immense majority of cases of hemorrhage and softening affecting the points of the encephalon we have mentioned.

Undoubtedly, gentlemen, that is what takes place, in the great majority of cases. But, the chapter of exceptions accompanies the rule. There are cases, and I have myself observed several of this kind, in which sensibility is affected in a predominant manner, and in which anæsthesia persists, even after the recovery of motion.

Such alterations of sensibility may present themselves with the following characters. The anæsthesia affects one entire half of the body and stops just at the median line. The corresponding half of the face, both as regards the skin and the mucous membranes, shows insensibility, exactly as in hysterical hemianæsthesia. Then also *analgesia* and *thermo-anæsthesia* may be observed, with conservation of tactual sensibility, as MM. Landois and Mosler[1] have ascertained. Finally, there are also cases, though of more rare occurrence and as yet imperfectly described, but having still their own importance, which render it probable that, under such circumstances, alterations of the special senses may exist on the side opposite to the encephalic lesion, or, in other words, on the same side with the hemianæsthesia.

The physicians of the last century have already remarked these exceptional phenomena. Borsieri, among others, relates the history of a patient who, three months before, had been stricken with apoplexy, and in whom anæsthesia still persisted, although the power of movement had returned. He quotes some other cases of the same kind from different authors.[2]

Analogous cases have been mentioned by Abercrombie, Andral, and, in later days, by Hirsch,[3] Leubuscher, Broadbent, Hughlings-Jackson,[4] and especially by Türck. The latter alone has been able to furnish decisive data in reference to the position occupied by the encephalic lesions in such cases.

When the hemianæsthesia presents itself with these characters, the optic thalamus is almost always affected in a predominant, if not in an exclusive manner. For my own part, I have seen hemianæsthesia superadded to hemiplegia, in many patients affected with cerebral hemorrhage, and, in such cases, on post-mortem examination I always found the lesion of the optic thalamus, the existence of which during life I had ventured to announce.

From what precedes, gentlemen, should we conclude that the lesion of the optic thalamus is the real organic cause of the hemianæsthesia observed in all these cases? That is a question deserving of discussion. I am thus led to speak of the physiological theory, which may be called the *British theory*, because it was, I believe,

[1] Landois et Mosler, 'Berliner Klin. Wochens.,' 1868, p. 401.
[2] Borsieri, 'Inst. pract.,' vol. iii, p. 76.
[3] Hirsch, 'Klinische fragments,' I. Abth., p. 207, Kœnigsberg, 1857.
[4] H. Jackson, 'Note on the Functions of the Optic Thalamus.' In 'London Hospital Reports,' 1866, t. iii, p. 373.

first published and maintained by Messrs. Todd and Carpenter, two British authors. According to this theory, the *optic thalamus* is the centre of perception of tactual impressions; it would, in some degree, correspond to the posterior cornua of the gray substance of the spinal cord. The *corpus striatum* would be the terminal of the *motor tractus* and connected with the execution of voluntary movements; it would be analogue of the anterior cornua of the cord.

This theory of which Schrœder van der Kolk[1] has shown himself the avowed partisan, is, if we might use the word, the antipodes of the French theory, which you will find set forth in a very complete manner in M. Vulpian's Lectures. According to the latter view, the centre on which sensitive impressions are transformed into sensations would not be in the brain proper, because an animal, from which the brain, including the optic thalamus and the corpus striatum, has been removed, continues to see, to hear, and to feel pain, &c. The centre of sensitive impressions would therefore reside lower down, in the protuberantia and perhaps also in the crura cerebri.

Under this hypothesis, the following is the manner in which its advocates regard, in the pathological domain, those authentic facts which show a lesion of the optic thalamus coinciding with the decrease or abolition of sensibility on the side of the body stricken with hemiplegia. They say, and their allegation is perfectly correct, that, in such cases, we have frequently to do with recent lesions, such as *intra-encephalic hemorrhage*, or *ramollissement*, or *tumours*—lesions by which the optic thalamus is extremely distended and which, consequently, may have the effect of determining the compression of the adjacent parts,—of the crura cerebri for instance. It is, on the other hand, well established that, in a number of cases, the optic thalamus may be injured, even gravely and throughout a large portion of its extent, without being followed by any special disorder in the transmission of sensitive impressions.

To the last argument, the British authors, M. Broadbent[2] among others, oppose the plea that the optic thalamus, the presumed centre of sensitive impressions, should doubtless be assimilated to the gray axis of the spinal cord—the latter, it is known, continues to transmit these impressions even when it has suffered the most serious derangement, if only a small remnant of gray matter persist, capable of connecting its lower with its upper extremities. I confess that the comparison seems to me far-fetched, especially from the moment it is laid down as a principle that the optic thalamus should be considered a centre; for, so far as regards the transmission

[1] Schrœder van der Kolk, 'Pathol. und Therapie der Geistenkrankheiten.' Braunschweig, 1863, p, 20.
[2] Broadbent, 'Medical Society,' London, 1865, and 'Med.-Chir. Review.'

of sensitive impressions, the gray axis of the cord is manifestly merely a conductor.

However this may be, gentlemen, such is the state of the question. In my opinion, the disputed points cannot be definitely solved, except by means of careful clinical observation, verified by studious anatomical investigations, the chief aim of which should be to establish, with great precision, the seat of the encephalic lesions, to which the symptoms recorded during life might be correlated. And the circumstances of the case should be such that the influence of compression, or any other phenomenon, acting by contiguity, would be completely eliminated. Now, gentlemen, in the present state of the science, the cases which include all these conditions are extremely rare, so far, at least, as my knowledge goes. We may, however, mention as approximating to this ideal, the cases which were presented by L. Türck to the Academy of Sciences of Vienna,[1] to which I have already alluded. They were four in number.

In the instances recorded by L. Türck, there had been, gentlemen, either old hemorrhagic foci, then represented by ochreous cicatrices, or ramollissement foci arrived at the stage of cellular infiltration. In all the cases, the hemiplegia resulting from the presence of foci had disappeared long before death, but the hemianæsthesia had persisted until the fatal end. The portions of the encephalon affected by the alteration are carefully mapped out.

The German nomenclature of the different parts of the encephalon, however forbidding it seems to us, on account of its multiplicity of strange terms, yet presents in my opinion, an incontestable advantage, that, namely, of supplying a very complete topographical map, if I may make use of the comparison, where the smallest hamlet receives a name. The French nomenclature has, no doubt, the advantage of tending to simplification, but this is sometimes to the detriment of absolute exactness; it is often incomplete. Now, with respect to the question which occupies us, there is no detail, however minute, which ought to be neglected. We must, at all hazards, take heed of the slightest details, for we are quite ignorant, in the actual state of the science of the brain-physiology, whether some little point, which has no name in the French nomenclature, may not be a position of primary importance.

Availing ourselves, therefore, of the nomenclature in use beyond the Rhine, let us endeavour to become familiar with the topography, in order that we may accurately recognize the seat of the lesions, in the observations recorded by L. Türck.

I place under your observation, a frontal section taken across the cerebral hemispheres, immediately behind the corpora mammillaria

[1] 'Sitzungsber. der Kais. Akademie der Wissenschaften zu Wien,' 1859. *V. infra*, the analyses of these cases.

(Fig. 18). You recognize on this section, just exterior to the middle ventricles, the *nucleus caudatus* (or intra-ventricular nucleus of the corpus striatum), which, in this region, is merely represented by a very small portion of gray matter,—beneath and interior to it, the *optic thalamus*, here largely developed; external to this lies the *capsula interna*, formed principally by bands of white substance which are simply the prolongations of the lower stage of the crura cerebri; these proceed to expand in the centrum ovale to assist in constituting the corona radiata:—external to this is the *extra-ventricular nucleus of the corpus striatum*, in which you distinguish three secondary nuclei denominated by the numbers 1, 2, 3; the third, or outermost, is sometimes designated by the term *putamen*. Still more external is a thin lamina of white matter, the *capsula externa*, and finally, a small band of gray substance, the rampart (or claustrum) (*Vormauer*).[1]

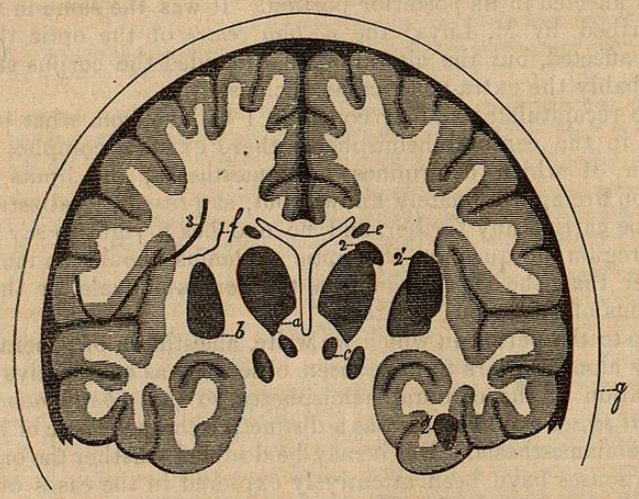

FIG. 18.—Transversal section of brain,—*a*, optic thalamus;—*b*, corpus striatum, lenticular nucleus;—*c*, corpus striatum, caudate nucleus;—*f*, indication of the radiating corona of Reil;—2, 2′, 2″, apoplectic foci (Obs. ii, in 'Türck's Memoir,' *v. infra*, pp. 212–14);—3, vestige of an apoplectic focus (Obs. iii, in 'Türck's Memoir').

Now, gentlemen, in the cases recorded by Herr Türck, the lesions had invaded alike the superior and external portion of the optic thalamus, the third nucleus of the extra-ventricular portion of the corpus striatum, the superior portion of the capsula interna, the

[1] The *vormauer* of German anatomists, the *avant-mur*, *rempart*, or *noyau rubané* of the French, is a band of gray matter which, arising from the superior portion of the amygdala, curves round towards the white substance of the convolution bounding the fissure of Sylvius. (S.)

corresponding region of the radiating corona, and the adjacent white substance of the posterior lobe.

We have consequently complex lesions to deal with here, but they, at all events, allow the region which requires investigation to be circumscribed. Further researches, when sufficiently numerous, will enable us soon to ascertain the fundamental lesion, to which the existence of the hemianæsthesia should be attributed.

Some other cases of hemianæsthesia, of cerebral origin, which have been published since those of Türck appeared, testify to lesions occupying the same circumscribed regions of the encephalon; they make, however, no important additions to the results obtained by that observer. Such amongst others, is the case recorded by Dr. Hughlings Jackson,[1]—here again the alteration was not confined to the *thalamus;* it extended to the extra-ventricular nucleus of the corpus striatum, and consequently the capsula interna must have been affected in its posterior portion. It was the same in the case described by M. Luys,[2] the *median centre* of the optic thalamus was affected, but the alteration had invaded the corpus striatum, (probably the extra-ventricular nucleus).

To recapitulate, we may conclude I believe from what precedes that, in the cerebral hemispheres, there exists a complex region, lesion of which determines hemianæsthesia; the limits of this region are approximately known, but, at present, localization cannot be carried any further, and no one has a right to say that in the region in question, the optic thalamus should be inculpated rather than the capsula interna, the centrum ovale, or the third nucleus of the corpus striatum.

Up to the period in which we write, anæsthesia of general sensibility alone appears to have been observed, as consecutive on an alteration of the cerebral hemispheres, so that *obnubilation of the special senses* would remain as a distinctive characteristic of hysterical hemianæsthesia. But it may be doubted whether the organs of these senses have been attentively explored in the cases of hemianæsthesia of cerebral origin, hitherto published; the records are silent with respect to it.[3]

[1] The disease was not strictly limited to the thalamus. . . . Outwards the disease extended through the small tongue of corpus striatum which curves round the outside of the thalamus, and thence up to the gray matter of the convolutions of the Sylvian fissure. ('London Hospital Reports', *loc. cit.*, t. iii, p. 376.)

[2] Luys, 'Iconographie photographique des centres nerveux,' p. 16.

[3] At the period when this Lecture was delivered, we were only acquainted with the observations of L. Türck by the brief mention made of them in Rosenthal's 'Treatise on Diseases of the Nervous System.' Since then, thanks to the courtesy of M. Magnan, we have been enabled to procure the complete translation of Türck's memoir (Ueber die Beziechung gewisses Krankheitsherde des grossen Gehirnes zur Anasthesie,' Aus dem xxvi Band, S. 191, des Jahrganges, 1859, des Sitzungsberichte der Mathem. Naturw. Classe der Kais. Akademie der Wissenchaften). We think it useful to give the substance of this work. After recalling

For my own part, I am inclined to believe that the participation of the special senses will be one day recognized, when care shall

the fact that, usually, in hemiplegia caused by the formation of apoplectic foci in the brain (hemorrhage and ramollissement), the sensibility re-appears very promptly as a general rule, the author relates four cases where, on the contrary, the anæsthesia persisted in a high degree of intensity.

Case 1.—Fr. Amerso, æt. 18. In August, 1858, left hemiplegia, speedy re-appearance of motor power. 12*th. Nov.*—The movements of the left upper extremity are rapid and energetic; those of the corresponding inferior extremity exhibit slight paresis. Very intense anæsthesia exists on the left side (limbs, body, etc.). Facial sensibility is diminished, on this side only. Formications from time to time through all the left side. Died, 18th March, 1859.

Autopsy.—At the base of the corona radiata of the right hemisphere, immediately outside of the tail of the corpus striatum, appears a lacuna of the size of a pea (*cellular infiltration*). The anterior wall of this lacuna is two lines behind the anterior extremity of the optic thalamus. Two or three lines farther off, another lacuna is seen, of smaller dimensions, which extends to four or five lines behind the posterior extremity of the thalamus, so that as the usual length of the optic thalamus is eighteen lines, the portion of the corona radiata which lies immediately adjacent to the tail of the corpus striatum was perforated, fore and aft, by the old focus of ramollissement for an extent of eleven lines. A similar focus involves the external portion of the third part of the lenticular nucleus. It commences nearly two lines behind the anterior border of the optic thalamus and ends at about four lines from the exterior extremity of the optic thalamus. In its course of one inch long, it occupied the greater length of the internal side of the third part of the lenticular nucleus, and part of the capsula interna. In the posterior half of their course, then two foci were not farther part, in one place, than the distance of one line. It follows, that in this place, almost all the corona was separated from the internal capsule and the optic thalamus. *Spinal cord.*—Collection of granular bodies somewhat abundant in the left lateral column, rare in the anterior column.

Case 2, S. J., æt. 55.—Apoplectic attack, followed by hemiplegia, Oct. 25, 1851. Two months after, the paralysis of the extremities disappeared to such an extent that the patient could extend the arms, grasp objects with some strength, and walk without help, but lamely. *Oct.*, 1855.—Anæsthesia of the left extremities (face and body also benumbed, but in a less degree) persisting since the attack. Power of motion recovered, but the limbs of the left side are more feeble than those of the right. Died Oct. 31, 1858.

Autopsy.—Old flat cicatrix, about five lines in breadth and eight in length, situated at the superior and external part of the right optic thalamus. The cicatrix begins four and a half lines behind the left anterior extremity of the optic thalamus, and ends eight lines farther off. Lying parallel to this cicatrix is another, an inch long, occupying the third part of the lenticular nucleus; it begins two lines behind the anterior extremity of the thalamus and ends nearly three lines in front of its posterior extremity (fig. 18, 2, and 2′). There was, besides, a lacuna in the right inferior lobe (fig. 18, 2″), another in the anterior lobe of the same side, two as big as a pin's head in the anterior part of the right optic thalamus; two in the pons Varolii, and finally, one in the right and superior portion of the left hemisphere of the cerebellum. No secondary degeneration of the cord was observed.

Case 3.—Fr. Hasvelka, æt. 22. November 1st, 1852. Apoplectic attack, hemiplegia on the right, with intense anæsthesia of the corresponding half of the body. At the end of five weeks, diminution of motor paralysis. *February* 3, 1853.—Motion quite free, on the right side. The entire right half of the body is the seat of very marked anæsthesia (scalp, ear, face, and body). The anæsthesia is equally noticeable in the eyelid, nostril, left half of the lips, and not only on the outer but also on the inner side. The right conjunctiva is less sensitive than the left. When the right nostril is tickled, the sensation is less felt than in the left. Same difference between the right meatus auditorius, and the left. In the right half of the mouth (tongue, palate, gums, cheek), the sensation of heat is

have been taken to seek for it. My opinion is founded on the following basis:—

There exists in the clinical history of the organic diseases of the nervous centres a symptomatic sign but little known, and little remarked as yet, which I shall have occasion some day to discuss in detail before you. This is a kind of rhythmical convulsion which occupies an entire half of the body, including the face (in many instances at least), and which assumes sometimes the appearance of the clonic jerking of chorea, sometimes that of the tremor of paralysis agitans. This hemilateral trembling occasionally presents itself as a primary affection; at other times it supervenes consecu-

less vivid than in the left. At the tip of the tongue on the right, and over the space of an inch in length, the patient does not feel the *taste* of salt Same result as regards the right half of the dorsum and root of the tongue On the right side also, the sense of *smell* is weakened, and *vision* is less distinct. When the pupils have been made to contract by bringing a light close to the eyes, the right pupil afterwards dilates more than the left, the sense of hearing is normal on both sides. *February* 26.—The anæsthesia has diminished; the movements are more energetic. *March* 15.—Temporary improvement of vision: no difference between the two eyes. *April* 3.—The anæsthesia still exists over the right half of the body (on touch. and pinching). Debility of vision augmented on the right. Died April 4.

Autopsy.—In the white substance of the left superior lobe, is found a focus of ramolissement two inches in length and one in breadth. It dipped into the inferior convolutions of the operculum, and attained the surface of the brain. Its posterior extremity corresponded to that of the optic thalamus; its anterior part greatly exceeded that of the thalamus. In its broadest portion, the focus was only separated by three lines from the tail of the corpus striatum. The convolutions lying beneath, were to the extent of a florin, yellow, softened, and depressed. (Fig. 18, 3.) Optic thalamus healthy, perhaps a little fragment of the third part of the lenticular nucleus has been touched. The focus had destroyed a somewhat considerable length of white substance, and the two external thirds of the foot of the corona radiata. *Spinal Cord.*—Slight agglomeration of nuclei in the most posterior part of the lateral column.

Case 4.—Anne B—, an aged woman, died 22d February. For many years she had right hemiplegia, with intense anæsthesia of the same side. In addition, sensorial anæsthesia (sight, smell, taste) of the same side, and formications.

Autopsy.—Old apoplectic focus, pigmented brown, situated along the outer part of the left optic thalamus, and quite close to the tail of the corpus striatum. It commences six lines behind the anterior extremity of the thalamus and extends two or three lines in front of its posterior extremity. Anteriorly, it is half a line, and posteriorly two or three lines beneath the superior surface of the thalamus, which is considerably depressed in this direction. An inch long and four or five lines deep, the focus touches a large extent of the posterior part of the radiation of the crus cerebri, a part of the internal capsule, and, perhaps, a part also of the lenticular nucleus *Spinal Cord.*—Accumulation of granular bodies in the posterior part of the right lateral column.

To sum up: the foci were seated at the external periphery of the optic thalami, they extended, from before backwards, in the longitudinal axis of the cerebrum, without, in most cases, reaching the extremities of the thalamus. They were from eight lines to an inch in length, reaching even two inches into the white substance. The regions affected were: the superior and external part of the thalamus; the third part of the lenticular nucleus; the posterior part of the internal capsule, comprised between the thalamus and the lenticular nucleus; the corresponding portion of the white substance of the superior lobe opposed to it. Several of these regions were always affected together. The fibres which proceed from the white substance of the hemisphere into the external part of the optic thalamus were constantly affected.

tively on a hemiplegia, whose invasion was sudden. In the latter case, it commences to appear at the epoch when motor paralysis begins to improve. The lesion consists in the presence either of a focus of hemorrhage or of ramolissement, or in that of a tumour. In all cases of this kind, which I have hitherto observed, and in the analogous facts collected from various authors, the lesion in question occupied the posterior region of the ophthalmic thalamus and the adjacent parts of the cerebral hemisphere exterior to it.

Now, hemianæsthesia is a tolerably common—but still not a constant—accompaniment of this group of symptoms, and it occupies the same side of the body as the tremor.[1]

It existed in a high degree of development, in a male patient whose history M. Magnan has recently communicated to the *Société de Biologie;* in his case, the form of tremor of which I have tried to give you a summary notion, showed itself in a most marked manner. Everything tends to show (I cannot be more positive as there was no autopsy) that the encephalic lesion was, in this man, of the same sort, with respect to position, as that which I found in my patient. Now, in this case, M. Magnan ascertained, in the clearest manner, that tactual sensibility was not alone involved; the special senses were themselves affected, as they are in hysterical hemianæsthesia. On the side stricken with hemianæsthesia, the eye was affected with amblyopia, the sense of smell was lost, and taste was completely abolished.

Hence, it becomes probable, if I am not mistaken, that complete hemianæsthesia, with derangements of the special senses,—and consequently, such as is presented in hysteria,—may, in certain cases, be produced by a circumscribed lesion of the cerebral hemispheres.[2]

[1] See a Lecture of M. Charcot (in 'Le Progrès Médical,' 23 Janvier, and 6 Fevrier, 1875), on 'Hemichorée post-hémiplégique.' (Note to 2d edition.)

[2] The views expressed in this Lecture, relative to hemianæsthesia of cerebral origin, have received further clinical confirmation from the incidents of a case, which we noted, in M. Charcot's wards. (Progrès Médical,' 1873, p. 244), and from the experiments on animals conducted by M. Veyssière (Recherches cliniques et experimentales sur l'hemianesthesie de cause cérébrale,' Paris, 1874). This work also contains some interesting clinical notes. (Note to the 2d edition.)