meningitis, with strong inflammation at the convexity, is manifested by violent headache, vomiting, ordinary delirious excitement, convulsive appearances, changes in the pupils; it is always accompanied by high fever," &c.; and adds, "Now and then recent and rapidly fatal cases are actually brought to asylums as cases of mania." Griesinger's description in no way suits the case I have given, but his latter remark may apply more closely. For my own part I can only say I treated the case as one of acute mania, without suspecting one-sided meningitis, and have no anxiety to defend or excuse my opinion, but I think the case is very instructive in a clinical point of view, and for that reason worth recording.

On the present State of our Knowledge regarding General Paralysis of the Insane. Part II. By Dr. C. WESTPHAL, Physician to the Lunatic Wards of the Charité, and Lecturer on Medical Psychology in the University of Berlin. Translated from the German by James Rutherford, M.D., F.R.C.P. Edin., Assistant Medical Officer, Borough Lunatic Asylum, Birmingham.

(From Griesinger's Archiv für Psychiatrie. No. I.; concluded from the Journal of Mental Science for July, 1868., *p.* 192.*)*

The theories which have hitherto been advanced regarding the nature of the morbid process in general paralysis of the insane are based essentially on actual or supposed anatomical changes in the brain and its membranes. The spinal cord was, as a rule, very seldom examined. In the many records of *post mortem* examinations which have been published, it is scarcely ever referred to; and only in a few isolated, carefully-observed, and well-described cases (especially by H. Hoffmann), which remain almost unnoticed, has—if we exclude the uncriticisable cases of "softening"—any palpable disease of the spinal cord been established. Thus it came to pass that in the framing of theories regarding the nature of the paralysis, the spinal cord was either entirely ignored, or the purely *cerebral* character of the disease was expressly and emphatically inculcated as distinguishing it from other *spinal* affections.

It must be admitted that the assumption has already been made by Joffe,* that in all cases of general paralysis of long standing, if the spinal cord be minutely investigated, morbid conditions (new formation of connective tissue) of the spinal marrow will be found to exist, but it is not made manifest in how far this general statement is justi-

* Zeitschr. der Gesellsch. der Aerzte 24 Wien. 1860. Nr. 5, p. 74.

fied by the results of actual observation. In the cases adduced, he only once makes mention of that condition of the spinal marrow—minute details are omitted. Nevertheless, the merit of having first directed attention, in a general way, to the occurrence of disease in the spinal cord in general paralysis is incontestibly due to Joffe. His observations, however—perhaps for the very reason that they were not supported by special observations—remained absolutely unheeded. The great majority of asylum physicians examined the spinal cord no more frequently than formerly; the motory disorders continued to be referred to changes in the brain, and the theory of the disease remained the same. Diseased conditions of the spinal cord are now, however, as I think I have shown,* quite common in general paralysis of the insane, and may be considered as amongst the best constituted facts. In so far as they have as yet been observed, these affections present various forms and degrees; sometimes the membranes are involved, sometimes they are not.

On the dura mater inflammatory processes are occasionally observed (pachymeningitis, also of a hœmorrhagic character). Affection of the pia mater is recognised by general opacity and thickening of its tissue: thickened bands and retiform lines are also frequently seen projecting from its surface, and, moreover, filiform or more membranous adhesions pass between it and the dura mater. In regard to the disease of the spinal cord itself, there may be distinguished, anatomically, the following forms:—1st. Affection of the posterior columns throughout their whole length from the cervical to the lumbar regions. 2nd. Affection of the posterior section of the lateral columns likewise throughout their whole extent. 3rd. Mixed affection of the posterior columns, and of the posterior portion of the lateral columns. The isolated affection of the posterior columns assumes a form somewhat different anatomically from the other varieties. It consists in a considerable loss of nerve elements (atrophy), in the place of which there has entered a connective tissue-like substance, which is sometimes plainly seen, when longitudial sections are made, in the form of completely developed fibrous connective tissue. When transverse sections are made, it is seen that this connective tissue lies imbedded here and there in irregular plates, of larger or smaller size, between the transverse sections of the nerve tubes. Where the process is further advanced these plates unite with each other, so that when still further advanced, merely a connective tissue-like substance is apparent, in which here and there the transverse section of an isolated nerve tube may still be seen. The nerve tubes themselves appear partly very small, partly of ordinary diameter, and occasionally very broad. Atrophy and hypertrophy of the nerve tubes have therefore been spoken of; but this point demands further investigation, as, even

* Virchow, Arch. Bd., 39 u 40. Compare also Allgem. Zeitschr. für Psych. XXIII., p. 709; XX., p. l.c., XXI., p., 361 u 450. More recently Magnam (l.c.) has also recognised affections of the spinal cord.

in the normal spinal cord, considerable differences occur in the diameter of the nerve tubes. The mode of preparation (for example, unequal hardening) also plays a part. I consider, therefore, that this question is not yet fully elucidated.

The process is usually most markedly developed at the periphery of the posterior columns, especially in the region of Goll's tracts; but there occur many kinds of irregularities, in particular the parts at the side of the middle portion of the posterior longitudinal fissure are often strongly affected. By comparing the superior and inferior portions of the spinal cord in regard to the intensity and extent of the disease, no constant distinctions can be made out. Nevertheless, in the cervical portion very frequently Goll's tracts only are affected, while in the dorsal and lumbar portions the changes extend over the whole area of the posterior columns. The most anterior portions of the posterior columns situated next to the posterior commissure always remain most intact. Such are the appearances presented after hardening of the spinal marrow. In fresh preparations matters appear differently, inasmuch as, in some cases, numerous fat cells may be observed free and upon the walls of the vessels, while in others—in other respects not distinguishable from the first—the fat cells are altogether absent. Further, there is observed, though relatively seldom, a great number of pale cellular elements containing nuclei, and very frequently numerous corpora amylacea.

The picture assumes a different appearance in the second and third modes of development—viz., with affection of the lateral columns, and of the lateral and a portion of the posterior columns. Here there are always found, in fresh conditions, free nucleated cells lying in the tissue. In hardened preparations the individual nerve tubes containing nerve substance, or groups of these, are seen to be surrounded by diffused lines of connective tissue, which, as a whole, present the appearance of a network with knotted points. The lines of the network are much broader than those which in normal conditions surround the individual nerve tubes, and its meshes are occupied sometimes by one, sometimes by several transverse sections of nerve tubes, containing nerve substance. In the knotted points just mentioned, nucleolar structures are occasionally distinctly observed which extend also into the lines of the net. This picture differs distinctly from that first described (grey degeneration), through the appearance and preservation of the reticular outline, the absence of the large irregular plates of connective tissue, and the constant presence of nucleated cells. I, in accordance with the dominant appearances, characterise this form as *chronic myelitis*. If the lateral columns only are affected, the disease is always confined to the posterior section, and it diminishes towards the lumbar portion, so that there only the parts in the neighbourhood of the apex of the posterior cornua are usually attacked. In the cervical portions the change either affects the entire posterior section of the lateral columns, or it is limited to one locality, corresponding to

the angle between the anterior and posterior cornua and along the latter. In the dorsal portion the change appears (the number of the nucleated cells) in general to be most pronounced.

If the posterior columns, and the posterior section of the lateral columns, are simultaneously affected,* the latter are found to be, in regard to the relative extent of the disease upwards and downwards, as has been described. The posterior columns are, on the contrary, generally affected only from the cervical to a division of the dorsal portion, and occasionally in such a manner that again in the cervical portion Goll's tracts, and in the dorsal portion the whole extent of the posterior columns are involved. From this point downwards the disease of the posterior columns diminishes, and frequently the parts situated nearest to the posterior longitudinal fissures are the first to become free. Occasionally, however, only the *cervical portion* of the posterior columns is affected. In the cases which I have hitherto carefully investigated, the mode of extension of the disease was confined to the way we have just mentioned. It is nevertheless certain, as I can affirm after the experience of further recent cases, that still other combinations occur. Even in the affection of the posterior and lateral columns just described there were also found here and there nucleated cells in the *anterior columns*. In the cases, however, which have recently come under my notice, the latter were strongly involved, and the affection of the posterior columns descended further. I have not as yet been able to undertake a more minute investigation of these cases.

I either did not find the appearances of grey degeneration (atrophy) as seen in isolated affection of the *posterior columns* in the forms classed under 2 and 3, or merely found traces of them in circumscribed spots.

It is an interesting fact that the degeneration may be followed still farther through the medulla oblongata. The affection of the posterior columns terminates very soon, and does not extend beyond the commencement of the 4th ventricle. It can be shown in the expansions of the slender tract that the grey substance radiating there (mediales hinteres nebenhorn, *Reichert*) is not involved. The disease of the lateral columns may be traced by means of the nucleated cells through the decussation of the pyramids, the pyramids, and the longitudinal fibres of the pons Varolii to the foot of the cerebral peduncle, the external section of which will be affected by it.

To these facts is immediately associated the question whether there exists a direct connection between the pathological conditions of the spinal cord and of the brain. Such a connection has not yet been anatomically demonstrated. The nucleated cells are not found beyond the foot of the peduncles of the cerebrum, not in the inner capsule,

* Perhaps Calmeil, in a few of the cases described by him in which he examined the spinal cord, has also seen the nucleated cells, although, for the forementioned reasons, he could not well form an opinion regarding them.

the grey substance of the lenticular bodies, corpus striatum, thalamus, &c. In my investigations I have not, as yet, discovered anything which seemed to deviate from the normal condition in these parts; and, in particular, no changes of the ganglia of the nerves. This, nevertheless, is saying very little if the difficulty of investigating the grey substance of these portions of the brain be borne in mind. Almost each portion would require to be made the subject of special study before any authoritative opinion could be given. One might, perhaps, on first thoughts, expect to find an affection of the parts we have mentioned (internal capsule, &c.,) in cases of disease of the lateral columns extending into the peduncles of the brain. A comparison with cases of so-called secondary degeneration of the spinal cord from primary cerebral apoplexy, shows that the disease pursues exactly the same course through the medulla oblongata and spinalis in both circumstances; then, also, in cases of apoplexy and softening affecting the inner capsule, the lenticular body, &c., it is in the pyramids and posterior of the lateral columns that the formation of nucleated cells takes place (Türck). The idea that in general paralysis we have likewise to do with an affection of the brain, which produces a secondary disease of the spinal cord, extending *downwards*, may, for the present at least, and until further investigations are instituted, be abandoned, as no evidence in favour of it exists. On the contrary, the degeneration of the *posterior columns* in its form and mode of development (diminishing towards the cervical region), reminds us very much of cases in which a primary disease extending from the lumbar region *upwards*, must be assumed (as in certain cases of grey degeneration of the posterior columns in persons not insane), or of cases where, in consequence of *localized affection* of the spinal cord (from compression in consequence of tumours, &c.,) disease of these columns extending upwards is met with. Still, as we have just seen, even in such cases, no extension beyond the medulla oblongata, either towards the cerebrum or cerebellum, could be demonstrated. There is nothing, therefore, in the meantime, to justify the assumption of a direct continuation of the pathological process into the brain.

Neither do the *symptoms observed during life* necessarily point in that direction. Here the leading question must be regarding the order of succession of the symptoms of the cerebral and spinal affections, and in how far these symptoms indicate an ascent of the malady to or descent from the brain. There are now on record whole series of cases in which manifest and undoubted *spinal* symptoms *preceded* the cerebral. These comprehend all those, who, years before the outbreak of a cerebral affection, presented well-marked symptoms of Tabes Dorsualis, including the characteristic gait, which can be referred to no other than a spinal affection. It must be admitted that here, at an early period of the disease and before any psychical anomalies show themselves, symptoms frequently appear which might be considered due to a cerebral malady of longer standing than the spinal

affection; for example, occasional incomplete paralysis of certain muscles of the eye, and more or less pronounced atrophy of the papilla of the optic nerve. We have, nevertheless, no satisfactory grounds for referring these affections of individual cerebral nerves *per se* to a central malady of the brain; pathological anatomy, in particular, offers in this respect merely negative results. There might easily be some exclusively peripheral affection of these nerves, to which, perhaps we might refer a state of atrophy; but, on the other hand (in the incomplete and transitory paralysis of the muscles of the eye) no palpable lesion can be discerned.

In those cases, also, where decided cerebral symptoms (psychical disorder) do not appear until after an undoubted spinal affection has existed for a long time, we have every reason to regard the disease of the spinal cord as the *primary* affection. If it be further admitted that in those cases where the lower extremities are affected by the motory disorder before the upper, the disease is first located in the lower section of the spinal cord—which, however, might be disputed—an ascending course of the disease from the lumbar region towards the neck might be assumed; for, in reality, the lower extremities are generally first affected, and as the disease advances, they are also the parts which are most under its sway. If the process should advance further upwards, we would have to expect phenomena referrible to the medulla oblongata, and very soon cerebral phenomena. Alas! according to the present position of our knowledge, symptoms indicating the anatomical locality are here absent. Of course we see the sphere of the hypoglossal (disorder of speech) and facial nerves (tremor, incomplete paralysis) attacked; but we have no sure basis by which to judge in how far these phenomena are referable to changes in the medulla oblongata, or in the brain itself. When, however, on the other hand, we consider that the *cerebral disease* (psychical disorder) following upon the spinal affection is, in many of these cases, highly developed before the appearance of disorders in the sphere of the hypoglossal (disorder of speech) or facial nerves, we can scarcely believe that the disease makes *direct* progress within the medulla oblongata along a course which includes these nerves or their so-called central nuclei. We can, therefore, primarily only show that the cerebral phenomena (psychical disorder) and those of the spinal disease succeed each other, without being able by the *symptoms* to prove a continuous *progression* of the process upwards.

It is still more difficult to prove that the reverse is ever the case. We all know that in a great number of cases the first symptoms observed are undoubtedly those of cerebral disease (psychical disturbance), and not until long afterwards do motory disorders of the extremities follow. Even if we assume for the present that the latter are in reality due to a spinal affection, still we could not—because of the motory disorders appearing subsequently to the psychical—conclude that the spinal cord did not become affected until after the

existence of disease in the brain, and particularly for the reason that, as we shall afterwards show, the spinal affection may progress so latently as not to be recognised by well-marked symptoms, particularly by characteristic motory disorders of the extremities, rendering it possible that the disease was already present before the appearance of the symptoms. The appearance of the disorder of speech also affords no secure point of departure. It occurs, in cases of this kind, at very various periods, sometimes subsequently to the entrance of the psychical disorder, sometimes coincidently with its first symptoms; and it bears no constant relation to the extent of the motory disorders of the extremities. It cannot serve as a guide to the locality of the morbid process, chiefly because we do not know where to seek its special cause.

If we consider all the circumstances, we must *for the present* regard the cerebral and spinal diseases which simultaneously exist in general paralysis of the insane as, in-so-far, existing *per se*, and, in certain respects, independent of each other, as it is impossible for us to define more minutely the nature of the cerebral malady, and to establish a connection between it on the one hand, and the processes of grey degeneration and chronic myelitis of the spinal cord or medulla oblongata on the other.

To show the complete independence of the spinal disease as regards the cerebral malady, those cases might, perhaps, be adduced, in which grey degeneration of the spinal cord (Tabes Dorsalis) has existed for a long series of years, even until death, without any alteration of the psychical faculties ever having occurred. Such cases have hitherto been viewed as the sole examples of the type of disease characterised as Tabes Dorsalis, from which there was expressly excluded a central disease of the brain. This independency of the spinal affection is, however, as our investigations have repeatedly shown, in-so-far only relative as the same symptoms occur in peculiar connection with a typical cerebral disease—a connection which has hitherto been almost entirely mistaken, as in general paralysis observers continually recognised a purely cerebral malady.

We cannot now advocate such an absolute independency of grey degeneration of the spinal cord as formerly. We can do this still less if we take into consideration another circumstance. There occur, as we have seen, in the paralytic insane, both grey degeneration and a disease of the spinal cord resembling myelitis; we may, therefore, assume as highly probable that these two forms, on account of their common relations to a disease of the brain clinically of the same nature, and common to both, will also possess anatomically certain relations to each other; indeed, that it is, perhaps, the same process, which, under different conditions which are of course unknown to us, may be modified in its development, and observed in various stages. Thus grey degeneration would lose its independence, clinically as a spinal affection, and anatomically as atrophy, and be brought in con-

nection on the one hand with a cerebral malady, and on the other hand with chronic myelitis.

Certain morbid phenomena, which are always observed, firstly in grey degeneration without paralytic insanity, secondly in it when connected with the latter, and thirdly in paralytic insanity in connection with chronic myelitic processes, point to such a community. To these belong, besides other phenomena, the affection of individual *cerebral* nerves, especially the *optic*, and the *apoplecti* and *epileptiform attacks*.

It is known that in the ordinary form of Tabes Dorsalis (without paralytic insanity),—in those cases where there is found, on examination after death, grey degeneration of the posterior columns—atrophy of the optic nerve is by no means a rare occurrence. The same atrophy, however, also occurs *in general paralysis*, in those, too, who do not present the tabic form of gait, and where, after death, there is found not grey degeneration of the posterior columns, but the fore-mentioned chronic myelitic processes in various columns.* According to Virchow (Arch. X, p. 192), it also occurs in the so-called speckled grey degeneration, so that in all these forms of disease of the spinal cord, although they assume appearances which are anatomically different, we cannot but recognise a common element. Regarding the incomplete paralyses of individual *nerves supplying the muscles of the eye*, that which has been said of the optic nerve is applicable, in so far as they likewise occur, not only in paralytics with grey degeneration of the posterior columns, but also in cases where the disease has the myelitic character. In these cases it is very difficult to make correct observations, as the disorders of movement of the eye occur generally at a period before the patients are subjected to minute clinical observation; when the psychical malady is further developed, they can only with

* Amongst fifty-six male patients investigated in one day in May this year, there were fourteen in which, owing to the psychical phenomena alone, or in connection with motory disorders, general paralysis was diagnosed. Of these fourteen so-called paralytics, two had atrophy of the papilla of the optic nerve, of the ordinary form occurring in spinal diseases. Sight was, so far as could be judged, only moderately altered; but of course a minute examination could not be made. Neither of these two patients had exhibited, up to the day of the examination, any disturbance of the motory apparatus, of speech, or of muscular movement. One of them died soon afterwards, and by the aid of the microscope nucleated cells (myelitis) were found in a portion of the lateral column of the spinal cord; the optic nerves were, in parts, of a grey translucent appearance. Another patient, who died before this one, with complete amaurosis and bilateral atrophy of the papillæ, had only disturbance of speech during the course of the disease, and during the last few days exhibited mere symptoms of slight disorders of innervation in the extremities; in him there was likewise found myelitis (nucleated cells) in the anterior, posterior, and lateral columns; the optic and olfactory nerves were grey, translucent, and atrophied. In about fifty women examined in one day, no case of general paralysis was discovered, and none had atrophy of the papilla. In every case the ophthalmoscopic appearances were confirmed by Dr. Von Græffe, who also had the kindness to give complete descriptions of them. In no case were traces of neuritis diagnosed.

difficulty, if at all, be made out; and afterwards, it is only in rare cases that the nerves in question present any palpable change.

The apoplectiform or epileptiform attacks, already mentioned, are likewise *common phenomena*, which occur both in grey degeneration without mental disturbance, and in the paralytic insane with the various forms of spinal disease. The same remarks are applicable to the occasional sudden and transitory *paralyses* in connection with these attacks.

As we have seen, the morbid process in the spinal cord itself presents different forms on examination after death. It either assumes the appearance of a considerable loss of nerve-substance, and substitution of connective tissue (atrophy, grey degeneration), or this change is more of the nature of a chronic interstitial process with fatty degeneration (spreading of the interstitial connective tissue and formation of nucleated cells, chronic myelitis). In the latter form a contingent loss of nerve-substance is not a prominent feature, and the process cannot well be called atrophy. Concerning the finer changes lying at the foundation of these processes, very little is known. According to one view, there is in atrophy a new formation of connective tissue, owing to which the nerve elements are gradually destroyed. According to another view, there is a primary atrophy of the nerve-substance contained in the nerve-tubes, and the connective tissue which contained the nerve-substance is viewed as a remainder, and its increase only relative. The grounds advanced for both the first and second views are extremely meagre, and look like makeshifts. In reality the mode in which the nerve-elements are destroyed and the atrophy originates, is unknown. The chronic myelitic processes, too, are, I believe, not yet sufficiently elucidated. There, is, however, probably an increase of the connective tissue elements, which become filled with fat, and in part permit the recognition of an increase of nuclei. Both forms, however, grey degeneration and chronic myelitis, in spite of the different anatomical aspects, appear to stand in a certain relation to each other, which, of course is, as yet, not quite clear. The cases of atrophy, in which still greater numbers of nucleated cells are met with, would then present, as it were, transition cases. Add to this also, that in hardened preparations of myelitis, there are occasionally seen limited spots of the character of grey degeneration, and that the so-called *secondary* affections of the spinal cord (through pressure upon it, &c.) resemble sometimes chronic myelitis, sometimes grey degeneration. Nevertheless, how the processes actually proceed, whether and to what extent we may thereby speak of *inflammation*, are questions which will not be clearly elucidated until our present vague notions regarding the processes which form the basis of the so-called chronic inflammation are rendered more clear.

After what has been said, we may now come to the conclusion regarding the relations of the cerebral and spinal affections in general paralysis, that in those patients there exists a certain disposition of

the nervous system, owing to which, according, indeed, to the nature of the unknown influences of causation, sometimes the spinal, at other times the cerebral section of the nervous system, at others again, peripheral cerebral nerves, are attacked by the morbid process, either in succession or simultaneously.

It has already been stated that encephalitic processes have not hitherto been discovered in any part of the brain in this disease. Consequently, therefore, it is, as yet, impossible to form an analogy between the disease of the spinal cord and the nature of the co-existing cerebral affection. On the other hand, a connecting link is seen in the frequent occurrence of chronic meningitis simultaneously in the brain and spinal cord. This is, of course, neither in the one nor in the other, a constant appearance; nevertheless, it is so frequent, that we may consider both processes, the unknown affection in the brain on the one hand, and the chronic myelitis and atrophy of the spinal cord on the other, to be most generally accompanied by chronic meningitis; we cannot, however, regard this as the basis of the malady. Likewise pachymeningitic processes in the dura spinalis and cerebralis are more frequently accompanying phenomena.

In conclusion, the question now arises in how far can we explain the symptoms observed during life by the changes found after death? Bayle has already attempted to explain the psychical phenomena by considering the delirium and the agitation to be dependent upon the irritation exerted by the inflamed meninges upon the cortical substance. Indeed he thus made a quite untenable attempt to explain the delirium of greatness, which, according to him, occurs exclusively in this disease. This explanation given by Bayle—though not in relation to the *subject* of the delirium—remains even to the present time influential in regard to conditions of maniacal excitement, and, as we have seen, has been subsequently adopted, especially by L. Meyer, in a somewhat different form. Others, on the contrary, attach more weight to the alleged inflammation of the cortical substance itself, and Meschede believes that the psychical disorder can be best accounted for by parenchymatous inflammation of the ganglion cells, which he thought he had discovered. This, he considers, progresses in a corresponding ratio to the exacerbations of the mental phenomena in various kinds of delirium; with the destruction of the ganglion cells is destroyed likewise the mental life.

I must express myself most decidedly against these anatomical explanations of the psychical phenomena,—quite independent of the circumstance that the anatomical facts even are not sufficiently founded. It is, in short, thorough trifling. We should, I think, have at the present day, a greater conception of the complexity of the psychical processes than to occupy ourselves with such rude attempts to explain them, and actually believe that in so doing we are advancing science. I openly express my aversion to this, and hope that, in future, observers will occupy themselves more with actual facts than with taxing the

imagination with theories of this kind. In the meantime, we have no knowledge in any way established of the process upon which the psychical symptoms of this disease depend.

It is more to our purpose to inquire regarding the immediate causes of the motory disorders and of the *apoplecti* or *epileptiform* attacks. Bayle attempted to account for the motory disorders by compression of the brain which, in the first two stages of the disease, would be produced by the congestion of the vessels of the pia mater, and in the latter stages, by the serous infiltration of the latter, and the copious serous effusion. Others attached importance to the changes in the cortical substance adherent to the pia mater, while others again would attribute the disorders of movement to softening of the inner parts (corpus callosum, fornix, &c.) All, however, thought exclusively of a cerebral cause.

From our investigations we must first of all, so far as the lower extremities are concerned, draw a distinction between the two varieties of disturbance of motion in walking, already described. That the one variety, constituting the tabic form of gait, is to be traced to spinal (grey degeneration of the posterior column), and not to cerebral causes, no one can longer doubt, especially as the same motory disorder may also occur in patients who have no cerebral (mental) disorder. It, moreover, cannot be denied that it is the degeneration of a great number of nervous elements in the posterior roots and posterior columns which gives rise to this gait, and that whether the fundamental motory disorder be attributed to simple loss of sensibility or not. I will not here enter into a discussion of the question why the disorder has the special character which we have designated tabic.* At all events, grey degeneration of the posterior columns is always found in the insane in whom this gait is characteristically devoloped, although all in whom this anatomical lesion is found did not present the same motory disorder.

More difficult in regard to the purely anatomical cause is that form of gait which we have termed *paralytic*, in which the disturbance is, during the greater part of the course of the disease, more insignificant and less characteristic, so that, frequently, for a long time there is merely a slight degree of awkwardness, clumsiness, and slowness of movement. These are the cases in which the process appears in the form of a chronic myelitis, and no appreciable loss of nerve tubes in the spinal cord and the roots of the nerves can be discovered. One might, on first thoughts, assume that the extent of the motory disorder would here stand in a certain relation to the degree and extent of the myelitic process, which apparently cannot exist without the nerve tubes being somehow affected by it, although no marked disappearance of

* Also in regard to the staggering with shut eyes in these patients, I will only here mention that I do not consider this phenomena as yet satisfactorily explained, but nothing essentially new can be adduced. Most probably it is the degree and nature of the loss of sensibility which plays the most important part.

nerve substance is observed. Yet that there must be a certain compression of the nerve tubes by the growth of the intermediate connective tissue-like substance infiltrated with fat (not to speak of other unknown influences), can scarcely be doubted, and may for the present be assumed in explanation of the hindrance to the movements. Nevertheless it is very difficult to form an estimate of the degree of motory disturbance existing in life from the extent of the changes found after death. There are, as I have shown, many cases of myelitic disease of the lateral and posterior columns without the motory functions having been appreciably involved during life, so that the diagnosis of general paralysis could only be established by the character and the course of the psychical disorder alone or in connection with certain accompanying phenomena, such as apoplectiform attacks, atrophy of the papilla of the optic nerve, &c. In these cases of *latent* disease of the spinal cord, therefore, integrity of the motory apparatus cannot be considered as identical with integrity of the spinal cord. We must, on the contrary, constantly bear in mind that an individual whose psychical symptoms are characteristic of general paralysis may already be suffering under spinal disease, though no objective symptoms are apparent. On the other hand, however, a subjective sensation of slight *lassitude* frequently seems to exist in these cases; this, of course, in well marked mental disorder is most difficult to establish. Why it is that in both the tabic and paralytic forms of the disease, sometimes the most striking improvement almost resembling recovery, and at other times relapses, spontaneously occur, we cannot anatomically explain.

It has already been mentioned that towards the end of the disease the power of movement in the lower extremities (also in the trunk) may be reduced to a minimum, so that while lying in bed the legs can scarcely be voluntarily moved; therefore contractions are apt to set in. This occurs both in grey degeneration of the posterior columns, and myelitic disease of the posterior and lateral columns. The degree and extent of the disease afford no certain explanation; for example, a patient may possess almost complete capability of movement of the extremities, and at the autopsy the same changes (grey degeneration of the posterior columns) might be seen to exist as in a patient who presented the tabic gait and, before death, was totally incapable of voluntary movement of the extremities. Of course it is impossible minutely to determine the amount of the destroyed nerve substance, but in both cases, according to a general estimate, the degree and the extent of the disease are often apparently similar. That the cause of the immobility is here to be sought in an atrophy of the muscular elements, by no means applies to every case.

Whether the frequently occurring muscular tremour, the stronger convulsive tremour, which is sometimes spontaneous, sometimes apparent in complicated movements (especially of the upper extremities), and the involuntary muscular contractions, are to be attributed to direct

irritation of motory nerve tubes, and whether some of these phenomena depend upon a disproportion between the impulses of will and the capability of voluntary contraction of the muscles, remains uncertain. At all events, the same phenomena are observed in analogous affections of the spinal cord without cerebral disease; therefore there is no apparent reason to attribute these phenomena to any other than the spinal disease.

One of the most frequent and most striking symptoms, the disorder of articulation in speech, admits as yet of no interpretation, as neither in the peripheral nor in a portion of the central tract (medulla oblongata, hypoglossus) can any change be discovered. Likewise the frequent cessation and disappearance of the disorder of speech, occasionally with the entrance of maniacal excitement, is incapable of explanation.

In conclusion, we have still a few words to say regarding the *epileptiform* and *apoplectiform attacks* with their accompanying *paralytic phenomena.* Bayle drew a distinction between the two kinds of attacks founded upon their mode of origin, inasmuch as he considered the apoplectiform to be caused by sudden congestions of the pia mater and of the brain, and the epileptiform (also other convulsive phenomena) by the inflammation of the cortical substance (softening of it and adhesion to the pia mater). He bases the latter supposition on the fact that he found inflammation of the cortex in every case in which epileptiform attacks or convulsive phenomena had occurred. This hypothesis is nevertheless refuted by numerous observations, and it is but an arbitrary interpretation of negative facts when Bayle imagines that in these the convulsive appearances merely escaped observation, and must have occurred at some former period, &c.

In more recent times, the apoplectiform, as well as the epileptiform attacks of the paralytic, have been very generally, and in France almost exclusively, referred to congestions towards the brain (medulla oblongata, congestion cérébrale); congestions à forma apoplectique, or à forma convulsive, are spoken of. Indeed, this view of the nature of these attacks has gradually become so general that it scarcely occurs to any one to think of anything else.* Nevertheless, there does not exist a single proof of the correctness of this view, and the circumstance, constantly referred to, of the congested condition of the brain after death is so far from being a fact that often the very opposite condition is met with in individuals who have succumbed to these attacks. In such cases, a certain state of fulness of the veins has generally been confounded with actual arterial hyperæmia. Likewise, the alleged rapid and certain influence of blood letting is not in accordance with facts, as the attacks, which as a rule quickly pass off without interference, are either rendered more persistent by abstraction of blood,

* See, besides, the argument between Bouilland and Trousseau concerning congestion cérébrale apopleticforme in its relations to epilepsy. Bullet. de l'acad. impér. de méd., 1861, XXVI.

or at all events not modified by it. If these phenomena, loss of consciousness, convulsions, &c., are to be referred at all to disorders of circulation, it would be more in accordance with recently discovered physiological facts (experiments of Kussmaul and Tenner) to think of sudden anæmia of certain parts of the brain. This idea, at all events, would not be in direct opposition to pathological anatomical facts. Considering that we have been studying a series of changes in certain columns of the spinal cord, in the medulla oblongata, the pons Varolii, and the crus cerebri, the supposition is not improbable that through a temporary excitation (due to the morbid process) of the vasomotor nerves proceeding from these parts, the anæmias we have mentioned may be produced.* Further speculation on this point, however, can lead to nothing, until further physiological facts have been obtained.

The paralytic phenomena which so frequently succeed these attacks are very remarkable, and most difficult to explain. As we have seen, they are, in the majority of cases, very transient; they affect the face or the tongue, the trunk, and extremities; they have occasionally the character of complete or incomplete hemiplegia, and are sometimes associated with contractions. They can never—or only in rare and exceptional cases—be referred to intermeningeal apoplexies, to effusions of blood in the cerebral substance, or other appreciable occurrences of this nature. It surprises us, after having observed almost complete hemiplegia, to find, when the patient dies after the attack, absolutely nothing in the brain to account for the paralysis; we ought not, under such circumstances, as the uninitiated are so apt to do, to make a diagnosis of intermeningeal apoplexy, hœmorrhagic pachymeningitis, &c.† Baillarger‡ has attempted to explain these hemiplegias on the theory of cerebral congestion. By weighing the brain he makes out that the hemisphere opposite to the paralysed side weighs less than the other—is atrophied, and he supposes that in the sudden congestions (in the attacks) it is always the same hemisphere which is chiefly affected (unilateral congestions), in consequence of which it becomes more atrophied. Moreover, the congestions might also take place, not suddenly, in the form of attacks, but gradually and permanently, and so produce unilateral atrophy without the occurrence of congestive attacks. If we put out of the question the more than problematical

* This reminds us that Burge, from experiments upon rabbits, assumes that the pedunculus cerebri is a central point of excitation for the vasomotor nerves of all the arteries in the body. Centralbl. für die Med. Wissensch, 1864. No. 35.

† When, after attacks with unilateral paralytic phenomena a slight degree of hœmorrhagic pachymeningitis is occasionally found, we ought not to refer the paralysis, without further evidence, to the hœmorrage, which is often very trifling and may be quite accidental. Indeed, when limited to one side, its position often does not correspond with the side affected by the paralysis (that is, it is not on the *opposite* side).

‡ De la cause anatomique de quelques hémiplégies incomplètes. Ann. Méd.-psych, 1858, p. 168.

view, that the presumed congestions lead to atrophy of the cerebral substance, there remains, as the basis of this explanation of the hemiplegias, merely the difference in weight of the two hemispheres. The number of cases adduced by Baillarger in support of this theory amounts to four; in all, he has observed eight; subsequently Baume * added four. Exclusive of other objections which might be raised against these observations, inequality in the weights of the hemispheres (in the numbers, too, given above) is not a condition which is restricted to hemiplegic paralytics, but occurs in individuals who have never presented paralytic symptoms. Thus, amongst others, Follet and Baume find that inequality of the hemispheres is a common condition in epilepsy; Boyd, Wells, † in epilepsy and idiocy; Koster, ‡ in cases of periodical insanity, &c. Baillarger has left his work uncompleted; has he since arrived at other conclusions? The inequality of the hemispheres is evidently dependent upon very various circumstances, and especially upon the relations of the cranium. No proposition can be recognised as a scientific fact which does not take these circumstances into account, nor so long as numbers of individuals present this peculiarity who have no cerebral malady. With these precautions, the pursuit of this investigation may, perhaps, not be without interest, although it must be confessed—having regard to other conditions of loss and atrophy of the hemispheres—the whole theory seems very improbable.

The occurrence of "serous apoplexy," in contradistinction to sanguinous effusion, has been insisted on by the older physicians in explanation of certain cases of death occurring apparently from apoplexy, but in which no satisfactory post mortem appearances can be discovered. This theory has also been extended to the attacks and consequent paralytic phenomena at present under discussion, inasmuch as it was supposed that sudden effusions of serum occur, particularly in the cerebral ventricles, which might occasion the attacks of loss of consciousness with convulsions, and likewise (through pressure) to the subsequent paralytic phenomena. The unilateral character of the latter would be accounted for by the presence of a greater amount of secretion in the ventricle of one side, and the usually rapid disappearance of the symptoms by resorption of the fluid, or accommodation of the brain to its presence. It is difficult to speak positively on this point, as, on the one hand, we have no satisfactory basis on which to found an opinion regarding the standing of the hydrocephalus internus found in those who die in the attacks, and, on the other hand, sometimes such a condition does not exist. I can, however, deny on the

* Baume, De l'inégalité du poids des hémisphèris cérébraus, etc. Ann. Méd.-psych., 1862, p. 540.

† Boyd, Wells. "Observations on the measurement of the Head and the weight of the Brain in 696 Cases of Insanity." Med. Times, Sept. 24th, 1864.

‡ Koster, Untersuchungen über den Einfluss des Mondes auf das periodische Irresein. Allgem. Zeitschr. f. Psych., p. 709.

basis of special observations, that in those cases where there is a greater amount of fluid in the ventricle of one side, there exists a definite relation to unilateral paralytic phenomena, as I have found the side of the body corresponding to the more dilated ventricle paralysed, instead of the opposite side, as would be expected did this relation exist.

We must conclude, therefore, that nothing positive is known concerning the causes either of the persistent, or transient paralytic symptoms of a hemiplegic character. To the *latter* we can indeed adduce certain analogies, as similar transitory paralyses occasionally appear after the attacks in epilepsy, and, moreover, in patients who presented no other symptoms indicative of an apoplectic disease of the brain, and in whom none could be discovered after death. Nevertheless, these cases remain quite as obscure as the former, and, notwithstanding their analogy, present nothing by which they can be explained.

Regarding the *more persistent* hemiplegias or hemipareses, we must, in the first place, look principally to the spinal affection (grey degeneration and chronic myelitis), although it is extremely difficult to establish a standard of degree and extent of change deciding the occurrence of these paralytic phenomena. In particular, in those cases where one half of the body is predominantly affected, we cannot discover any predominant affection of one half of the spinal cord, as, in general, the columns of both sides seem to be equally involved.

But again, it is almost impossible to distinguish with certainty whether there is more disease of one side, as, owing to the extent and complexity of the organ, a perfectly exact investigation can scarcely be made. But even although the persistent unilateral paralytic phenomena could actually be so interpreted, yet the relation which they hold to the apoplectiform and epileptiform attacks must still remain problematical—the paralyses being frequently initiated or markedly aggravated by these attacks.

The foregoing paper affords an explanation of many facts which formerly stood unconnected, and were the occasion of much discussion. I have hitherto left out of the question the disputed points concerning the relative order of succession of the psychical and motory disorders, and other matters connected therewith, particularly the theory of the existence of "General Paralysis without Mental Disturbance," which has been raised in France. These cases, exclusive of cases of apoplexy and muscular atrophy, which do not belong to the same category—refer chiefly to patients with motory disorders similar to those occurring in the paralytic insane, but accompanied by only very slight, and easily overlooked psychical weakness, and very often only by slight weakness of memory, to which, owing to the great preponderence of the motory affection, no importance is attached. It

is evident that in these cases there exist spinal affections analogous to those just described, associated with the first symptoms of mental disturbance.

Another disputed point, viz., whether motory disorders might supervene upon other forms of insanity, more chronic in their course, and which hitherto have not been considered as paralytic, because they are not from the commencement characterised by mental weakness, will furnish a future subject for discussion. I will not, therefore, touch upon it at this time.

I would fain hope that, by directing attention in this paper to the peculiar connection of certain chronic, cerebral, and spinal diseases, and the frequent latency of the course of the latter, I have opened up a new and fruitful sphere of investigation.

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