ON THE EFFERENT RELATIONSHIP OF THE OPTIC THALAMUS AND DEITER'S NUCLEUS TO THE SPINAL CORD, WITH SPECIAL REFERENCE TO THE CEREBELLAR INFLUX OF DR HUGHLINGS JACKSON AND THE GENESIS OF THE DECEREBRATE RIGIDITY OF ORD AND SHERRINGTON.

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Attention has long been drawn to the question of the contracture which occurs with lesions of the nervous system. This contracture has been noticed in lesions of the pyramidal tracts, whether acquired or congenital, in cerebellar lesions, and in inflammatory conditions at the base of the brain, as in posterior basic meningitis.

With cerebral lesions the rigidity that occurs is classified as: 1. Initial Rigidity. 2. Early Rigidity. 3. Late Rigidity. 4. Structural Rigidity.

The initial rigidity occurs in the first few hours after the occurrence of the lesion affecting the pyramidal tracts and is supposed to be due to irritation due to the lesion.

The early rigidity begins a few days after the onset of the lesion and lasts a few weeks. It is generally supposed to be due to inflammatory irritation of the parts around the lesion.

Late rigidity occurs in enduring paralysis and may be permanent. It is always associated with descending degeneration in the pyramidal tracts and is influenced by the irritative nature of the lesion. It depends a great deal on active muscular contraction, since it is less after sleep and with warmth and is overcome by passive movements. It affects the arms more than the legs. In the arms the rigidity preponderates in the flexors, in the legs the incidence affects the flexors and extensors more equally.

This condition finally passes on to structural rigidity if the paralysis is enduring.

In disseminated sclerosis marked rigidity occurs, and is at first marked passive rigidity. It occurs when the paresis is only slight, and finally becomes permanent. In this the legs are more affected than the arms, and at first is an extensor spasm, later it may become flexor.

Disease of or injury to the motor centres of the cortex of the brain, and the pyramidal tracts which descend from them through the cord, are regarded as causes which give rise to contracture. Slight interference with the healthy condition of the cortical cells and pyramidal fibres results in the loosening of the reins which inhibit the spinal centres, and an increase in tendon reflexes is the first evidence of the change. A little more disease of these tracts and clonus is developed, and finally with grosser disease contracture occurs. The rigidity early and late depends on the overaction of the spinal centres. The centres which thus overact are probably those on which normal muscular tone depends, and the rigidity is an excessive degree of this tone due to the removal of the inhibitory influence exerted by the motor tract.

Bastian[1], however, showed that in complete transverse lesions of the spinal cord this increased tonus is absent, that the tendon reflexes are absent, and the paralysis is a flaccid one. From this he concluded that the cerebellum exerts an adjuvant influence and the pyramidal an inhibitory influence, and that the anterior cornual cells have only a slight inherent tonic influence.

Dr Hughlings Jackson[2] in his Hughlings Jackson Lecture to the Neurological Society, 1897, put forward his views as regards rigidity as follows:

"Speaking very roughly the cerebellum represents movements of the skeletal muscles in order, trunk, leg, arm, preponderatingly extensorwise; the cerebrum represents movements of the same muscles, in order, arm, leg, trunk, preponderatingly flexorwise. It is also supposed that impulses from motor centres of the higher levels of each sub-system continuously act upon the motor centres of the lowest level, that the impulses from each set of higher levels antagonise or inhibit one another in different degrees upon different lowest motor centres; that the degree in which the cerebral and cerebellar impulses antagonise one another is the same as the order of the degree of their different representations of movements of the muscles of the body. In accordance with this hypothesis the rigidity in hemiplegia results because, cerebral influence being cut off the lowest

[1] Bastian, *Trans. Med. Chir. Soc.*, London, 1891.

[2] Quoted in Allbutt's *Med.* vol. III. p. 540.

motor centres, as the cerebrum represents movements in order, arm, leg, trunk, cerebellar influence upon the lowest motor centres is no longer antagonised; there is cerebellar influx into the parts which the cerebrum has abandoned. There is another way of considering the hypotheses of the relations of the cerebral and cerebellar sub-systems to one another, by their having the lowest level in common. Certain cerebral and cerebellar maladies may be contrasted and compared with one another as being complementary inverses (corresponding opposites). The best marked complementary inverse is a case of extensive cerebellar paralysis (trunk, legs, arms) and rigidity, as the corresponding opposite of the double hemiplegia (arms, legs, trunk), and rigidity of an advanced case of paralysis agitans. In the former case the attitude is opisthotonic, in the latter slightly emprosthotonic." Those who hold the other views as regard rigidity explain the rigidity occurring in cerebellar lesions as due to irritation of the pyramidal tracts.

In these experiments it was determined to investigate the matter from the point of view of tracing the relations of the thalamus to the mesencephalon and hind brain.

Now the thalamic grey centres are in association with the bulb and spinal cord by the thalamo-bulbar and thalamo-spinal tracts. Placed on and around these are the tectospinal and rubrospinal tracts together with the Deiter spinal tract lower down and the cerebellum, the lateral lobe of which is directly connected with the contralateral pontine nuclei. As the pyramidal tracts run through these latter it became necessary to exclude the influence of the pyramidal fibres. The investigation therefore became as follows:

1. Localisation of the genesis of decerebrate rigidity. This was arrived at by making successive coronal slices through the thalamus, mesencephalon, pons and bulb.

2. An endeavour to arrive at some idea of the effect of the cerebellum or pontine nuclei or Deiter's nucleus by the stimulation of the middle and superior peduncles, and with the view of excluding the pyramidal system and its effects a large number of control experiments were made in which the pyramidal fibres had been caused to totally degenerate by means of suitable lesions made in the middle zone of the cerebral hemispheres.

3. Stimulation of the cut surface of the mesencephalon and optic thalamus with or without previous complete pyramidal degeneration.

These experiments were conducted under anæsthesia, the anæsthetic used being chloroform or ether.

In cases where the brain was removed in slices to note the decerebrate rigidity both carotids were previously tied to diminish the bleeding. In cases where the pyramidal fibres were caused to degenerate the animal was anæsthetised, trephined under strict antiseptic precautions and the tracts divided by coronal division of the hemisphere.

In some cases this was performed on the left, in others on the right side, and in a few cases bilateral section was performed. The animals rapidly recovered power after this lesion and had the typical movements *de ménage*.

The results will now be dealt with in the following order:—

1. Localisation and genesis of decerebrate rigidity.
   (*a*) The influence of the cerebellum.
   (*b*) The condition of the knee jerks.
2. The results of excitation of the cerebellar peduncles.
3. The results of excitation of the mesencephalic tracts.

DECEREBRATE RIGIDITY.

a. Influence of Cerebellum.

Decerebrate rigidity was first studied by Sherrington[1] and later by Löwenthal[2] and Horsley.

This condition arises after transection of the mesencephalon. In it the respiratory and cardiac functions after a time go on quietly, but certain groups of muscles become rigid. It affects the muscles of the forelimb more than those of the hind limb. The elbow becomes quite rigidly fixed, the wrists less, and the digits not at all. The homologous joints of the hind limb are affected in the same manner as those of the forelimb. The tail may be erect, the head and neck are retracted. Passive movement increases the rigidity, brings it out if not present, or re-establishes it if passed off.

Sherrington also showed that after semisection of the mesencephalon the rigidity was present on both sides but most on the side of the lesion, and that it could be inhibited on the side of the lesion by stimulation of the appropriate areas of the motor cortex of the contralateral side. He also demonstrated that the rigidity could be abolished on one

[1] Sherrington. *Schäfer's Physiology*, II. pp. 914—915; *Proc. Royal Soc.* LX. p. 411, 1896; This *Journal*, XXII. p. 319. 1897.

[2] Löwenthal and Horsley. *Proc. Roy. Soc.* LXI. p. 20. 1897.

side by unilateral transection of the bulb in its lower third, and by section of the afferent spinal roots of the region implicated. Similarly stimulation of the anterior surface of the cerebellum at points extending from the middle line to far on to the lateral lobe inhibits the rigidity, especially on the homolateral side. Sherrington further demonstrated that in an animal in this condition of decerebrate rigidity following transection through the mesencephalon reflexes can be obtained which actuate the neck, trunk, and the limbs as a whole, and treat them as components of a single motile apparatus. The reflexes thus elicited by putting one paw into hot water are such that the fore paw so heated is flexed and protracted, the contralateral is extended and retracted, the hind limb on the homolateral side is extended and contracted, the contralateral limb is flexed and protracted. The whole body thus appears to move as in the movements of walking.

Langendorff[1] has shown that in birds the inhibitory influence from the optic lobes is crossed.

In the present investigation, which was carried out in cats and monkeys, the method employed was in some animals the removal of the cortex with subsequent removal of the cerebral hemispheres, mesencephalon and hind brain by successive coronal slices till rigidity apeared and again disappeared.

In others the cerebellum was removed piecemeal before and after the production of rigidity by mesencephalic transections. In some of these cases the tentorium was first removed, in others the tentorium was untouched.

In another series again the fibres from Deiter's nucleus were divided to note the effect of their division on the occurrence of decerebrate rigidity.

Finally in another series of animals, unilateral lesions were made at different levels in the thalamus and mesencephalon to study unilateral effects and to endeavour to obtain contralateral rigidity.

As the result of these methods it was found that decerebrate rigidity did not commence till the level of the section passed through the posterior part of the optic thalamus. The rigidity thus obtained was not so marked as that which occurred with lesions farther back. The most marked rigidity occurred when the section was made at the level of the posterior corpus quadrigeminum.

It was always noted that so long as the level of the section did not

[1] Langendorff. *Archiv für Physiol.* 1877.

go beyond the pontine fibres the rigidity remained in great intensity; if, however, the level of the section was through the trapezial fibres the rigidity was absolutely abolished.

In most cases the cat continued to breathe spontaneously, if not, then artificial respiration was performed till the cat breathed spontaneously. The animal was kept alive after this under anæsthesia for a short time and during this time the rigidity never redeveloped. Although, as sometimes happened, transection at a higher level caused arrest of respiration and loss of rigidity, the rigidity always came back when respiration was re-established.

When this was found to be the case it was surmised that the abolition might really be due to the severance of the Deiter spinal tract.

This was subsequently found to be so. Unilateral separation of Deiter's nucleus from the medulla or division of the tract as it passes down through the medulla produced relaxation of the rigidity on the homolateral side.

In most cases the momentary stimulation of the nucleus or its fibres by the knife caused a temporary increase in the rigidity. When the division of the tract was performed after mesencephalic transection the contralateral rigidity was unaffected, whilst the homolateral was abolished, and this continued as long as the animal was kept alive. In all cases the relaxation was not complete, and in some a certain amount of rigidity recurred after an interval. In most of these cases unilateral transection of the medulla at the level of the lower third of the calamus scriptorius did not cause this still existent rigidity to become less.

The rigidity remaining was therefore supposed to be due to the independent vitality of the anterior cornual cells.

According to these results it appears that the pontine nuclei have no part in the production of rigidity in these experiments, since by these successive coronal sections many pontine nuclei are removed without causing loss of rigidity, but when the section goes through the trapezium the rigidity vanishes, although a large part of these nuclei still remains. In fact successive transverse slices through the pons tend to increase the rigidity, which vanishes as soon as the trapezial level is reached.

These experiments further demonstrated that the cortex and pyramidal tracts have no relation to the causation of decerebrate rigidity. In no case where the pyramidal tracts had been caused to degenerate by previous section was there any difference in the nature and extent of the rigidity following mesencephalic transection.

Piecemeal removal of the cerebellum by horizontal slices produced no influence on pre-existing decerebrate rigidity until Deiter's nucleus was included in the section, when as before the rigidity was abolished.

In those cases in which the cerebellum was first removed before any lesion was made in the mesencephalon, the removal of the cerebellum was in some cases performed with or without previous removal of the tentorium.

During removal of the tentorium it was noticed that extensor rigidity always developed, and that the rigidity was of exactly the same nature as that following mesencephalic transection. Removal of the tentorium over one lobe of the cerebellum also produced bilateral rigidity, which was usually most marked on the same side. This rigidity was, however, frequently noticed to be more marked on the opposite side, although it subsequently increased on the same side.

In cases in which the tentorium was removed in this way, piecemeal removal of the lateral lobe of the cerebellum usually caused a temporary diminution of the pre-existing rigidity on the homolateral side, and an increase on the contralateral. The rigidity on the homolateral side also came back after a time unless nearly the whole of the lateral lobe was removed, when the rigidity did not recur on the homolateral side.

If, however, the tentorium was not previously removed then removal of one lateral lobe by successive sagittal slices caused at first the development of a slight degree of bilateral rigidity, which was usually at first more marked on the contralateral side, but it subsequently increased homolaterally. In other cases the homolateral rigidity was the most marked from the beginning. With greater removal the rigidity increased still more, till finally when nearly the whole of one lobe had been removed the homolateral rigidity diminished greatly or completely disappeared.

If the operation of transection of the mesencephalon was performed subsequently to the above-mentioned procedures then it was found that if Deiter's nucleus was uninjured the rigidity developed on the side of the cerebellar lesion as well as on the opposite side; if, however, it was injured then the rigidity did not appear on the side of the lesion.

Sherrington[1] in his description of the condition of decerebrate rigidity mentions the fact that the rigidity is abolished by transection

[1] Sherrington, *loc. cit. supra.*

of the bulb at the level of the lower third of the calamus scriptorius and that the influence therefore descends from somewhere higher up.

It would appear from these experiments that the influence comes down the Deiter's spinal tract.

These experiments also agree with those of Sherrington, in that section of the posterior roots of the limb affected by the rigidity caused abolition of the rigidity, and that previous section of the roots prevented its occurrence on division of the mesencephalon.

This being so, I thought it would be interesting to see the part played by the posterior columns and the direct cerebellar tracts. Rigidity was accordingly produced in the usual way, the cord was then exposed and these tracts divided just above the cervical enlargement. It was then seen that division of either of these tracts was without effect on the rigidity. In these experiments I was also able to produce unilateral rigidity due to unilateral mesencephalic transections. Not only was homolateral rigidity produced as Sherrington describes, but if the transection was made far forwards in the region of the optic thalamus and the anterior corpora quadrigemina marked contralateral rigidity was produced, the homolateral limbs were scarcely at all affected and then the rigidity in them occurred later and only on manipulation of the limbs.

When the lesion was made further back in the region behind the anterior corpora quadrigemina the homolateral rigidity was obtained with only slight contralateral.

These facts point to the conclusion that the influence descending from the region of the thalamus is a crossed one, and that the decussation takes place high up in the region of the decussation of the rubro-spinal tract.

Investigations were also made as to the influence of the cerebellum by way of the anterior cerebellar peduncles on the rigidity, with a view to see what relation the afferent and efferent parts of the cerebro-cerebellar circuit bear to this condition. Sherrington[1] says that the division of the anterior cerebellar peduncles and the cortico- and thalamo-pontine fibres produces decerebrate rigidity, a condition closely resembling that obtained by removal of the cerebellum, a fact which lays stress on the importance of the cerebro-cerebellar circuit as a factor in the cerebellar action.

Now the efferent channels of this circuit are the afferent nerves and

[1] Sherrington. *Schäfer's Physiology*, II. p. 908.

tracts of the cord, the Vth and VIIIth nerves acting through the cerebellar cortex, the corpus dentatum, the anterior cerebellar peduncle to the crossed rubro-thalamic nuclei, and the crossed cerebral cortex.

The efferent channel is the cortico- and thalamo-pontine fibres and the ponto-cerebellar fibres which have their nuclei in the half of the pons contralateral to each cerebellar hemisphere. In these experiments the superior cerebellar peduncles only were divided. The operation was performed under anæsthesia in the following way:

The posterior part of one cerebral hemisphere was exposed and drawn forwards and upwards from the tentorium, which was thus exposed; in other cases to avoid any traction the posterior part of the hemisphere was excised. The tentorium was separated from its attachments by a small sharp chisel and removed with as little disturbance as possible. The posterior corpus quadrigeminum was thus exposed, was separated from the cerebellum, and the superior peduncle was divided in the interval between these structures. In all cases, no matter how gently the tentorium was removed, rigidity followed this removal. As before, it was bilateral and usually most marked on the same side, though occasionally the contralateral rigidity was greatest.

The separation of the cerebellum from the posterior corpora quadrigemina also further increased the rigidity following removal of the tentorium.

The effect of division of the anterior cerebellar peduncle was that the rigidity only became slightly increased on both sides. In several cases the contralateral rigidity was more marked than the homolateral.

The resulting rigidity was usually not very intense, and was always increased by subsequent transection of the mesencephalon in front of the line of division of the superior peduncle. In one case in which the peduncle was merely scratched marked bilateral rigidity occurred. In another case the division of the peduncle was followed by flexor rigidity on the same side and extensor rigidity on the opposite; the flexor rigidity, however, was only temporary and gave place to the usual extensor rigidity.

It thus appears that the mere removal of the tentorium causes bilateral extensor rigidity, which is increased by manipulation of the cerebellum and mescencephalon, and still more by division of the superior peduncle of the cerebellum.

Removal of the tentorium cannot directly affect the peduncles, since they are completely protected by the close adhesion of the anterior part of the cerebellum and the posterior corpora quadrigemina.

In two cases, one a cat, the other a dog, fairly well-marked bilateral rigidity occurred with most marked homolateral effects as the result of a superficial incision into the side of the left crus, whereby some fibres of the cerebro-pontine tract were divided.

Again, from the previous results mentioned above relating to the production of unilateral rigidity, it is seen that in order to produce contralateral results the division must be made far forwards immediately in the region of the anterior corpus quadrigeminum, that is, the tract whose division causes the rigidity decussates anterior to the decussation of the superior cerebellar peduncles.

Taking these results together it would appear that as far as decerebrate rigidity is concerned the cerebellum does not exercise a marked controlling function over the opposite thalamus by way of the anterior peduncle; division of this afferent channel of the above-mentioned cerebro-cerebellar circuit is only slightly if at all concerned in the production of decerebrate rigidity. The question then arises as to the action exercised by the optic thalamus on the opposite cerebellar hemisphere by way of the afferent channel above described, as is evidenced by unilateral lesions in decerebrate rigidity.

As has been shown above the usual result of unilateral mesencephalic transection is the production of homolateral rigidity with much less marked contralateral rigidity; almost pure contralateral rigidity only occurred when the lesion was far forwards. From these results it would appear that it may be possible that the contralateral effect in the case of unilateral transection of the mesencephalon below the level of the anterior corpora quadrigemina is due to the abolition of a continuous cerebral control upon the opposite cerebellar lobe by way of the thalamo-pontine and ponto-cerebellar tracts, but it must also be remembered that it has been shown that some of the mesencephalic descending tracts from the region of the red nucleus have a set of fibres which do not decussate.

The conclusion that therefore appears to follow is that the inhibitory influence descends from the optic thalamus, and is mainly exerted directly upon the anterior cornual cells of the opposite side along a tract which decussates in the anterior part of the mesencephalon; and on the homolateral cells, along the part of the tract which does not decussate.

In the course of these experiments it was shown that removal of the tentorium, removal of the vermis, and bisections of the vermis produced an extensor rigidity exactly like decerebrate rigidity.

Removal of part of one lateral lobe of the cerebellum also produced bilateral extensor rigidity; when however the whole of one lateral lobe was removed the rigidity on the same side was very slight or completely absent. This latter result differs from that which occurs when the animal recovers from the anæsthetic. The animal then developes bilateral rigidity, most marked homolaterally. This rigidity comes out especially on exertion.

In monkeys, however, Ferrier[1] has shown that on the homolateral side there is no rigidity, but the forelimb is flexed and flaccid. The difference in these animals and between animals which are and are not under anæsthesia appears to be due to the different degrees of independent activity of the anterior cornual cells in cats and monkeys when not under anæsthesia, and the depression of their activity by anæsthesia. In these experiments the absence of homolateral rigidity was due to removal of the Deiter's control; in the animals not under anæsthesia the rigidity in the homolateral side may be partly due to the anterior cornual cells and the paralytic thalamic hyperactivity as suggested by Sherrington.

The question then arises as to the cause of the extensor rigidity which occurs after cerebellar ablation, and the cause of the bilateral effect after unilateral ablation.

The condition cannot be an irritative one since it is the reverse of that obtained by stimulation of the cerebellar peduncles, and again the removal was always performed by a clean incision. Sharply cut incisions always produce paralytic effects in other parts of the nervous system.

Sherrington[2] suggests that these results with the other transitory phenomena following cerebellar ablation may be due to paralytic over-activity of the centres in the posterior part of the optic thalamus due to removal of the cerebellar control. He points out that with each variety of cerebellar lesion there is repeated the same class of effects; effects which can be produced by stimulation of the posterior part of the thalamus.

The difficulty of regarding this as the whole solution appears to lie in the fact that the division of the superior peduncle has very little immediate effect as regards rigidity, and that this rigidity is greatly increased by division of the mesencephalon higher up, by which, as has been shown above, tracts which come from the thalamus are divided, and the tracts whose division causes rigidity are not the thalamo-pontine fibres.

[1] Ferrier. *Brain*, XVII. p. 1. 1894.
[2] Sherrington. *Schäfer's Physiology*, II. p. 907.

Hence the rigidity while under the anæsthetic cannot be due to a paralytic hyperactivity of the thalamic centres.

It appears possible to explain the rigidity occurring after mesencephalic section and cerebellar ablations in the following way.

The thalamus has been shown to exert a continuous inhibitory control over the anterior cornual cells of the opposite side by way of the crossed thalmo-spinal tract.

Deiter's nucleus appears to exert a continuous adjuvant influence on the homolateral anterior cornual cells by way of the Deiter's spinal tract. Section of this tract causes the homolateral abolition of rigidity due to mesencephalic transection. The cortex of the cerebellar hemispheres and vermis normally exerts an inhibitory control over the Deiter's nucleus of the same and opposite sides, but most over the homolateral one. In support of this latter part are the following observations.

Sherrington[1] and Löwenthal[2] have independently shown that stimulation of the dorsal surface of the cerebellar hemispheres over a large area from the mesial part onwards and the vermis causes a relaxation of rigidity due to previous mesencephalic transection. The relaxation is more marked homolaterally. Ferrier[3] and Russell[4] have shown that fibres from the vermis degenerate towards the vestibular and Deiter's nuclei. Removal of the vermis causes marked extensor rigidity, which can be explained by the removal of the control over the Deiter's nuclei. Similarly bisection of the vermis causes bilateral rigidity just like decerebrate rigidity. This is of very short duration, and can be explained by depression of function, to shock, and vascular lesion. Both the above results have been obtained during the course of these experiments.

On this assumption the rigidity following cerebellar ablation can be explained. Removal of the greater part of a hemisphere causes rigidity since the cerebellar cortical control is cut off. Removal of Deiter's nucleus as well causes the rigidity to remain on the opposite side, with relaxation on the homolateral side.

The rigidity due to removal of the tentorium can be also explained in this way. The removal interferes with the vascular supply of, and causes shock to, the cerebellar cortical cells and so the Deiter's influence is uncontrolled. No fibres however have been seen to go from the dorsal

[1] Sherrington. This *Journal*, XIX. p. 26.

[2] Löwenthal and Horsley. *Proc. Roy. Soc.* LXI. p. 20. 1897.

[3] Ferrier and Turner. *Proc. Roy. Soc.* 185, B, p. 743. 1893. *Phil. Trans.* CV.

[4] Russell. *Proc. Roy. Soc.* p. 57. 1894.

part of the cerebellar cortex towards Deiter's nuclei, but the control may be exerted through stations in the pons.

Then as far as decerebrate rigidity is concerned it would appear that the hypothesis of Dr Hughlings Jackson is upheld. The thalamo-spinal system is the tract along which the inhibitory influence normally exerted upon the anterior cornual cells passes, and the Deiter's spinal tract is that along which the adjuvant influence normally is transmitted. It would however appear further that the cerebellum normally exerts an inhibitory influence on the Deiter's nucleus since removal causes rigidity. These experiments also show that the cerebellar influence is exerted mainly upon the muscles working the larger joints and is mainly extensor.

The results, however, differ from the hypothesis in that the arms are more affected than the legs.

The results are in accordance with the results of clinical and pathological observations. Bastian[1] and Bowlby[2] have shown that in man with complete transverse lesions of the cord rigidity and increased reflexes do not occur, but that the resulting paralysis is a flaccid one and the knee jerks are absent. In these cases the cerebellar influx is cut off from the anterior cornual cells. In animals, however, this does not occur, for in them after complete transverse division of the spinal cord rigidity and increased reflexes occur. The difference between the results obtained in animals and man is due to the great degree of independent vitality of the anterior cornual cells in animals. This has been demonstrated by Ferrier and Goltz. The latter in fact even succeeded in teaching dogs on whom transection of the cord in the mid-dorsal region had been performed to walk with their hind limbs. In animals after transection of the cord in the mid-dorsal region the knee jerks now become increased; in monkeys, however, the knee jerks only come back 7—10 days after the operation, the independent vitality of the cord being much less than in the dog or cat.

The difference therefore between the results in man and animals is wholly due to the difference in the independent vitality of the cord.

In these experiments it was also seen that the independent vitality of the anterior cornual cells of the cat is very great, since after transection below the calamus scriptorius a fair amount of tonus was present in the muscles, and in some cases the tail, where it was not previously elevated, became erect and stiff.

During the course of these experiments it occasionally occurred that

[1] Bastian, *loc. cit. supra.*

[2] Bowlby, *loc. cit. supra.*

transection of the mesencephalon produced a primary flexor rigidity which subsequently gave place to an extensor rigidity.

In some cases also when Deiter's influence was completely removed by transection at the level of the trapezium the extensor rigidity gave place to a very marked flexor rigidity; the animal was crouched up, the back was arched, the head and neck drawn in, the limbs were flexed and drawn up under the animal, and a good deal of traction had to be employed to overcome the flexor spasm.

The following summary can be drawn from the examination of the above phenomena.

1. Decerebrate rigidity is not due to removal of a cortical influence, but a thalamic one.

2. Decerebrate rigidity does not appear to be caused by division of the anterior cerebellar peduncle.

3. The thalamic influence is transmitted along a tract which decussates high up in the mesencephalon.

4. The rigidity is abolished by complete removal of a cerebellar hemisphere or by division of the Deiter's spinal tract. The abolition is unilateral.

5. The maintenance of the reflex arc is necessary for the existence of rigidity, since division of the afferent nerve abolishes it or prevents its occurrence.

Division of the afferent tracts in the cord has no influence.

6. Rigidity can be inhibited by stimulation of the cerebral cortex or cerebellar cortex. Stimulation of the cerebellar cortex inhibits the condition, on both sides, but chiefly homolaterally.

7. Rigidity is uninfluenced by previous degeneration of the pyramidal tracts.

8. The rigidity is extensor and affects the trunk and girdle muscles especially.

9. A rigidity like decerebrate rigidity occurs after removal of the tentorium, division of the superior vermis.

10. A rigidity like decerebrate rigidity but most marked homolaterally occurs under anæsthesia after partial unilateral cerebellar ablation.

b. The Knee Jerks.

Seeing the effect of the removal of the cerebellar hemisphere and Deiter's nucleus on the condition of decerebrate rigidity, it was thought interesting to note the changes in the knee jerks after removal of the

cerebellar hemisphere in successive slices and also after section of the superior cerebellar peduncle. The experiments were performed on dogs and cats.

Russell[1], experimenting on dogs, has shown that removal of one cerebellar hemisphere, the removal in some cases involving and in others leaving Deiter's nucleus intact, produces immediately, if the anæsthesia be not too deep, a marked increase in the knee jerk of the homolateral side, and a diminution of the contralateral. The increase on the homolateral side in his experiments was so marked as to give rise to clonus and tonic extension on tapping the tendon.

He further showed that removal of the homolateral cerebellar hemisphere caused a further increase in the knee jerk which had already been exaggerated by previous removal of the contralateral cerebral hemisphere; and if the cerebral hemisphere was removed on one side, to give rise to exaggerated reflexes on the contralateral side; removal of the cerebellar lobe on the same side as the cerebral removal gave rise to a great diminution in the increased jerk.

Removal of half of the posterior part of the vermis gives rise to an increase in the knee jerk of the same side.

Ferrier[2] noted that removal of one cerebellar hemisphere produced increased knee jerks, but he was unable to detect any difference on two sides. He experimented with monkeys.

Russell[3] mentions the fact that when experimenting with monkeys the same marked increase in the knee jerk is not obtained after cerebellar ablation, as it is with dogs.

In the case of dogs Dr Russell's results were easily verified, viz. the increase of the knee jerk homolaterally and the diminution contralaterally. Removing the lateral lobe of the cerebellum in successive sagittal slices showed that this marked change did not occur till the greater part of the lateral lobe had been removed.

In cats the alteration in the knee jerks was never so marked as in the dog after cerebellar ablation. The most usual result was that when about half of one cerebellar hemisphere was removed by a sagittal section the knee jerks became increased on both sides; the increase was not usually very great. When nearly the whole lobe had been removed the knee jerks were usually brisker. In some cases the contralateral jerk was diminished. In no case was the increase so great on the homolateral

[1] R. Russell. *Proc. Royal Soc.* LIII. p. 430. 1893.
[2] Ferrier, *loc. cit. supra*; also *Brain*, XVII. p. 14. 1894.
[3] R. Russell, *loc. cit. supra.*

side as to give rise to clonus of the rectus on tapping the infrapatellar tendon. Subsequent mesencephalic transection always equalised the two jerks, which became still more exaggerated as the result of this than with the cerebellar ablation alone.

Removal of half of the posterior part of the superior vermis always gave rise to an increase in the knee jerk on the same side, in agreement with the result obtained by Russell.

Sections of the superior cerebellar peduncle just after emerging from the cerebellum produced in the majority of cases a marked increase in the knee jerk of the same side. In some cases so brisk a jerk was obtained that rectus clonus was elicited. In some cases, however, the lesion gave rise to a diminution of the homolateral jerk. In one of the animals there had been a lot of hæmorrhage, in others a large amount of shock; in cases where the tentorium had been very carefully removed the increase always took place.

In the cases of section of the superior peduncle there was never any change in the contralateral jerk. Subsequent mesencephalic transection equalised the jerks, but the exaggerated homolateral jerk remained about the same.

From the results it would appear that,

1. In dogs removal of one lateral lobe of the cerebellum causes the homolateral knee jerk to become increased, the contralateral to be diminished, in total agreement with Risien Russell's results.

2. In cats the same operation produces bilateral increase with occasional instances of the condition as in (1) above.

This is in confirmation of Ferrier and Turner and Russell in monkeys.

3. Removal of one lateral half of the posterior part of the vermis causes an increase in the homolateral knee jerk, as was shown by Russell to occur in dogs.

4. The increase in the knee jerk after piecemeal ablation of one cerebellar hemisphere occurs when about more than half of the lateral lobe has been removed.

5. Section of the superior peduncle causes an increase in the homolateral jerk.

6. Removal of the cerebral influence after removal of the influence of one cerebellar hemisphere produces a further exaggeration of the knee jerk.

These results must be regarded as paralytic. In these experiments they were all made with a sharp knife and no irritation could exist after the moment of section.

As regards the production of these phenomena there are two possible hypotheses. One is that the cerebellar hemisphere of one side exercises an energising control over the opposite cerebral hemisphere, enabling the cerebrum to exercise a more marked control over the opposite anterior cornual cells.

According to this, removal of one cerebellar lobe or section of the superior peduncle should produce an increase in the homolateral reflex, which occurs experimentally. The hypothesis is, however, insufficient, since it will not explain the further increase in an already increased jerk following removal of the cerebral or cerebellar control by subsequent removal of the other control. It has also been previously shown in this paper that as far as decerebrate rigidity is concerned the cerebellum does not exercise a large amount of continuous control over the opposite cerebral hemisphere as is evidenced by the results in the rigidity following section of the superior peduncle.

The other hypothesis is that the cerebellum exercises a direct control over the anterior cornual cells of the same side.

Thus removal of the cerebellar control causes an increase in the reflex of the same side and also still further increases a reflex already exaggerated by removal of its cerebral control. It will, however, not explain the diminution of the contralateral jerk in dogs or the contralateral increase in cats and monkeys.

The experiments quoted here show that this increase is obtained when a large part of one lateral lobe has been removed or when half the vermis has been taken away. It does not appear that removal of Deiter's influence has any further effect on the knee jerks already exaggerated by partial removal of a cerebellar hemisphere.

Ferrier and Turner[1], and Russell[2], also noticed that the increase occurred in the jerk whether the Deiter's spinal tract was degenerated or not. It would therefore appear that the exaggeration of the jerks is not due to abolition of the function of Deiter's nucleus. Therefore the facts as brought out by experiments on knee jerks seem to warrant the same conclusion as was brought out by consideration of decerebrate rigidity, viz., that the cerebellar cortical cells exert an inhibitory control probably upon Deiter's nucleus, and so removal of this control causes increase in the knee jerks of both sides; the homolateral increase being the most constant.

[1] Ferrier and Turner, *loc. cit.*

[2] R. Russell, *loc. cit.* Both these papers say in some cases the Deiter's nucleus was uninjured, in others not.

EXCITATION OF THE CEREBELLAR PEDUNCLES.

In these experiments it was endeavoured to arrive at some idea of the effect of the cerebellum or the pontine nuclei and Deiter's nucleus by stimulating the middle and superior peduncles. With the view of excluding the pyramidal system and the effects of its stimulation, in several cases the pyramidal tracts were divided on one side or on both sides by transection of the cerebral hemispheres and allowing them to degenerate. The peduncles were exposed by removing some of the calvarium over the posterior part of the cerebrum, then removing the tentorium and part of the posterior fossa of the skull. The dura was turned off the cerebellum and cerebral hemisphere, the cerebral hemisphere was drawn upwards and forwards, and the flocculus and adjoining parts were drawn backwards and away from the middle peduncle, the anterior part of which was thus exposed.

To expose the superior peduncle the middle was cut through at its anterior part. This operation was a difficult one and was frequently attended by a large amount of shock and hæmorrhage.

Anatomy of the region stimulated. In exposing the middle cerebellar peduncle, the posterior corpus quadrigeminum is at the upper part of the area of the brain stem exposed, the crusta cerebri just comes in at the anterior and ventral angle, the lateral fillet occupies the interval between the corpus quadrigeminum and its brachium, the crusta cerebri and the middle cerebellar peduncle. The IVth nerve passes from behind the posterior corpus quadrigeminum ventralwards over the fillet to the base of the brain.

During the course of the experiments the IVth nerve was frequently stimulated and always the same result was obtained, namely, rotation of the homolateral eye upwards and outwards.

Ferrier states that stimulation of the posterior corpora quadrigemina gives rise to barking and other cries, dilation of the pupils, direction of the eyeballs and head to the opposite side and upwards, retraction of the ears, trismus, retraction of the angles of the mouth and ears, and ultimately opisthotonos.

In these experiments stimulation of the lateral aspect of the posterior corpora quadrigemina gave rise to no movements of the eyes or body movements. A cry, if there was one, could not be heard because of the tracheotomy. Stimulation of the fillet, however, gave rise uniformly to pricking and turning back and down of the contralateral ear, rotation of the eyes to the contralateral side. The head movements of course were

not examined since the head was fixed. This result is entirely in accordance with the anatomical relations of the fillet and the posterior corpora quadrigemina, the movements being of a similar nature to those that would naturally occur from the stimulation of the ends of the contralateral auditory nerve; the animal of course turning its head and eyes to the side from whence the sound came and pricking the ear towards it to localise it.

From an examination of the results obtained in this investigation it appears that the stimulation of the middle peduncle produces rather varying results as regards the ocular positions. In the majority of cases the movements are bilateral; when the eye of one side only was affected it was usually the homolateral eye, less frequently the contralateral eye only was affected. The usual movement was a conjugate movement towards the side of the stimulation with accompanying upward or downward rotation. In a few there was conjugate deviation to the contralateral side. As the point of stimulation is very near the lateral fillet it might be assumed that these latter results were due to stimulation of these fibres, but the characteristic accompanying ear movements which occur with stimulation of the lateral fillet were not observed.

In many cases nystagmus was produced, which in some cases persisted after removal of the stimulus.

The stimulation of the superior cerebellar peduncle also produced ocular deviations which were most frequent in the homolateral eye. In some cases they were merely a repetition of those obtained by stimulation of the middle peduncle; in others the direction was somewhat altered, an upward rotation in one case being replaced by a downward in the other, or an inward by an outward. In some the direction was the exact opposite of that produced by stimulation of the middle peduncle.

Ferrier showed that stimulation of the cerebellar hemispheres, flocculus, and vermis give rise to bilateral movements of the eyes, both eyes moving in the same direction. He also noted contraction of the pupil on the side of stimulation[1].

Russell[2] as the result of ablation of one cerebellar hemisphere found also that at first the ocular deviations are bilateral, but they are skew deviations, the homolateral eye is rotated up and out, the contralateral down and in. When only one eye is affected it is the contralateral eye, the eye in this case is rotated out with a varying degree of downward rotation.

[1] Ferrier, "Functions of the Brain," p. 187.
[2] Risien Russell. *Phil. Trans.* 185, B, p. 839.

The effects on the homolateral eye are not permanent. Russell showed that they pass off very soon in this eye, and after a few days they pass off the contralateral eye.

The effects obtained by stimulation of the peduncles in these experiments are not uniformly the reverse of those of section, in some cases the deviation in the homolateral eye was the reverse of the results obtained by Russell after unilateral ablation. These experiments thus tend to confirm those of Ferrier and Russell, that the cerebellum exercises a bilateral control over the eyes.

In all cases stimulation of the middle peduncles produced facial and nasal contractions. These were usually bilateral and occurred most markedly on the homolateral side.

The muscular movements elicited by stimulation of the middle peduncle were very definite. The pectorals and trapezii were always thrown into a state of contraction. The effect was as above usually bilateral with most marked homolateral effects. In many cases definite contractions of the erector spinæ were noted so that the neck was drawn up and back, and the trunk extended. The forelimbs were always more affected by the stimulation of the peduncle than the hind limbs. As before, the results were usually bilateral with the same homolateral preponderance, the contralateral effects were usually very slight or were absent.

Stimulation of the middle peduncle produced also protrusion of the forelimbs, with elevation and flexion at the elbows. The paws were only occasionally influenced, the movement then was opening out and extension of the claws. In the hind limbs the movements were not very marked, there was usually some twitching of the thigh muscles of or flexion of the homolateral limb.

These results, especially the facial, pectoral and trapezial contractions, were not due to escape stimulation since they were only produced by the stimulation of the peduncle.

Stimulation of the superior peduncle was not successfully performed in many cases since the amount of shock produced by the operation was so great that stimulation produced no result. In those cases in which results were obtained from stimulation of the superior peduncle the movements were nearly the same as from the stimulation of the middle peduncle. Bilateral shoulder girdle and trunk muscle movements were obtained. The contractions as before were most marked on the homolateral side. In one case the animal's body rotated towards the side of the stimulation. In almost all cases there were movements of

the homolateral forelimbs, protrusion at the shoulder and flexion at the elbow; movements of the contralateral forelimbs were less marked. In the hind limbs, movements were not frequently obtained, the homolateral limb was usually flexed.

It is to be observed that these movements were obtained in cases where the pyramidal tracts had been previously caused to degenerate on one, or other, or both sides, and that they were the same as when the tracts were intact. It must therefore be concluded that the movements were due to the stimulation of the cerebellar peduncles and not due to stimulation of the pyramidal tracts.

From these results it appears that the cerebellum exercises a control over the muscles of the body and that the control is in the main exerted on the homolateral side, though as these experiments show the contralateral muscles are also influenced, but to a less degree. Again the control appears to be chiefly over the muscles of the trunk and body girdles, the finer muscles of the limbs do not appear to be much influenced by stimulation of the peduncles.

It was also frequently noticed during the operations of removal of a cerebellar hemisphere or of section of the superior peduncle that at the moment of section both forelimbs were protracted, drawn up, or flexed at the elbows.

These experiments do not agree with those of Binswanger[1] who called attention to the existence in the tegmental region of the pons of a convulsive centre which on excitation produces convulsive movements of the trunk, neck, and limbs. He also said that this centre could be brought into action reflexly by stimulation of the fifth nerve. Never in the course of these experiments were convulsive movements obtained by stimulation of the middle peduncle.

Sherrington[2] and Löwenthal[3] have called attention to the fact that stimulation of the vermis and the adjacent part of the dorsal surface of the cerebellum gives rise in animals which are in a state of decerebrate rigidity to a relaxation of the spasm, most markedly on the same side and in the forelimbs. This result has also been noted in the experiments, but in addition it has been seen that stimulation of the middle peduncle produces a relaxation of the rigidity which has been previously described as occurring after removal of the tentorium and manipulation of the mesencephalon and adjacent parts of the cerebellum, and which is exactly

[1] *Archiv f. Psychiat.* XIX. 1888.
[2] Sherrington. *Proc. Roy. Soc.* LX. p. 382. 1896.
[3] Löwenthal and Horsley. *Ibid.* LXI. p. 20.

like typical decerebrate rigidity. The experiments above mentioned are confirmed and expanded by these, and they confirm the fact that stimulation of the cerebellum can cause an inhibition of the spasm, and again the effect is chiefly homolateral.

When the position assumed by stimulation of the middle peduncle and the immediate transitory effect of division of a cerebellar peduncle is compared with the position assumed in decerebrate rigidity it appears that the two positions are as nearly as possible the converse of one another.

In decerebrate rigidity the limbs are in a position of extreme extension, the head is retracted, the chest is frequently in a state of full inspiration, whereas with stimulation of the middle peduncle the limbs become flexed at the elbow and knee, become drawn up, the body becomes extended. It would thus appear that the immediate results following cerebellar ablations, etc., are paralytic and not irritative.

From the study of the results of these experiments it appears that,

1. The cerebellum is capable of producing through its peduncles muscular movements.
2. The movements are in the main homolateral.
3. They affect chiefly the trunk and girdle muscles.
4. They are independent of the pyramidal tracts.

EXCITATION OF THE MESENCEPHALIC TRACTS.

In dealing with this subject the known descending paths in this region and their course may be mentioned. These are the quadrigemino-spinal or tecto-spinal, the rubro-spinal, the thalamo-spinal, the thalamo-bulbar.

The following general results were obtained on attempting to stimulate these tracts.

The brain as before was removed in slices by successive coronal sections, and after removal of each slice the cut surface was stimulated with electricity, and as before the animal was under the influence of the anæsthetic and the carotids had been ligatured. The experiment was performed both on animals in which no previous lesion of the pyramidal tracts had been made and on those in which one or both pyramidal tracts had been previously caused to degenerate.

It was then found that stimulation of the cut surface of the optic thalamus gave no result till the section passed through the posterior

end of the thalamus. Then stimulation of a large area produced movements like those of locomotion.

It was found that the homolateral forelimb was retracted, while the contralateral limb became extended, even more than it was from the decerebrate rigidity due to the section. The hind limbs on the contrary were usually affected so that the homolateral was extended, the contralateral flexed. Sometimes both hind limbs were flexed and the trunk muscles involved so that there was curving of the body to the homolateral side.

These results were obtained also in cases where one or both pyramidal tracts were degenerated.

In sections farther back the area which gave rise to these results was more localised, it was below and to the side of the posterior longitudinal bundle and could be traced as far as the medulla.

The current required to bring it into action was usually weaker than that required to elicit pyramidal movements. Continued stimulation gave rise to a coarse clonus.

These results therefore show that:—

1. There is another motor tract beside the pyramidal tract.

2. The act of walking is not wholly dependent on the cerebral cortical cells, but the function is subserved by basal centres.

3. The centre lies in the posterior part of the optic thalamus and in the mesencephalon.

4. The centre appears to lie in the medium nucleus of the thalamus, and the tract is the rubro-spinal tract or one that passes down with it.

That the act of walking is not dependent on the integrity of the cerebral cortex has long been proved experimentally. These are the well-known experiments on the lower vertebrates, and in mammals the same has been shown.

Thus according to Christiani, rabbits are no longer capable of progression if the optic thalamus be removed as well as the cerebral cortex. Goltz removed the cerebral hemisphere in a dog and kept it alive for 18 months after the operation; the animal was able to walk restlessly about its cage. At the autopsy it was seen that all the cerebral hemispheres had been removed except the basal part of the temporal lobes and the cuneus, and these with the corpus striatum, the optic thalamus, and left colliculi were in a state of yellow softening.

The experiments also showed a marked difference between the animals in which only the cerebral cortex had been removed and those in which the thalamus had been removed as well. In the former the

animal appeared to be able to perform apparently spontaneous movements, which also appeared to be rhythmically repeated. These actions were probably not spontaneous, but reflex responses to excitations of the retinæ and auditory nerves, and it would appear that if the centre were once set in action it would go on automatically till the animal was exhausted.

In the case of animals in which decerebrate rigidity has been caused by transverse sections of the mesencephalon Sherrington has shown that reflexes can be obtained which actuate the trunk, neck, and limbs as a whole and treat them as parts of a single motile apparatus. He concludes that there must exist behind the diencephalon a nervous mechanism which can co-ordinate the action of the great regions of the skeletal musculature, and the character of the combined movements leaves little room for doubt that the combination is subservient of quadrupedal progression. The movements thus obtained have a diagonal relation; if the arm of one side is flexed the contralateral hind limb is flexed, and the other two limbs are extended. These movements are exactly like those obtained by stimulation of the tract above described. In the course of the experiments on decerebrate rigidity this experiment was also performed and the above results were obtained. It was found that the reflex could best be obtained when the section was far forwards, when it was made farther behind the reflex did not spread to the hind limbs but was only obtained in the forelimbs. In all these cases the long latent period was very marked.

SUMMARY [1].

From the consideration of the results of the experiments discussed above, the following summary and conclusions may be drawn:

1. The optic thalamus contains in its posterior part on each side a centre which controls the co-ordinated movements of walking.

It is possible that some of this centre extends to the corpora quadrigemina.

The path by which control is effected appears to be the thalamo-rubro-spinal.

The function of this tract is quite independent of that of the pyramidal tracts since the centre and tracts cause the same movements when the pyramidal fibres are completely degenerated on one or both sides.

[1] See also the summary at the end of each section.

This observation confirms those obtained by Probst, Starlinger, and Rothmann, who obtained movements in the limbs on stimulating the cerebral cortex after complete pyramidal degeneration.

It also confirms the results of the co-ordinated reflexes obtained by Sherrington in cases of decerebrate rigidity.

2. The optic thalamus exerts an inhibitory control over the opposite anterior cornual cells. The path along which this control is exerted decussates high up in the mesencephalon and is probably along the thalamo-rubro-spinal tract.

3. The optic thalamus does not appear to exercise any marked continuous control over the opposite cerebellar hemisphere by way of the thalamo-pontine, ponto-cerebellar fibres.

4. The cerebellum does not appear to exert a continuous controlling function on the opposite optic thalamus by way of the superior peduncle, and results so interpreted may be really due to injury of the deeper mesencephalic spinal tract.

5. The cerebellar cortical cells appear to exert an inhibitory action over Deiter's nucleus of both sides, but the effect is most marked homolaterally.

6. Deiter's nucleus appears to exercise a continuous adjuvant action on the homolateral cornual cells along the Deiter spinal tract. This action is normally kept in check by the cerebellar cortical cells, since removal or depression of the function of these cells causes rigidity to occur.

Stimulation of these cells causes the relaxation of pre-existing rigidity.

7. The cerebellum through its peduncles governs the skeletal muscles and is capable of producing movements. The control affects both sides of the body, but the homolateral control is the most important. The muscles especially affected are the trunk muscles and the girdle muscles.

8. For the maintenance of the tonus of the muscles the reflex arc is necessary. The ascending spinal tracts do not appear to be necessary.

From these results it is again proved that movements of great complexity such as those of progression can be carried out in the absence of the cerebral cortex altogether, and that this mechanism exists in the optic thalamus at its posterior part. The muscular mechanisms thus centred together are of wide spatial distribution and employ the limbs of both sides and the trunk, thus differing from the highly specialised

cerebral cortical mechanisms which employ much more limited groups of muscles.

The broad structural linkage of the cerebellum with the thalamus according to these experiments appears to function in keeping those two important structures in co-ordination.

In conclusion I have great pleasure in thanking Sir Victor Horsley for his constant advice and assistance during this investigation, which also owes its inception to him.

APPENDIX[1].

It was always observed that the movements produced by faradisation of the mesencephalic tracts were much coarser and slower than those obtained from pyramidal excitation. The movements were also elicited with a weaker current than that necessary to produce movements by stimulation of the pyramidal tracts.

In the cases of rigidity the forelimbs were always more affected than the hind limbs.

In cases where the rigidity was less marked, it was noticed that in the forelimb the rigidity was present in the triceps and not in the biceps, and in the hind limbs the mass of the vasti was especially affected.

In the course of these experiments it was frequently noticed that the respirations became affected.

Division of the mesencephalon frequently produced exaggeration and acceleration of the respirations.

A similar result was obtained by stimulation of the cut surface of the mesencephalon and of the middle cerebellar peduncle.

In some cases of decerebrate rigidity the chest was in a position of full inspiratory spasm, the animal being in an apnœic state for some time, the heart continued to beat quietly, then respiration would take place at long intervals till normal quiet respiration was established.

During the course of these experiments other structures were stimulated with very definite results.

In all cases where the mesencephalic tracts were stimulated the posterior longitudinal bundle was stimulated. In no case did stimulation of the distal end of the cut bundle give rise to any movements. According to Ramón y Cajal and Van Gehuchten the mesial longi-

[1] A few miscellaneous observations may here be put on record as they do not immediately fall under the main headings of the previous pages.

tudinal bundle is in part a descending path from visual and auditory centres to the spinal cord subserving especially cervical motor mechanisms.

In several cases the posterior end of the ventricular surface of the optic thalamus has been stimulated. No movements of the limbs were obtained, head movements could not be observed owing to fixing of the head. In all the cases horizontal nystagmus occurred to the side opposite the stimulation.

In other cases in the experiments in which stimulation of the middle peduncle has been performed, the lateral fillet was stimulated. The results were conjugate movements of the eyes to the opposite side, pricking and turning the opposite ear down and out.