two conditions well known and referred to in all standard works as signs of great significance—namely, certain disturbances of breathing, especially the peculiar form of breathing known as Cheyne-Stokes respiration, and a rapid rise of temperature. Disturbances of respiration in cases of apoplexy are, I have no doubt, of grave omen. So long as the respiration remains regular, however noisy and stertorous it may be, other things not being unfavourable, I believe the patient may recover, but when irregular breathing sets in, and above all when undoubted Cheyne-Stokes respiration is present, the patient is, I believe, in the greatest danger. I am speaking of course of Cheyne-Stokes respiration along with insensibility; without insensibility it has no serious meaning at the moment.

A certain, always moderate, amount of pyrexia occurs in many cases of cerebral hæmorrhage after the initial depression due to shock has passed away, but a temperature rapidly rising to 104° , 106° , or even 108° is of absolutely fatal significance. Hyperpyrexia is not, I believe, of any great value in showing the locality and extent of the lesion; it indicates probably an extension to, or involvement of, the basal ganglia and pons, and so probably means an invasion of the ventricles.

There are then, as I have tried to show, in my opinion at least, three important prognostic indications to be looked for in any given case of apoplexy due to hæmorrhage into the substance of the hemisphere—namely,

1. Renal disease.
2. Cheyne-Stokes respiration.
3. Hyperpyrexia.

By far the most important of these is renal disease.

If, in conclusion, I may state my opinion in the form of a rule for prognosis, I would say this. In any case of apoplexy due to hæmorrhage into the hemisphere, if renal disease, Cheyne-Stokes respiration, or hyperpyrexia, any one, two, or all three be present, the patient will in all probability not recover. If no one of these is present, and does not make its appearance, he may, and probably will, recover, however long insensibility may last and however deep it may be.

The presence of other serious conditions, such as diabetes, chronic alcoholism, typhoid fever, idiopathic anæmia, will, I have no doubt—for I have seen examples to this effect—exert just as fatal an influence as renal disease upon the course of sanguineous apoplexy.

THE VALUE OF EXPERIMENTAL EVIDENCE IN THE DIAGNOSIS OF DISEASES OF THE CEREBELLUM.

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A REVIEW of the literature in connection with diseases of the cerebellum makes it evident that considerable confusion exists with regard to the symptoms on which we are to depend for the diagnosis of such morbid processes, and for their localisation in one rather than another part of the organ. Much of this confusion appears to be due to the following causes: (1) The discrepancies which exist in the results obtained by different experimental physiologists who have investigated the functions of the cerebellum. (2) A disregard on the part of the clinical observers, of experimental evidence bearing on the question. (3) Faulty attempts to apply experimental evidence in the elucidation of clinical phenomena. (4) A preconceived idea that such and such symptoms cannot be directly due to a lesion of the cerebellum, and that therefore some other explanation for their occurrence must be sought for.

THE ABSENCE OF CHARACTERISTIC SYMPTOMS.

One of the first points which calls for our attention is the fact that extensive disease of the cerebellum may exist in man with next to no clinical evidence pointing to the existence of any morbid condition of this part of the central nervous system. We find it at first difficult to reconcile this with the fact that in animals ablation experiments on the cerebellum result in most profound disturbances of function,

even when comparatively small portions of the organ have been removed. And yet nothing is more simple than the explanation of what at first appears to be so irreconcilable a discrepancy. It is perfectly true that the most pronounced disturbances of function result when a part or the whole of the cerebellum is removed; but it must not be forgotten that recovery takes place with marvellous rapidity, each disordered function being in turn compensated for with such exactitude that no one unfamiliar with this would believe that an animal presenting all the disorders of function observable directly after the operation could recover to the extent that it does even in the course of a month. Now, if after removal of a large part of the organ such complete restoration of function can be brought about by compensation through other parts of the central nervous system, what is more likely than that the slow processes of disease, destroying the substance of the cerebellum only gradually should produce no symptoms as a consequence of the loss of function of the part destroyed, owing to the fact that compensation goes on *pari passu* with destruction, the latter being so slow as to allow the former to keep pace with it, and thus no recognisable evidence of the destructive process in the cerebellum is forthcoming?

We need not look to the experimental side of the question alone for evidence in support of the above proposition, for in morbid processes of the cerebellum occupying precisely the same situation in the organ we meet with the most varying symptoms and all degrees of the same symptom. I am convinced that these various differences depend not so much on the extent of the morbid process as on the rate of its development; the more sudden its onset and rapid its development, the more pronounced are the evidences of disordered function, the more gradual its onset and slow its progress the more likelihood is there to be only slight evidence of disordered function or a complete absence of such. In support of this contention I would adduce the following three considerations: 1. The most sudden of all lesions of the cerebellum in man is a hæmorrhage into some part of its substance, an event which, as we are all aware, is attended by disorders of function of a pronounced character. 2. Abscess of the cerebellum may be of rapid or slow development, and correspondingly we find such cases presenting the most varying evidences of disordered function from pronounced to trivial or completely absent. Of course it is, with some exceptions, almost impossible to fix the exact time that the morbid process has been in existence, so that these cases are only of value in supporting the argument when taken in conjunction with cases of the first and third group. 3. Tumour cases more than any others show how trivial the evidence of disease of the cerebellum may be in conjunction with the most gross changes within that organ. It is no uncommon experience to find extensive new growth in the cerebellum with an almost total absence of symptoms which we are in the habit of associating with tumour in that part of the central nervous system. In this statement I of course exclude such general symptoms as headache, vomiting, and optic neuritis—symptoms common to intracranial tumour in the most varied situations. With this—the most slowly progressing of morbid processes to which the cerebellum is prone—we most commonly meet with an absence of or only trivial indications of disorders of function peculiar to the organ under consideration.

Thus, while with the most sudden of lesions of the cerebellum (hæmorrhage) we meet with the most pronounced symptoms of disordered function, with that of slowest development (tumour), we find least evidence of a similar nature, and with a process which may be rapid or slow (abscess) we meet with variations in symptoms which justify our attributing their variations, in great measure, to the rate at which the morbid process is developed.

There is one other factor which has to be taken into account, more especially in connection with tumours of the cerebellum, and that is, that the morbid process may displace without actually destroying the cerebellar tissue, so that unless the process of extension is rapid the organ is able to accommodate itself to the altered pressure relations for a considerable time.

Just as all symptoms characteristic of disease of the cerebellum may be absent, so it often happens that we have

to depend on one or two symptoms alone for diagnosis. The typical picture, comprising all the various phenomena which may be associated with disease in this part is of exceptional occurrence, so that in any given case we must be prepared to depend on the detection and correct interpretation of one or two phenomena to aid our diagnosis, rather than refrain from diagnosis because the typical picture is not before us. It will, therefore, be instructive to consider separately each symptom in which experimental evidence aids us, and on which we may have to depend largely for the correct localisation of disease in the cerebellum.

INCO-ORDINATION.

Probably no symptom is more closely associated with cerebellar disease than co-ordination. This is not the occasion to discuss the nature and mode of production of this phenomenon; it is sufficient for our present purpose that we recognise that it occurs, and attempt to diagnose the seat of the disease in the cerebellum from a consideration of the characters of the inco-ordination. It may be said at once that experimental, and, in the main, clinical evidence, are at one in ascribing to lesions of the front part of the vermis or middle lobe of the cerebellum instability of such a nature that the tendency is to fall forward, whereas when the posterior part of this lobe is affected, the tendency is for the animal or human subject to fall backward. And yet, that the direction of the fall cannot be relied on absolutely as indicating the precise locality of the morbid process in this lobe in the human subject is made evident by such cases as that recorded by Handford,¹ in which the patient tended to fall backward, and yet the whole of the middle lobe of the cerebellum was destroyed by a sarcoma.

When we turn to consider lesions of the lateral lobes of the cerebellum we find a great want of unanimity of opinion; experimental physiologists and clinical observers are alike at variance as to the direction in which the animal or human subject reels or falls when one or other lateral lobe of the organ is affected. According to one class of observers, in destructive lesions of one lateral lobe the tendency is for the subject to rotate, reel, or fall to the opposite side from the lesion; whereas the other class of observers hold that the instability results in a rotation, reel, or fall to the same side as the lesion. For my own part I have always adopted the former view, and I cannot help thinking that we all mean the same thing, but express ourselves differently. In discussing the subject with Dr. Beevor recently, he made a suggestion which appears to me calculated to clear up a good deal of the confusion which at present exists with regard to this subject. Dr. Beevor's suggestion was that we should describe the direction of rotation as compared to a corkscrew as it is turned in going into the cork of a bottle, or in coming out of one.

Now according to my observations, which lead me to the conclusion that the animal rotates away from the side of the lesion; if the lesion happened to be in the right lateral lobe of the cerebellum the animal would rotate like a screw going into a bottle, whereas if of the left lateral lobe, then like a screw coming out of one. To make my meaning clearer, let us suppose that a man with disease of the right lateral lobe of the cerebellum is placed to stand facing the observer, he would tend to rotate in such a manner that his left side would next present itself to the observer, then his back, then his right side, and then his face once more; whereas a man with his left lateral lobe affected, similarly placed, would rotate in such a manner that his right side would first present itself to the observer, then his back, then his left side, and then his face once more.

In making the above remarks with regard to the direction of rotation, it must be clearly understood that I refer to the objective phenomena of reeling or rotation, and not to any subjective sensations experienced by the individual, for in experiments on animals it is of course on the objective phenomena associated with inco-ordination that we have solely to depend. Many of the records of clinical cases are wanting in this respect, inasmuch as it is not definitely stated whether the patient actually fell or tended to fall in a certain direction, or whether there was only a subjective sensation of falling in a given direction. Then again a point which appears to be often lost sight of by clinical observers

is the fact that implication of the auditory nerve, by pressure or otherwise, may give rise to inco-ordination. Now in animals² irritation or intracranial section of the auditory nerve results in rotation or reeling to the same side as the lesion, so that in the absence of middle-ear disease, deafness, or other auditory phenomena on one side, are of great importance quite apart from the evidence with which they supply us that the lesion of the cerebellum is probably situated on that side, for if neglected by those who consider that in an affection of one lateral lobe of the cerebellum the patient falls to the opposite side, a faulty localisation of the lesion would result if staggering, in reality due to implication of the auditory nerve, were attributed to the cerebellum.

It must further be remembered that, inasmuch as there may be motor paresis of the limbs on one side, the patient may, in such cases, tend to fall to that side in consequence of the inability of the lower extremity to support the weight of the body, and therefore this condition of things must be carefully distinguished from the instability which has already been discussed. The distinction is important, for, as we shall presently see, paresis of one side of the body may result as a direct consequence of a unilateral lesion of the cerebellum, in which case the paresis is on the same side as the cerebellar lesion, and the direction in which the patient would fall in consequence of failure of the lower extremity to support the weight of the body would be to the same side as the lesion, while, as I have already urged, the direction of the fall consequent on loss of balance associated with rotation or reeling, would be to the opposite side, that is, away from the side of the cerebellar lesion.

MOTOR PARESIS.

This leads me to speak of a symptom of cerebellar lesions to which little attention has been paid, and which has not been hitherto recognised as consequent on the cerebellar defect; I allude to motor paresis involving the same side of the body as the lesion of the cerebellum.

It has, of course, long been recognised that tumour or abscess involving one side of the cerebellum may, by pressure exerted on the pyramidal tract of the same side, occasion paresis of the opposite side of the body in consequence of the pressure being exerted on the pyramidal tract above its decussation. But the cases recorded in which there has been paresis on the same side of the body as that on which the cerebellar lesion has been found *post mortem* have met with no satisfactory explanation. In fact, where an attempt has been made to localise the exact seat of the lesion during life it has resulted in error, inasmuch as a lesion of the opposite side of the cerebellum pressing on the pyramidal tract has been diagnosed, or a lesion of the opposite cerebral hemisphere has been looked for as the probable cause of the motor paresis, which was in reality due to a destructive lesion of the side of the cerebellum corresponding to the paresis.³

My own observations on animals⁴ leave no doubt in my mind that such paresis on the same side as a lesion of the cerebellum is a direct consequence of the cerebellar defect, and is in no way indirectly induced by pressure on other parts. The precise way in which the cerebellar defect induces this paresis does not so much concern us in a paper of this kind as does the practical question whether motor paresis ever occurs on the same side as a unilateral lesion of the cerebellum. That it does is made evident by the results of my experiments, which are further supported by clinical and pathological facts that have been placed on record, and also by others which have come under my own observation. Among recorded cases of the kind one by Ogle⁵ is especially interesting because a cyst the size of a walnut occupied the region of the corpus dentatum on the left side of the cerebellum, and there was loss of power in the left arm and leg during life.⁶ When, therefore a patient presents, in conjunction with other symptoms usually associated with disease of the cerebellum, paresis on one side of the body, this sym-

² Phil. Trans. Roy. Soc., 1894.

³ Cf. Drummond, *Lancet*, July 28th, 1894.

⁴ Loc. cit.

⁵ Brit. and Foreign Medico-Chir. Rev., 1865, vol. xxxvi, p. 233.

⁶ Such loss of power on the same side as a unilateral lesion of the cerebellum has been sometimes erroneously ascribed to pressure on the pyramidal tract below the decussation.

¹ Brain, 1892, vol. xv, p. 458.

ptom' properly interpreted becomes a localising sign of the highest significance. It is only when taken in conjunction with other evidences of disease of the cerebellum that we can hope to obtain any useful information from this symptom, however, inasmuch as it may signify a lesion of the same side of the cerebellum, or, if occasioned by pressure on the pyramid, then of the opposite side of the organ. Cases of the latter class are generally accepted, and call for no illustration, but it is otherwise with the former class, of which the following is a good example:

At a recent meeting of the Neurological Society of London, Dr. Acland and Mr. Ballance showed a case of right-sided cerebellar abscess which had been successfully operated on by Mr. Ballance. Before the operation this patient had, in addition to other signs of cerebellar disease, paresis of the right arm and leg, and conjugate turning of the eyes to the left. Now no other lesion of the cerebellum but one of the right lateral half of the organ could be responsible for this combination of symptoms, for, had the cerebellar lesion been on the left side, causing the motor paresis on the right side by pressure on the pyramid, the eyes, if deviated, would have been directed to the right and not to the left.

Another instructive case in this connection is one recorded by Vulpian,⁷ in which a tuberculous tumour of the right side of the cerebellum was responsible for paresis of the right extremities together with turning of the eyes to the left.

There is one point of difference between animals (more especially dogs) and man, and that is that in the former the hind limb on the side of the lesion appears to be the most paresed, while in the latter, when any details have been given in the record, it is the upper limb which has been noted as chiefly paresed, the lower limb being less so, or apparently escaping in some cases.⁸ Instances are not wanting, however, in which the paresis has involved both inferior extremities alone in man, and that even in cases of unilateral lesion of the cerebellum.⁹ Similar cases have also been recorded in which a tumour growing from the inferior surface of the middle lobe has exerted pressure on the medulla, and in which the movements of the superior extremities have been perfectly preserved.¹⁰

Before passing from the subject of motor paresis I must refer briefly to the fact that Dr. Hughlings Jackson has long maintained that in affections of the middle lobe of the cerebellum weakness of the spinal muscles results—an opinion which was also held by Niemeyer.¹¹ It is not easy to judge of the condition of the spinal muscles in most animals, but I found that monkeys deprived of the whole cerebellum were unable to sit up,¹² a condition probably depending on weakness of the back muscles. A year ago I had the privilege of recording a case, in conjunction with Dr. Hughlings Jackson, in which there was undoubted evidence of weakness of the spinal muscles in a patient the subject of a cyst of the cerebellum which involved its middle lobe chiefly.¹³

OCULAR DEVIATIONS.

Closely associated with this question of motor paresis of the trunk and limbs is that of the displacement of the eyes, which I believe to be of a paralytic nature also. Formerly the abnormal positions of the globes were attributed to overaction of certain of the eye muscles, that is, they were classed among the so-called irritation phenomena of cerebellar lesions, in which category they have been included even by Luciani¹⁴ in his comparatively recent work on the cerebellum. I have contended that these abnormal positions of the eyes are due, not to overaction of any muscles, but to want of action of certain of them. In other words, that they ought to be classed with the paralytic and not with the irritation phenomena of cerebellar lesions, in support of which contention I have adduced arguments elsewhere.¹⁵

Experimental and clinical evidence point to turning of the eyes from the side of the lesion, as the abnormal position

which is met with in an uncomplicated unilateral lesion of the cerebellum. Among other cases illustrating this is one of cerebellar abscess, successfully operated on, which has been recently recorded.¹⁶ Luciani¹⁷ found that both eyes turned to the opposite side as a result of ablation of one lateral lobe in animals, while, according to my observations, it is the opposite eye which is chiefly displaced in that direction.¹⁸ In a certain number of cases which have been published convergent strabismus has been noted in which the eye on the opposite side from the lesions has been the more displaced, or in which it was alone displaced.¹⁹ As far as I have been able to ascertain, however, the disease has not been limited to the lateral lobe in such cases, but has extended up to the middle line, or even across to the opposite lateral lobe. But we have another point to consider with regard to these cases and that is generally recognised that, from the length of their intracranial course, the sixth nerves are peculiarly liable to suffer in cases of increased intracranial pressure, so that even where no obvious changes have been detected in these nerves, the pressure may have been sufficient to interrupt the transmission of impressions along them, which would account for the convergent strabismus met with, an explanation especially applicable when both external recti are weak.

It is my belief then that the displacement of the globes, which depend on an uncomplicated defect of one lateral lobe of the cerebellum in man, is that in which both eyes are turned to the opposite side, but instead of the eyes being turned to one side, they may appear to be normal until the patient is asked to look to the side of the lesion, when it is found that such a patient is unable to turn the eyes in that direction, or that the resulting movement is very feeble, and that there is a difficulty in keeping the eyes turned to that side.

Ocular displacements differing from those just described probably indicate defect of the middle lobe if they are directly due to the cerebellar disease, and not secondarily induced by pressure on adjoining parts or extension of the morbid process to these. When there is any evidence of pressure on the auditory nerve it ought to be remembered that this in itself is capable of producing ocular displacement and nystagmus.

NYSTAGMUS.

Nystagmus commonly occurs in association with ocular displacements, the result of weakness of the external muscles of the eyes, or it may occur apart from any such displacement. The experimental evidence points to lateral nystagmus, with the jerks from the opposite side towards the side of the lesion, as being that met with in lesions of one or other lateral lobe, and clinical evidence supports this. My experimental results lead me to conclude that any irregular or rotatory nystagmus ought to direct our attention to the middle lobe as the probable seat of the disease, and clinically I have not met with a case in which nystagmus of this nature, depending directly on the cerebellar defect, was present, in which the middle lobe was not involved, whether the lateral lobes were also involved or not. In these statements I, of course, exclude all cases in which the nystagmus is not a direct result of the cerebellar defect, but is secondarily induced by pressure, etc., on other parts—for example, the auditory nerve.

THE ATTITUDE.

In animals there is a curious rotation of the neck, so that the side of the face corresponding to the lesion is turned upwards, and at the same time the chin is directed to the affected side. So too the trunk is curved with the concavity to the side of the lesion. Similar twisting of the neck with retraction of the head and curving of the trunk with the concavity on the side of the cerebellar lesion has been met with in man, especially in cases of abscess.²⁰ These conditions no

¹⁶ Winter and Deanesly, *Lancet*, December 8th, 1894.

¹⁷ *Loc. cit.*

¹⁸ It is necessary for me to call attention to the fact that I observed "skew deviation" only when the animals were under the influence of the anæsthetic, as I have evidently been misunderstood on this point. Reliance must only be placed on the effects observed after the influence of the anæsthetic has passed off when comparing the effects in animals with those resulting from disease in man.

¹⁹ Cf. Hadden, *Trans. Path. Soc.*, 1890, xli, 17.

²⁰ Cf. Ballance, *Clinical Journal*, 1893.

⁷ *Compt. Rend. Soc. Biol.*, 1861, iii, p. 29.

⁸ Cf. Macewen, *Pyogenic Infective Diseases of the Brain and Spinal Cord*.

⁹ Cf. Davidson, *Med. Times and Gaz.*, 1878, ii, p. 218.

¹⁰ Cf. Poole, *Lancet*, 1891, ii, 664.

¹¹ *A Textbook of Practical Medicine*, 1876 (Transl.), ii, 244.

¹² *Loc. cit.*

¹³ *BRITISH MEDICAL JOURNAL*, February 24th, 1894.

¹⁴ Il Cervelletto; *Nuov. Stud. d. Fisiot. Norm. e Patol.*, Firenze, 1891.

¹⁵ *Loc. cit.*

doubt depend on spasm of the muscles on the side of the lesion, of which we have next to speak in connection with muscular rigidity in the limbs.

MUSCULAR RIGIDITY.

Muscular rigidity is most marked in the limbs on the side of the lesion,²¹ and especially in the fore limb,²² which is rigidly extended in animals. In the human subject such rigidity has been noted chiefly in cases of hæmorrhage, and also in cases of abscess. When it exists on one side only, it deserves careful consideration in conjunction with the other evidences of unilateral disease of the cerebellum, for it may be either the direct result of the cerebellar defect, or it may be consequent on the morbid condition in the cerebellum exerting pressure on the pyramidal tract above the decussation on one side, and thus causing rigidity in the muscles of the opposite side of the body. So that, like motor paresis, this symptom is only of value in diagnosis when taken in association with others, inasmuch as alone it may indicate disease of either side of the cerebellum according as we interpret it as due directly to the cerebellar defect, or as the result of pressure on the pyramidal tract above the decussation.

CONVULSIONS.

Closely associated with this last symptom is a form of convulsion which has been observed to affect the limbs on the same side as a lesion of the cerebellum.²³ As a rule, the convulsions resemble tetanus seizures rather than those which result from discharges having their origin in the cerebral cortex.

I have not myself made any observations with regard to excitation of the cerebellum in animals; but the results obtained by Ferrier²⁴ are in accordance with the condition of things occasionally met with in man, for this observer found that movements of a sudden and spasmodic character occurred in the limbs on the same side as the hemisphere of the cerebellum excited. Of course, to both muscular rigidity and convulsion of supposed cerebellar origin it has been objected, and fairly so, that these phenomena may result from pressure on the pons, and where both sides of the body are involved, either in rigidity or convulsion, it must be admitted that there is no satisfactory argument to adduce against this view. The same may be said, and more, when the rigidity or convulsions involve the opposite side of the body to that on which the lesion is situated. Under such circumstances there can be little doubt that these symptoms are consequent on pressure on the side of the pons corresponding to the cerebellar lesion. But it is otherwise when, with a lesion limited to one side of the cerebellum, we meet with rigidity or convulsions involving the muscles of the same side of the body as that on which the cerebellar defect exists. In such cases I am convinced that these phenomena are directly due to the cerebellar defect, and that they are not in any way indirectly induced by pressure on adjacent structures, which could only result in the symptoms being general, or, if unilateral, then limited to the opposite side of the body.

THE TENDON REFLEXES.

The tendon reflexes present curious differences according as they are observed in animals after experimental destruction of portions of the cerebellum, or in man where morbid processes are responsible for the alterations met with. In animals exaltation of the tendon reflexes is the rule, for, with the exception of the fact that immediately after the operation of removal of the lateral half of the cerebellum the opposite knee jerk is greatly diminished if not abolished, they are always exaggerated. The knee jerk on the same side as the lesion is greatly increased in activity immediately after the operation, after which it gradually becomes less so, but even three months after it is still brisker than normal, and the opposite knee-jerk, so much diminished at first, becomes very active also, in fact, almost as much so as its fellow; but at a remote period while it is still more active than normal, that on the side of the lesion can be demonstrated to be a little more active than it is.²⁵ This last characteristic was well

shown in a case of cerebellar abscess, successfully operated on by Mr. Dean,²⁶ and shown at a recent meeting of the Neurological Society of London. In this case, although the abscess formerly existed on the right side of the cerebellum, both knee-jerks were exceedingly active two years and a half after the operation, and Mr. Dean was unable to say which was the more so.

While exaggeration of the knee-jerks occurs in cerebellar disease in man, it is quite as common an experience to find them diminished or abolished; and beyond stating that in those cases in which there is evidence of pressure on the pyramidal tract, causing paralysis, the knee jerk on the paralysed side is exaggerated, it cannot be said that we have any satisfactory clinical data to rely on in attempting to come to a correct conclusion as to what significance exaltation or diminution of the knee-jerks has in conjunction with cerebellar disease. All that can be said is that the experimental evidence points to exaltation of the knee-jerk more marked on one side as being the result of ablation of the side of the cerebellum on which the knee jerk is the more active; whereas any marked diminution is to be associated with the opposite side. In either case these statements are only true when the altered condition of the reflexes is directly dependent on some cerebellar defect, and not indirectly induced by pressure on the pyramidal tract.

ANÆSTHESIA.

Anæsthesia is a symptom which, so far as I am aware, has not been met with clinically in any case in which it could be said to be directly due to a lesion of the cerebellum, whereas I have met with it in animals²⁷ after ablation of one lateral half of the organ, in which cases it has, like the motor paresis, involved the limbs on the same side of the body chiefly, the opposite posterior extremity being only slightly involved, and the anterior escaping altogether. So transitory is this symptom in animals, however, that it is not surprising to me that it is absent in cases of cerebellar disease in man, where, as I have already urged, the processes of compensation go on *pari passu* with the slowly destructive processes of disease.

The only lesions of the cerebellum in which we might reasonably expect to find this symptom present, apart from pressure on sensory tracts, are possibly abscesses of rapid formation, and hæmorrhage producing extensive destruction of the cerebellum. Now in both these classes of cases the probabilities are that the rapid or sudden onset of a lesion at all extensive would be attended with loss of consciousness, so that the patient would be unable to give us the necessary information; and probably by the time that consciousness is recovered the symptom, if even it existed previously, would have disappeared. Nevertheless that such cases are worthy of being carefully tested with regard to the perception of sensory stimuli is evident from what has already been said. Like many of the other evidences of cerebellar disease that have been dealt with in this paper, it is only when carefully considered in conjunction with other symptoms that anæsthesia, if present, can be expected to furnish us with any reasonable guide as to the probable seat of the disease in the cerebellum; or, what is practically the same, help us to decide whether the anæsthesia is a direct result of the cerebellar defect, or whether it is only a secondary result consequent on pressure on sensory tracts.

CONCLUDING REMARKS.

Throughout this paper I have attempted, as far as possible, to limit myself to the consideration of those clinical phenomena with regard to which we have experimental data for comparison. In so doing many important clinical aids to the localisation of cerebellar disease have not been discussed, as the space at my disposal would not allow of this. Besides which, the chief object of the paper has been to show how experimental results may aid us in the diagnosis and localisation of cerebellar disease in man, to call attention to certain phenomena not generally recognised as the direct results of defect in the cerebellum, and to offer a different explanation for the occurrence of some symptoms to that which has been formerly accepted.

²¹ Cf. Drummond, *loc. cit.*

²² Cf. Macewen, *loc. cit.*

²³ Cf. Drummond, *loc. cit.*

²⁴ Ferrier, *The Functions of the Brain*, second edition, p. 180.

²⁵ *Proc. Roy. Soc.*, 1893.

²⁶ Dean, *Lancet*, July 30th, 1892.

²⁷ *Phil. Trans. Roy. Soc.*, 1894.