Gentlemen,—In assuming the office of President for the ensuing session, I desire, in the first place, to express my high appreciation of the distinguished honour, and to say that it will be my endeavour to promote to the utmost the best interests of the Society, and to maintain, with your help and co-operation, the high standard which the discussions and communications to the Society have attained.

It seems appropriate that on this occasion, the opening day of a new session, we should, as a branch of the universal brotherhood of science, pay some tribute to the memory of one recently passed away, whom we all, without exception, acknowledge to have been one of the greatest masters of our science in this or any age—the late Professor Charcot. It is altogether unnecessary that I should, before such an audience, attempt to recount the services which Professor Charcot has rendered to Neurological science. There is scarcely a subject

1 Being the Presidential Address to the Neurological Society, January 25, 1894.
which he has not touched, and which he has not likewise adorned. His death, ripe in years and wisdom, and in the full enjoyment of his splendid reputation, has deprived the world of one whose place can never be filled, and our Society of one of the most illustrious of its small and select list of honorary members. Professor Charcot took much interest in our Society, in which he had so many personal friends, and on the last occasion when I saw him, shortly before his death, he promised to be present and take an active part at one of our meetings during the ensuing session. This, alas! to our grievous disappointment, was not to be. During the last year the Society has also lost in Dr. James Anderson, one of its office-bearers, esteemed alike for his qualities both of head and heart, and just as he was beginning to reap the fruits of the solid reputation he had established for careful and accurate clinical work; and in Dr. Walter Hadden, a neuro-pathologist of brilliant promise, an active participator in the discussions of the Society, and frequent contributor to its journal.

In inaugurating the work of a new session, it has been the custom of my predecessors to introduce some question of more or less present interest in Neurology in one or other of its various aspects. In accordance with this custom, which I consider well worthy of imitation, I have chosen as the chief subject of my discourse this evening one which, though old and worn, one would think, threadbare, is yet ever new and apparently as far as ever from being exhausted: to wit, the functional and anatomical relations of the cerebellum.

These have been practically reconstructed by the recent work of Luciani and Marchi, and it is more particularly in reference to the views expounded by these enquirers, that I wish to engage your attention and consideration for a brief space. I have selected this topic from others that offered themselves, for the special reason that it has been my latest study, and has formed during the last two years a joint research with my colleague Dr. Turner. (Proc. Roy. Soc., Dec. 14, 1893.)

1 "Il Cervelloto," Firenze, 1891; German Trans., Kleinhirn, 1893.
Luciani is the first who has, by skilfully contrived and well-executed experiments, succeeded in studying for long periods of time the symptoms consequent on complete or partial removal of the cerebellum in the higher animals; viz., dogs and monkeys; whereas, with the exception of fowls and pigeons, most previous experimenters had limited their observations to the more immediate effects of such lesions. But these, as the researches of recent years have more and more strongly emphasized, are of themselves inadequate data, and may even lead to erroneous conceptions as to the true functional relationships of any particular organ or nerve centre. The importance of Luciani's work cannot be overestimated, and his analysis and synthesis are worthy of the most careful consideration.

The immediate effects of cerebellar lesion, as described by Luciani, and amply confirmed by our own experiments, do not differ materially from those which are familiar to everyone in the classical descriptions of Flourens. The stationary phenomena, that is, the phenomena of deficiency, are those which are particularly illustrated by abundant richness of experimental detail. By overwhelming proofs, if further proof were necessary, he has disposed finally of the supposed relation of the cerebellum to the sexual instinct. Indeed, as he himself says: "In the history of my experimental animals, there were so many love episodes, of such a wanton and grotesque character that one might really ask whether the loss of the cerebellum was not the cause, perhaps indirectly, of an abnormal degree of licentiousness; and these love relationships were not without their natural consequences. On the contrary, pregnancies, lyings-in, and parturitions followed each other so frequently that my laboratory appeared to have been converted into a maternity hospital."

There is no proof that the cerebellum has any share, or takes any part in psychical manifestations. The total removal of the cerebellum causes no discoverable impairment of any of the special senses, or of any of the forms of cutaneous or of muscular sensibility. In this respect our experiments are entirely in harmony with his.
Luciani has also furnished additional and conclusive evidence to show that the influence of the cerebellum is direct, and not crossed, and that each half of the cerebellum exerts its influence mainly, if not exclusively, on the corresponding side of the body.

His experiments, with which our own entirely agree, have summarily disposed of the theory of Nothnagel that to the middle lobe alone belong those functions which are commonly ascribed to the cerebellum, and that on lesion of this lobe, directly or indirectly, the symptoms of cerebellar disease exclusively depend. This view, which has always appeared to me a very strained reading of the facts of disease, in accordance with a preconceived notion founded on imperfect data, has been shown by Luciani's experiments, as well as our own, to be without foundation; for the effects of removal of the middle lobe are not more pronounced, and are not more enduring than those which follow extirpation of the lateral lobe.

When the cerebellum has been totally removed from monkeys—an operation which we have found to be a most formidable one, and more frequently fatal than successful—the animals exhibit the most tumultuous disorders of equilibrium, so that station and locomotion are, for the time, altogether impossible. Gradually, however, these tumultuous disorders subside to such an extent that the animal is able to sit up, with the help of some support to which it clings, and ultimately regains such a degree of stability that it may dispense with extraneous aid, but is so tottery that it falls over on the slightest disturbance or excitement. Its gait is of a peculiar sprawling character; at first, barely raising its abdominal surface from the ground, and planting its limbs wide apart so as to increase the basis of support; these being in progression raised and set down in a brusque and characteristically ungraduated fashion, which character the movements of the limbs retain for an indefinite period. Notwithstanding this extraordinary unsteadiness and instability of equilibrium, the animals are, however, able to grasp tenaciously with both hands and feet, so much so that it is sometimes more easy to drag the chair to which
they may cling than loosen their grip; and they are able to climb a rope with agility hand-over-hand, in no way differing in this respect from perfectly normal animals. But the most noteworthy and persistent feature is the remarkable astasia or unsteadiness of the head, trunk, and limbs, which are either agitated by constant fine tremors, apart from obvious muscular exertion, or exhibit oscillations of the typical disseminated sclerosis type on volitional exertion; so that, for instance, an attempt to lay hold of a piece of fruit excites such wild oscillations of the hand and arm that the object aimed at is either knocked away, or, if seized, after many unsuccessful efforts, is with the utmost difficulty brought up and held to the mouth. This instability is a persistent feature, and, though lessening to some extent, never entirely disappears, and was clearly evident in one of Luciani's monkeys a whole year after the operation.

If only the lateral lobe, or one half of the cerebellum, is removed, the persistent symptoms, after the cessation of the primary disorders, namely, the sprawling, ungraded action of the limbs, and the tremors or instability on volitional effort, are confined to the same side as the lesion.

If, on the other hand, the middle lobe is destroyed, or extensively injured, as by antero-posterior division, the symptoms are essentially of the same character as those which follow destruction of the whole organ, but they do not affect one side more than the other, and, according to our observations, are more pronounced in the head and trunk than in the limbs. They also appear to be less persistent, and, in the course of a few months, pass off to such an extent that, except on careful examination, it would be difficult to distinguish an animal so operated upon among its normal companions.

In the course of our investigations, Dr. Turner and I have succeeded in dividing the individual peduncles of the cerebellum with comparatively little injury to neighbouring structures at all calculated to complicate the symptomatology. When the primary disturbances, which I will allude to subsequently, have passed off, the persistent phenomena—whichever peduncle is cut—are essentially of
the same character as those following destruction of the lateral lobe, and are confined to the limbs on the side of lesion.

Such is a brief outline of the symptoms, temporary and permanent, following total or partial extirpation of the cerebellum and section of its peduncles. The question is, how are these to be explained in accordance with the anatomical connexions and relationships of the cerebellum? In answer to which I fear that, notwithstanding the valuable researches of Luciani, the opinion of Vulpian that "the problem of the functions of the cerebellum is still far from being definitely solved" is not inapplicable to the present state of our knowledge. Luciani's views are, briefly, as follows:

The cerebellum, with its annexes, constitutes a more or less independent system. It is not an organ interpolated in the course of the cerebro-spinal tracts, but is an "end-organ," or, so to speak, an appendage to the cerebro-spinal axis, directly or indirectly related to certain peripherical sensory organs, and in direct efferent relationship with certain ganglia of the cerebro-spinal axis, and indirectly with the motor apparatus in general. It is functionally homogeneous, each part exercising the functions of the whole, but having special relation to the muscles on the corresponding side of the body. The symptoms following its destruction are, in the first place, irritative phenomena, opisthotonic or pleurothotonic in character, according as the lesion is general or unilateral, together with rotation round the longitudinal axis, and squinting, or deviation, of the optic axis; secondly, ataxic phenomena, or phenomena of deficiency (Ausfallserscheinungen), which are more or less compensated by volitional effort on the part of the animal (functional compensation), or by such parts of the organ as have escaped destruction (organic compensation). The phenomena of deficiency, or cerebellar ataxy, are ascribed by him to three principal conditions, which he has a special fondness for reiterating; to wit, asthenia, atonia, and astasia, due to the loss of a sthenic tonic, and static influence, which, according to him, the cerebellum normally exerts on the apparatus of
movement, and which is the essence of its function. In addition to this, he ascribes to the cerebellum a trophic function, direct on the efferent tracts which spring from it, and which, therefore, degenerate when the cerebellum is destroyed, and indirect on the nutrition of the body generally.

I should like, with your permission, to examine some of these views a little more in detail.

Touching the so-called irritative nature of the primary disturbances (Reizerscheinungen), which Luciani ascribes to inflammatory action set up by the lesion ("Kleinhirn," p. 151), I am persuaded that this is erroneous, and would prefer with Goltz to term them dynamic, or Hemmungserscheinungen, without attempting to offer a more exact explanation of the mode in which they are brought about. They certainly occur under conditions which entirely exclude inflammatory action, and under methods which, when applied to the cerebral cortex, cause paralytic and not irritative symptoms; and, moreover, there is reason to believe that the phenomena are entirely reversed when the conditions are such as to excite true vital irritation or inflammatory action. The so-called irritative phenomena occur, according to Luciani, more especially when the peduncles are injured, and this is, on the whole true; for, according to our experiments, rotation round the longitudinal axis certainly occurs more constantly when the peduncles are divided than when the lateral lobes are removed. But this is not an absolute rule, and one of the most marked instances of rotation round the vertebral axis which we observed was an animal in which only the cortex of the lateral lobe had been partially cauterized, and under conditions likely to excite irritation of neighbouring parts. In this case the rotation was exactly in the opposite direction to that which usually occurred. The direction of the rotation appears to be the subject of some confusion and difference of opinion. Magendie, as is well-known—and his observations have been confirmed by Hitzig and others—described the rotation after section of the middle peduncle as taking place towards the side of lesion; so that, for instance, if the left were cut
the animal would roll to this side with incredible force and rapidity. Luciani, however, describes the rotation as uniformly occurring from the injured to the sound side, so that, e.g., if the right side of the cerebellum is removed, the rotation takes place from right to left.

Our experiments on the cerebellar peduncles, effected in a precise manner by simple section, are absolutely in harmony with the results obtained by Magendie and Hitzig. We have found that, without exception, the animals roll towards the side of lesion.

The apparent discrepancy between our results and those of Luciani has been so uniform that we cannot but think that it is more in the manner of description than in the actual facts themselves. The facts, according to our observations, are that the animals, when placed on the ground, roll towards the side of lesion, as if carried by some irresistible force. And yet the impulse which causes the animal to roll towards the side of lesion, may be correctly described as a rotation round the vertebral axis from the injured to the sound side. Thus, if the left peduncle is divided, the animal will roll towards the left, but the impulse which causes this rolling movement would, if the animal were viewed in front, appear as a rotation round the vertebral axis from left to right. It might, therefore, be equally correct to say that the rotation is from the injured to the sound side, and that the rolling is towards the side of lesion. That this is the real explanation of the apparent discrepancy, I gather from a passage in which Luciani gives a detailed account of what he terms rotation from left to right. He says ("Kleinhirn," p. 139): "When the animal [a dog] was laid on its left side the torsion of the vertebral column was such as to bring the dorsal surface downwards, and the feet in the air. From this position, and with movements of all four limbs, it turned on its right side with its back to the right, and then on to its left side." It appears to me evident that if these movements were repeated several times in succession the result would be that of an animal rolling towards the left side. If this is not the true solution of the seeming discrepancy as to the direction of the rotation, as described by
Magendie and Luciani, we should say that our results are in agreement with those of Magendie, and that the direction in which animals roll after removal of the cerebellar hemisphere, or section of its peduncles, is towards the side of lesion.

The deviations of the optic axes, described by Magendie in connection with the rotatory phenomena, we have found in monkeys to be far from uniform, and we have not been able to establish any constant relation between lesion of any particular portion of the cerebellum, and any particular distortion or deviation of the optic axes. Nor have we found those indications of tonic contracture of the limbs in monkeys on the side of lesion such as Luciani appears to have uniformly observed and recorded as signs of irritation.

A special and characteristic attitude is assumed by animals after section of one or other peduncle, namely, curvation of the vertebral column, with the concavity towards the side of lesion, adduction and flexion of the limbs on the same side, and abduction and extension of the limbs on the opposite. The chin, as a rule, deviates towards the sound side, that is, in the direction of the rotation which causes the animal to roll towards the side of lesion. But the flexion of the limbs on the side of lesion is not a rigid flexion or contracture, and offers no resistance to passive movements or change of position.

Let us now examine what Luciani considers to be the essential factors of cerebellar ataxy, namely, asthenia, atonia and astasia.

First, as to asthenia, by which is meant a defect of energy, due to the loss of some influence supposed to be exerted by the cerebellum, which heightens or reinforces the energy of muscular contraction. Of the existence of any such influence, such as has been contended for by Luys, Weir-Mitchell and others, or that the phenomena of cerebellar ataxy can in any way be ascribed to such a condition, I have discovered no satisfactory evidence, either from a study of Luciani's experiments, or in the facts of our own, or in the phenomena of cerebellar disease in man. On the
contrary, clinical observation has abundantly shown that in cases of atrophy or disease of the cerebellum, of such a nature, and of such extent, as to render station and locomotion altogether impossible, the patients may in the recumbent position perform all movements of the limbs with energy and vigour. Of one of his patients, who was suffering from atrophy of the cerebellum, and who could not walk except in the most bizarre fashion, pushing a chair in front of her and often falling down, Vulpian remarks: "Yet, and this is very remarkable, she retained such muscular vigour that when one held out a hand to save her from falling she gripped so firmly as to cause pain."

When a monkey, within a few days after complete removal of its cerebellum, and so unstable as to be unable to sit up without support, or progress except in a sprawling fashion with its abdomen in contact with the ground, is, notwithstanding, able to climb a rope or lattice work hand-over-hand with apparently the same agility as a normal monkey, and grips so firmly with hands and feet as to require considerable force to detach it, I do not think the term "asthenia," or defect of muscular energy, is at all applicable to such an animal. And when a monkey, within an hour of removal of the left lobe of the cerebellum, and still unable to sit up, is able to grip so firmly with its left hand as to bear the whole weight of its body on it, I do not think that its left limbs can at all properly be described as being affected by asthenia. Yet these and similar facts might be multiplied from our observations.

In no case in which either the lateral lobe was removed, or a peduncle cut, could we detect any appreciable difference in the grip of the two sides. We found it, however, difficult to make exact dynamometric observations, as the animals were not always in the humour to make that trial of their strength which alone would permit of accurate comparison, and without which tests of this kind are altogether fallacious. There is, however, no doubt that an animal after removal of one lateral lobe prefers to use the limbs on the sound side, but this is obviously more in consequence of the instability of the limbs than from any real want of strength, and such,
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in my opinion, is the real explanation of the apparent want of strength in the limbs on the side of lesion which Luciani has so frequently noted. For the most part, he speaks of a semblance of weakness only, as evidenced by the doubling up of the limbs and tendency to fall on the injured side. But when the same monkey which exhibits this defect is able to climb well, and a dog to swim vigorously, as he admits, the symptom cannot be explained by loss of muscular energy.

One of the proofs relied on by him as indicating relative weakness of the limbs on the side of lesion is of so remarkable a character as to deserve special mention. Thus, he says that dogs deprived of one-half of the cerebellum, when swimming, tend, almost invariably, to the opposite side. Thus, if the right half of the cerebellum has been removed, the animal swims towards the left, in consequence, he says, "of the greater energy of movement of the limbs on the left side." ("Kleinhirn," p. 171.) Any swimmer, however, would say that in order to swim to the left he would require to make more vigorous efforts with his right limbs, and the very fact mentioned by Luciani would appear to prove exactly the opposite of what he says. In further corroboration of this may be mentioned another observation of his, viz., a dog in which the right side of the cerebellum had been removed, together with the motor centres (sigmoid gyrus) of the left hemisphere, thereby causing paresis of the right limbs. This dog in swimming, swam towards the right side, because, as he says, and truly, "the left limbs struck the water more vigorously than the right." (Ibid., p. 133.)

With these remarks I would dismiss Luciani's theory of asthenia as an element in the causation of so-called cerebellar ataxy, as being unsupported by valid evidence. That animals after complete removal of the cerebellum may, as Weir-Mitchell says, show signs of exhaustion after sustained muscular effort sooner than normal animals, is, I believe, more easily explained on my own hypothesis that the efforts at functional compensation, effected, as Luciani has verified, by the cerebral motor centres, are a considerable strain on
the animal's power, and, in consequence, lead to exhaustion much sooner than under normal conditions.

Next, as to the evidence of atonia, or loss of tone in the muscles. The usual test of the tone of muscles is the readiness, or otherwise, with which they respond to mechanical tension, such as is constantly exemplified in the usual method of eliciting the patellar-tendon reaction. Luciani has, however, apparently not investigated the condition of the tendon reflexes in his experimental animals. As a proof of the loss of tone, he speaks of the muscles of the limbs on the side of lesion as appearing to him more flaccid, and of the leg occasionally giving way, as if the tone suddenly ceased, &c., all of which appear to me more matters of constructive speculation than actual realities.

That the tone of the muscles, so far at least as indicated by the tendon reactions, is not diminished by cerebellar extirpation, complete or unilateral, or after section of its peduncles, has been amply proved by our own experiments, as well as by those of Dr. Risien Russell. (Proc. Roy. Society, vol. liii., p. 430.) We have not, however, observed the marked exaggeration of the knee-jerk on the side of lesion, which Dr. Russell appears to have found in dogs. As a rule, little or no difference could be made out after extirpation of the lateral lobe, or section of the peduncles on one side, immediately after the operation. If anything, the difference was in favour of the reaction on the side of lesion, but it showed a distinct tendency to increase the longer the animals survived; and when the whole cerebellum was extirpated, the knee-jerks were, after some months, decidedly increased, as compared with the normal, but there was no rigidity of the limbs.

These facts lead me to diverge for a moment to comment on the views propounded by Drs. Hughlings Jackson and Bastian as to the mechanism of the development of rigidity in limbs paralysed by transverse lesions of the spinal cord. Dr. Bastian has adduced weighty evidence (Med. Chir. Trans., vol. lxxiii., p. 151), and he has been amply supported by Bowlby (Ibid., p. 313), and others, to show that in total transverse lesions of the spinal cord the tendon reflexes are
abolished and never return, and that this condition cannot be satisfactorily explained on the hypothesis of a coincident lesion, or mere dynamic disturbance of the centres situated below the lesion. Though I think the point is one which may well bear further investigation, I am prepared, for the purposes of argument, to admit Dr. Bastian's law as being universally applicable. But his further proposition to the effect that the development of rigidity in limbs paralysed from cerebral disease, or partial lesion of the cord, is due to the "unrestrained," or, as Dr. Jackson would say, the "unantagonized" influence of the cerebellum passing downwards, perhaps through the grey matter of the cord, I cannot allow to pass without question.

First, I can find no experimental proof that the cerebellum exercises any such influence as that contended for by Dr. Bastian. It is no doubt true that in some cases of disease in the posterior fossa, involving the cerebellum, the tendon reflexes are abolished. I am not acquainted with, and am myself unable to offer any satisfactory explanation of this fact, but, certainly, it is not the rule that the knee-jerks are abolished in cerebellar disease. And when we find that in monkeys, after complete removal of the cerebellum, the knee-jerks instead of being lost become actually increased, it is clear that the mechanism for the increase of the knee-jerks still exists. That this is independent, both of the cerebrum and cerebellum, is further evident from the fact that in monkeys the knee-jerks may become increased; and late rigidity ensue, when the spinal cord has been totally divided transversely. In illustration of this point, I will here briefly mention the facts of two experiments made by Dr. Turner and myself.

In the first case, the spinal cord was divided transversely and completely at the level of the eighth dorsal root in March, 1891. Immediately after the operation the knee-jerks were capable of being elicited as before the operation. The animal exhibited the usual paraplegic symptoms, both as regards total paralysis of motion and sensation, and continued so until its death on July 24, that is, fully four months. During this period there
gradually developed well-marked increase of the knee-jerks, as well as of the superficial reflexes, together with considerable rigidity of the limbs. The animal was exhibited before its death to Drs. Hughlings Jackson and Bastian, who both satisfied themselves as to the condition here described.

In a second case, the cord was completely divided at the level of the sixth dorsal nerve on July 21, 1891. Both knee-jerks were readily elicited immediately after the operation, if anything, more markedly than before. The animal lived till September 29; that is, two months. This animal, however, exhibited some noteworthy differences as compared with the former. Instead of a gradual increase of the knee-jerks and development of late rigidity, no reaction could be obtained on August 4, that is, twelve days after the operation. The knee-jerks proper remained in abeyance till the 20th, that is, for sixteen days. During this time the muscles of the limbs had undergone considerable wasting, and the faradic contractility of the quadriceps extensor became decidedly diminished as compared with the other muscles, and more so on the left than on the right. After the 20th the right knee-jerk re-appeared and continued to increase. The left remained in abeyance until September 10 at least, after which date the knee-jerks were not examined, as the animal died during the holiday season.

The first of these two cases shows conclusively that in the cord itself there exist the conditions for the presence and increase of the knee-jerks, and the subsequent development of rigidity or contracture of the limbs. And the second case shows also, in a highly instructive manner, that in the cord itself conditions may occur which, either temporarily, or it may be permanently, prevent the manifestation of these phenomena. In both these cases, the influence of the cerebrum and cerebellum was, from the first, entirely excluded. But it will probably be said that though this may be true of monkeys, it is not applicable to man. That the symptomatology of total transverse lesions in man and the lower animals is widely different, I readily admit. Indeed, we have every reason for believing that the independence of the
spinal centres is much greater in the lower animals than in man. We all know the extraordinary adaptiveness and apparent intelligence of the spinal cord of a frog; and the experiments of Goltz on dogs have shown that, even in animals so high in the scale as dogs, the hind limbs, after total transverse section of the spinal cord, remain capable of a number of the most complex, reflex or responsive actions. In answer to some enquiries I made of him in regard to his animals, Professor Goltz wrote to me: "Several of my dogs learnt to stand on their hind legs, and even to walk a few steps. While so standing, the hind legs became stiff in consequence of a general contracture of the muscles of a reflex character, conditioned by the position of the animal." Such things are, of course, impossible in monkeys and, a fortiori, in man.

The point, however, which I desire to emphasize is, that the increase of the knee-jerks, and development of rigidity, can occur in monkeys altogether independently of the cerebellum, owing to conditions in the cord itself, in which also the opposite conditions may occur, either temporarily or permanently. The difference between the symptomatology of total transverse lesions of the cord in man and the lower animals is, in my opinion, due to the greater independent vitality of the spinal centres in the latter as compared with the former. In these, total separation from the higher centres leads to such impaired vitality, that they are no longer capable of manifesting that increased excitability, which is the primary cause of the phenomena in question.

Let us now return to the third cardinal factor of cerebellar deficiency; namely, the condition which Luciani terms "astasia." This term very aptly describes the primary instability, both of station and locomotion, as well as the subsequent, and more persistent, tremor and unsteadiness, not only of the body as a whole, but of the limbs, which is so noteworthy a feature after cerebellar extirpation in monkeys and dogs. The reeling gait and the various degrees of what most nearly represents alcoholic intoxication, are familiar and generally recognised symptoms of cerebellar
disease in man, but the tremor and unsteadiness of the limbs, though occasionally noted, and more particularly in cases of cerebellar atrophy, are not so common, and yet they are sometimes present, as I have myself had occasion to observe. When they occur more particularly on one side, they ought to furnish a reliable guide to the determination of the position of the lesion, inasmuch as we have seen that they occur specially on the side of lesion. It would appear, therefore, that the instability of cerebellar defect is not limited to the muscles of the trunk, but affects the muscles, in general, and manifests itself not only in the unsteadiness on volitional effort, but also in the ungraded or dismetric (Luciani) manner in which the limbs are raised and planted in progression, in spite of the most complete compensation which the voluntary motor centres are capable of effecting. But the term "astasia" is, after all, only a description, better perhaps than any other that has been applied to the phenomena of cerebellar deficiency, and not an explanation; and the problem still remains, how is the astasia conditioned? To explain this is to my mind to explain the true mechanism of the cerebellum.

Luciani sees in the tremors and oscillations, &c., a defective summation of the individual impulses on which the muscular contraction depends, and he looks on the abruptness of the movements of the limbs as due to a sudden flaccidity (Erschlaffung) of the antagonists while the flexors or extensors are acting respectively; or, it may be, owing to collateral muscles intervening which are not generally concerned in the action in question. And there is much to be said in favour of a hypothesis of this kind. And yet I question whether this is any improvement on the hypothesis of Herbert Spencer, that the cerebellum is the organ of "doubly compound co-ordination in space," while the cerebrum is the organ of "doubly compound co-ordination in time," so often and so ably expounded and applied by Dr. Hughlings Jackson. "According to this hypothesis," as James Ross has lucidly stated it, "all the muscles of the body are innervated both by the cerebrum and cerebellum,
but in an inverse order. The cerebellum regulates the muscular contractions necessary for the maintenance of our attitudes in space, while the cerebrum regulates the contractions necessary to effect all the change of attitude which are made in response to the successive impressions occurring in time. Now, so long as a particular attitude is maintained in opposition to gravity and other forces, the contractions of the various groups of muscles concerned must be continuous and in equilibrium with each other, while each change of attitude necessitates the overthrow of this equilibrium, involving the preponderance of the contractions of some groups of muscles over those of others, so that the change of attitude involves alternate muscular contractions and relaxations. Speaking broadly then, the cerebellum regulates continuous or tonic muscular contractions. It will be seen, therefore, that every compound muscular adjustment necessitates the co-operation of both these organs. No change of attitude can be effected by the cerebrum, except in so far as a certain attitude was previously maintained by the cerebellum, and no steady movement can be produced by the alternate contractions of some groups of muscles except in so far as other groups of muscles are maintained in a state of continuous contraction; hence it may be inferred that all the movements of the body are co-ordinated both in the cerebellum and cerebrum." (Ross, "Diseases of the Nervous System," vol. i., p. 57.) The cessation of the tonic or continuous influence of the cerebellum should, therefore, exhibit itself in an unsteady, ungraded character of all the motor adjustments, whether of the body as a whole, or of its individual parts, and would, theoretically, produce a state of affairs which agrees well with the actual effects of cerebellar extirpation in animals.

Even, however, if we assume that this is the true formula for the influence of the cerebellum, it still remains to be determined how its activity is called into play and brought to bear on the muscles, either in association with the cerebrum, or independently. That it is a more or less independent responsive mechanism is clearly shown by the fact that the stability of motor adjustments is still
capable of being maintained after the cerebrum has been removed. That the cerebellum stands in complex afferent and efferent relations with the cerebro-spinal system is undoubted, but we, as yet, know little as to the true functions of the respective centripetal tracts, and less, perhaps, with regard to the centres and tracts through which it acts centrifugally.

The superior peduncles proper appear to be efferent in function; that is to say, after cerebellar extirpation they degenerate from the cerebellum up to the opposite red nucleus and thence to the optic thalamus. On the dorsal aspect of the brachium conjunctivum, however, there is a tract which degenerates towards the cerebellum, a tract which the observations of Loewenthal, Mott, Tooth, and our own, prove to be the termination of Gowers' antero-lateral spinal tract. As yet, however, there has been no exact determination of the functions of the red nucleus, or of Gowers' antero-lateral tract.

The middle cerebellar peduncle appears also to be efferent from the lateral lobe of the cerebellum to the opposite nucleus pontis. It is, no doubt, through the intermediation of the cells of the nucleus pontis that the cerebellar hemisphere of the one side is brought in relation with the cerebral hemisphere of the opposite. But here again we are in ignorance as to the true signification of those tracts, which are described as connecting the frontal and temporo-occipital regions of the cortex with the cell groups in question.

The inferior cerebellar peduncle contains both efferent and afferent fibres, efferent to the opposite inferior olive, by way of the external arcuate fibres; and afferent, viz., the direct cerebellar tract from Clarke's vesicular column, largely also of fibres from the posterior median and postero-external columns of the spinal cord by way of the clavate and cuneate nuclei, and probably also of the vestibular branch of the eighth, and, perhaps, also some of the other cranial nerves. But as to the exact functions of these different afferent

1 Rev. Med. d. l. Suisse Romande, 1885.
2 Brain, vol. xv., 1892.
3 Ibid.
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tracts to the cerebellum, we know very little. No discoverable loss of sensation ensues when they are divided. In particular, destruction of the clavate and cuneate nuclei, though leading to temporary disturbances of equilibrium, causes no discoverable impairment of any of the forms of cutaneous or general sensibility. And as the great bulk of the fibres which spring from the clavate and cuneate nuclei decussate in the interolivary layer, and ascend as the mesial fillet, which degenerates completely when they are destroyed, it is clear that this is not the path of transmission of sensations proper to the cerebrum. A tract which forms the internal division of the restiform body, and termed by Edinger "the direct sensory cerebellar tract," has been supposed to be the path by which the fifth, eighth, and probably also the vagus and glosso-pharyngeal nerves form connections with the middle lobe of the cerebellum. We have found, however, after extirpation of the middle lobe, that the tract in question degenerates downwards, or in an efferent direction, towards Deiters' nucleus. This is the only tract which appears to degenerate centrifugally from the middle lobe, a fact which shows that this lobe has no direct connection with the efferent tracts, which emerge in the cerebellar peduncles. Beyond those already mentioned, there is no proof of the existence of any other efferent tract from the cerebellum to the spinal cord. Marchi, however, has described a tract which degenerates downwards in the antero-lateral region of the cord after extirpation of the cerebellum, which he derives specially from the middle lobe by way of the posterior longitudinal bundles and fillet. The tract described by Marchi occupies the periphery of the antero-lateral region, with an offshoot posteriorly projecting into the lateral column. Our observations, however, lead us to believe that Marchi's views as to the origin of this tract are erroneous; for in a case of total extirpation of the cerebellum, which lived several months, we found no evidence of any degeneration in the position assigned by Marchi to his tract. And yet in one case of removal of the lateral lobe we found a degeneration in the periphery of the anterior

1 Sul. Orig. e. Decor. dei Ped. Cerebell., 1891.
column corresponding in position with the anterior division of Marchi's tract; and in another we found a well-marked descending degeneration in the lateral column in the region of the posterior division of the tract described by him. A further careful examination, however, of these cases, indicated that the degeneration in the anterior column was due to lesion of Deiters' nucleus, which would thus be an internode between the middle lobe and the anterior marginal column of the spinal cord, and that the degeneration in the lateral tract stood in relation to lesion of the lateral fillet, which forms a tract connecting the posterior corpus quadrigeminum with the lateral column of the cord. But while so much obscurity still prevails as to the functional significance of the various afferent and efferent tracts which connect the cerebellum with the rest of the cerebro-spinal system, we are not yet in a position to go much beyond mere speculation as to the mechanism of cerebellar co-ordination. In this some of us have indulged with greater or less plausibility, but I do not propose on this occasion to enter on the field of speculation, as I think we may more profitably wait for the dry clear light of further exact research.

If I may trespass on your patience a little longer, I should like to say a few words on a question which, though not directly related to the main subject of my discourse, has yet formed the subject of some inquiry in the course of our other experiments on the connexions of the cerebellum. Thus, on section of the inferior peduncle, it not unfrequently happened that the immediately subjacent, so-called, "ascending root" of the fifth nerve was wholly or partially severed. The result of lesion of this root was more or less complete anaesthesia of the cornea, and of the region of distribution of the sensory branches of the fifth nerve of the head and face. And by other experiments, not necessary to be detailed here, it was proved that this so-called ascending root is the true sensory root of the fifth nerve, and is, in reality, a descending root springing from the Gasserian ganglion, and passing through the substantia gelatinosa Rolandi towards the cells of the posterior cornu as far, at least, as the origin of the second cervical nerve.
On dividing the superior peduncle, one usually severs also the fibres of the so-called "descending root," which courses underneath this peduncle towards the crescentic layer of cells which surrounds the grey matter of the aqueduct of Sylvius. After such section, as well as after section of the motor root within the pons, but not after section of the sensory root, there followed atrophy of the fibres of this root, and of the crescentic layer of cells above-mentioned. The atrophy, however, was not a degenerative, but a simple atrophy, similar to that which occurs, as has been shown by Bergmann, Darkschewitsch, &c., in the central end of a motor root after division of the trunk. This is the root which has been supposed by some to join the sensory, and by others to join the motor division of the fifth: while Merkel has described it as being the trophic root of the trigeminus. This also is the root which Mendel has described as being atrophied in a case of old-standing facial hemiatrophy. A recent experiment of ours points to the descending root as being the motor root par excellence of the fifth nerve. No loss of sensation was caused in the cornea or elsewhere by its division, nor did any trophic disturbance occur in the eyeball during the many weeks which the animal survived. On the other hand, atrophic paralysis ensued in the muscles of mastication.

A point which we kept specially before us in our experiments on the fifth nerve, was the so-called trophic influence of this nerve on the eyeball, a question which is still the subject of dispute, and which has come more recently again into prominence in connection with surgical operations on the Gasserian ganglion, with a view to the relief of inveterate trigeminal neuralgia.

The question is, as to the exact causation of the ulceration of the cornea and subsequent destructive inflammation of the eyeball, which have been so frequently observed both in man and animals after lesion or section of the fifth nerve. Mere anaesthesia, with the consequent loss of the protective

1 Obersteiner's Arbeiten, 1892.
2 Neur. Centralblatt, 1893.
3 Neurol. Centralbl., 1888.
influence conferred by the adaptive reactions of the eyelids, does not seem capable of explaining the facts, for the eye may be insensible from cerebral lesion, or even from lesion of the fifth nerve itself, and yet show no sign of corneal ulceration or destructive inflammation, though the anaesthetic eye has not been more protected from external influences than the other. An interesting case of this kind has been recorded by Hutchinson (Ophtal. Hosp. Reports, vol. iv., p. 191), and many others might be alluded to. And the acute inflammation has been seen in cases of lesion of the fifth nerve, not sufficient to cause complete anaesthesia; facts which have led Meissner and others to postulate the existence of a separate set of fibres exercising a trophic influence on the eyeball, apart from those which confer sensibility on it. Nor can the inflammation be satisfactorily accounted for by mere vascular paralysis, for the inflammatory disorders first show themselves in the cornea, the extra-vascular part of the eyeball; and Sinitzin states, though in this he is opposed by Eckhard,¹ that section of the cervical sympathetic, and consequent vaso-motor paralysis, actually prevents the inflammatory consequences otherwise following section of the fifth. Snellen found that when the eyeball was completely protected no inflammation occurred; and he, therefore, attributed the whole of the phenomena to external influences. But these conclusions have been contradicted by others (Decker, Laborde, Büttner, &c.) who have observed speedy destructive inflammation follow section of the nerve in spite of protective precautions; and also cases in which the eye did not become inflamed, though no special precautions were taken to protect it. There remains, therefore, some factor requiring elucidation which is of considerable importance from a practical point of view.

Magendie first pointed out, and in this he was confirmed by Longet and others, that the effects of section of the fifth nerve differ according as the lesion was made behind or in front of the Gasserian ganglion. Thus, whereas, section between the ganglion and the pons did not, or rarely, cause

inflammation of the eye, section anterior to it was usually followed by destructive panophthalmitis. The latest experimenter in this field, Gaule (Centralblatt für Physiologie, 1892, p. 419) confirms the statements of Magendie, and holds that only lesion of the Gasserian ganglion itself, or section of the ophthalmic division, is effectual in inducing trophic disorders. These, he holds, occur immediately on section of the nerve, and altogether irrespective of external influences. These trophic disorders consist, according to him, in necrosis of certain portions, with cell proliferations in other portions of the cornea, as well as changes in the corneal corpuscles, Descemet's membrane and aqueous humour, which necrotic and correlated changes, in consequence of external influences, such as drying, mechanical injuries, septic infection, &c., advance to the well-known destructive neuro-paralytic ophthalmia.

Our experiments have included section of the so-called ascending root in its course above and through the tubercle of Rolando; section of the trunk of the nerve between the ganglion and the pons; and section of the ophthalmic division in front of the Gasserian ganglion. No special precautions were taken to protect the eye on the side of lesion, which was simply left under the same conditions as the normal one.

Of twelve experiments in which the nerve was divided behind the ganglion, the duration of life was from two days to four months. With the exception of two, which deserve separate mention, none of these cases exhibited any progressive ulceration of the cornea or destructive panophthalmitis. Of those that exhibited any alteration, there was, at most, some degree of conjunctivitis, and haziness of the cornea, as if from drying of the surface. In the majority the cornea remained quite clear and transparent. In one of the exceptional cases above alluded to, in which the eye remained in a perfectly normal condition for eight days after the operation, a drop of collodion accidentally entered the anaesthetic eye. This occasioned intense conjunctivitis, oedema of the eyelids, and a milky white opacity, which overspread the whole of the cornea. Mr. Gunn, who examined this case
with us, attributed the opacity of the cornea to the shedding of the superficial epithelium, similar to that produced in man by an irritant, such as lime. The opacity, however, disappeared in the course of a fortnight, and the cornea regained its transparency with the exception of a minute ulcer surrounded by slight opacity, which remained till the death of the animal, six weeks after the operation.

These cases show, therefore, that there is no necessary tendency to progressive inflammation of the eyeball after division of the fifth nerve, behind the Gasserian ganglion, and that such changes as occur do not differ in their process of repair from those that occur in a perfectly normal eye. In one case, after division of the trunk of the nerve, the cornea on the second day exhibited a depressed ulcer and some degree of opacity, which, however, cleared somewhat at the end of a week, but again set in accompanied by a purulent discharge, which resulted in complete disorganization of the eyeball in the course of six weeks. In this case, however, there were indications that the wound was not strictly aseptic, and the temperature remained throughout higher than normal. The probability is, therefore, that the progressive destructive inflammation of the eye was due to septic irritation of the trunk of the nerve.

The most instructive and interesting of our experiments are those relating to division of the fifth, between the Gasserian ganglion and the eyeball. This is the lesion which most observers have found to be specially calculated to excite inflammatory changes in the eye, apparently independent of external influences. We have divided the ophthalmic division in front of the Gasserian ganglion in five cases. In three of these animals, which lived respectively two days, fourteen days, and six weeks, the cornea remained perfectly clear and transparent during the whole time of the animals' survival. Microscopical examination, which was carried out by Dr. Bulloch, showed that the epithelium, corneal corpuscles, and substance of the cornea presented a perfectly normal appearance. In the one which lived six weeks, the anaesthetic cornea did not take on the chloride of gold stain like the normal, but as regards other re-agents
there was no change. Occasionally the cornea, when examined by a magnifying glass, was seen to exhibit minute, pin-pointed depressions, such as described by Gaule and Eckhard, as if from partial shedding of the superficial epithelium, but similar depressions not unfrequently occurred also in the sound eye, and they did not appear to have any pathological significance.

In a fourth case the cornea, which had remained perfectly clear and transparent until the fourth day, was irritated by a minute speck of nitrate of silver, and exactly the same condition was established also in the normal eye. This gave rise in each eye to a superficial ulcer with mucopurulent conjunctivitis, but there was no progressive ulceration of the cornea in either case, and the process of repair occurred in the anaesthetic eye as well as in the normal one. In a fifth case, however, the cornea, which had remained clear for two days after the operation, exhibited at the end of that time a central depression with opacity, which progressed until it overspread the whole surface. In this case, however, there were indications of septic irritation in the wound with febrile disturbance.

It results, therefore, from these experiments, that the separation of the eye from the Gasserian ganglion by division of the ophthalmic branch of the fifth, does not necessarily induce any trophic disturbance in the eyeball, or interfere with the processes of healthy nutrition and repair. The only strictly trophic influence of the Gasserian ganglion is, I conceive, on the cellulipetal processes of the Gasserian ganglion cells and their annexes, which probably undergo atrophy, though we have not as yet been able to demonstrate this microscopically. There seems little doubt that the so-called trophic disturbances, which so commonly occur after division of the ophthalmic branch, are due to inflammatory irritation of the nerve, and are of the same character as those which occur in connection with similar irritation of sensory nerves generally. By this irritation, changes are indirectly excited in the corneal tissues, which, under the influence of external agencies and septic infection, result in general panophthalmitis. If, however, proper measures are
taken to avoid setting up inflammatory irritation, there is no reason, so far as I can see, why extirpation of the Gasserian ganglion, or section of the ophthalmic division, should excite inflammation of the eye any more than that section of any sensory nerve should be followed by so-called trophic disorders in the region of its distribution, which we know practically is neither a necessary nor, indeed, a common occurrence.