Randschicht*), covered from the outside by a thin sheath of glia cells (membrana limitans externa). Changes appear in this structure at the level occupied by the callosum (*cf.* figs. 9 and 9A). The matrix layer becomes thinner, those cells arranged tangentially in respect to the surface of the pallium wall disappearing first, those radially placed disappearing next. The breadth of the intermediate layer increases, and at the same time this stratum gets richer in nuclei, especially around the fibres of the commissure; these nuclei surround this structure like a cap (C.n.). The cortex layer, like the matrix, becomes thinner, bends somewhat inwards and ends in a sharp edge at a little distance from the under border of the mesial wall of the pallium. Now, in this stage of development this ending of the cortex is not so evident as it is afterwards (figs 9 and 9A). The difference in the density of the nuclei between the intermediate layer, the cortex and the marginal zone, at the level of the edge of the cortex, is trifling, so that it is difficult here to recognize the cortex layer. That the cortex really does end may be deduced from the fact that the nuclei of this stratum show a radial arrangement, whilst this is not the case with the nuclei of the two other strata. Therefore I cannot agree with the statement of His [8 (p. 129, figs. 84 and 86)] that the cortex layer is enlarged and forms a prominence at the external surface of the pallium.

Neither have I been able to find a fissura arcuata, but at about the same level, where it is described by Goldstein [5], the inner surface of the mesial wall shows a notch. In the frontal region this indentation begins very flat, but towards the level of the lamina terminalis it grows deeper and sharper. With the indentation of the inner surface corresponds a notch at the outer surface, which lies a little lower; at the same time the wall of the pallium grows thinner, so that the place between the two notches is the most slender part of the pallium wall. In my sections there is at this place a beginning of folding in of the wall, which is doubtlessly artificial. I therefore agree in this matter with Hochstetter [9], Mall [14], Retzius [20], Goldstein [6], and others who consider the folds of the wall of the pallium in these stages of development purely artificial.

The brain of the embryo of the middle of the fourth month was not so well preserved as the first one. The embryo, when born, showed symptoms of luetic infection. In the frontal region, at the place of the arched fissure, the wall of the pallium on both sides is folded in exactly as is drawn by His [8] in fig. 86. This fold,

in the region of the callosum, goes over into a rupturing of the wall. At the place of the rupture the walls are bent in, so that these parts of the mesial walls, which are adjacent to the callosum, diverge abnormally. This divergence influences the form of the interhemispherical tissue, but not in such a degree as entirely to disturb the normal relation of this structure to the mesial wall of the pallium. It appears, from a comparison of the various dimensions, that this brain agrees in all respects with that described by Goldstein [5]. The



Frontal section through the zone of union at the more posterior part of the callosum. Human embryo of the middle of the fourth month. Outline of a photograph, enl. $\frac{12}{1}$.
C. ant., anterior commissure.
C.c., corpus callosum.
F., fornix.
F.c., fornix commissure.
I.H.T., interhemispherical tissue.
L. tr., lamina trapezoidea.
M.W., mesial wall of the pallium.
Str., striatum.
U., zone of union.
V., lateral ventricle.

FIG. 4.

sections I made varied in thickness; some are 15 μ, some are 20 μ, according to the region of the cerebrum.

In this brain the anterior commissure has a diameter of 0·6 mm. The more frontal part lies in the zone of union, the more posterior part in the lamina terminalis, covered from behind by the ependyma. It protrudes into the cavity of the third ventricle, a relation which is also often met with in the adult state. Large glia cells from the zone of union are spread between the various fasciculæ of the commissure. The corpus callosum lies entirely in front of the foramen of Monro, its posterior border agreeing exactly with the frontal border

of the foramen. The tissue of the commissure is further histologically differentiated, having at this stage a more fibrous structure. This is also the case with the fornix. The fronto-occipital diameter of the callosum amounts to 2·4 mm. The most posterior part of it still lies in the lamina terminalis, but this relation is by no means so evident as it is in earlier stages of development. The lamina itself has become thicker, attaining now to a diameter of about 0·5 mm.

Fig. 4 gives the outlines of a section through the zone of union at the more posterior part of the callosum. The great commissure (C.c.) forms a closed system, which occupies the most dorsal part of this zone. On both sides the fibres of the commissure pass over into

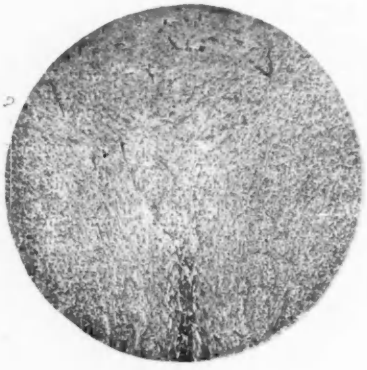


FIG. 4A.

Photograph of that part of the section which is indicated by a black circle in fig. 4. Photograph Zeiss obj. B.B., enl. $\frac{36}{1}$.

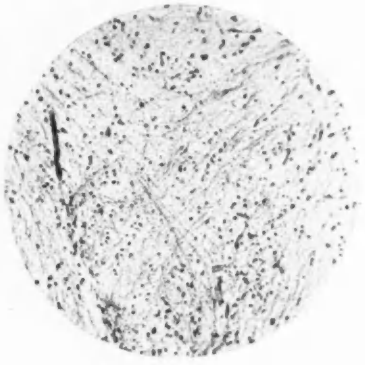


FIG. 4B.

Same section as fig. 4A; fornix commissure more strongly magnified. Photograph Zeiss obj. D.D., enl. $\frac{70}{1}$.

the mesial wall (M.W.) of the pallium in the same way as in the foregoing stage the fornix (F.) lies laterally of the callosum. The bundles of the fornix spread out as they pass over into the lamina trapezoidea (L. tr.). They may be followed in the section as far as the anterior commissure (C. ant.), when they bend sideways in crossing this system.

At the angle where callosum and fornix meet, a bundle of fibres appears, coming from behind. This bundle spreads towards the median plane and crosses a bundle coming from the opposite direction. This crossing system belongs to the fornix, and represents the fornix commissure (F.c.). These relations may be illustrated by fig. 4A, which

is the photographic reproduction of that part of the section that is indicated by a black circle in fig. 4; and by fig. 4B, which shows the structure of the fornix commissure more strongly magnified.

The crossing of the fornix fibres lies entirely in the zone of union, as the glia nuclei can be followed dorsally of this commissure and even in the adjacent layers of the callosum. The fronto-occipital diameter of the fornix commissure is 0·24 mm. In respect to the callosum, the fornix commissure is situated in such a way that the posterior border of the callosum exceeds the posterior border of the fornix commissure by 0·6 mm. Fig. 5 represents this relation diagrammatically.

In the region in front of the fornix commissure the callosum lies in the zone of union, whilst the fornix is confined to the mesial wall of the pallium and its continuation, the lamina trapezoidea. Only the most internal fibres of the fornix leave the wall of the pallium and pass

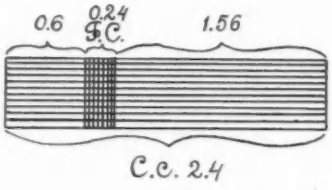


FIG. 5.

C.c., corpus callosum.
F.C., fornix commissure.

through the glia tissue of the zone of union. From the relation I described between the callosum, the fornix and the fornix commissure, I should be inclined to conclude that the more posterior part of the corpus callosum in this stage of development already agrees with the splenium, as in the adult stage only this part of the commissure shows the same relation to the fornix commissure; it follows from the topographical relation of the more anterior part of the corpus callosum to the fornix bundles that this part of the large commissure agrees with the corpus and the genu of the callosum in the adult stage. Now, this view is further supported by the course of the fibres of the callosum. The more posterior fibres passing over into the mesial wall of the pallium bend in occipital direction, exactly in the same way as do the fibres from the splenium.

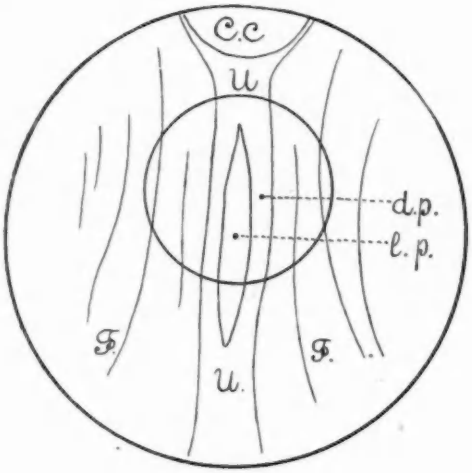
The same relation exhibits the more frontal fibres in respect to the frontal region of the brain, whilst the fibres of the middle part of the

callosum have a more transverse direction. Moreover, the position of the fibres is such that in the posterior part of the callosum the fibres coming from more posterior regions lie nearer the surface than those coming from more anterior regions. Now the fibres, in proportion as they come from more occipital regions, have a more horizontal direction in the wall of the pallium, and this is the reason why in the preparations the frontal section of the callosum is triangular. The anterior part of the callosum shows a symmetrical condition in respect to the frontal region of the brain. In a schematic figure His [8] gives the same relations for a somewhat older embryo (fig. 93); only I have not been able to find the free ending fibres that His represents.

The zone of union of the pallia, in the frontal section, is triangularly shaped, with the base turned downwards (U, fig. 4). From above it is bordered by the callosum, laterally by the fornix bundles, and from below by the anterior commissure. It extends also a little below this commissure. This field is also triangular, the base resting against the commissure and the top turned towards the ependyma of the third ventricle. The tissue of which it is composed consists of glia cells. These cells are flat and mostly square or hexagonal. The nucleus is round and large and lies within a cell body that stains deeply with eosin. The cells form a meshwork with small meshes. The glia cells penetrate between the fibres of the callosum and also between the more internal bundles of the fornix. At the place where the anterior commissure passes through the zone of union they enter in large numbers into the substance of this commissure.

In the more posterior part of the zone of union, at the level of the commissure of the fornix, the structure of this tissue becomes loose in its middle portion (figs. 6 and 6A, l.p.). Here the nuclei are more distant, some of them are larger, and stain badly with hæmatoxylin; at the same time the meshes become larger. Adjacent to the fornix bundle the tissue is more dense (d.p.); the transitions between the looser and the denser parts are pretty abrupt. Now in my opinion we have here to deal with the beginning of a cleavage in the glia tissue as a rudiment of the cavum septi lucidi. The process extends in a fronto-occipital direction over a distance of 0·25 mm. As may be observed in the photographic reproduction (fig. 6A), the difference in the structure of the tissue does not give the impression of being artificial, because the shrinking of the tissue has only caused rents and fissures which are easily recognized. Independently of the mode of origin of the cavum, it is evident that the septum lucidum comprises more than the original

mesial wall of the pallium, for the most internal fibres of the fornix pass through the glia tissue of the zone of union. The process of



Frontal section through the more posterior part of the zone of union, at the level of the fornix commissure.
Human embryo of the middle of the fourth month. Outline of a photograph enl. $\frac{4}{1}$.
C.c., corpus callosum.
d.p., dense part of the glia tissue.
F., fornix.
l.p., loose part of the glia tissue.
U., zone of union.

FIG. 6.

Photograph of that part of the section which is indicated by a black circle in fig. 6. Zeiss obj. D.D., enl. $\frac{124}{1}$.

FIG. 6A.

union of the two pallia does not differ in its general features from the process I described in the younger stage. Only the atrophy of the

interhemispherical tissue and the ingrowth of capillaries coincide at several places and makes an analysis of the process illusory. At these spots at first sight a distinct line of demarcation between the interhemispherical tissue and the mesial wall seems to fail, but a close observation seldom fails to reveal some vestige of this line of demarcation. By the ingrowth of the capillaries the mesial wall gets, in the frontal section, a serrated appearance, and this causes distinct demarcation to disappear.

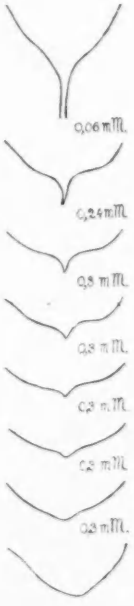
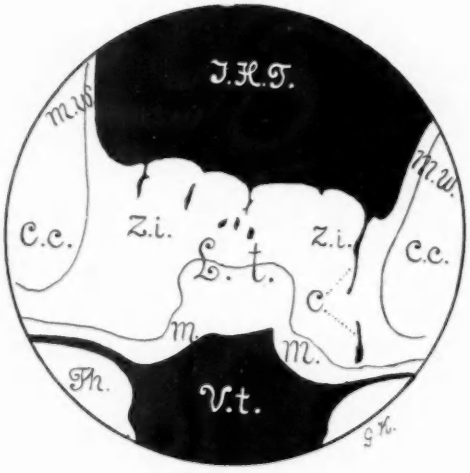


FIG. 7.

Successive profiles of the zone of union ; the numbers indicate the fronto-occipital distance between the successive profiles.
Human embryo of the middle of the fourth month.

In the more frontal part of the zone of union the walls of the pallium meet at an acute angle, into which the interhemispherical tissue penetrates. The fibres of the callosum sharply bend round the top of the connective tissue. I have not seen any fibres ending free near the edge of the pallium wall in front of the zone of union. Going backward the notch between the two pallia gets more and more flattened and superficial (fig. 7). The fibres of the callosum show an equivalent relation and are still pressed against the notch.

From the combined observations I should be inclined to believe that the enlarging commissure pushes forward the interhemispherical tissue,



Frontal section through the lamina terminalis behind the corpus callosum.
Human embryo of the middle of the fourth month. Enl. $\frac{4}{1}$.
C., capillaries.
C.c., corpus callosum.
I.H.T., interhemispherical tissue.
L.t., lamina terminalis.
M., matrix.
M.W., mesial wall of the pallium.
Th., thalamus.
V.t., third ventricle.
Z.i., intermediate zone.

FIG. 8

Frontal section through the lamina terminalis behind the corpus callosum.
Human embryo of the middle of the fourth month.
Photograph Zeiss obj. B.B., enl. $\frac{4}{1}$.
Fig. 8 is the outline of this photograph.

FIG. 8A.

and partially brings it to atrophy. The question often put forward, whether the union of the pallia takes place in consequence of the

protrusion of the lamina terminalis or otherwise by a more direct process, is a very subtle one; my preparations are not sufficient to enter upon this question.

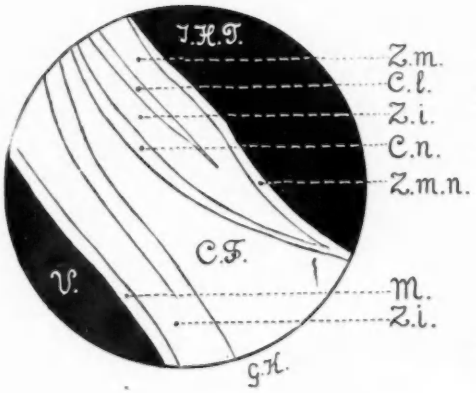
Behind the corpus callosum the lamina terminalis (figs. 8 and 8A) is continued over a short distance (0·6 mm.). The posterior border of the lamina is folded in and goes over into the lamina choroidea of the third ventricle. It forms the roof of the foramen of Monro, and has no direct relation to the callosum. In its structure this part of the lamina agrees with the adjacent mesial wall of the pallium. The lamina consists of an ependymal layer turned towards the foramen of Monro, then a matrix (M.) and thereupon an intermediate layer (Z.i.). A distinct cortex and marginal zone do not seem to exist. The diameter of the lamina attains 0·44 mm. From the outside a large number of capillaries (C.) penetrate into the lamina, and this produces in a frontal section the curled appearance of the border of the lamina. This curling has therefore no direct relation to the process of union, as this part of the lamina terminalis never unites with any part of the mesial walls.

The structure of the mesial wall (figs. 9 and 9A) agrees with the description I have given of the younger stage, only the different layers are more clearly differentiated. At the place of inflexion and rupturing the mesial wall is most slender; towards the inferior border the wall increases in thickness, producing a club-like appearance in the frontal section. In this thickened part of the wall the callosum and fornix (C.F.) are situated.

Concerning the relations of the different layers, I have but little to add. In the more frontal region the cortex layer, at the level of the callosum, becomes looser and is continued into the cortex layer of the lamina trapezoidea. About the fore-edge of the zone of union, instead of loosening, the cortex layer becomes discontinuous, ending with a free, sharp edge (C.l.). At a single place, however, this edge is a little swollen, but in no case have I seen the protrusion of the surface of the pallium wall by the cortex layer. In this stage of development it may clearly be seen that the nuclei of the intermediate layer penetrate into the marginal zone (Z.m.n.) round the edge of the cortex layer, and cause this zone to disappear as a distinct layer. These nuclei are continued over the dorsal surface of the callosum. Also in this stage of development the commissure is strictly confined to the intermediate layer of the pallium wall; that this is really the case is clearly demonstrated by a section passing behind the foramen of Monro; there the under border of the pallium shows very distinctly a marginal zone, and nowhere fibres of

the callosum are to be found piercing this zone and reaching the surface of the wall of the pallium.

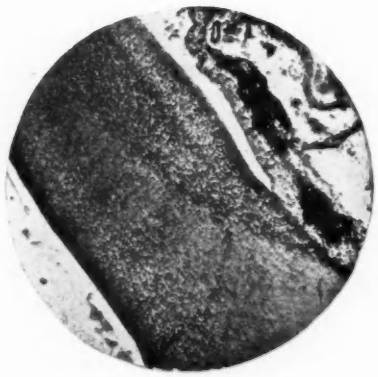
In the same way as described for the posterior part of the lamina terminalis, the mesial wall is also curled by the ingrowth of capillaries



Frontal section through the mesial wall of the pallium at the place indicated by a circle (partly black, partly white) in fig. 4. The section from which the photograph is taken lies well behind the section of fig. 4. Enl. $\frac{25}{1}$.

C.F., corpus callosum and fornix.
C.l., cortex layer.
C.n., cap of nuclei surrounding the callosum fornix.
I.H.T., interhemispherical tissue.
M., matrix.
V., lateral ventricle.
Z.i., intermediate zone.
Z.m., marginal zone.
Z.m.n., nucleated part of the marginal zone.

FIG. 9.



Photograph of the mesial wall of the pallium; fig. 9 is the outline of this figure. Photograph Zeiss obj. BB, enl. $\frac{25}{1}$.

FIG. 9A.

from the interhemispherical tissue. In its structure this tissue does not differ from the description given in the younger stage, and I only wish to call attention to the abundance of large blood-sinuses in the connective tissue in this stage of development.

I have no material of the stage of development when the mesial walls

begin to unite behind the foramen of Monro, so that for these later stages I can only add some superficial observations I gathered on inspection of median sections.

As a consequence of the enlargement of the corpus callosum the zone of union also increases. Dorsally of the foramen of Monro the posterior border of the callosum expands in an occipital direction and causes the mesial walls of the pallium behind the foramen to unite. At the same time with the enlargement, the callosum and the fornix bundle diverge, forming together a V lying on its side with the top turned backward. In frontal direction as well the callosum enlarges, forming the rostrum; under the influence of the expansion of the commissure in this direction the zone of union also increases. In the relative position of the systems of fibres no changes occur. The fornix bundle remains in a position lateral to the callosum in the reconstructed mesial wall of the pallium; the fornix commissure still lies ventrally of the callosum in the glia tissue of the zone of union. This latter relation is clearly seen in fig. 10, which is the reproduction of a drawing made of the median section of a brain of the end of the fifth month. Here the postero-inferior border of the callosum goes over into a beak, which is prolonged into a lamina (L.i.f.) that unites the two fornix bundles. In the beaked prolongation and adjacent to the callosum lies the fornix commissure (C.f.). If a frontal section were made at an appropriate level, this section would show the same relations as the sections reproduced in fig. 4, with the sole difference that a continuation of the cavum septi extends between the two commissures. The cavum septi, once formed, enlarges in a frontal as well as in an occipital direction, in correspondence with the enlargement of the callosum. On both sides of the median plane the posterior part of the cavum septi shows a protusion laterally of, and underneath, the callosum (R.pl.). In the same way as this recessus postero-lateralis there is a recessus antero-lateralis (R.a.l.), but this niche is by no means so well developed as the posterior one.

Now, from the fact that the fibres which compose the callosum show a regular arrangement in respect to the regions of the pallium they connect, it is obvious that the development of the pallium influences the form of the callosum. This is shown by the form of the splenium, which at the stage of development shown in the drawing is not yet rolled in. The rolling in of the splenium seems to be caused by the further differentiation of the lobus occipito-temporalis, to which the larger number of the fibres of the splenium belong. Now, the splenium follows the temporal

lobe when bending round the thalamus in frontal direction, and consequently is rolled in. By the rolling in of the splenium, callosum and fornix bundle approach each other, so that the posterior part of the cavum septi becomes smaller and the recessus postero-lateralis is reduced

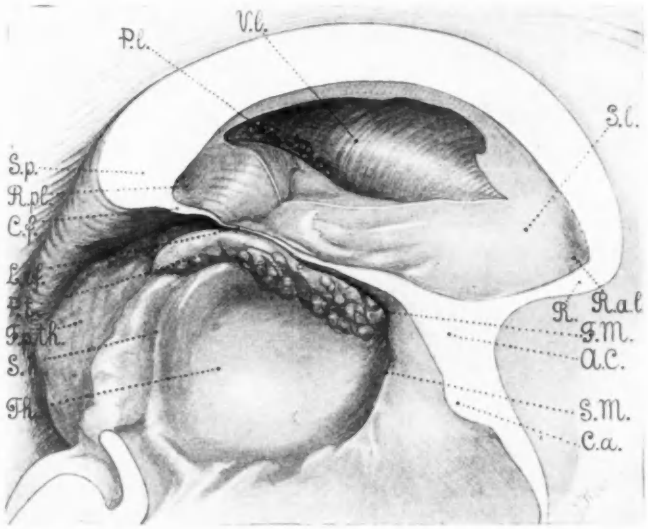


FIG. 10.

Drawing of the median section of a brain of the end of the fifth month, enl. 1.
A.C., anterior part of the zone of union.
C.a., anterior commissure.
C.f., fornix commissure.
F.M., foramen of Monro.
F.p.th., posterior surface of the thalamus.
L.i.f., lamina interfornicaria.
P.l., choroid plexus of the lateral ventricle.
P.t., choroid plexus of the third ventricle.
R., rostrum.
R.a.l., antero-lateral recesses.
R.pl., postero-lateral recesses.
S.h., sulcus habenularis.
S.l., septum lucidum.
S.M., sulcus of Monro.
S.p., splenium.
Th., thalamus, mesial surface.
V.l., lateral ventricle.

to a transverse slit. The posterior narrowest part of the cavum—the ventriculus Vergæ—in most cases in the new-born still communicates with the anterior part of the cavum septi.

From these stages of development as reproduced in the drawing it

seems evident that the lamina that unites the two fornix bundles (L.i.f.)[1] is a remainder of the zone of union, for this lamina in frontal direction continuously goes over into the anterior part of this zone (A.C.), in which the commissura anterior (C.a.) is situated. Occipitally the lamina is continued in the prolongation in which the fornix commissure is located, and this tissue is also doubtlessly a remainder of the posterior part of the zone of union. The uniting lamina between the two fornix bundles in the adult stage shows exactly the same relation, only these parts of the zone of union into which it is continued are more reduced. Therefore this lamina, for which I should propose the name of "lamina interfornicaria," belongs neither to the corpus callosum nor to the fornix bundle, but is a remnant of the zone of union. Consequently the union of the fornix bundles in the median plane is a secondary one, and this explains, I believe, the relation that the lamina interfornicaria shows in respect to the fibres of the alveus, a connection to which Dejerine [3 (p. 278)] has given much attention.

Summary.

(1) The anterior commissure precedes in its histological differentiation the corpus callosum and the fornix commissure.

(2) In a stage of development in which the diameter of the corpus callosum amounts to but 0·5 mm., about one-third of the commissure lies in the lamina terminalis and two-thirds in the zone of union.[2]

(3) From the relation of the corpus callosum to the fornix and to the fornix commissure it follows that the callosum at its first appearance shows all the essential features of the commissure in the adult stage.

(4) The interhemispherical tissue atrophies before the enlarging commissure.

(5) The true histological nature of the process of union of the walls of the pallium I must leave undetermined.

(6) The corpus callosum remains strictly confined to the intermediate layer of the wall of the pallium.

(7) In the region of the callosum the cortex layer of the mesial wall ends in a sharp edge.

(8) The cavum septi lucidi is probably formed by a cleavage in the glial tissue of the zone of union.

(9) The lamina interfornicaria is a derivative of the zone of union.

[1] This structure is described by Marchand [15] under the name of "Bodenlamelle des Cavum septi"; by Martin [16] as "Verlängerte Schlussplatte"; and by Dejerine [3] as the "lamina intertrigonalis."

[2] Unless we consider the zone of union a direct derivative of the lamina terminalis, this result is partly in contradiction with the explicit statement of Elliot Smith in "Morphology of the True Limbic Lobe." [25 (p. 198)].

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THE STATE OF THE BRAIN DURING HYPNOSIS.

BY WILLIAM McDOUGALL, M.A., M.B.

THE attempt to formulate a theory of the hypnotic state has been approached by two very different roads. On the one hand there is the road along which the late F. W. H. Myers so brilliantly led the attack. Those who follow this road fix their attention on the most strange and perplexing of the phenomena of hypnotism; they seek to exhibit the gulf between the normal and the hypnotic states as very wide; they incline to regard the hypnotic condition as essentially one of extended faculty and increased mental power; and they value hypnotic experiment as one way of approaching the study of alleged supernormal and marvellous phenomena, belief in which orthodox science regards as a delusion bred by erroneous observation and engendered by an incorrigible yearning after empirical evidence of a realm of purely psychical existence.

The other more sober way in which the task of explaining hypnotic phenomena may be approached is to concentrate attention upon the simplest and least astonishing of them, to seek out and establish the affinities of hypnosis with better-known mental states, and to try to find some hypothesis that will account for these simpler facts and will bring our knowledge of hypnosis into line with the facts of our normal mental life and with theories that have proved their value in other departments of research; for, if any such working hypothesis can be formulated, it may be hoped that its field of application may be gradually enlarged and that the more perplexing phenomena may be brought within its range as our knowledge of them increases.

Though I would not disparage or undervalue speculations of the former kind, I believe that the second way of approach is more consistent with scientific principles, and the following pages present the results of my own gropings along this road.

The aim of this paper is to render a little clearer and more definite in detail a hypothesis which has been gradually taking shape during recent years under the hands of a number of writers, and which goes by

the name of the *theory of cerebral dissociation;* for certain unorthodox views as to the nature of cerebral processes set out by me in former numbers of this Journal [6] and elsewhere seem to lend themselves very well to this purpose.

The current loose usage of the phrase "theory of dissociation" tends to the confusion of several different hypotheses which have little in common with one another, and some of which are held by authors who follow the former of the two roads distinguished above. There is the doctrine (or group of allied doctrines) which teaches that each normal personality comprises two selves, two personalities or strata of personality, which normally are in some sense fused together—the waking and the subwaking selves of Boris Sidis [9], the *Doppel-Ich* of Max Dessoir [2], the waking consciousness and the dream-consciousness, the primary and secondary personalities, of other authors[1]; and that mental dissociation somehow consists in the freeing of the lower, or normally submerged and hidden, stratum from the dominance and control of the upper stratum. I do not think that at the present time we are compelled to make, or are justified in making, the assumption that the normal human mind comprises any such submerged and coherent system of faculties as is implied by each and all of these allied doctrines.

More nearly allied to the form of the theory of dissociation that I am concerned to support is Pierre Janet's doctrine of mental disaggregation [4]. Janet speculates only in terms of psychical material, and refuses to attempt a corresponding neurological hypothesis; but I am very decidedly of the opinion that here, if anywhere, attempts at purely psychological explanation are out of place. I believe that any explanation or theory of the hypnotic state and phenomena must be psycho-physiological; for it seems clear that, during hypnosis, the brain is in some abnormal condition and functions in some manner different from that of the normal waking state, and that therefore we cannot claim to understand hypnosis until we learn the nature of this abnormal brain state; that, in short, a description of the abnormal state of the brain must be an essential, if not the essential, feature of any explanation of the facts.

Th. Lipps [5], of Munich, also has proposed an explanation which, although he states it in terms of psychical dispositions only, and so limits its usefulness, seems to me very helpful; and it can easily be translated into terms of psycho-physical dispositions and developed on those lines.

[1] Possibly the doctrine of the Sublimal Self, elaborated by the late F. W. H. Myers, belongs to this group of theories, but I do not feel myself competent to class it.

Lastly, I would mention the speculations of Oscar Vogt [3] (accepted and endorsed by Forel) as those with which my own views, although independently reached, have the closest affinity.

It is held by many authors, notably by Bernheim—rightly, I think—that hypnosis is a state closely allied in many respects to normal sleep, although it presents important differences. The onset of the two states is very similar in many (if not all) cases, and it is favoured in both cases by the same influences and conditions, namely, by the withdrawal of all strong sense-stimuli, by restful position, by monotonous gentle stimulation of one or more of the senses, by expectation and habit, and by the banishment of exciting thoughts and the concentration of attention on some unexciting object or sense-impression.

Another point of similarity of the onset of the two states is the heaviness of the eyelids, which is generally the first symptom of both sleep and hypnosis, and which, in both cases, is generally accompanied by a general drowsiness and disinclination to make any effort or movement.[1]

In both states the subject lies inert and passive; his mind is less responsive than during the waking state to most of the sense-impressions from the outer world; and, although it is not completely shut off from their influence, it is apt to interpret them falsely. In both states the subject may entertain the most fantastic ideas, ideas absolutely incompatible with his actual situation or his best-established convictions, without recognizing their absurdity; and such ideas are apt to undergo some fantastic elaboration in keeping with any prevalent emotion; in both cases these ideational processes are characterized by a lack of the voluntary and critical control which is habitual and normal in the waking state. In both cases the subject not infrequently desires to move his limbs, but finds himself incapable of doing so. In both cases, again, the subject is very apt to have, when awakened, no spontaneous recollection of the ideas of the period (of sleep or hypnosis), though recollection can generally be evoked by

[1] I am, of course, aware that some subjects exhibit while in hypnosis a degree of liveliness and excitability apparently greater than the normal, and that some of their faculties may seem even more alert and active than during their normal waking life. But these are distinctly exceptional cases, and usually, I think, this lively state is only arrived at after some training. We have to concentrate our attention on the more usual type of hypnosis; for the symptoms of the state are so bewilderingly diverse and numerous that, if we attempt to form a theory which shall account at once equally well for all of them, it is obvious we shall fail. Very much depends upon the handling of each case by the operator. In my own experience I have generally aimed at inducing a sleep-like condition, and have always obtained this result, except where I have failed to induce any degree of hypnosis.

appropriate questioning. Some subjects may be induced to display an abnormal suggestibility during normal sleep, and in some the cataleptic plasticity of the limbs, which is a so frequent symptom of hypnosis, may be induced during normal sleep.

Lastly, if the subject in hypnosis is left to himself, he is apt to pass spontaneously into normal sleep, the transition from the one state to the other being perfectly gradual, so that no line can be drawn between them; and, conversely, the state of normal sleep may be converted into hypnosis.

The resemblance between hypnosis and sleep is, in some respects, still closer in the case of ordinary sleep-walking or somnambulism, which may perhaps be regarded as a state intermediate in character to these two.

On the other hand, hypnosis differs from normal sleep in that it has certain positive characteristics that are not found in the latter. A large proportion of these may be summed up by saying that the subject remains peculiarly sensitive to the influence of the hypnotizer, and that ideas introduced to his mind by the hypnotizer operate with unusual energy and effectiveness, or, as we say, the subject in hypnosis is very suggestible towards the operator, or readily takes suggestions from him.

The obvious line of approach is, then, to find a good working hypothesis as to the state of the brain in normal sleep; and, having formulated this, to try to understand what further peculiarities of the state of the brain are connected with the positive features in which hypnosis differs from sleep.

Unfortunately we have as yet no generally accepted theory of sleep. We have a number of partial theories or views—the view that sleep is due to the action of waste products of metabolism on the nervous tissue; the view that it is due to cerebral anæmia; the view that it is due to auto-suggestion; the recent view of Claparède [1] that the onset of sleep is an instinctive process.

There is probably some truth in all of these views, as well as in others; but neither individually nor collectively do they give an answer to the essential question: How does the state of the nervous tissue of the brain during sleep differ from its state during waking life?

It seems clear that during sleep the nervous substance of the cerebral hemispheres is either less excitable than during waking life or transmits any excitement less readily from part to part; and in all probability both these statements are true. A high degree of excitability and great freedom of transmission of excitement between all parts —these seem to be the essential positive characters of the waking brain

that are lacking to the sleeping brain. Our definition of the state of the brain during sleep must, then, be a negative definition; it must consist in denying it the condition which underlies this proneness to activity characteristic of the waking state.

What, then, is this peculiarity of the waking state? The clue to the answer to this question is given, I think, by those rare cases of very widespread anæsthesia, of which the most striking and most frequently cited example is the case described by Strümpell [10]. The patient was a boy completely anæsthetic as regards all his sense-organs and sensory surfaces, with the exception of one eye and one ear. Whenever the sound eye was closed and the sound ear stopped, this patient fell almost immediately into deep sleep, and could then only be wakened by flashing a light into the sound eye or shouting into the sound ear, or otherwise stimulating violently one or both of these organs. That is to say, in this case the waking state was maintained only by the constant incidence of impressions on these sense-organs.

Now, it seems in the highest degree probable that the constant rain of stimuli upon our sense-organs plays a similar part in maintaining the waking state of the normal man; but the fact is obscured in two ways: firstly, by the great variety and sensitivity of the sense-organs of the normal man and the impossibility of shielding all of them from stimulalation; secondly, by what may be called the capacity of the human psycho-physical organism for maintaining an endogenous excitation. We must briefly consider these in turn.

Even if one lies still on a soft bed in a quiet dark place, the nervous system is yet liable to receive many stimuli, especially through the sense-organs of the kinæsthetic and visceral systems. Under these conditions we often become aware of these impressions, and often they excite some sense of discomfort, some need of movement or change, so that one may be kept turning restlessly without sleep. The most striking example of the power of these vague sense-impressions arising within the body to prevent sleep is afforded by those which excite the sensation of hunger.

Nevertheless, save in conditions of great fatigue, the withdrawal of stimuli from the principal sense-organs is a favourable and, for most of us, an essential condition of the onset of sleep. When sleep comes, its continuance depends upon the absence of strong sense-impressions; it may normally be terminated at any time by a sufficiently strong sensory stimulus, or by the summation of effects of a series of sensory stimuli, each of which is too weak to produce any marked change of state;[1] and

[1] I have described and discussed this process in some detail in the pages of *Mind* [7].

even after sleep is thus banished we do not usually feel ourselves to be fully awake and alert until a further stream of stimuli has impinged upon our sense-organs.

Now, unfortunately, our knowledge of the immediate effect of stimulation of a sensory nerve is still very imperfect; but there is much to be said for the view that the immediate and essential effect is a katabolic process which liberates chemically stored energy in the substance of the neurone, and that the spread of excitation consists in the discharge of this freed nervous energy from neurone to neurone across the synapses or places of junction of the neurones.[1] This distinction between chemically stored or potential nervous energy and the liberated active nervous energy is, I feel sure, one of the first importance for neurological speculation, although but little attention is commonly paid to it. Oscar Vogt has recognized its importance and has proposed to mark it by calling the freed nervous energy "neurokyme." Some years ago I, in ignorance of Vogt's work, proposed to call it "neurin." What name we use does not much matter, so long as we hold fast to this distinction and to this conception of liberated active nervous energy; but for this purpose some name is essential; since Vogt's proposal was prior to my own, and since my proposed name is so similar to neurine, the name of one of the chemical compounds found by the chemists in nervous tissue, I adopt Vogt's term "neurokyme" in place of "neurin."

During waking life, then, stimuli rain unceasingly on all the sense-organs and liberate in all the sensory nerves streams of neurokyme, which ascend by the sensory tracts of the cord and lower brain to the cerebellum and cerebrum. The brain is thus fed and its activity is sustained by these streams of energy, which keep it charged with

[1] Some arguments in support of this view are adduced in two papers in former numbers of this Journal [6]. In a recent paper, privately printed (*Proceedings of the Junior Scientific Society*, Oxford), I have ventured to suggest that this way of conceiving the immediate effect of a stimulus upon a nerve affords a possible explanation of the fact that the amount of nervous response varies with the intensity of the stimulus applied. We may suppose that the katabolic change propagates itself along the substance of a neurone on the all-or-nothing principle, to which an explosive decomposition may be expected to conform; but we may suppose that the quantity of such change in unit of time varies with the energy of the incident stimulus, as in the case of a train of damp gunpowder. The distance through which the change will propagate itself, or, in other words, the length of nerve fibre, and hence the amount of nervous substance which undergoes the katabolic change, will, then, vary with the intensity of the stimulus; and since the quantity of energy liberated will vary with the amount of nervous substance which undergoes this change, it also will vary with the intensity of the stimulus. A maximal stimulus will, then, cause the katabolic process to propagate itself throughout the whole substance of the neurone, and every submaximal stimulus will cause it to propagate itself through a part only of the neurone substance, the length of which part will be proportional to the intensity of the stimulus. I throw out this suggestion with the greatest diffidence for the consideration of those who are better qualified than myself to say whether it is a tenable hypothesis.

neurokyme at a varying tension or potential; and this charge of free energy is constantly being worked off by thought or mental activity of any kind, for all mental activity involves the discharge of neurokyme from the sensory to the motor side of the brain, in accordance with James's law of forward conduction.

Now consider the second condition that obscures the importance of sense-stimuli for the maintenance of the waking state—what was called above the capacity for endogenous or automatic maintenance of the state of excitation.

The organism comprises certain hereditary pyscho-physical dispositions, which, in the evolutionary sense, are essentially continuous with, or to be identified with, the instincts of the animals. In what each such disposition exactly consists we do not yet know, though no doubt an essential feature of it is a complex system of sensori-motor arcs. In the present connexion the important fact is that each such disposition is a great spring of nervous energy; when anyone is excited in any way it liberates a great quantity of neurokyme that raises the activity of the brain to a higher level, a fact which manifests itself in symptoms of general excitement, in very energetic thought and action and, subjectively, in the form of impulse, desire, and emotion.

These dispositions can be excited by way of sense-presentation; hence sense-impressions contribute to the maintenance of the state of general excitation of the brain, not only in proportion to the intensity of the stimuli and the extent of sensory surface affected, but also in proportion as they lead to the excitement of any of these special springs of energy. In most of the animals these dispositions can be excited only by sense-impressions, but in the human being they can be excited also by way of representative or ideational processes. Hence the human mind and brain do not necessarily come to rest as soon as all sensory stimuli are withdrawn; the activity of an excitable brain may continue to be sustained by this process of endogenous liberation of energy, by the power of the impulses awakened through ideas and recollections. When this is the case, sleep can only be secured by the avoidance of emotionally exciting ideas, *i.e.*, by turning the attention to indifferent things—to sheep jumping through a gap in a hedge, to counting, or to some faint bodily impression.

The presence in the brain-neurones of a store of free energy or neurokyme derived from these two sources is, then, a prime condition of the waking state; but there is a second important condition, dependent in large measure upon this one, which underlies the freedom of

transmission of excitation from point to point of the brain, the free interplay of all parts of the brain, that is characteristic of the waking state; this is the state of the synapses.

We can confidently infer that the neurones that make up the nervous tissue of the cerebrum are connected together to form functional groups, the members of each group being so intimately connected that excitement of any one member of the group tends to spread at once throughout the group to every member of it.

Such a group of neurones is a functional unit, and we may call it a psycho-physical disposition; of such groups the hereditary dispositions mentioned above constitute a very important and peculiar class. We may infer also that these dispositions are connected with one another with various degrees of intimacy to form systems; these, again, with less degrees of intimacy to form larger systems; and these yet again with still less intimacy to form still larger systems; and so on, until we reach the most comprehensive system, which is the whole of the central nervous system.

Each disposition is an intricately woven chain of neurones making up a complex sensori-motor arc or system of arcs. All thought, all perceptual or ideational mental process, involves the perpetual shifting of the main nervous current from one disposition to another, at any one moment some one disposition being the main path of discharge of neurokyme from the sensory side of the brain, where it is constantly accumulating, to the motor side; and, while any one disposition thus predominantly active is the principal focus of excitation, those most intimately connected with it are in a state of subexcitement. When any one disposition thus becomes the main path of discharge, it is because, owing to a favourable conjunction of circumstances, it has become for the moment the path of least resistance from sensory to motor side of the brain. The discharge through any one disposition is the neural concomitant of the rise to consciousness of a corresponding presentation or idea; and the shifting of the main stream from one disposition to another is the neural concomitant of the play of ideas, of the succession of presentations at the focus of consciousness, which continues so long as we are awake.

An essential feature of the view I am expounding is that the various degrees of intimacy of connexion between neurones and between groups and systems of neurones are held to be functions of the synapses or junctions of neurones. If excitement spreads readily from one group to another, it is because the synapses on the path

connecting those two groups present at that moment a low degree of resistance; if it spreads less readily to another group, it is because the synapses on this connecting path present a greater resistance. Now there are many good reasons for believing that the resistance presented by any synapse is not a fixed quantity, and that it is not only permanently diminished in some degree by repeated transmission of the excitation-process, but that it is a quantity which varies from moment to moment under a number of influences, of which the most important are fatigue of its own substance, chemical influences from the blood, and the charge or potential of charge of neurokyme in the neurones between which the synapse forms a junction. That is to say, it is maintained that each synapse (in the resting condition of the part) presents a certain normal degree of resistance which varies from synapse to synapse, and is in each case a fixed quantity (or one only slightly or slowly changeable) determined by heredity and the course of individual experience; but that this normal degree, to which the resistance of each synapse returns when the brain is at rest, is constantly liable to be modified by the influences named above, being raised by fatigue and anæsthetic drugs such as alcohol and chloroform, diminished by strychnine and tetanus-toxin and by the excitement of the neurones between which the synapse lies.

This last condition is the most important one in view of the problem in hand. I assume, and the assumption is not without positive evidence in its favour, that the resistance of the synapse falls as the potential of charge of neurokyme rises in both, or in either one, of the neurones between which it lies, and that it rises as this potential falls.

In the waking state, then, the hemispheres being constantly supplied with large quantities of neurokyme from the two sources indicated above, the main mass of its neurones is kept moderately charged with this free energy, the result of which is that all synapses, and therefore all connecting paths, are kept in a state of partially lowered resistance; and there is, therefore, a constant free interplay between all parts of the brain, the main current of energy shifting freely from one disposition to another and from one system to another, each disposition tending to draw to itself a maximal stream of energy, each competing with all the rest for the fullest share of energy according to the principle of drainage.

Now, when we lie down to sleep in a quiet dark place, we shut off as nearly as possible all stimuli from the sense-organs, and we divert our thoughts from all emotionally exciting topics. The supply of neurokyme

to the brain is thus diminished, the charge present in, or banked up in, the neurones of the afferent side of the brain falls to a lower potential, and, consequently, the resistance of the synapses in general rises. When sleep ensues from great general fatigue, another factor probably plays the principal part, namely, the waste products of metabolism, which, accumulated in the blood and lymph that bathe the synapses, act upon them, like chloroform or alcohol, as poisons which diminish their metabolism and so raise their resistance. Also, in the production of the sleep that ensues from deficient energy of the heart's action or from diminished circulation of blood in the brain, however produced, this second factor probably plays a large part, the waste products being allowed to accumulate locally. In normal falling asleep these two conditions—the general accumulation of waste products in the blood and the general slowing of the circulation—coöperate with the diminution of supply of neurokyme to raise the resistances of the synapses of all parts of the brain.

This general raising of the synaptic resistances throws the whole brain into a condition of *relative dissociation* or functional dissociation; that is to say, the dispositions and systems of dispositions, as well as the neurones comprised within any one disposition, become in some degree functionally isolated or separated from one another. And this functional discontinuity will be most complete in the case of the least intimately connected systems, less complete between the more intimately connected dispositions of any one system, and least between the neurones that are united in one disposition; for the resistance of each synapse will be reduced to, or near to, its normal resting degree.

Normal sleep implies, then, a state of relative dissociation of the brain, and the many points of similarity noted above between sleep and hypnosis indicate that hypnosis also involves relative dissociation of the brain; on the other hand, some of the phenomena of hypnosis, to be noticed below, afford positive evidence that such dissociation obtains, and so confirm the indications afforded by the foregoing consideration of the general physiology of the brain and of sleep.

We have to inquire: How does the state of the brain during hypnosis differ from this state of general relative dissociation of normal sleep? The answer to this question suggests itself when we consider the way in which hypnosis is commonly induced. The onset of hypnosis is favoured by the influences which favour sleep (with the exception, possibly, of fatigue), namely rest and quiet (*i.e.*, the withdrawal of sensory stimuli), the slowing of the circulation, the banishment of emotionally exciting

thoughts, and by the expectation of sleep. How expectation operates remains a very obscure problem, but it is clear, I think, that in neither case is it an essential factor. The important influences brought to bear in the induction of hypnosis, in addition to those which normally produce sleep, are: (1) monotonous stimulation of sense-organs, either continued (as by visual fixation of a bright point) or intermittent (as by passes); such monotonous stimulation is favourable also to the onset of normal sleep; (2) the personal contact of the hypnotizer, who, by speech, by verbal suggestions, and by manipulations, keeps the subject constantly aware of his presence.

The monotonous stimulation seems to aid in bringing the whole brain to a quiescent condition, by facilitating the continued direction of attention to an object or impression of an unexciting uninteresting character, and thereby preventing the free play of ideas which otherwise may maintain itself for a considerable period in the way noted above. In terms of neural process we may say that the monotonous stimulation tends to keep some one minor disposition or small system of dispositions in dominant activity, keeps open this one path of discharge, so that this one channel, constantly draining off from the sensory side of the brain the supply of neurokyme, depresses, or tends to prevent, the activity of all others.[1]

The personal contact of the operator contributes to produce the same result. His passes, his manipulations, his verbal suggestions, all serve to keep the idea of the operator present to the mind of the subject, to keep the subject's attention (no doubt an attention of low grade or potential) directed to the operator; *i.e.*, in terms of neural process, they serve to keep in a state of excitation one system of psycho-physical dispositions, the system whose activity underlies the presence to consciousness of all thought of the operator; or, again, they tend to keep the main current of nervous energy shifting from one disposition to another within this one system. In this way, while all the rest of the brain is allowed to sink into a state of quiescence and of relative dissociation similar to that which obtains in normal sleep, this one system is kept active and waking, so to speak. It thus serves as an open channel through which ideas can be introduced to, or evoked in, the mind of the subject, as a single focus of nervous activity in a quiescent brain, from which focus other parts of

[1] That inhibition within the nervous system is always and at all levels a process of drainage of energy from one path or system to another in virtue of the lower resistance presented by the inhibiting path is a view that, as I have tried to show in a previous paper (*Brain*, vol. xxvi.), is compatible with all the facts and seems to be the only tenable working hypothesis.

the brain may be brought into play. Any proposition made by the operator to the subject is then accepted uncritically and acted upon because accepted with belief—that is the essence of suggestion—whereas the subject is blind and deaf to impressions from all other persons and objects, except in so far as they are connected in his mind with the operator, *i.e.*, except in so far as they belong to the same system of ideas.

Now a leading feature of hypnosis is that ideas or propositions suggested by the hypnotizer not only are accepted, but, being accepted, operate with a quite unusual force or effectiveness in the mind and on the body of the subject. The state of the brain described above, the state of relative dissociation of all systems except the one, enables us to suggest an explanation of this feature also. In the normal waking state any proposition about any topic or object is received more or less critically, and is only accepted with conviction if it is not incompatible with the organized body of knowledge or belief about that topic or kind of object already established in the mind. Every idea, we may say, has to withstand or overcome the inhibiting tendencies of these other ideas connected with the same topic before it is fully accepted, before it can prevail stably and determine action in the way characteristic of belief. But in the state of relative dissociation, any idea introduced to the mind by the operator prevails stably and determines action—is, in fact, accepted with belief—just because the ideas which could check or weaken its operation are not aroused, are not brought to bear upon it in criticism, owing to the state of relative dissociation which renders all interplay of ideas more difficult, more sluggish, than in the waking state. Further, in the waking state not only contradictory ideas, but all ideas whatsoever that have any tendency to rise to consciousness at the moment, play a similar part, weakening to some extent the force with which the dominant idea at the focus of consciousness operates in the mind and on the body.[1] The refined experimental researches of G. E. Müller upon reproduction and association seem to have established this fact [8].

We may try to express these relations in physiological terms. We must remember that in the waking state of the brain all dispositions and systems of dispositions are in relation of reciprocal inhibition with one another, such that the activity of any one tends to inhibit the activity of every other; and we may fairly suppose that between dispositions whose activities underlie incompatible or contradictory ideas about any object,

[1] I am, of course, using the word idea to denote the whole psycho-physical system of activity which reveals itself in consciousness as a presentation.

this relation of reciprocal inhibition is peculiarly intimate and direct.[1] All dispositions, then, compete with another, except those that form a harmonious system and tend to express themselves in some particular mode of coördinated bodily activity. In the waking state, then, the energy with which any idea tends to express itself, or realize itself through bodily action, is thus diminished by the competition of all other ideas that have any tendency to rise to consciousness; or, in neural terms, the energy with which any disposition functions is normally to some extent depressed by the competition of all other dispositions that are in any state of subexcitation, and especially of those of contradictory ideas. In hypnosis, on the other hand, this depressing, weakening influence, this partial inhibition, is abolished or diminished in virtue of, and in proportion to the degree of, relative dissociation or functional isolation of dispositions from one another. Hence, any idea suggested by the hypnotizer is not only accepted uncritically, but operates with greater force than any idea accepted with conviction in the waking state.

The absence or diminution of all such inhibitory weakening and restraint, and the correlative concentration of all available neurokyme along the channels of one disposition, seem to be the principal factors to be taken into account when we seek to explain all the commonest and most easily produced results of hypnotic suggestion, namely, the illusions, positive hallucinations,[2] delusions, the control of the voluntary

[1] The hypothesis of inhibition by drainage seems to lend itself well to the explanation of this kind of inhibition, although, of course, it is not possible to apply it in detailed fashion. We may liken the relation between the dispositions of contradictory ideas to the relation (so brilliantly studied by Sherrington—*see* "Integrative Action of the Nervous System") obtaining between the reflex arcs innervating antagonistic muscle-groups; and this case is not only truly analogous, but is probably more than analogous; it is probably the simplest example of the same type of functional relation. Contradictory ideas about an object tend to issue in opposed systems of muscular activity.

[2] It is perhaps worth while to expound the application of the hypothesis to one of these cases a little more in detail. Take the case of induction of a positive hallucination. It is suggested to a subject that a blank card carries a photograph of some familiar building. Perhaps the subject says he can see no picture on the card. A few irregular lines drawn on the card will then often facilitate the induction of the hallucination, and on repetition of the suggestion the subject may then admit that he sees the picture, and may describe it in some detail, and may even continue to see it on the same card when awakened, without post-hypnotic suggestion to that effect having been given. The influence of the lines drawn on the card in facilitating the induction of the hallucination (which in this case approximates to the type of illusion rather than pure hallucination) is significant. There is good reason to believe that the lines, serving as *points de repère*, are worked up among the hallucinatory lines of the subject's picture. We may suppose that the verbal suggestion of the picture of the familiar object evoked, in the mind of the subject, an image or visual representation of the building, which, when he directs his gaze to the card, is projected upon the card with hallucinatory or sensory vividness. I have sometimes been able to follow this order of events—the formation of a visual representation of an object while the eyes are closed and the subsequent projection of this image, with increasing vividness and distinctness, when the subject is directed to open his eyes upon a card. The neural events we may conceive as follows: the rise to consciousness of the visual image results from the spread of excitation to the

muscles and (to some extent) of the involuntary muscles and of the visceral processes, secretion, nutrition, and so forth. While, as Pierre Janet has suggested, cataleptic plasticity of the limbs may be equally well regarded as due to the functional isolation of the cerebral tracts by which the afferent impulses ascending from the organs of the "muscular sense" return to those motor elements of the cortex from which the same movements and positions of the same parts are voluntarily effected (in accordance with the principle of the upper motor circuits). The weakening or abolition of reflexes, which, I believe, occurs only in very deep stages of hypnosis, may be regarded as due to the dissociation having attained so great a degree as to affect the functional continuity of the neurones composing the reflex arc.

To negative hallucinations, and to the execution of post-hypnotic suggestions by a subject who remains unaware of the nature of the suggestions given, these principles of explanation are not so easily applicable; that is to say, while cerebral dissociation is implied by them, the principle is not in itself adequate to shadow forth an explanation of them; some further principle is implied. But these processes are especially interesting from the present point of view because they prove, more clearly than any other of the phenomena, that some functional dissociation of the brain is really present. In both cases we have unmistakable evidence that some process goes on in the brain independently of, and without affecting or being involved in, the main stream of psycho-physical process. Such, for example, is the deliberate ignoring by the subject of an object of which he has been told that it is no longer present, for although he certainly does not perceive the object

corresponding visual disposition from the auditory disposition directly excited by the verbal suggestion. If the subject were in the normal state and directed his gaze to the card, the lines upon it would be seen for what they are and the image would fade from consciousness as the attention was diverted to these lines; that is to say the disposition concerned in the production of the image would cease to function, because the main stream of energy would be diverted to another and simpler disposition, that which subserves the appreciation of the lines drawn on the card. But in the hypnotic state, owing to the relative dissociation and quiescence of all parts of the brain, save the disposition concerned in the production of the image, and owing to the abnormal preponderance of the excitation of this disposition over all others, the sensory impulses initiated in the retina by the lines on the card and passing up to the brain are diverted from the channels they would normally follow and are led into this preponderantly excited disposition. This disposition thereupon receives its stimulus, not only from other parts of the cortex in the way characteristic of ideational process, but directly from the sensory nerve tract; and this direct reception of sensory impulses seems to be in all cases the condition of the perceptual character or sensory vividness of a presentation. This view of the process of induction of the hypnotic hallucination brings it into line with the "recurrent sense-impressions" which are apt to follow on prolonged work with the microscope on some one kind of structure, or on other constantly repeated sense-impressions; and also with the pathological hallucinations that seem to arise from morbid irritability of some brain tract. For in all such cases the hallucination may be plausibly regarded as due to the diversion of sensory impulses from their normal channels and their attraction into the over-excited system or disposition.

in normal fashion, and is apparently not conscious of it, his neglect and active avoidance of it show that the object is in some sense recognized ;[1] and when a post-hypnotic suggestion is executed after a given number of repetitions of some signal, the signals clearly have been in some sense counted ; and yet the subject remains unconscious of them. It is the facts of this order that have led so many authors to postulate a co-consciousness, a secondary stream of consciousness split off from the primary consciousness and flowing independently of it ; for the processes involved seem to be distinctly mental processes, such as normally involve consciousness. There is much to be said for that view, and also there are difficulties in the way of its acceptance. But, though we may leave the reality of such co-consciousness an open question, we are compelled by the facts of this order to believe that a complex and orderly sequence of nervous processes, dissociated from the main stream of brain activity, is involved in the execution of such tasks ; the dissociation seems to circumscribe the independently operating systems.

Anæsthesia also affords good evidence of dissociation ; the sensory areas concerned with the reception of afferent impulses from the anæsthetic part seem to be profoundly dissociated from the rest of the brain ; though whether this neural dissociation suffices in itself to account for the anæsthesia, and whether we are justified in assuming, as Janet does, that the sensations of the anæsthetic limb exist, or occur, as isolated sensations or sensations of a slender stream of secondary consciousness—these are very obscure questions which also we may leave open, while we accept the anæsthesia as positive evidence that at some point in the sensory path from the anæsthetic organ, probably a point within the cortex of the brain, resistance is abnormally increased in the way which constitutes dissociation.

The discontinuity of the memory trains of the hypnotic and the waking states, which so commonly obtains, is another piece of direct evidence of the reality of dissociation ; although here, as in the preceding cases, it remains a very obscure problem : how can verbal suggestion determine the position of the line of cleavage or dissociation that separates the two systems ?

A few words may be ventured as to the bearing of the views set forth above on the therapeutic applications of hypnotic suggestion. If these views represent an approximation towards the truth, it follows that the therapeutic value of hypnotic suggestion consists principally in the fact that it is a means of concentrating powerful currents of nervous

[1] The subject usually avoids turning his gaze directly upon such a forbidden object.

energy in any required direction and of withdrawing them from other parts. By thus withdrawing the nervous currents from an overworked or unduly irritable nervous centre or bodily organ, and by isolating it through induction of a relative dissociation of the centre, rest may be secured and a bad habit of over-action may be suspended, as, *e.g.*, in neuralgia; while, by repeatedly directing a powerful stream of innervation through some other channel, a too sluggish organ (*e.g.*, the bowel) may be brought back to a more active and healthy functioning, a habit that has become disordered or unduly weakened may be restored, or a new habit may be set up to supplant, counteract, or suppress some undesirable habit. Under these two heads, the increase or the diminution of the metabolism and functioning of organs, most of the therapeutic effects of hypnotic suggestion may, I think, be classified.

There are a number of phenomena that remain very obscure, and it is not claimed that the theory of cerebral dissociation as here presented provides a complete explanation of any of the facts. But that cerebral dissociation of some degree is at least one of the essential features of the hypnotic state can, I think, hardly be doubted; and though it may be questioned by some whether even a complete account of the cerebral changes would afford anything like a complete explanation of the facts, it can hardly be disputed that any complete theory of hypnosis must take the cerebral changes into account. I venture to think that the foregoing hypothetical description of the state of the brain during hypnosis may render the conceptions of cerebral dissociation, of the peculiarity of the hypnotic dissociation, and of the process of its induction, a little clearer and more definite than they have hitherto been. It is but just to point out that the hypothesis of cerebral dissociation which I have endeavoured to develop seems to have been first suggested by Hughes Bennett,[1] and that therefore, if it should ever attain to the rank of a generally accepted theory, the credit of having first enunciated it must be assigned to him.

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[1] I am indebted to Dr. Milne Bramwell for having drawn my attention to this fact on the occasion when the substance of this paper was read to the Medical Society for the Study of Suggestive Therapeutics over which he presides. It would seem that the theory was first implied by Bennett in a lecture entitled "The Mesmeric Mania of 1851," delivered and published at Edinburgh in the year 1851. I have not been able to refer to a copy of this lecture.

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THE NATURE AND QUANTITY OF DISSOCIATION CHANGES PRODUCED IN SALINE SOLUTIONS BY KNOWN QUANTITIES OF ELECTRICITY.

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OWING to the fact that electro-chemical action has now assumed such a prominent position in the investigation and accurate definition of the minute topographical anatomy of the central nervous system, the following observations may prove of interest.

These investigations have been undertaken with the object, primarily, of estimating the quantities of alkali and acid which are produced in living brain tissue under the influence of electrolysis in the neighbourhood of the kathode and anode respectively as one of the residual products of electrolytic dissociation, brought about by means of feeble constant currents of known milliamperage.

Through the kindness of Sir Victor Horsley and Dr. R. H. Clarke, I have been present during many of their experiments, and subsequently had the opportunity of examining numerous microscopical sections of portions of brain substance, wherein minute lesions have been produced by electro-chemical means.

Although the kathodal lesion has been attributed mainly to the action of sodium hydroxyl, yet an examination of these lesions, both kathodal and anodal, affords striking presumptive evidence that in addition to mere chemical agencies a stronger and more potent factor is called into play in their production; for in every instance a marked mechanical element stands out pre-eminent. This mechanical factor is undoubtedly due to the pressure of the gases evolved during electrolysis, the effects of which are not alone dependent upon the volume and quality of such gases, but also upon the density of the current which has been responsible for their production.

A detailed description of these lesions is here superfluous, as they have been fully described in an article on this subject by Sir Victor Horsley and Dr. R. H. Clarke [1].

For the purpose of these investigations, however, there is one point upon which I desire to lay especial stress, namely, the fact that it is blood and blood-clot, and not actual brain tissue, which is invariably found in the immediate vicinity of the electrodes; and consequently it must of necessity be within this substance that the major portion of electro-chemical action takes place, although, as will be subsequently shown, the reaction is by no means confined to this area, for certain of the products of dissociation are undoubtedly disseminated through the tissues into which the gases evolved make their way.

The fact that alkali and acid are produced in brain tissue in the neighbourhood of the kathode and anode respectively under electrolysis is undoubted, and is but the natural outcome of electro-chemical laws. For brain substance may be regarded electro-chemically as a saline electrolyte, since it is but an aqueous jelly, holding immeshed in every part inorganic salts (derived from the blood and lymph); under the influence of electrolysis, therefore, it offers but little more resistance to the ionic migration of these substances than does a simple electrolytic solution of these salts to which Faraday's laws are applicable.

The presence of alkali and acid may be demonstrated on their production in brain tissue by bathing the neighbourhood of the kathode and anode with weak solutions of phenolphthalein and dimethylamido-azo-benzol; on closure of the current, gas is immediately evolved, and simultaneously the colour index, purple red and rose pink, appear at the kathode and anode as evidence of the presence of alkali and acid respectively. It is worthy of note that the purple red colour increases in intensity in proportion to the duration and strength of the current, and that it is chiefly conspicuous in the immediate vicinity of the electrode, but yet is in no way confined to this area, for it will be seen to spread rapidly in all directions in which the gas evolved may travel; this colour, therefore, may in many instances be seen upon the surface of the brain though the electrode is buried beneath its surface and not apparently connected therewith. But on closer examination it will be seen that minute bubbles of gas have permeated the brain substance, and even after so doing they retain the power of conveying with them an alkaline atmosphere, as is shown by this phenomenon.

This fact is important in proving the statement previously made

that the products of electrolytic dissociation are not confined to the immediate vicinity of the electrode, but, on the contrary, are universally distributed over the area permeated by the gases evolved during electrolysis.

Similarly evolution of gas occurs at the anode, but in far less appreciable quantity than that evolved at the kathode. Almost simultaneously with its appearance will be seen a faint rose pink tinge at a minute distance from the electrode, showing, therefore, the formation of an acid; the colourless zone seen in the immediate vicinity of the electrode is due undoubtedly to the bleaching effect of the chlorine there liberated, which can be readily identified by its odour even at some little distance from the electrode. To this phenomenon allusion will be made when dealing with the quantitative estimation of acid, as compared to that of the alkali.

Having shown that acid and alkali are produced in brain tissue during electrolysis, it is of interest to trace the chief source of their origin. In this connection it is important to remember that the electrodes (as already stated) are surrounded by blood. It follows, therefore, of necessity that from the inorganic constituents of the serum (*vide* note 1) an acid and alkali must be produced as the outcome of its electrolytic dissociation.

Note 1.

Inorganic constituents in 1,000 grm. human blood-serum.[1]

Total inorganic material ...	8·57 grm.
Potassium sulphate	0·283 grm.
Potassium chloride	0·362 grm.
Sodium chloride	5·591 grm.
Sodium phosphate	0·273 grm.
Soda	1·545 grm.
Calcium phosphate	0·300 grm.
Magnesium	0·220 grm.

From the above figures it is clearly evident that blood-serum is a highly ionized saline electrolytic solution, within which preponderates to a very marked degree the readily dissociable inorganic salt sodium chloride. Consequently, under the influence of electrolysis, of the many

[1] C. Schmidt's estimation (*vide* Limbeck, "New Sydenham Society," 1901, vol. cxlvii., p. 87).

metallic radicals present, sodium is readily and freely liberated at the kathode, and, being incapable of individual existence in the presence of water, it reacts upon it to form sodium hydroxyl, liberating thereby an equivalent quantity of hydrogen.

Similarly under these circumstances chlorine, which is the chief acid radical, is liberated at the anode, which, in its turn, reacts to a variable extent upon the water present, thereby setting free an equivalent quantity of oxygen, an acid being simultaneously produced.

With regard to the exact nature of the acid here formed, it is impossible to make any definite statement, owing to the numerous and complex subsidiary reactions which occur in chlorine dissociation, a fact which has been ably illustrated in the works of Förster and Müller.

It is, however, certain that during anodal dissociation of such an electrolyte, a concentration of hydrogen ions takes place in the neighbourhood of the electrode, a phenomenon which must contribute materially to the increased acidity which takes place during electrolysis.

It is also equally certain that though a minute quantity of chlorine escapes during electrolysis, yet a large proportion remains in the electrolyte, either free or in a loose chemical combination, as is shown by the marked bleaching propensities of the same long after electrolysis has ceased. It is possible, therefore, that some of this chlorine reacts upon the hydroxidion and hydrion present, with the production of hypochlorite and hydrochloric acid.

This hypothesis is further strengthened by the fact that the total volume of gases actually estimated at the anode varies considerably with the density of the current; and such hypothesis would likewise account for the marked divergence subsequently shown in the total volumes of the gases liberated at the anode and kathode respectively when examined under identical conditions.

It is most probable, however, that the acidity subsequently estimated in these experiments is due to the first-named causes, *e.g.*, concentration of the hydrogen ions and hypochlorite, for the acidity therein ascertained proved of a very unstable and evanescent quality. This was shown by bringing moistened blue litmus paper in contact with the electrode during electrolysis, when the vivid red border surrounding the central bleached area so produced faded rapidly if the paper was immediately removed from the solution and washed and immersed in pure distilled water; this phenomenon was not so readily obtainable when the paper had been touched, even with a very weak solution of hydrochloric acid.

In addition to these substances many acids and alkalies, organic and

otherwise, and other complex bodies are produced as the outcome of electrolytic dissociation of blood; with these, however, I am not concerned, for my primary object is but to estimate approximately the total residual quantities of alkali and acid, the chief of which are in all probability those above specified.

With this object in view estimations were attempted in living brain tissue, portions of which were treated *in situ* within identical glass cylinders under precisely similar conditions. Such a method, however, proved totally inadequate, for though the conditions adopted were identical, the contents of these cylinders varied widely, and, consequently, neither comparative nor even approximate estimation could be made; and the investigation, for this reason, had to be relinquished.

Since, however, the electrodes in brain substance are intimately surrounded by blood, I have adopted—for the purpose of comparison and estimations—in the following experiments two saline electrolytic solutions, (A) and (B) (*vide* note 2), the one being but a simple 0·685 per cent. sodium chloride solution, the other a compound saline electrolytic solution similar in many respects to blood-serum in inorganic salts. By this means—for reasons subsequently shown—some approximate idea may be gained of the amounts of alkali and acid which are produced in brain substance under analogous (*i.e.*, electrolytic) conditions.

Note 2.

Solutions.

(A)	Sodium chloride	...	3·888	grm.
	Water (distilled)	...	568·340	c.c.

Thus = 0·685 sodium chloride solution.

(B)	Sodium chloride	...	3·24	grm.
	Sodium sulphate	...	0·162	,,
	Sodium carbonate	...	0·162	,,
	Sodium phosphate	...	0·1296	,,
	Potassium chloride	...	0·1944	,,
	Water (distilled)	...		568·340 c.c.

3·888 grm.

(Compare with note 1, blood-serum.)

50 c.c. (A) solution equivalent in alkalinity to 0·00008 grm. NaOH.
50 c.c. (B) solution equivalent in alkalinity to 0·0036 grm. NaOH.

(Though the above represents the average alkalinity of these solutions, they are subject to slight variation under atmospheric conditions, and were consequently examined prior to any series of experiments.)

In order to carry out the following estimations, I had made for me by Messrs. Müller and Co. the glass electrolytic apparatus shown in fig. 1, consisting essentially of two limbs connected by a narrow glass tube fitted with a stopcock. Each limb of the apparatus is capable of containing 50 c.c. of fluid and is graduated in cubic centimetres and $\frac{1}{10}$ c.c.,

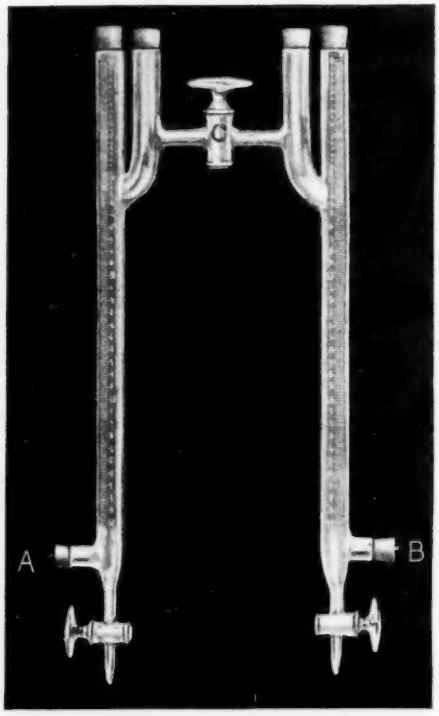

FIG. 1.
(One-third original size.)
A and B, platinum electrodes. C, stopcock.

and fitted with stopcocks and rubber corks through which insulated platinum electrodes are inserted.

By this arrangement mechanical disturbance in the fluids, due to the evolution of gases during electrolysis, is prevented, and no diffusion occurs between the products of dissociation at the anode and kathode.

In this respect the instrument proved accurate for estimations extending over periods of twenty minutes when using currents up to 5 ma. The simplicity of its construction permits of thorough cleansing of every part, the chief disadvantage being its high internal resistance, necessitating as it does a powerful battery of 12 volts to 15 volts to obtain currents of 4 ma., even in electrolytes so highly ionized as the solutions under estimation. (In a normal solution of sodium chloride at 18° C. a little over two-thirds of the salt is split up into its constituent ions.)

My first experiments were conducted with solutions (A) and (B), in order first to estimate the actual residual quantity of alkali which is formed in the two solutions by currents of known milliamperage; and secondly, by comparing the results thus obtained, to ascertain to what extent they were influenced by subsidiary reaction.

In order to make these estimations, the original alkalinity of the solution having been estimated, the apparatus was filled with one or other of the solutions to be examined, and then fixed rigidly so that no movement could take place; the stopcock, C, being opened, the solutions were then subjected to electrolysis, the current being regulated by means of a rheostat and measured by a galvanometer.

On completion of electrolysis the stopcock, C, was closed and the whole or part of the contents of either limb was withdrawn for examination. This was carried out by adding a known quantity of neutral litmus solution and titrating with $\frac{1}{100}$ N NaOH or H_2SO_4 solution, the end reaction being indicated by comparison with a standard colour.

1 c.c. NaOH $\frac{1}{100}$ N = 0·0004 grm. NaOH.
1 c.c. H_2SO_4 $\frac{1}{100}$ N = 0·00049 grm. H_2SO_4.

(Owing to the composition of solution (B) it must of necessity follow that in addition to the sodium hydrate and hydrochloric acid, other alkalies and acids are produced as the outcome of electrolysis. Consequently, throughout this article, wherever solution (B) was utilized the figures are but relative, signifying only that an amount of alkalinity or acidity was estimated which is equivalent to an amount of NaOH or H_2SO_4 which is shown by these figures.)

In order to attain my first object, namely, to ascertain the quantities of alkali which are produced in the solutions (A) and (B) under electrolysis, a series of twenty experiments was made, a current of 1 ma. being utilized for a period of ten minutes, the results of which experiments are shown in the following table:—

TABLE I.

Estimations with regard to the amount of Alkali which is produced in Solutions (A) and (B) under the influence of Electrolysis.

	50 c.c. of Electrolytic Solution. (A)			50 c.c. of Electrolytic Solution. (B)		
Conditions. Kathode	Original alkaline equivalent of the electrolyte prior to electrolysis	*Estimated gain in alkalinity after electrolysis*	Such amount when reduced to terms of 1 ma. × 5 m.	Original alkaline equivalent of the electrolyte prior to electrolysis	*Estimated gain in alkalinity after electrolysis*	Such amount when reduced to terms of 1 ma. × 5 m.
	Grm. NaOH	Grm. NaOH	Grammes	Grm. NaOH	Grm. NaOH	Grammes
Same in both electrolytic solutions, namely, 50 c.c. of solution, 1 ma. current, 10 m. duration, 1 cm. electrode, 23 gauge S.W.G.	0·00008	**0·00026**	0·00013	0·0032	**0·00032**	0·00016
	0·00008	**0·00024**	0·00012	0·0032	**0·000213**	0·0001065
	0·00008	**0·00024**	0·00012	0·0032	**0·000266**	0·000133
	0·00008	**0·00028**	0·00014	0·0032	**0·00024**	0·00012
	0·00008	**0·00026**	0·00013	0·0032	**0·000266**	0·000133
	0·00012	**0·00024**	0·00012	0·00512	**0·00032**	0·00016
	0·00012	**0·00024**	0·00012	0·00512	**0·0002932**	0·0001466
	0·00012	**0·00024**	0·00012	0·00512	**0·00024**	0·00012
	0·00012	**0·00024**	0·00012	0·00512	**0·00024**	0·00012
	0·00012	**0·00024**	0·00012	0·00512	**0·000266**	0·000133
Total:		**0·00248**			**0·0026642**	

For purposes of comparison in the above and all subsequent experiments all estimations have been reduced to a common denomination, signified in the right-hand column of each series of experiments.

A review of the above figures definitely proves three points: First, that in both solutions an alkali is produced; secondly, that in both instances the amount is very small; and, thirdly, that the difference in the respective quantities is exceedingly minute.

It is seen that in solution (A) the total gain in alkali is equivalent to 0·00248 grm. NaOH, as against 0·0026642 grm. in (B), representing, therefore, a total difference of only 0·0001842 grm. in ten experiments extending over a period of ten minutes, or an average per estimation of 0·000248 grm. in (A), as against 0·00026642 grm. in (B), a difference of only 0·00001842 grm. NaOH in individual estimations.

These quantities are exceedingly small, although the period of electrolysis was of comparatively long duration. Now since it was my object to ascertain what amount of alkali is actually produced in brain tissue during the formation of the lesions above alluded to, I have still further reduced these amounts proportionately for currents of 1 ma.

acting for thirty seconds, which for reasons subsequently shown may be done by dividing these figures by 20 (*vide* Tables I. and IV.). By so doing we ascertain that 0·0000124 grm. NaOH is produced in (A), as against 0·000013321 grm. in (B), a difference therefore in each estimation of 0·000000921 grm. only NaOH in the two electrolytes under these conditions, which quantity may be looked upon as non-existent.

Since, therefore, this difference is practically inappreciable in two solutions varying so essentially from one another, though alike in their main inorganic content, NaCl, it is seen that subsidiary reaction may be disregarded, and, therefore, an approximate estimate may be gained of the amount of alkali which is formed in blood-serum under the influence of electrolysis; for there exists between it and solution (B) a marked similarity in inorganic content, and in both of them sodium chloride preponderates, from which substance the alkali is doubtless produced in brain tissue.

If such analogy be admitted the following experiments may be of service in giving some approximate idea of the amounts of alkali and acid which are produced in brain tissue under similar conditions; and also an insight with regard to certain other phenomena which contribute to the production of these lesions.

In the experiments enumerated under Table II. I have estimated the amount of alkali which was produced by means of currents of 2 ma. in solution (B) acting for periods of five, ten and twenty minutes.

TABLE II.

Estimations with regard to Duration of Electrolysis.

Current, 2 ma.; electrolytic solution (B).

Gain in NaOH in 5 minutes	Reduced to 1 ma. for 5 m.	*Gain in NaOH in 10 minutes*	Reduced to 1 ma. for 5 m.	*Gain in NaOH in 20 minutes*	Reduced to 1 ma. for 5 m.
0·00028	0·00014	**0·00052**	0·00013	**0·00104**	0·000125
0·00024	0·00012	**0·00052**	0·00013	**0·00116**	0·000145
0·00032	0·00016	**0·0004**	0·0001	**0·00128**	0·00016
0·00024	0·00012	**0·0006**	0·00015	**0·00124**	0·00013
0·00028	0·00014	**0·0006**	0·00015	**0·00116**	0·000145
Total: **0·00136**		**0·00264**		**0·00588**	

Results here obtained point to a definite proportional relation between the amount of alkali produced and the duration of the current, or as

may be otherwise expressed, the amount of alkali is directly proportional to the amount of electricity which flows through the solution in a given time.

Under Table III. will be seen the results of experiments conducted with solution (B), under identical conditions as to time and milliamperage of the current, the only difference being the calibre of the electrodes.

TABLE III.

Estimations with regard to the Calibre of Electrodes.

(Here synonymous with density of current.)

Current, 2 ma.; duration ten minutes; electrolytic solution (B).

Gain in NaOH Electrode 1 mm. long Gauge $\frac{1}{6}$ mm.	Gain when reduced to 1 ma. for 5 minutes	*Gain in NaOH Electrode 2 mm. long Gauge $\frac{1}{6}$ mm.*	Gain when reduced to 1 ma. for 5 minutes	*Gain in NaOH Electrode $\frac{1}{2}$ cm. long Gauge 23 S. W. G.*	Gain when reduced to 1 ma. for 5 minutes	*Gain in NaOH Electrode 1 cm. long Gauge 23 S. W. G.*	Gain when reduced to 1 ma. for 5 minutes
0·00064	0·00016	**0·00052**	0·00013	**0·0006932**	0·0001733	**0·00048**	0·00012
0·0004264	0·0001066	**0·0006**	0·00015	**0·000532**	0·000133	**0·000532**	0·000133
0·000532	0·000133	**0·0006**	0·00015	**0·00048**	0·00012	**0·000532**	0·000133
0·000532	0·000133	**0·0006**	0·00015	**0·0004264**	0·0001066	**0·000672**	0·000168
0·000532	0·000133	**0·00052**	0·00013	**0·00048**	0·00012	**0·000532**	0·000133
0·00064	0·00016	**0·0006**	0·00015	**0·0006932**	0·0001733	**0·00064**	0·00016
0·0005864	0·0001466	**0·00052**	0·00013	**0·000672**	0·000168	**0·000782**	0·0001955
0·0005864	0·0001466	**0·0006**	0·00013	**0·00052**	0·00013	**0·00048**	0·00012
Total: **0·0044752**		**0·00456**		**0·0045368**		**0·00465**	

Seeing that the currents, time and solutions are identical, the above experiments may be regarded as an estimation wherein the density of the current is examined, with regard to its effect upon the quantities of alkali produced.

These figures show that no virtual difference is appreciable in the residual amounts of alkali, though such estimations have been made with electrodes varying markedly in size. Consequently, the density of the current does not virtually affect the amount of alkali produced provided the quantity of electricity be the same. (*Compare* Table VI.)

The next investigations were conducted in order to ascertain to what extent the quantities of alkali produced were influenced by the milliamperage of the current in any given time, namely, currents of 1 ma., 2 ma. and 4 ma.

TABLE IV.

Estimations with regard to the Milliamperage of Currents of 1 ma., 2 ma. and 4 ma. acting for a period of ten minutes in Solution (A).

(Herein synonymous with Quantity of Electricity.)

Duration, ten minutes; electrolytic solution (A).

Gain in NaOH Current 1 ma.	Gain when reduced to 1 ma. for 5 minutes.	*Gain in NaOH Current 2 ma.*	Gain when reduced to 1 ma. for 5 minutes.	*Gain in NaOH Current 4 ma.*	Gain when reduced to 1 ma. for 5 minutes
0·00024	0·00012	**0·00052**	0·00013	**0·00092**	0·000115
0·00024	0·00012	**0·00048**	0·00012	**0·001**	0·000125
0·00024	0·00012	**0·00048**	0·00012	**0·001**	0·000125
0·00024	0·00012	**0·00048**	0·00012	**0·00112**	0·00014
0·00024	0·00012	**0·00052**	0·00013	**0·00084**	0·000105
Total: **0·0012**		**0·00248**		**0·00488**	

For these experiments solution (A) was selected in preference to solution (B), since previous experiments proved that the delicacy of the end reaction was more apparent, and the results thus obtained more constant (*vide* Table I.); a fact which may be attributed to the simplicity of its composition, and its faint original alkalinity.

Fifty cubic centimetres of solution (A) represented prior to estimations an alkalinity equivalent to 0·00012 grm. NaOH, the electrode used being 1 cm. of platinum 23 Imperial Standard Wire Gauge.

It will be seen that a very definite proportional relation is established between the amount of alkali produced and milliamperage of the current in a given time.

A survey of the above Tables I. to IV. proves that the resultant product of electrolytic dissociation of these electrolytes—in so far as the alkali—is virtually dependent upon the amount of electricity which flows through them, and upon no other factor.

That this relative proportionality is maintained throughout the whole series of these experiments is quite apparent, for the results, in so far as the alkali produced, are almost invariably alike; it does not matter whether currents of high or low density be used, or whether 2 ma. for five minutes or 1 ma. for ten minutes be utilized, the results are virtually the same. From this fact it may be assumed that the slight variation in the two solutions, as regards the amount of alkali produced, is not so materially due to the quantity or quality of electricity which has been utilized as to the original nature of their composition, and that subsidiary reaction apparently in no way influences the results.

With regard to the estimation of the acid I was unable to obtain the same definite results, this being, I consider, chiefly accounted for by interference with the delicacy of the indicator, owing to the bleaching effect of the chlorine in solution after electrolysis.

This difficulty was in a measure overcome by adding to the content of the anodal chamber, prior to titration, a known and sufficient quantity of $\frac{1}{100}$ N NaOH, in order to make the solution alkaline, thus combining any free chlorine present. The solution was then titrated against a known quantity of $\frac{1}{100}$ N acid solution, allowance being made in the estimations for the previous alkalinity of the solution, together with the quantity of alkali added subsequent to electrolysis.

The following Table V. shows the results obtained in five simultaneous estimations of the respective amounts of acid and alkali produced in solution (A) under the influence of currents of 2 ma. of ten minutes duration, the results being expressed in terms of equivalent quantities of NaOH, H_2SO_4 and HCl respectively.

TABLE V.

Estimations of the respective amounts of acid and alkali produced simultaneously in Solution (A).

		Kathode.	*Anode.*				
Experiment	Conditions	*Gain in alkalinity NaOH*	*Original equivalent alkalinity of electrolyte 50 c.c.*	*Amount of alkali added NaOH*	*Amount of H_2SO_4 required for titration*	*Equivalent gain in H_2SO_4*	*Equivalent gain in HCl*
1	1 cm., 23 S.W.G. electrode, 2 ma. currents, 10 minutes duration	0·00052	0·2 c.c. $\frac{1}{100}$ NaOH	2 c.c. $\frac{1}{100}$ N	1·1 c.c. $\frac{1}{100}$ N	0·000539	0·00040095
2		0·00048	0·2 c.c. $\frac{1}{100}$ NaOH	2 c.c. $\frac{1}{100}$ N	0·9 c.c. $\frac{1}{100}$ N	0·000441	0·00032805
3		0·00072	0·2 c.c. $\frac{1}{100}$ NaOH	3 c.c. $\frac{1}{100}$ N	1·3 c.c. $\frac{1}{100}$ N	0·000637	0·00047385
4		0·00056	0·2 c.c. $\frac{1}{100}$ NaOH	4 c.c. $\frac{1}{100}$ N	0·8 c.c. $\frac{1}{100}$ N	0·000392	0·0002916
5		0·00052	0·2 c.c. $\frac{1}{100}$ NaOH	4 c.c. $\frac{1}{100}$ N	0·8 c.c. $\frac{1}{100}$ N	0·000392	0·0002916
Total:		0·0028				0·002401	0·00178605

The addition of any substance to the solution subsequent to electrolysis must of necessity give rise to the possibility of error, and consequently I do not attach the same importance to these estimations as I do to the results obtained in previous experiments.

It is seen that the same uniformity in the amounts of alkali is not found as in previous experiments, a conspicuous difference being noticeable in experiment 3. If, however, these figures be compared with Table IV., experiment 2, wherein the same conditions obtained, it will be noticed that the results are almost identical, except in this one

instance; the difference therefore is most probably due to experimental error.

With regard to the acid formation a comparatively wide variation is evident, but yet a marked relative proportionality exists between these quantities and those of the alkali simultaneously produced. For this reason, together with the fact that in different experiments different quantities of alkali were added without materially altering this proportion, I contend that the above figures may be regarded as a fairly accurate estimate of the actual residual quantities of acid which were produced.

It is further seen that the total amount of acid formed is very small having regard to the milliamperage of the current and duration of the electrolysis, the total amount formed being equivalent to 0·00178605 grm. HCl in five estimations, or an average per estimation of 0·00035721 grm. in ten minutes, produced by a current of 2 ma. Assuming the same analogy to exist in this as in the estimation of alkali, we ascertain that the amount produced under the influence of a current of 1 ma. acting for thirty seconds is 0·00000893025 grm. HCl (equivalent).

Now, since the equivalent of 0·0028 grm. NaOH is 0·0025506 grm. HCl it is apparent that equivalent amounts of acid and alkali have not been produced in these experiments, the total difference being equivalent to 0·00076455 grm. HCl in five estimations, and consequently an average difference of 0·00015291 grm. HCl in individual estimations, which represents, when reduced to terms of 1 ma. for thirty seconds, a difference only of 0·00000382275 grm. HCl.

This inequality in equivalent amounts of acid and alkali might have been anticipated from the fact that during electrolysis an appreciable quantity of chlorine was lost to the solution, and that a considerable quantity was invariably found throughout the experiments to remain free in the solution.

A similar phenomenon, namely, the escape of chlorine, has been already alluded to when proving the formation of acid in brain tissue under electrolysis; from analogy, therefore, a similar condition may be assumed to obtain in the neighbourhood of these lesions, a lesser quantity of acid being produced than alkali under identical conditions.

In comparing these results with those ascertained in experiments enumerated under Table I., in which the same electrolyte was examined, we find that the amount of acid produced is to the amount of alkali as 0·00000893025 grm. HCl is to 0·0000124 grm. NaOH, when currents of 1 ma. are utilized for electrolysis extending over a period of thirty seconds.

Since there is practically no difference in the quantities of alkali produced in the two electrolytes, and the object of this research has been to establish an analogy between the results herein ascertained and those which may be assumed to obtain in brain tissue under electrolysis, an average of eighty-seven experiments has been taken in which the estimation of alkali has been made, and the amount thus ascertained is found to be 0·0000133969 grm. NaOH.

It may be stated, therefore, that the amount of alkali produced is to the amount of acid as 0·0000133969 grm. NaOH is to 0·00000893025 grm. HCl, during electrolysis extending over a period of thirty seconds with 1 ma. current.

Certain definite conclusions are therefore arrived at from a consideration of these experiments: First, that in both solutions under the influence of electrolysis alkali and acid are produced at the kathode and anode respectively; secondly, that the amount in each is exceedingly small; and thirdly, that though there is a tendency to the slightly greater formation of alkali in the compound solution (B) than there is in (A), this difference is scarcely appreciable.

If it is allowed that there is an analogy between the results of these experiments and the changes which may be assumed to take place in brain matter under electrolysis, I contend that these figures afford strong presumptive evidence that in addition to chemical action other and more potent factors must be called into play in the production of these cerebral lesions. For although it cannot be doubted that they contribute somewhat to the result, yet having regard to their minute quantities and manner in which they are disseminated, it is difficult to conceive how they can have any definite bearing upon the production of these lesions.

For the above reasons I have carried this research a little farther, in order to ascertain to what extent the production of these lesions may be attributed not only to the volumes of gas evolved, but also to the nature of their evolution; for, from certain observations made during the preceding experiments, I was struck by the probability that such factors must have a very marked bearing not only on the production of the lesions, but also upon their nature and extent.

With regard to the evolution of the gases three facts were conspicuous: First, that the gases evolved had the power of carrying with them the product of dissociation, a point which has already been shown; secondly, that the gases produced at the kathode and anode differed very considerably in volume; and thirdly, that the density of the current had

a distinct bearing not only upon the nature of their evolution, but also upon the actual volume produced.

The first point may be further demonstrated by adopting the same procedure as in previous experiments, having added to the anodal and kathodal chambers respectively one or two drops of dimethylamido-azo-benzol (0·5 per cent. alcoholic solution) and phenolphthalein (1 per cent. alcoholic solution). On closure of the current gas will be seen to be evolved at the two electrodes, and a faint rose pink tinge will be seen to be developed in the solution in the anodal chamber, more especially just under the meniscus, as evidence of the formation of an acid; this colour, however, rapidly disappears as the fluid becomes more saturated with chlorine.

In the kathodal chamber a very much more vivid and definite effect is produced; for simultaneously with the closure of the current a vivid flash of violet red is seen as evidence of alkali formation, which colour is first noticeable in the immediate vicinity of the electrode, and is then conveyed within the column of bubbles to the surface of the fluid, where, beneath the meniscus, it becomes rapidly accentuated and then gradually falls down the sides of the tube, producing the phenomena known as streaking.

These phenomena might be looked upon as the outcome of mechanical convection; but that this is not altogether the case may be shown by breaking the current suddenly, before the gases reach the surface; the streaking is then produced in the centre of the column of fluid, as though the gases were thereby deprived of their inherent power of conveying with them certain products of dissociation. This point is of practical importance when it is considered that through this inherent power the gases generated in brain tissue during electrolysis are capable of disseminating in the minutest manner possible the products of electrolytic dissociation, bringing them into immediate contact with every part with which these gases come in contact.

The second point is best illustrated by a survey of the following table, wherein is given a comparative estimation of the total volumes of gas which are liberated at the anode and kathode respectively, as also the volumes generated by currents of different density.

For the purpose of these estimates, the apparatus was inverted and the gases were collected in small graduated glass cylinders inverted over the electrodes and immersed within the solution within the respective limbs of the apparatus, electrodes of the same and different calibre being used for the purposes of comparative estimations.

TABLE VI.

Simultaneous estimations of the total volumes of gas liberated at the anode and kathode respectively by means of a current of 2 *ma., the electrodes being platinum* 1 *cm. long,* 23 *Imp. S.W.G., and* $\frac{1}{2}$ mm., $\frac{1}{10}$ *mm. in diameter; barometer* 30·5*; thermometer* 14° *C. Solution* (*A*).

Kathode.			*Anode.*		
Large electrode 1 cm., 23 Imp. S.W.G.	*Volume of gas in c.c.*	Small electrode $\frac{1}{2}$ mm. $\frac{1}{10}$ mm.	Large electrode 1 cm., 23 Imp. S.W.G.	*Volume of gas in c.c.*	Small electrode $\frac{1}{2}$ mm., $\frac{1}{10}$ mm.
Time in minutes		Time in minutes	Time in minutes		Time in minutes
5	**0·05**	4	20	**0·05**	24
7·5	**0·1**	7·5	36	**0·1**	47
11	**0·15**	10·5	50	**0·15**	66
15	**0·2**	14	60	**0·2**	86
18·5	**0·25**	17	80	**0·25**	104
20·5	**0·3**	20	95	**0·3**	122
24·5	**0·35**	25	108	**0·35**	145
29	**0·4**	27	120	**0·4**	168
31	**0·45**	30·5	133	**0·45**	187
34·5	**0·5**	34·5	147	**0·5**	208
Average	**0·05 c.c.**	**in 3·45 minutes**	**Average 0·05 c.c. in 14·7 minutes**		**Average 0·05 c.c. in 20·8 minutes**

Owing to the length of time required to obtain appreciable quantities of gas a current of 2 ma. was used; and for the same reason extremes in the calibre of the electrodes were adopted in order to ascertain the effect produced in the volumes of the gas by currents of different density.

Three points are here clearly indicated:—

First, that an appreciable volume of gas is generated in all instances.

Secondly, that the volume generated at the kathode in each instance is far larger than that generated at the anode; and

Thirdly, that the volume generated at the kathode is unaffected by the density of the current, whereas at the anode a marked contrast exists in this respect.

It is seen that whether generated at the larger or smaller electrode the volume of gas at the kathode is the same, 0·5 c.c. being generated in 34·5 minutes under a current of 2 ma., or 0·05 in 3·45 minutes. The second point is a matter of practical importance when applied to the subject under review, a fact which is well illustrated by an examination of the cerebral lesions produced by electrolysis, wherein it is seen that

the kathodal lesion is large and diffuse when compared with the anodal lesion, which is small and defined (*vide* photos Horsley and Clarke [1]).

The third point is also of very practical interest as illustrating a means whereby the volumes of gas eliminated may be controlled.

Throughout the experiments enumerated in the above and following tables it was observed that the rate of evolution of these gases was in individual cases constant, and that a definite proportional relation existed between the volume of gas and duration of electrolysis. This is a point of further interest and importance, since in accordance with Faraday's laws of electro-chemical equivalents and definite proportions it affords conclusive evidence of the accuracy of the estimations enumerated in Tables II., III. and IV., wherein it was shown that the quantities of alkali produced were in no way dependent upon the calibre of the electrode, provided the quantity of electricity was constant, and further that the quantities of alkali were proportional to the duration and milliamperage of the current. The proportion here established likewise proves in accordance with these laws that the same relation exists between the milliamperage of the current and volume of gas generated under identical conditions. Consequently the amount of gas which was estimated in the above experiments is in every instance double that which would obtain under the influence of 1 ma. current. The following results therefore are ascertained: that the actual volume of gas which is generated within the electrolyte (A) in thirty seconds with 1 ma. current is respectively 0·00362 c.c. at the kathode under any circumstances, and 0·0008 c.c. at the larger anode, as against 0·0006 c.c. at the smaller anode.

Since the results herein ascertained are in accordance with the quantitative estimates of the alkalies produced, and also in conformity with certain definite conditions observed in the lesions by Horsley and Clarke [1], it may be assumed that the above figures are a fairly approximate estimate of the volumes of gas which are generated in brain tissue; for such estimations bear a distinct relationship to the nature and extent of the lesions produced under analogous conditions.

Although it is practically impossible to estimate the complexity of reactions which are actually responsible for the variation in the volumes of gas generated at the respective anodes, it is certain that such must, in this instance, be the direct outcome of the density of the currents, whereby subsidiary actions are produced, for in all other respects the conditions were identical.

This illustrates a point of the utmost practical importance when

considering the production of these cerebral lesions; for it is seen that for any given current the greater its density the smaller is the volume of gas liberated at the anodal electrode; and thereby is illustrated a means of confining within certain limits the nature and extent of the lesion.

In order to verify these results three further experiments were undertaken, from which it will be seen that the conclusions arrived at were almost identical with those enumerated in Table VI.

TABLE VII.

Simultaneous estimations with regard to the total volumes of gas generated at the anode and kathode respectively. Large electrode 23 *Imp. S.W.G.; small electrode* $\frac{1}{2}$ *mm. long and* $\frac{1}{10}$ *mm. in diameter.*

Kathode.			*Anode.*		
Large electrode 1 cm., 23 Imp. S.W.G.	*Volume of gas in c.c.*	Small electrode $\frac{1}{2}$ mm., $\frac{1}{10}$ mm.	Large electrode 1 cm., 23 Imp. S.W.G.	*Volume of gas in c.c.*	Small electrode $\frac{1}{2}$ mm., $\frac{1}{10}$ mm.
Time in minutes		Time in minutes	Time in minutes		Time in minutes
				0·05	24
				0·1	48
	0·5	35		**0·15**	66
				0·2	83
				0·25	102
			18	**0·05**	
			30	**0·1**	
35	**0·5**		43	**0·15**	
			56	**0·2**	
			70	**0·25**	
				0·05	23
				0·1	45
	0·5	35		**0·15**	65
				0·2	82
				0·25	98

Owing to the time required to produce appreciable quantities of gas at the anode, estimations in the above were only conducted for a period when 0·25 c.c. of gas had been generated. The thermometer in these experiments was 16° C.; the barometric pressure I failed to note; such, however, can have but little bearing on the point under review, since the volume of gases is so small that any difference would be inappreciable.

From the last two series of experiments as to the volumes of gases liberated at the respective electrodes under different conditions, the following definite conclusions may be stated:—

First, that the total volumes of gas vary considerably at the kathode and anode respectively, but in all instances are appreciable in quantity.

Secondly, that the volume of gas generated at the kathode in a given time is unaffected by the density of the current.

Thirdly, that in every instance the volume of the gas generated by a given current in a given time at the kathode is greater than the volume of gas simultaneously liberated at the anode, and

Lastly, that the volume of gas generated at the anode varies directly with the size of the electrode, and indirectly as density of the current.

The last conclusion may be briefly stated as follows :—

That provided the milliamperage of the current is constant, the smaller the anodal electrode the smaller will be the volume of gas which is generated. This is a point of much practical interest in the electrolysis of cerebral substance, and is amply borne out by the minute accurately defined lesions which are obtained when these conditions have been adopted.

From the above facts as to the actual volumes of the gases which are produced it must be granted that, apart from any chemical action which they may possess, their mere volume must have a definite mechanical effect in cerebral tissue, and this is illustrated by the character of these lesions (Horsley and Clarke [1]). But I contend that a more potent factor has still to be established in order to account for certain mechanical effects which are apparent in these lesions, wherein the actual volume of gas produced must be exceedingly minute.

The point to which I now allude is the marked mechanical effect which is produced, not so much by the actual volume of the gases as by the manner of their generation. This factor is undoubtedly dependent on the density of the current, owing to the initial impetus with which the gases are ejected from the electrode.

During all the previous experiments it was observed that although the gases might be developed in equal volume in a given time with a given current, yet the precise nature of this evolution differed essentially with the size of the electrode. This was most definite in the estimation of the volumes of gases liberated at the kathodes under currents of identical milliamperage, wherein comparatively large but equal volumes of gas were generated, though the electrodes varied widely in calibre.

This phenomenon was further well illustrated by using the apparatus above alluded to, and filling it with solution (A), to which three or four drops of phenolphthalein (1 per cent. alcoholic solution) had been added; the electrodes used were of different calibre, 1 cm. 23 Imp. S.W.G., and $\frac{1}{2}$ mm. by $\frac{1}{10}$ mm., and the instrument was inverted, the

current used being 2 ma., regulated and measured by a rheostat and galvanometer. The large electrode now being the kathode, the current was closed, when simultaneously a violet red atmosphere was seen to surround the electrode, the electrode itself momentarily assuming the appearance of a piece of muffed glass, a condition which was due to minute gaseous particles which were seen adhering to it. As the current continued, these, however, rapidly ran together and, assuming considerable size, were slowly discharged from the electrode and passed to the surface of the fluid. The apparatus was now shaken to disseminate any colour and gas from either electrode and the current reversed. The smaller electrode being now the kathode, the current was closed, and a very much more striking effect was produced, for simultaneously with the closure of the current a vivid flash of rose red appeared, and was rushed through the centre of the column of fluid by means of the jets of gas which were discharged from the electrode, which in itself was covered by a seething mass of minute bubbles, which were ejected from the electrode with marked initial velocity.

It was noticed that the indicator of the galvanometer was not absolutely stationary, a slight but regular ticking being seen as evidence of the polarizing and depolarization of the electrode due to the evolution and discharge of its gas, an effect which is well illustrated in the following photograph (fig. 2).

That mechanical energy is here displayed is undoubted, and to this factor I attribute many of the phenomena which are evidenced in the cerebral lesions produced by electrolysis. For such mechanical energy cannot but produce marked distension in a substance such as cerebral tissue : the effect produced being in direct ratio to the density of the current and volumes of gas liberated.

To this fact I have already alluded, and it is therefore only necessary for me to observe that the strongest evidence of this mechanical energy is invariably displayed in all the cerebral lesions which I have seen produced by electro-chemical means (*see* Horsley and Clarke, figs. 20 *et seq.*, p. 98 of this volume.

Such lesions as above alluded to were in every instance produced by currents of comparatively high density analogous to those above enumerated, for minute electrodes were invariably used, with currents such as were examined in the above experiments and generated by a battery of like voltage. The gases which produced these lesions were therefore generated with marked potential energy, thus giving rise to a distension of the tissues and laceration of the vessels in the neighbourhood of the electrode, thus producing hæmorrhage not only in the immediate

neighbourhood of the electrode, but also radiating into those perivascular spaces into which the gases had forced their way.

A similar, but less marked, mechanical energy was displayed on examining the anodal electrodes under identical conditions; and an examination of the sections of these lesions, together with the photographs, proves that this phenomenon bears a definite relation to the conditions therein observed, for though the gases are still generated at a high density, their actual volumes are less, and consequently less destructive lesions were produced, a fact which is clearly evidenced by their compact and defined outline.

A consideration of this mechanical energy impresses one with the importance and bearing which this factor must have, not only in the production of the lesions in brain tissue, but also upon their nature and extent. In order further to illustrate this point from a practical standpoint a simple experiment was performed; instead of a single kathodal electrode, two electrodes of "apparently" similar calibre were made responsible for the kathodal dissociation, and we thereby obtained some idea of the great difference which may actually take place during electrolysis when precisely identical conditions are not observed. A fine thin platinum wire $\frac{1}{10}$ mm. in diameter was doubled upon itself, one end being insulated within a fine glass capillary tube, which was sealed $\frac{1}{2}$ mm. from the tip of the wire; the other end was similarly insulated within a closely insulating glass cylinder of the same dimensions, which permitted $\frac{1}{2}$ mm. of the wire to project; this cylinder, however, was not sealed upon the wire, which was retained in place by a small piece of wax, which did not close the actual lumen of the tube. The loop in the centre of this wire was now connected with the negative side of a battery, and the two kathodal electrodes inserted in the bottom of a narrow glass cell, containing solution (A), to which a drop of phenolphthalein solution was added. The anode was now inserted in the top of the cell equidistant from each kathode, and a current of 1 ma. utilized for electrolysis. The effect produced on closure of the current is exceedingly well represented in the accompanying photograph (fig. 2), which was taken by Sir Victor Horsley one-fifth of a second after closure of the current.

Striking evidence of the conditions which were actually observed is shown by the photograph, for simultaneously with the closure of the current a vivid flash of colour and immediate rush of gas took place from B, whereas for an appreciable interval nothing was observed at A, after which a large bubble appeared at the mouth of the glass insulating tube, and a few minute bubbles were evolved from the point of the wire;

these passed slowly through the solution, carrying with them a faint violet red colour, a certain amount of which was also noticeable within

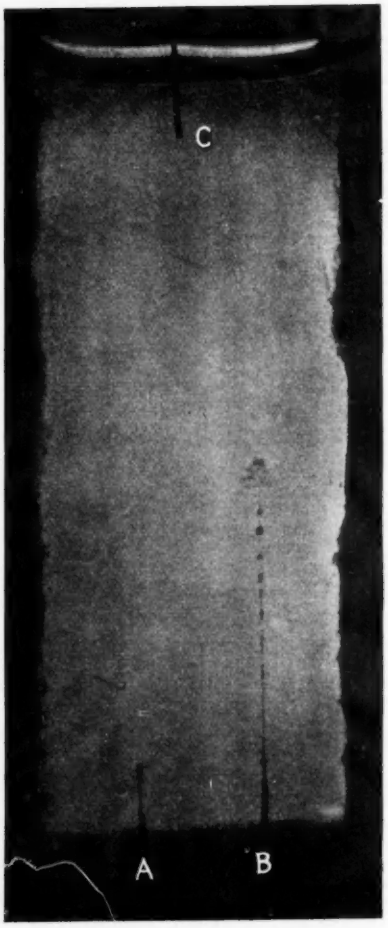

FIG. 2.

Glass cell and electrodes magnified two diameters: A, ineffectually insulated kathodal electrode; B, effectually insulated kathodal electrode; C, single anodal electrode. Current 1 ma. $\frac{1}{5}$ second after closure of current; $\frac{1}{2}$ mm. of wire projecting from each glass cylinder of the kathodal electrodes.

the glass insulating cylinder, from which a drop of coloured fluid subsequently escaped.

This experiment undoubtedly proves that a much more vivid electro-chemical reaction took place at B than at A, although it is probable that the quantity of electricity which passed through it was less than that which passed through A, owing to the greater resistance offered by the smaller area exposed to the electrolyte.

These phenomena clearly indicate that the electrolyte sustained considerable loss both in mechanical effect and chemical reaction, through wasted energy within the cylinder of the ineffectually insulated electrode.

From the "apparent" similarity in area of the actual electrodes exposed to the electrolyte within the cell, it might have been assumed that the ultimate results must be the same; but the above facts clearly prove the fallacy of any such supposition and show how great a difference may actually exist in instances where the conditions are not absolutely identical.

The experiment is of the utmost practical importance in proving the absolute necessity for accurate and effective insulation of the electrodes when uniform results, such as are essential in cerebral investigation, are most desirable.

The only means by which this can be attained is by accurate and effective sealing of the insulator at its distal extremity. When this is effected it is certain that the whole of the current which is actually registered must produce its products of dissociation within the brain substance, and not within the capillary container of the electrode, or even entirely without the brain substance through partial or complete short circuiting of the current; this is not only possible but very probable wherever it is essential to keep the tissues continually moistened during such investigation. What is here laid down with regard to electrolysis applies equally to faradic stimulation.

A practical difficulty is encountered in securing insulation of this nature when delicate glass cylinders are utilized for the purpose; for if they have been sealed upon the electrode, expansion during electrolysis and other factors are liable to crack the glass at the point of insulation; this difficulty is, however, readily overcome by utilizing a speck of rubber solution, or some such substance, in order to seal the distal extremity of the tube, for, unless this be done, capillary attraction draws within it the fluids of the part, and loss and uncertainty in the effects of electro-chemical reaction are inevitable.

That other factors than those above enumerated must inevitably contribute towards the formation of these cerebral lesions is only too

apparent, having regard to the complexity of reactions which must of necessity result from the electrolytic dissociation of a substance so complex as blood.

Yet, having regard to certain of the products of electrolysis as proved in the above experiments, many of the actual chemical changes which take place in blood-serum under analogous conditions may be ascertained by a reference to Starling's "Elements of Human Physiology" [2], from which an insight may be gained regarding many of the actual chemical reactions which are therein produced. Into so large a subject as this I regret I have not the ability to enter, and will now therefore but state certain conclusions which may be gathered from a consideration of the foregoing experiments.

Before doing so it is essential to state that for the purpose of these experiments only I have assumed that the acid formed as the outcome of electrolysis of these solutions was due to hydrochloric acid. Such presumption is inaccurate and justified only by the necessity for selecting some acid in respect of which comparative estimations might be made.

It is possible, as already pointed out, that a minute trace of hydrochloric acid is formed, but the major part of the acidity found in the solution at the anode after electrolysis of the solution (A) is not due to hydrochloric acid as such; for the chlorine herein liberated does not actually attack the water, but simply aids in its dissociation, remaining itself in simple solution as hydrolysed ions: the water is dissociated as hydrion and hydroxidion, the oxygen being then liberated from the hydroxidion, leaving a preponderating quantity of hydrion in solution; to this the whole or major portion of the acidity is probably accountable. That a like condition is also the outcome of electrolytic dissociation of solution (B) is evident from a consideration of its constituents.

With regard to other products of anodal dissociation, however, it has been shown that a very small, but yet appreciable volume of gas is produced; yet the point which is of the most practical importance is that free chlorine is undoubtedly liberated, and owing to its potency it must have a certain influence in the production of these lesions.

In kathodal dissociation, however, no doubt exists as to the formation of an alkali, which in solution (A) is sodium hydroxyl; this is the case also in solution (B), though other alkalies, notably potassium hydroxyl, are also produced, an equivalent quantity of hydrogen being liberated, which gas has been proved to have the power of conveying

with it during electrolysis these alkalies, which must also have a definite bearing upon the production of these lesions. The same alkalies are also formed in solution (B) and blood-serum as the outcome of its electrolytic dissociation owing to their similarity in inorganic content.

From the above experiments the following conclusions may be summarized :—

(1) That the definite alkalies sodium hydroxyl and potassium hydroxyl—and possibly a minute trace of other alkalies—are formed in brain tissue under the influence of electrolysis, the total amount produced with a current of 1 ma. in thirty seconds being equivalent to 0·000013396 grm. NaOH.

(2) That an acid is produced at a positive electrode which may be due to numerous causes ; the chief of these are most probably hydrion, hydrochlorite, and hydrochloric acid, the total quantity of which when eliminated at an electrode of 1 cm. long, 23 Imp. S.W.G., is equivalent to 0·00000893 grm. HCl, or 0·000012005 grm. H_2SO_4 with the current of 1 ma. in thirty seconds.

(3) That hydrogen is liberated at the kathode which preponderates in volume over the gases generated at the anode, in the proportion of 4 to 1 or 6 to 1, in accordance with the density of the anodal current.

(4) That the total volume of hydrogen generated is 0·00362 c.c. with the current of 1 ma. in thirty seconds no matter what be the density of the current.

(5) That the hydrogen gas has the power of conveying with it into the tissue other products of dissociation, *e.g.*, NaOH, and KOH.

(6) That the volume of hydrogen is constant, no matter what be the calibre of the electrode, provided the quantity of electricity is the same.

(7) That oxygen gas which is generated at the anode has the power of conveying with it into the tissues other products of dissociation, *e.g.*, chlorine.

(8) That the total volume of the gases liberated at the anode varies indirectly with the density of the current.

(9) That the total volume of gases generated at an anode 1 cm. long 23 Imp. S.W.G. is 0·0008 c.c., as against 0·0006 c.c. at an electrode $\frac{1}{2}$ mm., $\frac{1}{10}$ mm., with a current of 1 ma. in thirty seconds.

(10) That the density of the current in all instances has a marked bearing upon the mechanical energy of the gases generated during electrolysis, and consequently it is probable that it must have a similar bearing not only in the production of the cerebral lesions, but also in their nature and extent under analogous conditions.

(11) That provided the milliamperage of the current is constant, the mechanical effect produced is in inverse ratio to the size of the electrode; this effect, however, is somewhat modified at the anodal electrode, since the greater the density the smaller are the volumes of the gases liberated.

(12) That with a given current producing electrolysis, the smallest volume of gas is generated at the smallest anodal electrode in any given time.

From a review of the above facts as to the products of electrolytic dissociation, taken together with certain definite conditions ascertained and set forth by Horsley and Clarke [1], with which these results run parallel, it is most probable that, though chemical reaction bears a definite part in the production of these lesions, yet the mechanical element (due to the evolution of the gases) is the main factor in their production; more especially is this the case in the kathodal lesions, wherein comparatively large volumes of gas are generated, and to a lesser extent in the anodal lesions, wherein smaller volumes of gas are liberated, although such are here of a more potent nature.

The accuracy of this statement was tested by Horsley and Clarke, who injected the brain with similar quantities of alkalies as above estimated, without producing results in any way comparable with those obtained by electrolysis; the disintegrating effects of which, through the rush of ions to and from the electrodes, must aid materially in the production of these lesions.

As the outcome of facts elicited in the above experiments, it may be stated that with a current of certain known milliamperage, and an electrode of constant composition and accurate dimensions, provided short circuiting outside the actual brain substance be prevented, a means is afforded whereby in any instance it is possible to produce within that tissue lesions of uniform and definite dimensions: for the resistance in brain tissue may readily be ascertained prior to electrolysis although for all practical purposes it may be regarded as virtually constant. This accuracy, however, I maintain, can only be ensured when the electrode is "effectually insulated" at its distal extremity as above illustrated (fig. 2); otherwise loss of electro-chemical effect must result and irregular polarization and consequent variation in the density of the current are inevitable, whereby minute accuracy and uniformity in results are rendered impossible.

In conclusion I trust that the above observations may be of interest to those who are investigating the minute topographical anatomy of the central nervous system by electro-chemical means.

I have to thank Sir Victor Horsley and Dr. R. H. Clarke for having permitted me to be present during many of their investigations, and also Professor Vaughan Harley for the use of his laboratory in University College, where this research was carried out; and likewise Dr. Francis W. Goodbody for his kind assistance.

References.

[1] Horsley and Clarke. "The Structure and Functions of the Cerebellum investigated by a New Method," *Brain*, 1908, vol. xxxi., p. 45.

[2] Starling. "Elements of Human Physiology," chap. iii., pp. 47–78.

FAMILIAL ATROPHY OF THE HAND MUSCLES.

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WASTING of the muscles of the hands is a symptom common to many forms of nervous disease, but cases are rarely found occurring together in family groups. In the muscular dystrophies, where several members of a family are, as a rule, affected, wasting of the hand muscles is extremely uncommon. Again, in cases where the wasting of the hand muscles is due to disease of the spinal cord or peripheral nerves, it is very unusual to find the cases occurring in families.

An account of the Lewis family would therefore seem worthy of record. Out of sixty-four individuals forming five generations seven persons were affected with atrophy of the muscles of the hand.

Case 1.—Helen Lewis (No. 49 in the Table) came under my care at the Poplar Hospital for an attack of cardiac pain. She was a well-developed girl of 20 years of age. At 4 years old she was taken to a hospital for an attack of purpura; she had had occasional aching pains in the legs, but there was no definite history of acute rheumatism; she had never had scarlet fever, but had suffered from measles when a baby.

On examination, both the left and the right sides of the heart were somewhat enlarged. There was a systolic murmur at the mitral area, and a diastolic aortic murmur could also be heard; the pulse was somewhat collapsing in character; she had slight cough and the bases of the lungs showed evidence of engorgement; there was slight œdema of the feet, but the liver was not enlarged.

Under treatment the cardiac condition improved and she was discharged in three weeks time with good compensation.

While in the hospital the curious condition of both hands was noticed. The muscles of both hands were extremely wasted and formed a striking contrast to the well-developed muscles of her arms and forearms.

The patient herself noticed that her hands began to get thinner when she was about 16. She had no trouble with them until she went into domestic service at 17, when she noticed that she could not do needlework easily, nor could she pick up small objects without difficulty. She was, however, able to

do her ordinary work of scrubbing and cleaning well. Within the last year or two she had noticed that the hands were getting worse and that the fingers were becoming so bent that she could not straighten them.

In the right hand there was distinct wasting of both the thenar and the

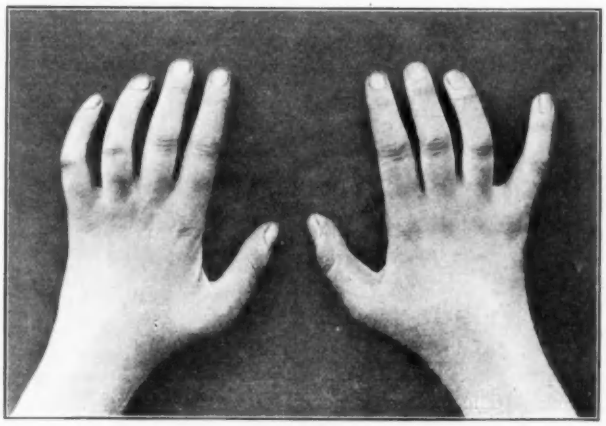


FIG. 1.

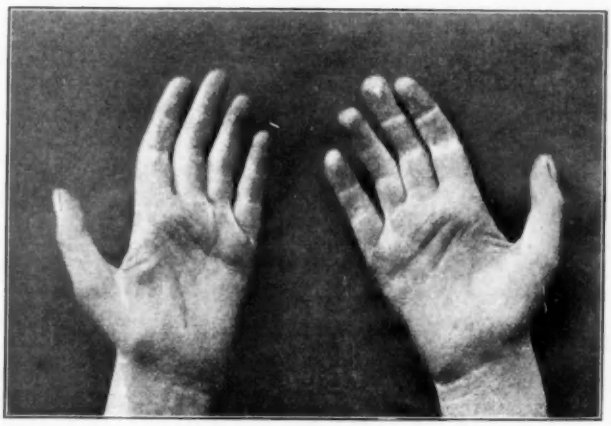


FIG. 2.

hypothenar eminences. The thumb was drawn back by the extensor thumb muscles to a level with the palm, and the flexor tendons showed up prominently upon the palmar surface of the hand. The fingers were hyperextended at the metacarpo-phalangeal joints and were flexed at the interphalangeal joints. As

regards the movements of the fingers, the thumb could be opposed to each of the four fingers in turn, but the movement was carried out slowly and with difficulty. The thumb could also be slightly abducted from the palm, but could not be moved in a direction perpendicular to the plane of the palm. The left hand was in a condition exactly similar to the right. The changes in the hands are well shown in the accompanying photographs (figs. 1 and 2), in which the extreme wasting of the interossei as well of the muscles of the thenar and hypothenar eminences can be well seen.

The muscles of the forearms and arms were strong and well developed, as were also the muscles round the shoulder-joints. There was no winging of the scapulæ. The face was natural in appearance and all the facial muscles acted well; the movements of the eyeball were normal, and the pupils reacted both to light and to accommodation. The tongue and the palate moved normally. The two sides of the chest expanded equally and there was no scoliosis of the spine. The muscles of the abdomen were normal, the leg muscles were strong and well developed, and there was no alteration whatever in the shape of the feet, nor was there any difficulty in walking.

Electrical reactions.—The muscular reactions in the left hand were very distinctly altered. None of the small muscles of the hand gave any response to the faradic current, and the majority of the muscles did not react to strong galvanic currents. The small adductor muscles of the thumb and of the little finger gave a slight response to a strong galvanic current, but the excitability of these muscles was evidently greatly diminished. A slight reaction was also obtained in the second interosseous muscle with the galvanic current. In the right hand this condition was even more marked, and no reaction could be obtained with either the faradic or the galvanic current in any of the muscles with the exception of the adductors of the thumb, which reacted slightly to the galvanic current.

Reflexes.—The knee-jerks were normal and the plantar reflex was of the flexor type; the triceps-jerk could be obtained easily on both sides, but the supinator-jerk was not obtained on either side.

Sensation.—During the whole time that the patient has noticed the condition of her hands there has never been any pain; she noticed, however, that they seemed to be worse in the cold weather. Sensation to touch, to heat, to cold, and to the pain of a pin-prick were perfectly normal in both hands, forearms and arms. The compass-points could be distinguished from one another over the hands and forearms within the normal limits. Thus over both palms the answers given were correct when the points were separated for a distance of 1·5 cm., while over the forearm the two points could be clearly distinguished from one point when separated for a distance of 3·5 cm.

A radiograph (fig. 3) of the upper part of the spinal column showed much enlargement of the transverse processes of the seventh cervical vertebra. On palpation, no tumour corresponding to these enlarged transverse processes could be made out at the root of the neck.

After her discharge from the Poplar Hospital her hands were treated by the

sinusoidal current bath for several weeks, and she thought that some improvement had taken place and that the fingers were not so much bent as they were before.

While in the hospital she stated that there were several cases of this wasting of the hands in her family, and that, as the family was a large one, its members were continually on the look-out to see if any of them would be affected with hand wasting. She said her father suffered in the same way as herself.

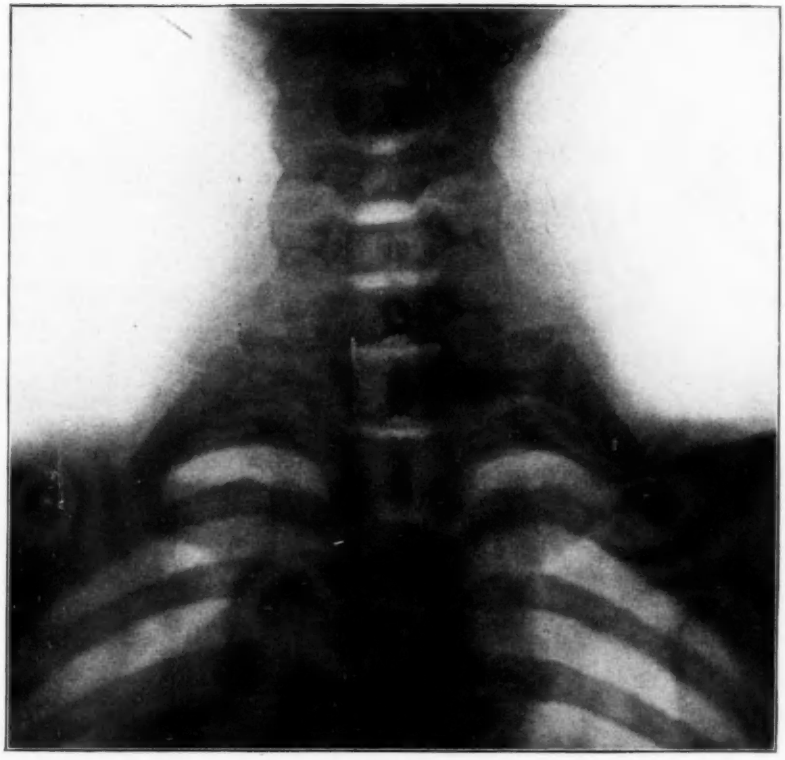


FIG. 3.

Case 2.—Frederick Lewis (No. 17 in the Table), father of Helen Lewis, kindly came up to the hospital and allowed his hands to be examined.

He was a powerfully built dock labourer. When 16 to 17 years old, he began to notice that the right thumb was becoming thin; the left hand became affected soon after. This wasting gradually got worse until most of the muscles

of the hands were affected, but the condition had not changed much of late years. There had never been any pain whatever, but his hands were apt to get numb when the weather was cold. He was able to do his work as a dock labourer well, and, with the help of his mate, could lift 2 cwt. of oil-cake, and he could

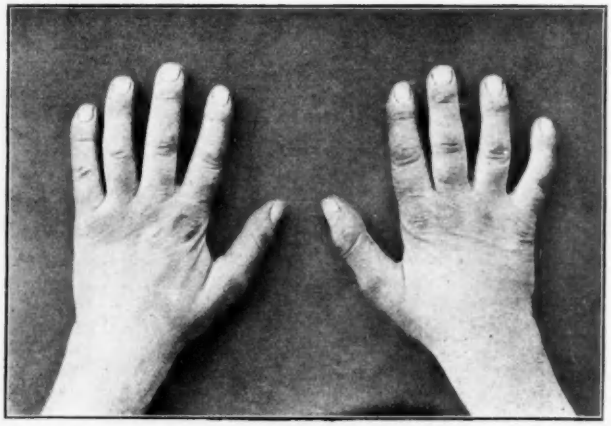


FIG. 4.

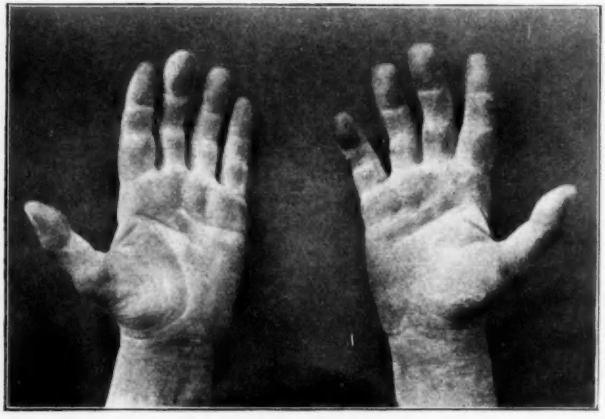


FIG. 5.

lift ½ cwt. by himself. He found, however, that he could not use his hands to pick up anything very small, especially when they were cold.

In his right hand the thenar eminence was much wasted, and the hypothenar to a less extent. The first interosseous muscle was profoundly wasted,

and to a less extent the other interosseous muscles. The thumb was extended and lay in the plane of the palm; the movements of opposition of the fingers and thumb were feeble, but could be performed in each case; abduction and adduction of the thumb were very feeble and the interosseous movements were absent. The wasting of the hands is well shown in figs. 4 and 5.

Electrical reactions.—All the intrinsic muscles of the hand reacted feebly

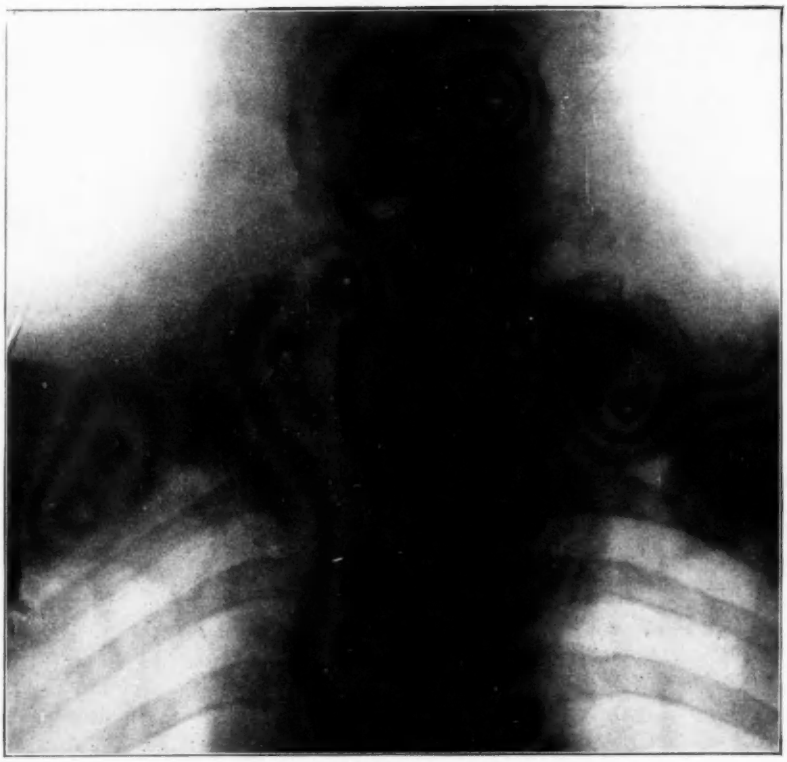


FIG. 6.

to galvanism and not at all to faradism. There is no response until the galvanic current is raised as high as 10 ma. to 20 ma. No response at all could be obtained from the abductor and the opponens muscles of the thumb, while the abductor minimi digitii reacted better than the other small muscles of the hand.

The left hand is in a condition similar to that of the right, but the wasting

is, if anything, more extensive, and on the left side the patient was unable to approximate the tip of the little finger to the end of the thumb.

The muscles of the arms, forearms and legs were perfectly normal, and there was no alteration in the shape of the feet and no difficulty in walking.

Reflexes.—The knee-jerks were normal, the triceps-jerks were easily obtained, but the supinator-jerks could not be obtained on either side.

Sensation.—There had never been any pain at any time, but he found that his hands got numb in the cold weather. No sensory changes could be found on examination.

FIG. 7.

A radiograph (fig. 6) of the upper part of the spinal column showed the presence of seventh cervical ribs, which appeared to be equally developed on the two sides. On palpation of the root of the neck it was impossible to feel any swelling corresponding to these cervical ribs.

Case 3.—Thomas S. (No. 6 in the Table) is now dead, but as he was well known to his nephew, whose case has just been recorded, it seemed best to refer to him in this place. He was known to have had wasting of the muscles of both hands, which came on when he was a young man, and which, though causing him some slight disability, never interfered with his work as a dock

labourer. He lived till the age of 82, and no muscles were ever affected except the muscles of his hands. By a fortunate chance I have been able to obtain a full length photograph of him two years before he died (fig. 7). This portrait shows distinct wasting of both hands.

The other members of this family have been collected in the accompanying table. I have only been able to examine the two cases already described where the wasting of the hand muscles existed. The other four affected cases could either not be traced or lived out of England.

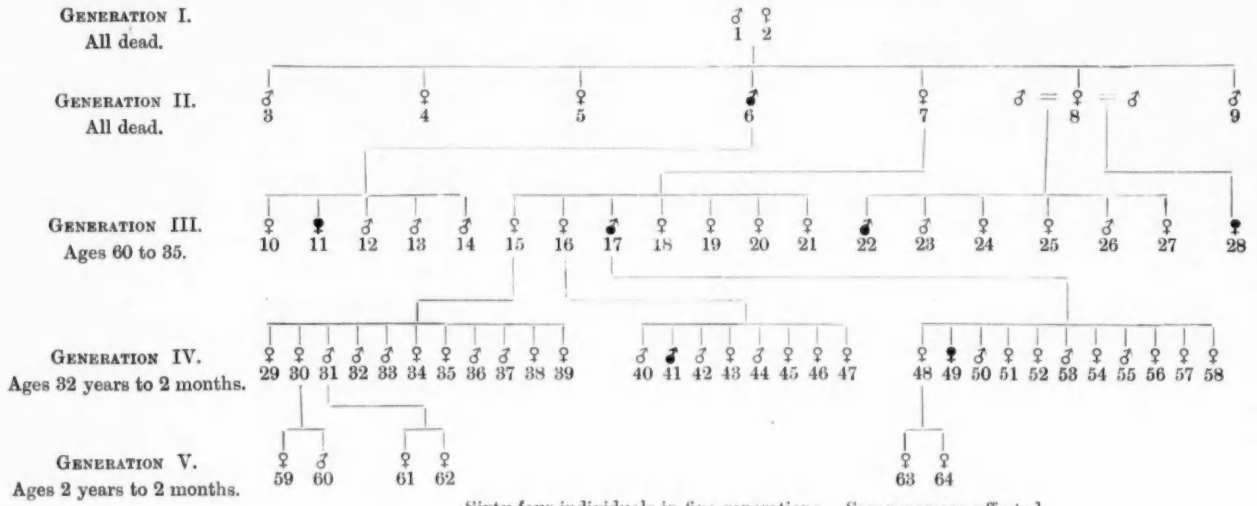
I have, however, been able to examine personally a very large number of the remaining fifty-seven cases, while for those who were either dead or inaccessible I have in every case been able to obtain first-hand evidence as to the condition of the hands.

Full details will be found in the explanation attached to the genealogical tree.

The condition here described as occurring in the Lewis family might be due to some defect in the muscular supply of the hand, or it might be caused by some other primary condition. It seems to me probable that the wasting of the hands is secondary, and that it is caused by the presence of the seventh cervical ribs, which are well known to occur in families. The two radiographs (figs. 8 and 9) are taken from two patients (father and son) under Mr. Sherren, who has kindly allowed me to reproduce them. The boy was brought by the father for some other complaint, and it was noticed that the child had prominent seventh cervical ribs. The ribs could also be felt in the case of the father. In neither case had the ribs produced any symptoms. These cases illustrate the familial characters of cervical ribs, and also the fact that the large and horizontally placed cervical ribs often produce no symptoms.

Against the hypothesis that the wasting of the hands is due to the presence of seventh cervical ribs the following objections may be urged:—

(1) That the condition is symmetrical. It has been pointed out by all observers on the conditions produced by cervical ribs that the symptoms produced are generally unilateral. On the other hand, the abnormality is practically always bilateral, being usually more marked on the one side than the other. Of the sixteen cases described by Hinds Howell [4], in all the deformity was bilateral, but in all cases, with one exception, the symptoms were unilateral. There seems, however, no reason at all why in certain cases the cervical ribs could not be equally developed and produce symptoms on both sides. In fact the cases originally described by Lewis Jones [6] in 1893 were considered under the title of "Symmetrical Atrophy affecting the Hands in Young People." It is interesting



Sixty-four individuals in five generations. Seven persons affected.

1. Long since dead. Three of the grandchildren (Nos. 15, 16 and 17) all declare that the hands were not affected.
2. Same evidence as No. 1 as to non-affection of the hands.
3. Died a single man. Said by Nos. 15, 16 and 17 to be unaffected.
4 and 5. Stated by Nos. 15, 16 and 17 to be unaffected. Lost sight of for many years.
6. Case 3. Description with photograph in the text.
7. Mother of Case 2 (No. 17). Nos. 15, 16 and 17 and the wife of No. 17 knew her to be unaffected.
8. Aunt of Case 2 (No. 17). Well known to be unaffected by No. 17 and his wife.
9. Known by No. 17 to be unaffected.
11. Seen by No. 17 for many years and known by him to have wasting of the hands.
10, 12, 13 and 14. All known to be unaffected by No. 17, but, like No. 11, have been lost sight of for years.
15. Personally examined. Unaffected. No cervical rib felt.
16. Personally examined. Hands unaffected. No cervical rib felt.
17. Case 2. Full details in the text.
18. Dead, of phthisis. Known by Nos. 15, 16 and 17 to be unaffected.
19, 20 and 21. Are all in Canada. Known by Nos. 15, 16 and 17 to be unaffected.
22 and 28. Both seen by No. 17 and his wife, and the wasting of the hands seen.
23, 24, 25, 26 and 27. All known to be unaffected by No. 17 and his wife. Lost sight of for years.
29. Personally examined. Hands unaffected. No cervical rib felt. Aged 32.
30, 31, 32, 33, 34, 35, 36, 37 and 38. Said by their mother (No. 15) to be unaffected. Ages 30 years to 5 years.
39. Personally examined. Hands unaffected. No cervical rib felt.
40. Stated by his mother (No. 16) and his cousin (No. 49) to be unaffected. Aged 23.
41. Now in Canada. Aged 21. His hands began to waste soon after he started work at 14 years old. He can still do heavy work, but cannot pick up small things. Well known to Nos. 16, 17 and his cousin (No. 49).
42, 43, 44, 45, 46 and 47. Ages 20 years to 7 years. All personally examined, and the hands found to be unaffected and no cervical ribs to be felt.
48. Aged 22. Personally examined. Hands and neck normal.
49. Case 1. Affected. Full details in the text.
50, 51, 52, 53, 54, 55, 56, 57 and 58. Ages 18 years down to 2 months. Each one personally examined and found to have hands unaffected and no palpable cervical ribs.
59, 60, 61, 62, 63 and 64. All babies under 2. No. 63 personally seen and is quite healthy.

to note that Lewis Jones states "in two of the cases a brother has had some similar symptoms." Moreover, in my cases the wasting of the hands was not exactly symmetrical, being slightly more marked on the one side than on the other.

(2) Another objection which may be urged against the cervical ribs being the cause of the condition is that the sensory changes in my cases are very little marked. This appears to have been the case in the patients first described by Lewis Jones [6] and already referred to, and which he has since [7 and 8] proved to be due to the presence of seventh cervical ribs. Moreover, in many of the cases reported the sensory changes have been very slight; thus in the sixteen cases described by Howell, in five no loss of sensation could be discovered on examination, while in six of the other cases the loss of sensation was described as slight.

(3) The third objection is that the appearances shown in the radio-

graphs are not conclusive. In the case of Frederick Lewis there is a definite though small and sharply curved seventh cervical rib on each side, and it must be remembered that it is in these cases rather than in the cases where the cervical rib is large and prominent that muscular atrophy is found. Lewis Jones [7] states that in the cases of cervical

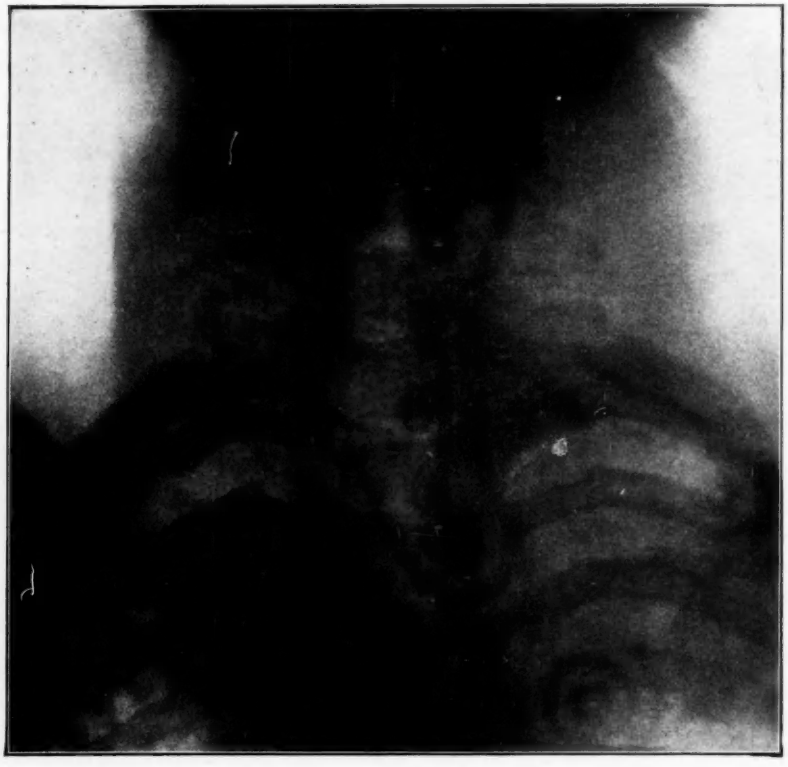


FIG. 8.

rib, where a bony prominence can be felt with ease, the brachial plexus is usually free from any pressure, and in these cases the radiograph shows a large and straight cervical rib. In the case of Helen Lewis the radiograph shows enlarged and prominent transverse processes of the seventh cervical vertebra. But even this, in the opinion of Howell [4],

may be sufficient to cause wasting of the hand. He says: "I think that an enlarged transverse process or rudimentary rib which extends beyond the first dorsal transverse process should be regarded as a potential factor for the production of pressure symptoms." Llewellyn Phillips's [9] dissection of a cervical rib supports this view.

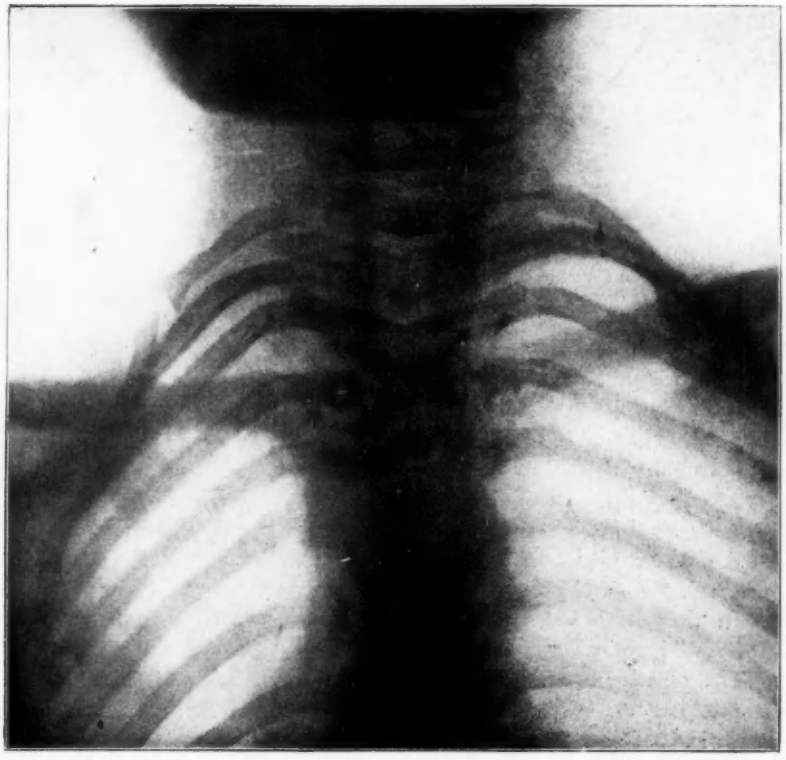


FIG. 9.

On the other hand, in favour of the condition being due to a cervical rib may be urged the early onset of the cases, occurring about the fifteenth or sixteenth year. Many of the cases of cervical rib begin to produce symptoms about this time, which coincides with the active growth and ossification of the cervical rib. Again, if the wasting of the

hand was part of some general muscular condition rather than secondary to a local cause, it is probable that other muscles would have been affected; and in the two cases which I have been able to study completely, no other muscular abnormality could be discovered.

The gradual onset of the wasting and its symmetrical distribution shows that the cases were not the result of anterior poliomyelitis or examples of those cases of uniradicular palsy of the brachial plexus due to vascular lesions described by E. F. Buzzard [1]. The non-progressive nature of the affection, which continued unchanged in one of the cases until the age of 82, and the absence of marked sensory changes negatives the possibility of syringomyelia, so that it may fairly be concluded that the wasting of the hands in these cases is due to the presence of cervical ribs.

Prefixation of Brachial Plexus in Cases of Cervical Ribs.

The method by which the brachial plexus is involved has given rise to some discussion. In the large majority of cases the lowest trunk of the plexus becomes kinked as it passes over the small and strongly curved seventh cervical rib. This condition was found by Coote [2], who was the first to operate on these cases. He states that the nerve was flattened upon the upper surface of the rib, and this was also the case in the patients operated upon by Keen [5] and Thorburn [10]. Weisenstein [12], who made a careful dissection of a case, described the lowest cord of the plexus as being lifted up upon the cervical rib, and Llewellyn Phillips [9] states that in his dissection the lowest trunk of the plexus formed by the eighth cervical and first dorsal nerves crossed the cervical rib and grooved it.

In the discussion on Thorburn's [10] paper before the Royal Medico-Chirurgical Society, it was remarked that it was difficult to understand why a seventh cervical rib caused a first dorsal pain. It is hard to see why the first dorsal nerve should pass over the seventh cervical rib instead of beneath it. Much light has, however, been thrown on this question by the careful study of the brachial plexus in a case of seventh cervical rib published by Hertzlet and Keith [3]. They found that the lowest trunk of the brachial plexus passed over the seventh cervical rib, but that the brachial plexus itself was abnormally constituted. Its constitution was as follows:—

The anterior division of the fourth cervical nerve gave a large branch to that of the fifth, and from this branch was given off the phrenic nerve.

The fifth cervical anterior division joined with the branch from the fourth to form a common trunk.

The sixth cervical anterior division formed a separate trunk.

The anterior division of the seventh cervical sent a branch to the eighth cervical anterior division, and this, with a branch from the first dorsal anterior division, formed the trunk which passed over the seventh cervical rib.

The first dorsal anterior division was distributed to the first intercostal space and gave a branch to the eighth cervical.

In this case the brachial plexus is seen to be prefixed, the fourth cervical nerve taking a large share in its formation, while the first dorsal took a comparatively small share. This preparation of the plexus will explain how it is in many cases that the lowest cord of the plexus passed over the supernumerary rib.

It must be remembered that a seventh cervical rib is merely an anatomical peculiarity, the seventh cervical vertebra approaching in its characteristics to the first dorsal vertebra. This being the case it is probable that in many instances the brachial plexus is also prefixed, each segment taking on the characters of the segment immediately below it.

It is not in every case of cervical ribs, however, that the brachial plexus is prefixed, for a few cases have been recorded in which the first dorsal root passed beneath the seventh cervical rib.

Thus Thomas and Cushing [11] described compression of the lowest cord of the plexus between the first rib and a dense fibrous band passing to it from the seventh cervical rib. A similar condition was found at operation in an unpublished case of Head's. Lewis Jones [7] describes a case operated upon by Rawling, in which the cervical rib had a groove above for the eighth cervical nerve, which was sharply kinked over the anterior part of the rib. The first dorsal nerve passed under the rib and was also slightly displaced and pressed on. Both cords were of the usual size and united together a little beyond the rib. We may therefore conclude that while in many cases there may be a prefixation of the plexus, yet this is by no means always the case.

The Inheritance of Cervical Ribs.

Interesting as the question is, the factors concerned in the inheritance of the abnormality are extremely complicated. Briefly, in the first generation, consisting of two persons, neither was affected. In the second generation, consisting of seven persons, three males and four females, one

male was affected. In the third generation, consisting of nineteen persons—seven males and twelve females—four persons were affected, two being males and two females. The inheritance took place in the case of one female through the only male affected in the second generation. One affected male was descended from an unaffected female of the second generation, while another unaffected female of the second generation gave birth to one affected male by her first husband and one affected female by her second husband. In the fourth generation, consisting of thirty persons, one male and one female were affected, the affected male being descended through an unaffected female of the second generation, while the affected female was descended from one of the affected males in the third generation.

It must, however, be remembered that many members of the fourth generation are under the age of puberty, before which time the wasting of the hands does not develop. Moreover, it seems clear that the wasting of the muscles is probably a secondary characteristic and that the primary inheritance is of the cervical ribs, which may have been present in other members of the family without producing any wasting of the muscles of the hands.

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MANIACAL-DEPRESSIVE INSANITY.

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THE term "maniacal-depressive insanity" was introduced by Kraepelin [16] in 1899 to serve as the expression of his generalization that not only the various forms of circular insanity, but also mania and melancholia (with the exception of the presenile and senile melancholias and of the melancholia which is symptomatic of dementia præcox), are merely different clinical types of one clinical entity. This generalization, though largely accepted in Germany, France and America, has hitherto not met with favour in England and Italy, and in this respect its fate resembles that of its elder compeer, dementia præcox.

Quite recently, with the general approval of Kraepelin, in whose clinic the investigation was conducted, Dreyfus [10] has further extended the connotation of "maniacal-depressive insanity" by including under it presenile melancholia. In this connection it is of interest to note that, in the sixth edition of his text-book, Kraepelin had already expressed the view that the considerably rarer presenile manias were late forms of maniacal-depressive insanity, and had thus, perhaps unwittingly, paved the way for the further inclusion of the presenile melancholias.

The following definition of maniacal-depressive insanity is given by Diefendorf [8] in his abridged translation of the seventh edition of Kraepelin's "Lehrbuch": "Manic-depressive insanity is characterized by the recurrence of groups of mental symptoms throughout the life of the individual, not leading to mental deterioration. These groups of symptoms are sufficiently defined to be termed the manic, the depressive, and the mixed phases of the disease. The chief symptoms usually appearing in the manic phase are psychomotor excitement with pressure of activity, flight of ideas, distractibility, and happy though unstable emotional attitude. In the depressive phase we expect to find psychomotor retardation, absence of spontaneous activity, dearth of ideas, and depressed emotional attitude; whilst the symptoms of the mixed phase consist of various combinations of the symptoms characteristic of both the manic and depressive phases."

As the term "maniacal-depressive insanity" implies, the fundamental characteristic of all the various types included in the group is unanimously considered, by such alienists as accept the generalization, to be an emotional or affective condition, to which such disorders of the intellect as may exist are merely secondary. The view that this emotional or affective state is conditioned by disorders of general sensation or by loss of the general sensory equilibrium, which normally occurs during healthy action of the various bodily organs (*dyscénesthésie*, Peixoto), may be considered to be merely a theoretical explanation of the emotional disturbance which is regarded as the essential feature of maniacal-depressive insanity.

The subject of maniacal-depressive insanity has recently been fully and lucidly treated by Deny and Camus [7] in their monograph, "La psychose manique-dépressive." Though primarily intended to demonstrate that the credit of initiating the generalization belongs to France rather than to Germany, their description of the manner in which the conception of maniacal-depressive insanity has been evolved is so excellent that the writer proposes to insert here its main features.

Deny and Camus divide the historical evolution of the idea of maniacal-depressive insanity into three periods :—

First period : up to the middle of the last century.

Second period : the French period, or the second half of the last century. This corresponds to the discovery of the *folie circulaire* and the *folie à double forme*.

Third period : the German period, commencing in 1899. This period is characterized by the synthesis of all the states usually described as mania and melancholic depression of many types—simple, intermittent, remittent, periodic, *folies alternés*, *à double forme*, circular, &c.

First period.—It had long been known that cases of mania and melancholia were subject to relapse at longer or shorter intervals, and also that these attacks might alternate and that mania might be transformed into melancholia and *vice versa*. Pinel and Esquirol merely confirmed these observations. Several authors, however—Dubuisson, Fodéré, Anceaume—were struck with the great gravity, in comparison with attacks of simple mania, of attacks of periodic and intermittent mania, and of mania complicated with melancholia. Later, in 1833, Guislain, and in 1845 Griesinger, remarked on the frequency of alternating attacks of mania and melancholia, but did not attach any especial importance to this observation. Up to the middle of the last century all authors without exception regarded mania and melancholia as two distinct entities.

Second or French period.—Pierre Falret [11], in 1851, described not an alternation of mania and melancholia separated by a more or less lengthy lucid interval, but a *forme circulaire*, consisting of a period of exaltation which alternated with a usually much longer one of depression. He, however, only described it as a distinct malady after Baillarger, in 1854, had indicated a type of insanity which is characterized by two regular periods, one of depression and one of excitement. Baillarger described this type as *folie à double forme*. He pointed out that this affection may appear as an isolated state, or may recur in an intermittent manner, or may be repeated uninterruptedly. Falret disputed the priority of Baillarger, and repeated that the *folie circulaire* consisted of three states: one of mania, one of depression, and a lucid interval. The only difference between the *folie à double forme* of Baillarger and the *folie circulaire* of Falret consists in the fact that the latter author considered the lucid interval as part of the attack, whilst the former excluded the lucid interval from it. Falret, indeed, seems to have introduced the lucid interval into his description in order to justify his term of *folie circulaire*. In brief, the malady was simultaneously described by both authors under different names. Falret, however, noted the importance of heredity, the greater frequency in the female sex, and the gravity of the prognosis. Morel [20], in 1868, saw no reason for the creation of a special type. Concerning his views Deny and Camus remark: "Et suivant encore une fois les mêmes errements qui l'avaient déjà empêché de considérer le *démence précoce* comme une entité distincte, il fit rentrer ces phénomènes d'alternance, de périodicité et d'intermittence dans la première classe de ses *Aliénations héréditaires*." Marcé, in 1876, proposed that the term *folie à double forme* should be applied to cases of mania-melancholia in which the phases are sharply separated by a distinct lucid interval, and that the term *folie circulaire* should be reserved for cases in which the phases of mania and melancholia succeeded one another without any interruption. These designations became generally accepted. They were, however, only finally perpetuated after the publication of the articles of Foville fils in 1872 and of Ritti in 1878. At this period French alienists generally, *e.g.*, Magnan and Cullerre in 1890, Régis in 1892, Gilbert Ballet in 1894, grouped these forms, together with recurrent mania and recurrent melancholia, into a special group, that of *periodic or intermittent psychoses.*

In Germany the majority of authors accepted the views of Falret and Baillarger. One of the earliest of these, Griesinger [13], insisted

on the frequency with which melancholia passes into mania, and then mania back again into melancholia. He remarks: "The disease, as a whole, thus consists of a morbid circle in which these two mental types often alternate in a regular fashion (this is the circular form which the French have discussed for some years). Other observers, and I among the number, have seen cases where regularly at a season, *e.g.*, winter, there followed a profound melancholia; then, in spring, this gave place to mania, which in its turn, in autumn, degenerated little by little into melancholia." [This description reminds one of the course at times taken by cases suffering from phthisis.]

L. Meyer, in 1874, sought to establish that the *folie à double forme* is due to trophic troubles. L. Kirn [15], in 1878, ranged the *folie circulaire* amongst the periodic psychoses. His example is followed by Krafft-Ebing, Schüle, and others.

Kraepelin, in 1893, in the fourth edition of his treatise, classed amongst incurable constitutional maladies of chronic type, by the side of systematized cases, periodic cases, of which he distinguished four groups: delirious (*i.e.*, delusional), maniacal, circular and depressive. This classification resembles that in use in France during the same period.

Third or German period.—In 1899 Kraepelin [16], in the sixth edition of his treatise, introduced a new classification. He observed that the more or less regular repetition and alternation of attacks of mania and melancholia are not characteristics of sufficient importance to constitute a basis for separate types. He showed in effect that the psychoses called intermittent, periodic, circular, *à double forme*, alternating, &c., all show the same mode of evolution, and that it is more logical to consider all these states as the manifestations and the equivalents of one fundamental malady, *maniacal-depressive insanity*. He noted that he had never seen a single case of mania which had not been followed by relapse, and also that it is impossible to distinguish between cases of simple mania and cases of relapsing and intermittent mania, and therefore he considered that classical mania ought to be removed from the list of mental maladies. This "mania" had previously been referred to by Pinel and Esquirol and had been ostracized by the two Falrets and by Morel. Kraepelin considered it convenient to describe apart those cases of melancholia which occur at the period of involution and in senility. He included melancholias of youth and of maturity, which always relapse or alternate with attacks of mania, under maniacal-depressive insanity, unless they should be symptomatic of dementia præcox.

Magnan [17], in 1890, had already grouped several of these forms under the term "intermittent insanities," but he excluded from this class degenerative maniacal exaltation and melancholic depression, and at the same time simple mania and simple melancholia. Deny and Camus therefore remark that Kraepelin, some years later, completed the essay at reconstruction initiated by Magnan.

These authors then produce further evidence pointing to the undesirability of regarding mania as a distinct clinical entity. Gilbert Ballet and Régis so regard it, but whilst the former indicates symptomatological differences between the simple and the intermittent forms of both mania and melancholia, the latter [23] considers such to be non-existent.

Erp Taalman, in 1897, stated that, of 107 cases of mania, in only four cases was the diagnosis confirmed by the course of the disease. Otto Hinrichsen similarly remarked that the proportion of cures in cases of mania dating for more than twenty years is only 4·7 per cent. Weygandt and also Thomsen follow Kraepelin, and the latter goes even further and throws doubt on the existence of periodic mania, at least in a state of purity, because it is always followed by a more or less marked period of depression. He thus includes periodic mania under *folie à double forme*, an opinion already formulated by Griesinger. In Belgium, Claus, in 1903, stated that pure cases of mania and melancholia are rare. Lambranzi and Perazzolo, in 1906, stated the same opinion, which had already been defended, in 1899, by Finzi and by Vedrani.

From these facts Deny and Camus conclude that the line traced by the older authors between simple mania and melancholia on the one hand, and periodic mania and melancholia on the other, is purely conventional, and that Kraepelin is right in placing the former in the old group of periodic psychoses, now called *maniacal-depressive insanity*.

Bianchi, Tanzi, Francoda Rocha, and others, however, include under maniacal-depressive insanity simply the double and circular forms, both of which show phases of excitement and depression; and at first sight it is not clear in what way intermittent and periodic forms of mania and melancholia can be included under the designation. Deny and Camus produce the following arguments in favour of the inclusion of the latter: Pure and unmixed periodic mania and melancholia are quite exceptional. The more often one observes two, three, and four attacks of mania, the more often one notices that an attack of depression replaces the expected one of mania (intermittent mania of irregular type [Arnaud]). Conversely, one sees a series of attacks of melancholia interrupted from

time to time by an attack of mania (intermittent melancholia of irregular type of the French authors). They thus consider that periodic manias and melancholias are really *abortive* examples of the double form, in which one of the phases is unrecognized. "In other words, attacks of intermittent mania are in reality attacks of mania-melancholia, but with a maniacal preponderance" (p. 18), and *vice versa*. They thus confirm the observations of Rogues de Fursac [12], who, in 1903, stated: "Each attack of mania or of melancholia thus contains in germ the elements both of excitement and of depression. An attack of *folie circulaire* becomes thus the prototype from which are derived all the others." Afranio Peixoto [21] holds the same view (1905). Gilbert Ballet [2], in 1902, suggested that circularity is a functional law of the nervous system, that we are all to some extent *circulaires*, and that the pathological state, which constitutes periodic insanity in its most characteristic form, is simply an exaggeration or amplification of the manner of bearing which is habitual to all of us. Deny and Camus accept this view and argue in favour of it. They point out that mixed states exist, described by Kraepelin but previously indicated by Guislain, in which excitement and depression coexist, are mixed, and are entangled, instead of *succeeding* as in the double form. They remark that, in spite of their opposed characters, the phenomena of excitement and of depression have the same origin and arise from the same psycho-pathological mechanism. They finally state: "But putting to one side these last two arguments . . . one can already conclude from the preceding considerations that the term maniacal-depressive insanity is doubly legitimate, since the association of excitement and depression is true, not only for the total series of attacks which constitute this psychosis, but still more so for each of these attacks when it is separately considered" (p. 20).

They then define maniacal-depressive insanity as "a constitutional psychosis, which is essentially hereditary, and is characterized by the repetition, the alternation, the juxtaposition, or the coexistence of states of excitement and of depression" (p. 21). They remark that this type of insanity can be differentiated only by its symptomatology and its evolution, and that it is impossible to assign to it a special etiology or to attach to it anatomical lesions. They regard it as "a simple *clinical entity*, which takes a natural place by the side of chronic systematized insanity, in the group of *constitutional psychoses*, similar to the natural position of dementia præcox by the side of general paralysis, in the group of *accidental psychoses*."

It should be remarked that these authors follow Kraepelin in specifically excluding the melancholia of involution from maniacal-depressive insanity.

The conception of maniacal-depressive insanity, as has already been stated, has finally been still further elaborated by G. L. Dreyfus [10], who, in his monograph entitled "Die Melancholie ein Zustandsbild des manisch-depressiven Irreseins" (1907), has proposed the inclusion of the melancholia of involution.

After a useful historical description of the evolution of the modern conception of melancholia, this author refers in detail to the views of Kraepelin [16] on melancholia. He notes how, in the different editions of Kraepelin's treatise, the application of the term "melancholia" is gradually more and more restricted, until in the fifth edition (1896) this term is applied by Kraepelin to a typical disease of the involution period of life which represents a transition to the senile mental disorders. The chief differences noted by Kraepelin between melancholia and maniacal-depressive insanity are the absence of psychomotor inhibition in the former and the tendency of this form to end in dementia. Kraepelin remarks that the depressive stage of maniacal-depressive insanity may exactly resemble presenile melancholia, and that the fact of melancholia occurring only once in a lifetime cannot be regarded as a ground for denying its relationship to circular insanity, since in the involution psychoses depressive states frequently recur. He, however, regards it as significant that a single attack of melancholia at the presenile period is common, but a single attack of mania is far rarer. Whilst, therefore, Kraepelin includes under the involution psychoses melancholia, presenile delusional insanity, and senile dementia, he considers presenile mania to be a late form of maniacal-depressive insanity.

Dreyfus, as the result of an exhaustive study of the material available at the Heidelberg clinic, has decided that these differential criteria lose their significance when cases of presenile melancholia are followed throughout their entire course. He states that it is common to meet in cases of presenile melancholia (1) with the qualities of mood which characterize the depressive stage of circular insanity, (2) with various kinds of partial inhibition, and (3) with variations and oscillations from day to day, &c., which are reminiscent of circular insanity. For the purposes of his investigation he has minutely studied the previous and also the subsequent histories of melancholic cases, with especial regard to the slight symptoms which occur from time to time during the periods when the patient is for practical purposes well and sane. He in

this connection remarks on the protracted course of the disease, of which the greater part is usually passed outside an asylum. With regard to the question of dementia in presenile melancholia, Dreyfus considers that it is less common than Kraepelin stated, and that when present it is due to cerebral arteriosclerosis.

As an indication of the way in which the proposition of Dreyfus has for a long time been foreshadowed at Heidelberg may be reproduced here the following figures, which indicate how, during the years 1892—1906 the number of cases diagnosed as "melancholia" has steadily decreased, although the number of annual admissions has more than doubled during this period:—

Year	Diagnosis of Melancholia, or "Depressive Wahnsinn"	Erroneous in	Total Admissions
1892	11	7	258
1893	10	1	240
1894	11	2	251
1895	12	2	254
1896	7	1	215
1897	7	1	280
1898	4	2	325
1899	4	2	367
1900	2	1	383
1901	3	0	366
1902	1	0	400
1903	5	1	520
1904	4	4	549
1905	0	1	531
1906	0	0	579
Total	81	25	5,518

As Kraepelin, in his preface to the volume, expresses his general agreement with the contention of Dreyfus, it may therefore be presumed that presenile melancholia will in the future be included under the maniacal-depressive group.

The above general review of maniacal-depressive insanity will now be concluded with a short reference to certain etiological and clinical characteristics.

Kraepelin considers that maniacal-depressive insanity is one of the most prominent forms of mental disease, and that it includes from 12 to 20 per cent. of admissions. Deny and Camus find the similar percentage of 17·5 at the Salpêtrière, and Peixoto the much smaller

percentage of 6·6 at Rio de Janeiro, where part of the asylum population is coloured.

Heredity figures as the prominent cause, and the various authors give percentages varying from 70—80 to 90. Relatives have often suffered from the same form of disease, and a defective constitutional basis, amounting in some cases to imbecility, is stated to be common.

About two-thirds of the cases are of the female sex, a fact first noted by Falret.

Kraepelin states that the first attack occurs before the age of 25 in two-thirds of the cases, and after the age of 40 in less than 10 per cent.; and that the onset may be as early as 10 or as late as 70.

Some difference of opinion (Deny and Camus) exists with regard to the mental condition of the patients during the "lucid" intervals. The majority of the French alienists follow Falret and Baillarger in considering that during the intervals the patient is sane, and has returned to his "normal" state. Krafft-Ebing, Schüle, and Kraepelin are of the opinion that during the intervals the patients as a rule exhibit such traces of the malady as distrust, increased emotivity, decreased energy and capacity for work, &c., and Dreyfus [10] largely bases his inclusion of presenile melancholia in the maniacal-depressive group on the existence of such symptoms during the course of the former disease. Magnan explains this divergence of opinion on the ground that the maniacal-depressive group as now constituted includes many degenerates suffering from mental debility and instability. Arnaud believes that lucid intervals of many months, and especially of years, represent a return to the normal, whilst such intervals, when lasting for days or weeks only, are of the nature of amelioration rather than of cessation of symptoms. Deny and Camus express a guarded opinion, though they are inclined to think on the whole that the German view is the more correct.

The above description of maniacal-depressive insanity, which is largely based on the monographs of Deny and Camus [7] and of Dreyfus [10], sufficiently indicates the nature of this generalization. The writer therefore does not propose to refer here to the numerous recent publications which deal with special details of symptomatology, &c. A paper by Albrecht [1] on the incidence of arteriosclerosis may, however, be mentioned, owing to its bearing on the views of Dreyfus, with regard to the development of dementia in presenile melancholia. Albrecht finds that 30 per cent. of cases of maniacal-depressive insanity exhibit this morbid appearance, whereas it occurs in but 40 per cent. of alcoholics and but 10 per cent. of cases of dementia præcox. He is

of the opinion that in the relatively rare elderly subjects, arteriosclerosis may precipitate the onset of mental symptoms, whereas in the larger number of cases the frequent variations in arterial tension which occur may be a cause of the arteriosclerosis.

Whilst the subject of maniacal-depressive insanity now possesses quite a respectable literature, the supporters of Kraepelin are chiefly drawn from Germany, France and America. Mendel [18], however, though he employs the term maniacal-depressive insanity, includes recurrent and periodic mania under mania, and periodic and remittent melancholia under melancholia, and groups other recurrent cases under the term "circular psychosis," after Hoche [14] and Pilcz [22].

Bianchi [3] regards pure mania as relatively rare (2 to 3 per cent. of admissions), and describes pure and periodic mania together. He considers melancholia to be more common than mania, and groups periodic melancholia with it. His "melancholia" somewhat resembles the melancholia of Kraepelin. Under the term "maniacal-depressive insanity" he includes maniacal-depressive, mixed, and circular forms.

The conception of maniacal-depressive insanity has hitherto not obtained any foothold in England. That this is due neither to insular conservatism nor to lack of interest in the study of mental diseases the writer, however, hopes to make clear during the remaining portion of this paper.

Clouston [5], in his chapter on "states of alternation, periodicity, remission and relapse in mental diseases," draws certain general conclusions concerning these states which well illustrate the broad conception of insanity held by the more prominent of English alienists. It is possible, in fact, that in the future foreign psychiatrists, by gradually increasing the breadth of their generalizations, will end where English psychiatrists have begun, by recognizing the unity of mental diseases. The remarks of Clouston are of sufficient importance to deserve quotation in full:—

"Looking at all those facts and considerations, therefore, I come to these conclusions: that periodicity, or a tendency to alternations of elevation and depression, is a very common characteristic of mental diseases; that it is much more marked where they are very hereditary than in any other cases; that it is more common in youth, puberty, and adolescence than at other periods; that it is in its essential nature the exaggerated or perverted physiological diurnal,

menstrual, sexual, or seasonal periodicities of the healthy brain; that the cases that have been called *folie circulaire*, katatonia, &c., are merely typical or exaggerated or more continuous examples of pathological periodicity. Another remarkable fact about the typical form of alternating insanity is, that by far the greater number of patients who suffered from it were persons of education, and far more than a due proportion of them were members of old families. I never met with a fine case in a person whose own brain and whose ancestors' brains had been uneducated. It seems to me that the tendency to alternation of mental condition, to energize at one time with morbid hurry and then with morbid slackness, is one of the forms of brain instability which specially results from too much 'pureness of blood' or from the heredity of many generations of gentlefolks, all of whose brains had been more or less educated. Possibly it is one of the modes by which Nature brings that kind of stock that has become degenerate by over-cultivation of the brain for many generations to an end. Real work can sometimes be done during the sane periods. D.D. has done some literary work in the intervals of his attacks, for the twenty-six years he has been ill."

Savage [24], in his Lumleian Lectures, expresses his views with regard to insanity as follows: "I still believe that what Hughlings Jackson said many years ago is true, viz., that we physicians connected with insanity resemble gardeners rather than botanists; that the fact must be recognized that we classify for convenience rather than on a scientific basis, because in point of fact no such basis, or finality of mode, has as yet been discovered. . . . The conclusion, then, follows that, there being no definite entity of insanity, there can be no one comprehensive definition of it, and that the person is sane or insane in relation to his own standard and not to any existent arbitrary one, except the conventional arbitrament of civilization. And as this varies, the truth of my former remark becomes evident, that what is reasonable conduct in one man under certain conditions may be plain madness in another differently situated. I often think of a splendid young animal whom I saw—the son of a distinguished father, who rightly judged his son to be an anachronism, out of place in fact; and considered that he would have made a fine knight in the Middle Ages, and perhaps even now might make a good cowboy in America." With regard to mental degenerates he remarks: "They generally come of insane stock and tend to transmit their neurosis, in one form or another, to their descendants. They are more liable than others to break down under slight stresses;

ceteris paribus, their neuroses appear at the critical periods of life, such as adolescence, puberty, and, in the case of women, with child-bearing and the menopause. These patients show a special liability to relapse after recovery, and, as a sequence, to establish a habit of recurring mental disorder—mania, melancholia, as the case may be. This morbid habit may become more and more organized and pass into regular circular insanity."

Savage and Goodall [25] describe recurrent melancholia and recurrent mania under chronic insanity, and make a brief reference to maniacal-depressive insanity in the following terms: "In the present state of knowledge there would not appear to be sufficient justification for abandoning the older method of classification and for regarding mania and melancholia as mere phases of one disease."

Mercier [19] thus states his views on the nature of insanity: "We have to recognize that in insanity there are not only those disorders of the object-consciousness—those delusions, doubts, obsessions, and so forth, which are described in the text-books—not only is there often an alteration in the feeling of well being—a melancholy or an elation—which is sometimes recognized to belong to the subject; but there is, in addition, a more profound and intimate change in the subject itself; a change in the mode of activity; a change in the capacities or possibilities of acting; a change in the direction of action; a change, in short, of the very self; which renders the insane man a different person from his sane self." As would be expected, therefore, Mercier recognizes "recurrent insanity" in his text-book, and makes no reference to maniacal-depressive insanity. He indicates the recurrent type of insanity described by foreign writers under "dementia præcox" in the following terms: "There is a variety of circular insanity in which the period of depression is replaced by a period of stupor, usually of the resistive type, and to this variety the name of 'katatonia' has been given."

Drapes [9], in a recent paper on the "Unity of Insanity," remarks: "The term 'manic-depressive' or 'maniacal-depressive,' which has come into vogue of late years, as denoting a special form of insanity, although not of any value if it is meant to imply some variety not yet described, is yet of some value as indicating an acknowledgment of the fact—hitherto ignored in all schemes of classification—that there is such a thing as mixed insanity. In reality such cases are merely those which constitute a large proportion of ordinary chronic insanity. We might extend this nomenclature still further and describe cases as

manic-depressive-stuporous, and even manic-depressive-stuporous-delusional-demented, if we want to give a more complete clinical description of quite a number of cases. The fact is, these fanciful so-called 'varieties' are all nothing but clinical descriptions of the one disordered mental condition, insanity, while it is passing through certain more or less transitory, or a succession of transitory, stages, and to attach a separate style and title to such temporary conditions, or to any combination of them, is nothing but to create confusion in our conception of insanity."

Maurice Craig [6], in his recent text-book, retains the terms recurrent mania, recurrent melancholia and *folie circulaire*. He remarks: "The student should clearly understand that such terms as mania and melancholia merely designate *groups of symptoms*"; and "The writer fully agrees with much that Kraepelin has stated, but the distinction that the latter makes in dividing his cases into groups is almost too fine for teaching purposes."

Whilst welcoming the generalization of maniacal-depressive insanity, in so far as it indicates a tendency to recognize that a large group of cases of insanity are of developmental rather than of obscure or accidental origin, the writer cannot regard it as indicative of a special type of mental disease. He would quote Gilbert Ballet against himself, and remark that we are all more or less "circulaires," whether we are sane or insane. We are circulars when sane; we are circulars when insane; we are circulars whether sane or insane, in cases where we are at one time sane and become at another time insane. Circularity is not insanity, but depends on periodic modifications in the functional activity of the cerebrum, whether this be working normally (sanity) or abnormally (insanity). The emotional tone of the sane varies in degree and kind according to circumstances. It is a quality rather than a special division of psychic function. The emotional tone of the insane also varies in degree and kind, but, except in the early stages of acute attacks, the change is always in the direction of decrease or loss, however much an increase of emotional tone may be suggested by the psychomotor symptomatology which is exhibited. Lest this statement should be misunderstood, it is perhaps desirable to remark that certain "chronic lunatics" are only permanently insane in the legal sense.

The space at his disposal not permitting of any discussion of the theory of the emotions, the writer thus merely indicates his opinion that the emotive or affective states, which are unanimously regarded as the

essential characteristics of maniacal-depressive insanity, form an entirely inadequate basis on which to erect an important group of mental diseases. As he has remarked in his series of articles on "Amentia and Dementia" [4], he considers insanity to be one disease, of which the various clinical "forms" depend, on the one hand, on various grades and types of cerebral subevolution, from idiocy up to the "normal" (amentia), and, on the other, on various grades and types of cerebral dissolution and involution (dementia). The more marked the degree of amentia, the less, in general, is the tendency to dementia, though the cortical neurones of even the idiot may undergo involution, and those of even the "normal" individual may suffer either dissolution or involution.

On this view the miscellaneous cases included under maniacal-depressive insanity are merely certain examples of such grades of amentia as preclude the application of sufficient "stress" to determine cerebral dissolution, for the cerebra of these cases, regarded as machines, so readily become deranged that asylum *régime* and the consequent absence of "stress" ensue before permanent injury to the cortical neurones results.

In such cases of mental disease, and also in many others, the functional disturbance of the cerebrum is of a relatively low order, and, considered from the general aspect, involves: (1) decreased action of the higher and latest evolved cerebral functions of control and coördination, which results in abnormalities of *immediate* cerebral activity, and in consequent emotional and psychomotor disturbances of various kinds, and (2) (in the more degenerate types) generally aberrant and subnormal cerebral activity. In other words, the writer considers that the cerebrum, as a machine, is working in a defective manner, and that all the "functions of mind," and not merely the emotions, are involved. This abnormal form of cerebral activity is, however, of an *immediate* type, and does not to any extent involve the revivification of complex and time-related portions of the subconscious content of mind, as often occurs in, *e.g.*, hysteria, epilepsy and paranoia.

Such examples (which include the whole maniacal-depressive group) are roughly classified by the writer into two groups: (1) Cases which at times may be regarded as "sane" or "normal," and (2) cases of greater cerebral degeneracy, which are never really so.

The former group includes all types of recurrent case, whether these be still capable of "recovery" or have become permanently insane asylum inmates. About two-thirds are of the female sex.

The latter includes the following general types: (*a*) "Moral"

cases; (*b*) simple "emotional" chronic mania; (*c*) chronic mania with incoherence and delusions; and (*d*) "cranks" and asylum curiosities. About three-quarters are of the female sex, the proportions varying from 8 to 1 in (*b*) to about 3 to 2 in (*a*) and (*d*).

The cases in these clinical subclasses show, respectively, the following prominent characteristics:—

(*a*) *Alteration of moral sense*, with a tendency to do desperate things, *e.g.*, to commit suicide or even homicide, to perform acts of self-injury or self-mutilation, to strike, smash or destroy, to irritate intensely those around them, to be sexually inclined in a normal or abnormal direction, &c.

(*b*) *Alteration of emotional and intellectual control*, *e.g.*, exuberance, instability, vanity, garrulity, childishness, and often violence, treachery, and destructiveness. The younger and adult patients usually display a more or less marked loss of control over the emotions and instincts. The older patients differ from these in the fact that the loss of control affects chiefly the intellectual functions. Their association of ideas is normal, except for its extreme rapidity and complexity. They talk continuously whenever a listener can be found, and they are frequently inconsequent and show a marked tendency to parenthesis during their descriptions.

(*c*) *Rapid and uncontrolled association of ideas*, with delusions of grandeur, which may or may not coexist with or follow delusions of persecution. These cases form a half-way house between subclasses (*b*) and (*d*), and shade gradually into each of these. They differ from the former in being, on the whole, less troublesome, and in showing an apparently complete incoherence in their association of ideas, and from the latter in the fact that their ideation is simply rapid and uncontrolled rather than grotesque or symbolical, and resulting in erratic and eccentric conduct.

(*d*) *Stereotyped, symbolical, or grotesque association of ideas*, which leads to weird actions and eccentric general behaviour. These cases are extremely conceited, vain, and grandiose. They are of many types, and may be simply asylum "show-birds," or may possess considerable artistic or intellectual talent. As a class, these cases only differ from certain "sane" individuals in the absurd and grotesque extremes to which they carry their ideas and their resulting behaviour and actions; and their stereotypism, which often suggests dementia, also only differs in degree from the stereotypism and prejudice which are often seen in the "cranks" of the outside world.

It appears to the writer that the cases of mental disease grouped under the term "maniacal-depressive insanity" all fall naturally into the above larger division. Experience has convinced him that the greater the degree of cerebral degeneracy the less evident is depression as a symptom or a phase of symptomatology, and *vice versa*. This emotional disturbance is therefore pre-eminently a characteristic of the former (recurrent) group, and is of subordinate importance, and often entirely absent, in the case of the latter (permanent) group. He has, in fact, often noted that cases which at one time were "circular" have later on partially or entirely lost the depressive phase, and he is disposed to regard this phase, when post-maniacal, as to some extent indicative of a still possible return to the "normal." He therefore welcomes the recent inclusion of presenile melancholia in maniacal-depressive insanity, for, in conformity with the above opinion, he has long been convinced that the capacity to develop a prolonged attack of melancholia is indicative of what is practically the mildest grade of cerebral insufficiency and of the onset in the majority of such cases of (senile or presenile) cerebral involution.

He would remark that any of the cases included in the above division of amentia may suffer from the accessory symptomatology of mental confusion, with disorientation and sensory symptoms, as the result of alcoholic or other form of intoxication, or of degeneration of the cerebral arteries; and from the latter aspect he partially agrees with Kraepelin, Dreyfus and others who consider arteriosclerosis to be the cause of the dementia which develops in many cases of chronic melancholia.

With regard to the recurrent types of insanity generally (*i.e.*, those recurrent types which do not develop dementia) he would again draw attention to the remark of Clouston: "Another remarkable fact about the typical form of alternating insanity is, that by far the greater number of patients who suffered from it were persons of education, and far more than a due proportion of them were members of old families. I never met with a fine case in a person whose brain and whose ancestors' brains had been uneducated." The possessor of one of the finest intellects the writer has met with was insane and in a condition of permanent and uncontrolled exaltation of cerebral function. He had earlier in life been a university professor, and a near relative had attained to eminence. This individual might be regarded as the owner of a cerebrum which was too elaborately developed to be properly controlled in its existing stage of evolution. Such brains, working under proper control, may be common

in the far distant future. From the "normal" aspect it is well known that fine pieces of work have been rapidly done whilst their authors were so intensely absorbed as to be practically in a condition of general cerebral exaltation or mild "sane" mania, for which, however, the subjects had afterwards to suffer in recuperative depression of the cerebral functions. The higher types of recurrent insanity thus grade upwards towards the cerebral hyper-activity of genius, whilst the lower types grade downwards into the analogous cases who are never really sane, although they may legally pass as such.

The writer is unable to regard mania and melancholic depression as simple and opposite motive states, though mere excitement and depression may be such. Mania, whilst at times outwardly indicative of general exaltation of cerebral function, is more often a sign of decreased action of the higher controlling and latest evolved portion of the cerebrum. Melancholia, on the other hand, whilst it is indicative at times of recuperative general depression of cerebral function, or of impending loss of higher cerebral control, is more often a sign of the onset of permanent general depression of the cerebral functions, and is thus the objective evidence of impending or developing (presenile) involution of the cortical neurones. Mania is thus of less grave prognostic import than is melancholic depression, and this statement is in agreement with general clinical experience and practice; for, alcoholic cases being excluded, patients with mania are more readily discharged "recovered" than those who suffer from melancholia (quite apart from the question of suicide).

As will have been gathered from the above remarks, the writer therefore considers that the maniacal-depressive generalization is untenable as a description of a special kind of mental disease, although he welcomes it as indicative of a tendency to decrease the number of "mental diseases," and thereby to make a further advance towards the conception that insanity is one disease, which is due on the one hand to various grades and forms of cerebral subevolution, and on the other to various grades and forms of cerebral involution and dissolution.

References.

[*The monographs of Deny and Camus and of Dreyfus, on which this review is based, contain references to most of the papers dealing with the subject of maniacal-depressive insanity.*]

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[25] Savage and Goodall. "Insanity and Allied Neuroses," 1907, p. 206.

Die Polyneuritischen Psychosen, by ALBERT KNAPP. (Wiesbaden: J. F. Bergmann, 1906.)

ALTHOUGH Bonhoeffer's "Akute Geisteskrankheiten der Gewohnheitstrinker" is still the most important and generally useful of works dealing with polyneuritic psychosis, this well-written little book of Dr. Knapp's is distinctly valuable as a supplement to it. Of its 140 pages, nearly ninety are devoted to a report of eight selected atypical cases, and the remainder to a general description of polyneuritic disorders, somewhat on the lines of a text-book.

The question when a psychosis may rightly be termed polyneuritic is answered thus (p. 116): "Doubtless when marked neuritic phenomena are combined with the amnesic symptom-complex. But it is generally recognized that in Korsakow's psychosis the neuritic phenomena may be in the background or may quite elude observation, and that the polyneuritic virus may attack the brain exclusively. . . . Just as the neuritic phenomena may be only slightly indicated, so, too, in many cases, the Korsakow symptom-complex is not present in its full development."

In his general description Knapp tells us nothing new about the Korsakow symptom-complex itself; he is content with a bare enumeration of its constituent features. In his reports of cases also he in this respect gives just sufficient facts only to indicate that this symptom-complex was present. He devotes his attention to the other very varied phenomena which may accompany it. Here are no results of that psychological analysis and experiment for which polyneuritic psychosis offers a field hardly to be equalled by any other pathological condition with which we are acquainted; but instead we are given an admirably pictorial account of a great variety of mental symptoms, familiar in themselves, but not commonly recognized as apt to occur in conjunction with polyneuritic psychosis. From this standpoint Knapp recognizes the following forms of polyneuritic psychosis:—Delirious, stuporose, demential, hallucinatory (systematized and unsystematized), and paranoic forms; *Angstzustände;* expansive, maniacal, and melancholic forms; polyneuritic motility psychoses; and cases with rarer mental anomalies. The maniacal form

is rare, and unrecorded before Knapp's fifth case here; but the writer of this notice has had under his observation a case which might well enough come under this head, as it presented marked *Ideenflucht*, with rhyming and clang-association, and a mood characterized by boisterous hilarity rapidly alternating with anger. Knapp's list may perhaps give fresh discouragement to those who look for a systematic classification of mental diseases on a basis of symptomatology.

The very interesting and important cerebral focal symptoms (aphasic, agnostic, and the like) are well discussed in a general way, but not in detail. In the section on bodily symptoms, especially those referred to peripheral nerves, a number of anomalies are discussed, such as the occasional occurrence of exaggerated knee-jerks with hypotonus. But sluggishness or rigidity of pupils to light is perhaps hardly so common as the wording of Knapp's remarks might lead one to suppose.

On the question of etiology, Knapp is not with those who regard polyneuritic psychosis as an essentially alcoholic disorder. In six of his eight selected cases he somewhat confidently excludes alcohol; and he says it is surely no accident that all polyneuritic psychoses of atypical course without alcoholic etiology have occurred in women, and he lays stress on menstrual disorders and the climacteric; he discusses also the various infections and toxæmias. Of course he agrees with Bonhoeffer and many others that cases of polyneuritic psychosis often begin with a condition indistinguishable from delirium tremens, but he goes further and says this may happen even in non-alcoholic cases. We look in vain for any mention of the remarkable curve of seasonal incidence of polyneuritic psychosis, which attains its maximum near the middle of the second half of the year, and thus resembles an exaggerated form of the curve for delirium tremens.

The book is sober, concise, and commendably quotable, its statements being put as positively as possible. Dr. Knapp has, on the whole, successfully contrived to give a general view with as little resumption as possible of old knowledge, and as complete a presentation as possible of what is new and original.

S. J. Cole.

Publications Recently Received.

[*Notes on a book under this heading do not preclude a subsequent review.*]

Vorlesungen über den Bau der nervösen Zentralorgane des Menschen und der Tiere. Zweiter Band: *Vergleichende Anatomie des Gehirns.* Von Ludwig Edinger. S. 334. Leipzig: Vogel, 1908.

The seventh edition of this remarkable book should be in the hands of every neurologist. It is easy to read, luminous, and illustrated with 283 figures. The author is to be congratulated on the character of his work and on his power of exposition.

The Science and Philosophy of the Organism. The Gifford Lectures, delivered before the University of Aberdeen in the year 1907. By Hans Driesch, Ph.D. Pp. 329. London: Black, 1908.

This volume is concerned with the chief results of analytical biology, morphogenesis, adaptation and inheritance. The author then passes to the principles of schematics, the theory of descent, and the logic of history. A most valuable work, dealing with the influence of present biological theories upon philosophic thought.

Diseases of the Spinal Cord. By R. T. Williamson, M.D. Pp. 432. With 183 illustrations and seven plates. London: Henry Frowde and Hodder & Stoughton, 1908.

This work is put together from a series of lectures by the author on a subject in which he has long been an authority. The pathological descriptions are unusually complete, and are illustrated with excellent plates. The clinical aspect is dealt with adequately. The book is admirably printed, and will be of use to the physician interested in the pathology of the cord-diseases which come under his notice.

Nervöse Angstzustände und ihre Behandlung. Von Dr. Wilhelm Stekel. S. 315. Berlin and Vienna: Wibau & Schwarzenberg, 1908.

A full account of those neuroses associated with fear, all those phenomena to which the name "phobias" has been given. The book contains a large number of clinical observations and is well worthy of study alike by the general physician and neurologist.

Transactions of the Ophthalmological Society. Vol. xxviii. Fasc. ii. London: Churchill, 1908. [These *Transcations* are now issued in three parts annually, each of which costs 4s.]

This number contains a paper on "Optic Neuritis in Cerebral Tumours," by Mr. Paton, based on observations made at the National Hospital. He concludes that there is no evidence to show that the changes in the disc are due to meningitis or to inflammation descending the nerve trunk; nor is it caused by pressure on the nerve sheath or local vasomotor changes. He prefers to believe optic neuritis with tumours of the brain is a local manifestation of a generalized œdema of the cerebral tissues. This paper was followed by an animated discussion.

Nursing the Insane. By CLARA BARRUS, M.D. Pp. 409. New York: Macmillan, 1908.

An excellent attempt to deal with a difficult subject. Care of the insane is treated not as a modified form of hospital nursing, but as worthy of independent study. The directions are clear and the index complete.

Education, Personality and Crime. By ALBERT WILSON, M.D. Pp. 296. London: Greening, 1908.

A popular book in which the work of modern neurologists, psychologists and alienists is dealt with superficially. It is full of crude generalizations from complex facts.