

# B R A I N .

OCTOBER, 1886.

## Original Articles.

### ON THE EXCITABLE AREA OF THE CORTEX, AND ITS RELATIONS TO THE COLUMNS OF THE SPINAL CORD.

*A Reply to Professor HORSLEY.*

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FIFTEEN years ago, when repeating the experiments of Hitzig and Fritsch on the excitable area of the cerebral cortex, I found some new properties of this cortex, and gave a new explanation of the well-known results of its electric excitation. These facts, previously announced in the 'Giornale Imparziale Medico' of Florence, were described first in the appendix to the second edition of my 'Lezioni di Fisiologia Sperimentale,' Florence, 1873, p. 523-540, and later in the first part of the 'Rivista Sperimentale di Frenatria,' for 1876, and in Pflüger's 'Archiv f. Physiologie,' vol. xxx. (1883) pp. 212-275, and in the same journal, vol. xxxiii. (1884) pp. 264-271.

I cannot repeat all, nor even the principal part, of what I said in the above quoted papers; but it seems to me that Prof. Horsley, before undertaking the criticism of my views on the subject, should have made himself thoroughly acquainted with these memoirs, where, I believe, I have plainly justified my opinions. He should not have directed his criticism against a single paper by Dr. Huggard, that was not intended to demonstrate the reality of my theories, but merely to record

some facts, which had struck the author whilst working in my laboratory.

I encouraged Dr. Huggard to publish his paper, not only because it contains some facts which could not yet be known to English readers, but also because I hoped that it would draw attention to my former publications, where the views to which he alluded have been justified by experiments, and in which my experimental methods are sufficiently set forth. If Prof. Horsley, after having read and repeated my experiments, still retains some doubt as to the reality of the facts, or to the validity of the conclusions, I am ready to enter upon a discussion with him, and to correct the errors that he can prove I committed, either in my method or in my reasoning.

But for the present I can only restate the most general results of my researches, giving merely one or two examples of the facts that have determined my judgment.

*Symptoms of the complete destruction of the Excitable Area.*

All the experiments I shall mention were made on the dog, when no other animal is specially indicated. The conclusions therefrom refer to the dog. In researches on the central nervous system, I never venture to generalise results obtained on one or two species of animals only. For other animals, such results point to possibilities which we must keep in view, but cannot take as realities, unless confirmed by special experiments. Hence I trust the reader will be able to accept the results, without being prejudiced by clinical or experimental facts, resulting from observations made on higher animals.

When we endeavour in a series of dogs to effect the destruction of all the excitable cortical area, we never can make in every case the conditions so exactly alike, that absolutely the same result is obtained in the early days after the operation. At this period traumatic irritation still exists; but after we have reached the stage which I have called "traumatic equilibrium," we distinguish certain symptoms which are uniformly present *in all the cases*; whilst others are shown only by animals with a deeper, more extended destruction of the cortical and a part of the white substance.

So we must distinguish traumatic from permanent symptoms; and among the permanent, those which are essential and general from those which, accompanying the essential ones, are present only in a certain number of cases.

I shall not enter here into an enumeration of the traumatic symptoms, observed before the so-called "equilibrium" has been reached; it may be sufficient to say, that in the first period the essential symptoms are not wanting, but they are complicated by others, which disappear after a certain time, varying from some hours to three weeks. It is evident that, contrarily to the views expressed by some authors, these do not depend upon the destroyed organs, but upon others, which are *probably* in the proximity of the wound. I take it for granted that the function of a destroyed organ is lost for ever, and cannot be re-established by compensation. It is not the loss of a function, but only the secondary effect of that loss, that can be diminished by the exercise of other organs. Hence we must expect that the true function of the cortex is lost for ever after its destruction, and that all such symptoms as disappear, either wholly or in part, are due *not to the loss of the organ*, but to the effect of traumatism. I must insist on this principle, which has been very often forgotten in the discussions on the functions of the brain.

Among the symptoms that disappear in the early period, sometimes within the first hour, after the operation, after the dog has recovered from the ether narcosis, is a diminution of all the sensations in the limbs, produced by pricking with a needle, by pressure, or by traction of the muscles. These sensibilities are not entirely lost (as sensibility for touch and cold really is), but they are feebler than in the normal state, feebler than on the corresponding parts of the other side of the body, even when on this other side there is no hyperæsthesia.

This state of the sensation (tactile anæsthesia) produces, on the side opposite to the operation, a surprising indifference to the positions of the limbs. This indifference has sometimes been taken for an expression of paralysis or paresis. It is much more evident when the dog is at rest, there being no motor current; here it cannot be *produced* by the weakness of this motor nerve-current. When the dog is walking, he shows

in a certain degree, the appearance which I have described on page 143 of my 'Physiology of the Nervous System' (Jahr. 1858-59), as the effect of the section of the posterior spinal roots, with perfect conservation of motor energy. And these observations, made first at Berne in 1856 and 1857, have been repeated and confirmed within the last few months by Dario Baldi<sup>1</sup> in a very interesting series of experiments, in which he was able to preserve for a very long time the dogs that had been operated upon. It is singular that this author, who arrives at no new conclusions, does not mention the experiments of Panizza, nor of Stilling, nor of myself, which have since been repeatedly demonstrated in the laboratory of Florence, where Baldi himself is now working.

When we stretch a limb, or bring it in a very inconvenient position, the animal—unless the equilibrium of its body be disturbed—does not resume its attitude before walking or making a general movement. Can this dog make a voluntary movement with a single limb, unless under the influence of the performance of a combined movement due to a spinal mechanism, like that of walking?

If I can prove that an intentional isolated movement is still possible, the want of such a movement in the case we consider here, can only be attributed to the want of intention. And the absence of intention can only be due to the fact, that the tactile insensibility, the existence of which is evident from other experiments, hinders the dog from being aware of the irregular position of his limbs.

A dog in which the state I am now describing lasted for six hours, was lying on the floor with the anterior left extremity stretched out at right angles with the body. I brought a metallic electrode, connected with a small Ruhmkorff coil, near the ball of the foot, and discharged three times into it a unipolar spark. After every discharge the foot was removed and brought forward, and hidden under the neck of the dog. I replaced the limb in its former stretched position, where it remained quiet till the following discharge. At the fourth time, the animal, seeing the movement of my hand and the electrode, did not wait till I was close to the foot, but before

<sup>1</sup> Baldi, 'Lo Sperimentale,' Florence, 1885.

the discharge occurred, removed the limb to hide it under his neck. Thus it is clear that a voluntary movement, a movement due to a mental impression, and not to the action of a general mechanism, was possible. It is exceptional that, as in this case, a cerebral movement occurs so early; but this exception is not a very rare one. I must remark, that the local impression which produced here a movement by a simple recollection was not of an extraordinary strength, because the dog was not put to flight, nor to a general movement; while the latter effect could easily be produced by firmly pinching the tail with my fingers. The phenomenon persisted for some weeks, till the same dog could almost give the paw when ordered.

*The indifference to position is but the effect of insensibility.*—When at a later period this insensibility is more and more reduced to want of sensibility for touch and for cold only, the indifference is less marked, though it persists indefinitely for those abnormal positions which the normal dog perceives only through the sensation of touch. And in the same manner the want of movement is reduced to those movements, which are normally excited by touch or by cold.

When the traumatic equilibrium sets in, the former are very quick, the latter become slower and slower. But before and during the return not a single muscle is paralysed, nor a single movement impossible. This is a point in which even Goltz and Munk, who were not always of this opinion, have for some years agreed perfectly with my observations. Lautenbach, who had assisted at some of my experiments, has published a paper in order to defend the same opinion. I must insist on the fact, that most of the dogs in which all the excitable cortex has been completely removed, begin after some weeks to use their anterior extremities as a hand. They give the paw at our request, if they have formerly been taught the trick. They hold a bone or a hard piece of bread, or rather they try again to hold it with the two anterior extremities. Cats, squirrels, and rats climb with the aid of the two or four extremities, and they place them, not as if they were directed by a pre-established mechanism, but in accordance to information given by visual and by central impressions generally. I have already insisted on all these points in my

first-quoted paper in Pflüger's 'Archiv,' where I have explained the remaining imperfections of these intentional movements by the loss of the direction given by the different organs of tactile sensibility. By reading this paper, Prof. Horsley may easily be convinced that the hiatuses he speaks of ('BRAIN,' *l. c.*, p. 45) exist less in my description than in what he knows of my observations. Most of the characteristic features with which Prof. Horsley tries to fill up these hiatuses must be again put out of the way, if we wish to know the real facts, and not Horsley's ideas about the facts.

It is true that in some, but far from all, cases, certain intentional and isolated movements of the fore and even of the posterior limb, did not return after several months. These are the cases in which I think (see Pflüger's 'Archiv,' 1883) that not only the cortex, which is only a channel for tactile (and thermic) impressions, but that the *centre* for these sensations, has also been damaged. I assume here a greater extension of the extirpation, though I must say that post-mortem examinations have not yet justified this opinion. I have found cases of this second category, in which the wound examined with the naked eye was only some millimetres deep, and have seen two examples of the first category, in which intentional movement returned, and in which the wound extended deeply into the brain, so as to destroy the upper part of the lateral ventricle. These examples prove, that the *loss of intentional movements is not one of the essential effects* of the complete destruction of the excitable area of the cortex.

Though the dog uses his anterior extremities for holding the food, he does not use them well. At first he makes the suitable movements to touch or to fix the bone. But while he is eating he must be guided by touch, in order to hold fast when he makes traction with the teeth. Our dog cannot feel the traction; he cannot feel whether he touches or not, and after a short time, still keeping his fore-paws fixed, he touches no more. The flexion of the toes, not being limited by the resistance of the object, becomes exaggerated, the paw is bent and kept in flexion. After a while we see the paw held in the same position, but at a certain distance from the object, the dog not being aware that he is no longer

touching it. Strength is not wanting, nay, at the beginning of the movement is sometimes *stronger* with the insensible paw; as can be shown in dogs, and better still in squirrels and cats, by offering them a piece of dry or half-dry bread hanging on a string. The impressions of the toes and claws of the insensible side, which cannot hold fast, are often more visible and deeper than the impressions of the other side.

As I said as early as 1858, in my 'Physiology of the Nervous System,' the movements which are not guided by sensibility are as often too strong as they are too weak. The same rule holds good for the strength of the movements with which the toes are placed on the ground.

It is more than ten years since I investigated this point by moistening the under side of the foot with oil, and observing the prints left by the animal walking along my long corridor in Florence. The sound produced by the contact may, on a resounding ground, give the same evidence in dogs, and still better in squirrels.<sup>1</sup>

When a dog or a cat is shaking itself (for instance, after having been sprinkled with water), the insensible extremities cannot quickly or adequately moderate and regulate the contact with the ground; they slip, and the animal may fall on the same side. On this point we must correct what Prof. Horsley (*l. c.*, p. 45) says of the position of our dogs that have been operated upon.

Prof. Horsley believes he must draw my attention to the position of the toes. But the possibility of making the dog or the cat stand with the toes of the anæsthetic side doubled up, so that they really stand, and somehow walk, on the dorsum of the foot, has been for me for a long time<sup>2</sup> the simplest and most evident sign of anæsthesia of touch. For many years I used to make my students try for themselves to double up the toes of the two sides, and feel the difference in the resistance. But I must add to what has been said by Prof. Horsley, that, when the dog has returned to perfect traumatic equilibrium, he no longer, or very seldom, takes this position by himself; like an ataxic man, he accustoms himself

<sup>1</sup> See Pflüger's 'Archiv,' 1883, p. 435.

<sup>2</sup> See my 'Lezioni di Fisiologia,' 2nd edition; Florence, 1873, p. 537.

to a stronger tension of the muscles, the movements of which he no longer feels.<sup>1</sup> But we can produce the aforesaid position with our hands, when the dog is standing quietly on the table, and observe the resistance of the dog, and the persistence of the position. I believe I have clearly and sufficiently proved<sup>2</sup> that the non-resistance of the dog to this tactile excitation does not depend, as Prof. Horsley still believes, on a paralytic state, and that there exists no trace of paralysis, either in the extremities or in other muscles of the body.

We can prove in an analogous manner, as I did it in the last quoted paper for the toes, the non-existence of paralysis and the existence of tactile anæsthesia in one half of the head, opposite to the decortication of the brain. This has been shortly indicated, as early as 1873, in my '*Lezioni di Fisiologia*,' p. 538.

Should I repeat here all that I have published on that matter, this paper would assume the length of a large volume; and such a repetition would be useless. I believe I have said enough to convince Prof. Horsley, that he must study the original articles and those of Goltz and Munk, if he wishes to have an idea of the actual state of our knowledge on this subject. As I said in the beginning, Dr. Huggard's valuable articles (and Prof. Horsley seems to know only these communications, which are and must be fragmentary) contain only the indication of some new facts, which must be misunderstood when all the premises are wanting.

I have spoken of Goltz, and I must add that this author, who now adopts nearly completely my explanation of some facts, holds that tactile sensibility is not completely wanting after decortication of the excitable area, but that it becomes much enfeebled. I will not enter here on the discussion of this question, which has for us only a secondary interest, and cannot change our explanation of the symptoms. Even though a trifling remnant of tactile sensibility still remains, it is not sufficient to provoke a motor reaction in the ordinary state of the animal.<sup>3</sup>

<sup>1</sup> See the outline figure of an ataxic cat in my paper in Pflüger's '*Archiv*,' 1883, p. 275, Fig. 2.

<sup>2</sup> Pflüger's '*Archiv*,' 1883, p. 264 et seq.

<sup>3</sup> *Ibid.* vol. xxxiv. p. 465.



Already in my paper of 1876 in the 'Rivista di Frenatria,' I remarked that a dog under the influence of strychnia, seems to be nearly equally excitable to tactile impressions on both sides, though on one side he has been deprived of the cortical excitable area. It is true he is excitable, but does he receive from both sides well-localised impressions? Some facts I have witnessed lead me to doubt that he knows that he has been touched on the anæsthetic side.

All I have said till now relates to full-grown or nearly full-grown animals. Is it the same for very young dogs? New experiments must be made on this subject. But I must add that in two instances, among more than 200 experiments, the state I suppose to exist in new-born animals was still existing in the latter part of the first year. And this *on one side only*. The other side of the brain showed the ordinary effect of decortication.<sup>1</sup>

All these facts, and those observed and analysed by other physiologists, justify the conclusions:

(1.) That the direct and only effect of the ablation of the excitable area in fully grown dogs and many other animals is tactile anæsthesia as observed by me, and insensibility for cold as first observed by Herzen.

(2.) That this effect persists without diminution as long as the animals live or can be observed.

(3.) That the simple and the co-ordinated muscular movements, or muscular innervations, are not involved so as to become either feeble or paralysed.

(4.) That some defect or some hesitation in the ordinary movements may arise in variable intensity through want of tactile sensation.

Such a state cannot be called paralytic, as Prof. Horsley would still call it. Seeking a name for it, the expression *Ataxy* presented itself very obviously. If I was wrong in adopting it (and Herzen followed me in that course, and Wernicke seems to approve of it), Prof. Horsley may propose a better one, and I shall accept it.

<sup>1</sup> See for one of these cases the paper of Herzen and Löwenthal, in one of the forthcoming numbers of the 'Revue Zoologique Suisse.' The other is still living in my laboratory, and will eventually be examined post-mortem by me or my friend, Prof. N. Löwenthal.

But in the meanwhile it seems to me that we must preserve it, unless we wish to create a new one. Thus the position of the toes remains for us the best and most expressive sign of ataxy. And a dog which has lost the excitable area is not only ataxic, but appears to me, in the words of Homer :

πόλλον ἀτάξιος ἄλλων.

I have endeavoured to find somewhere on the surface of the cortex a true motor centre. I have not found one. Even after a complete section of the internal capsule of one side, there exists no motor paralysis.

*Influence of the Posterior Columns of the Spinal Cord on the Excitable Area of the Brain.*

We see from the preceding exposition that, from the beginning of my experiments, the analysis of the facts led me to the conclusion, that the pretended motor centre does not exist in dogs, and that it must really be called a sensory centre, if it is a centre at all, and not, as I believe, the most central patch of afferent channels.

A new experiment I made some years ago led me to the conclusion, that what I have considered to be an analogy between the effects of decortication of the brain, and of division of the posterior columns of the spinal cord, is more than a mere analogy, that it is identity. The fifth day after the division of the posterior columns of the cord, the excitable area of the brain loses its influence, and no longer gives rise to reflex actions in the parts of the body posterior to the division of the posterior column.

Prof. Horsley, without being acquainted with our method of experimenting, believes that he has repeated our experiments. His results were in general negative, or nearly so. I must even regard as negative the result of the experiments in which the Professor found my result "marked" or "very marked," because, in order to confirm my experiment, I must demand that the result shall be confirmative in a quite *absolute* manner. It was not so, and this is not surprising. It would have been much more surprising, had he by his method been able to confirm my results.

I know only one method for making the resection of a part of the posterior columns without damaging the rest of the spinal cord. It is the method indicated in my text-book,<sup>1</sup> and to which I have always returned after having examined other apparently simpler and easier methods. By it all pressure is avoided on the so-called motor tract, when we make the first incision very obliquely and very incomplete. That even a slight pressure on the (true) motor tract is very fatal in the operations on the spinal cord, is not, as Prof. Horsley believes, a new discovery of his own, but has been largely studied and set forth in my text-book, p. 278. I had given there a list of the different authors who, up to that period (1858) had been led into error by the effects of unintentional pressure on the motor elements. The resistance of these latter to pressure is less than the resistance of the sensory elements. I believe that, by this remark, I have given the key for the explanation of some paradoxical facts met with in clinical observation.

Prof. Horsley kills his dogs about the fifth day, and excites the brain with an ordinary Du-Bois Reymond's coil worked with one Daniell's cell, and in one case with two. I think that currents of so high a tension and so short duration as induction currents, are not the best nor the surest means for exciting a large parenchymatous organ like the brain. Instantaneous currents must have a very great intensity to produce a reflex movement; and the so-called "motor centres" really act by a reflex process. The intensity of the current is a cause of extensive derivations, and so we are never sure to localise exactly an induction excitation in an organ the form of which is not linear. In another paper on a recently published case of Baldi, on the brain, I shall show that all his experiments have been lost by derivation into deeper parts of the brain, and even into the roots of the fifth nerve. It is for these reasons, that in experiments on the excitable area, I do not trust induction currents, and I have always made the decisive experiments with a simple galvanic battery with a rheocord. The makes and breaks are made with the hand by means of a Morse interruptor. And indeed, in my own paper

<sup>1</sup> 'Physiology of the Nervous System,' p. 242. See also 'Comptes Rendus,' 1854, p. 926.

on the experiment, which forms the object of Prof. Horsley's criticism,<sup>1</sup> and which the Professor seems to have overlooked, I recommend for the excitation the use of 4 to 16 Leclanché cells; and only exceptionally make use of a *single* induction opening current (the hammer of the instrument being fixed) as a corroborative experiment when it gives no contraction, and as an experiment of no account, when I had an effect which could not be obtained by the battery current.

We see that Prof. Horsley has been drawn into error when he says (*l. c.*, p. 47) that my method of experimentation, as far as he knows it, exists simply in exposing and dividing the posterior column on one side. I have always made, not the *division* but the *resection* of the two posterior columns, or of one. I never excited with an inductive current rapidly interrupted by vibrations of the hammer. I made the operation on various parts of the spinal cord, always by the method indicated in my book. I made and broke the current (even the induction current) with the hand,<sup>2</sup> never taking it too strong. I have much insisted on this method, which I think must give to Prof. Horsley, if he will really repeat my experiments, the same result as I have obtained in my own numerous experiments.

And this result is not, as Prof. Horsley supposes that I "would have him to believe," that the so-called motor centre becomes unexcitable, which are in strict reflex relation with the muscular groups, separated from the "centre" by the resection of the posterior columns. It is quite in harmony with my results, that after the division of the posterior columns within the fourth vertebra, he should still meet with what he calls flickerings in the trapezius, and even in the face, produced by excitation of these pretended centres.

But Prof. Horsley believes that *no* excitability is lost, but that his "centre" can no longer *transmit* its excitation, because a degeneration of the pyramidal, or, as he says, motor tracts, puts a block against the peripheral extension of the innervation. Let us now examine this opinion.

<sup>1</sup> Pflüger's 'Archiv,' xxx. p. 257 et seq.

<sup>2</sup> I must add, that during the last few weeks I have found a method of using a modification of the Clarke magneto-electric machine with mechanical interruption, which can be used for experiments on the brain, without exposing us to errors produced by derivation of the current.

*Can the Pyramidal Tract of the Lateral Columns be regarded as Motor?*

When Prof. Horsley first published his objections against my experiments and claimed them to be produced by a lesion of the motor tract, I did not understand what he meant by this expression of "motor tract," and with its unintentional lesion. The microscopical examination of transverse sections of my spinal cords, showed no accidental degeneration. That I made this examination long before Prof. Horsley could draw my attention to it, is proved by what I said in Pflüger's 'Archiv,' 1883, p. 264, where I concluded that the operation on the posterior columns did not produce a degeneration, neither total nor partial, of the pyramidal tract. But I must add here, that only a small part of the posterior grey horns was filled with white corpuscles, the nerve fibres being intact; and there was not even in these horns any trace of descending or ascending degeneration.

But why did I not understand the meaning of Prof. Horsley? I believe I have established, in my book of 1858 (and in the previous communication published by the Natural Society of Berne and the Institute of Paris), the first lines of an experimental physiology of the spinal cord. My researches have held their ground against many contrary results, which since that time have been announced, soon to disappear. So I was accustomed to my own terminology, and I did not acknowledge the existence of a special motor tract. In Prof. Horsley's last paper I find the explanation of his meaning, and it is on this occasion that he sets forth his discovery of the influence of pressure on the motor, or more properly *kinesodic*, elements of the spinal cord we have already spoken of. He had, by his method of operating, produced a more or less extended degeneration of the undivided, but compressed, lateral tract, and he erroneously supposes that I must have done the same, and that this assumed degeneration of the pyramidal tract of the lateral columns *can*, or as he thinks *must*, cause an interruption of the transmission of movement from the brain to the limbs.

The degeneration he produces enters into the category of what the French school has hitherto called a *sclerosis*. But I

have proved long ago that several forms of this so-called sclerosis do not destroy the cylinder-axis in the nerve-fibres, but are an expression of what I have called an Atelectasis of the nervous tracts.<sup>1</sup> Westphal's criticism of my view could not shake, but rather confirmed it, leading me to new confirmatory researches. In my paper on Atelectasis (1880), I arrived at the conclusion that, in many cases, the so-called sclerosis cannot interrupt the transmission of nervous influence—a conclusion which I applied at that period to the cases of posterior sclerosis described by Friedreich, Gowers, Pick, and others, where the sensibility was not at all, or scarcely, disturbed. Last year a new confirmation of my views arose from the discovery made in Paris, that a certain number of cases of sclerosis of the pyramidal tract presents no secondary descending degeneration. But a true block in the transmission, a transversal interruption, is always followed by descending degeneration of the pyramidal tract. Hence these cases of sclerosis do not produce a block. And this conclusion has been reached even by some neurologists, who formerly were my adversaries on the question of Atelectasis.

Degeneration of the pyramidal tracts is not produced in the experiments made according to my old method. I may regard as non-existing some experiments which I made after another method, with the "microtome" of Strauss-Durckheim,<sup>2</sup> in which the lateral tract was really injured.

But I can prove that this degeneration of inflammation of the pyramidal tract, even if we suppose with Prof. Horsley, and on the evidence of microscopical examination, that it has been produced in *all* my experiments on the posterior columns, *cannot be made responsible for the partial excitability I found in the so-called motor centres of the cortex.*

Prof. Horsley remarks, p. 47 of his paper, that "English neurologists, at any rate, will be interested to learn (*vide* 'Lancet,' 20th February, 1866) that Prof. Schiff is about to combat the general opinion, that the crossed lateral tract is motor." They will be still more interested to learn that I am

<sup>1</sup> See my paper on "Atelectasis Medullæ Spinalis" in Pflüger's 'Archiv,' xxi. p. 238, and also the criticism of Westphal in his 'Archiv.'

<sup>2</sup> See his 'Traité d'Anatomie comparative;' Paris, 1842, tome i. planche iv. fig. 59.

not about to combat, but I have combated for a long time this general opinion (in the case of dogs and cats), by a series of very clear and non-equivocal experiments.

My old opinion, resulting from a large number of experiments, that the lateral column does not contain fibres the section of which produces motor paralysis—an opinion which has since been fully confirmed by other experiments—may have been regarded as insufficient, as having been formed in a period (1850–58) in which no special attention has been paid to some special reflexes, which are distinguished as voluntary and unintentional, from these movements which depend upon a general and pre-reformed mechanism.

Pflüger's 'Archiv,' vol. xxx. (1883, pp. 243–248) contains a series of experiments with complete section of the pyramidal and partial section of the cerebellar tract (on one side), which prove that not only the general movements, but even the intentional ones (such as a dog giving the paw, holding the bone with both anterior legs, a cat climbing and catching objects with the limb on the operated side) are preserved, though there was not only interruption, but also a complete descending degeneration of Horsley's so-called motor tract. The degeneration, though it did not extend down to the origin of the roots of the sciatic nerve, but stopped somewhat higher, was *more complete* than that generally visible after the extirpation of the excitable area of the brain. These degenerations, and their differences in extent after injury of the brain or transverse section of the pyramidal tract of the spinal marrow, have been studied in the same animals by Prof. Löwenthal.<sup>1</sup>

Within the last few months these facts have been independently confirmed by Herzen and Löwenthal in a young cat, in which they had cut the pyramidal tract of the lateral column. This column showed very extensive degeneration.<sup>2</sup> Herzen has very well described the voluntary movements, though the cat was not kept alive long enough to allow them to be fully re-established with reference to their strength.

<sup>1</sup> See his communication in Pflüger's 'Archiv,' xxxi. p. 350, with the accompanying Table IV.

<sup>2</sup> See 'Archives de Physiologie,' April 1886, p. 20, and the plates.

Though the full energy was still wanting, it is clear that the movements were all present.

We cannot suppose that, in these animals, the preserved *direct* pyramidal tract in the anterior columns has in some degree compensated for the destruction of the lateral crossed tract, because we know now that, at least in dogs and cats, there is nothing of the nature of a direct pyramidal tract.

The experiments of mine I have just mentioned are not decisive. At the period I made them I was using the microtome of Strauss,<sup>1</sup> which must be introduced by stretching, and so I produced indeed extensive lateral compressions and degenerations outside and below the divided parts. Microscopical examination of the hardened marrow<sup>2</sup> revealed afterwards that these errors were due to the same conditions, which Prof. Horsley wrongly supposes complicated my sections of the posterior columns. And so I produced some symptoms, which at that time I attributed to the absence of the transmission in the pyramidal tract, but which really depend upon a concomitant alteration of the posterior columns, which has been found later in the hardened parts. But even before the microscopical examination I expressed my surprise (*l. c.*, p. 243) that what I took for the effect of section of the pyramidal tracts, resembled so much the symptoms of resection of the posterior columns, and that an intentional transversal section of the pyramidal and the posterior columns made together, and simultaneously, produces no other effect than section of the posterior column alone.

So I committed at that time the error recently imputed to me by Prof. Horsley, but quite in an inverse sense; I attributed too much to the pyramidal tracts, the section of which, as we know by later and more correct experiments, produces no visible symptoms, not visible at least with our methods of research.

But even in the former vicious experiments all the movements were present; voluntary, isolated movements were not wanting, though they were modified by insensibility, and so

<sup>1</sup> See Pflüger's 'Archiv,' *l. c.* p. 241.

<sup>2</sup> See Löwenthal, 'Des Dégénérationes secondaires de la Moëlle Épinrière.' Thèse; Genève, 1885.



true to the principle and the method indicated in my textbook of 1885, p. 231. I can give credit even to those former experiments establishing *that a complete degeneration of the pyramidal tract, and of all the posterior half of the lateral column, does not produce in our animals loss or diminution, either in the voluntary and isolated, or in the general movements of the parts behind the section and behind the beginning of the degeneration.*

There was still one question open, and it is for this purpose that I began two years ago a new series of experiments, conducted according to my old trustworthy method. Does excitation of the excitable area of the brain, opposite to a complete degeneration of the pyramidal tract, still produce movements of the limbs of the degenerated side? Some weeks or some months after the section of the posterior part of the lateral column (pyramidal tracts) of one side, I began the experiment by exciting the brain with a battery current, and in some cases a single induction shock. The (reflex) movements in the limb, which are normally seen by stimulation of the excitable area, were present by excitation on *both* sides of the brain. The section of the pyramidal tract did not hinder or enfeeble the effect observed in the experiments of Hitzig and Fritsch. The experiments of Ferrier (with quickly interrupted induction currents) probably do not fail in these cases; but if I had excited the brain immediately after having cut the pyramidal tract, the stimulation would have remained without result. It is essential to let the traumatic irritation pass off before testing.

When in a dog or a cat, in which one pyramidal tract has been divided and has long been degenerated (from the cervical region downwards), we make a deep and extensive extirpation of the so-called "motor-centres" of the opposite side, we can before traumatic equilibrium is reached, *i. e.* in the first weeks, observe all the symptoms which have been regarded as proving the absence of intentional movements on the side opposite to the operation, and the characteristic anæsthesia is present and maintains itself indefinitely. Hence all these symptoms produced after the second operation cannot be dependent upon the existence of the pyramidal tract, in which our previous operation had caused to degenerate

It is evident that the suppositions of Prof. Horsley, and his mode of viewing my experiments on the posterior columns, are quite erroneous. They cannot be admitted, even if it were true that I had compressed what he calls the motor tract. And they must be rejected *à fortiori*, because I had not compressed this tract.

The crossed pyramidal tract finds its nutritive centre in the neighbourhood or within the excitable area, but there is no known functional relation between this area and the lateral columns of the spinal cord.

When in 1853, after Waller's and my own experiments, I first proposed and defended the difference between the nutritive and the functional centres, there was among anatomists and physiologists much discussion and contention about this subject. Here we find another and a very striking example of this difference. This example must hold good, at least till we know something of the functions of these pyramidal tracts. It seems, after an observation of Beck, that they can be cut even in man without the loss of voluntary movement.

One of my pupils, Mr. Schopen, is now about to publish some observations of injury of the anterior part of the brain on one side accompanied by anæsthesia (for tactile excitations). In one of these cases (and in two similar I observed some years ago) the secondary degeneration was wanting in the pyramidal tracts. In two other cases there was degeneration of equal extension and intensity in the two crossed pyramidal tracts,<sup>1</sup> that is, on *both* sides, and notwithstanding anæsthesia and its allied symptoms were present only on the side opposite to the cerebral injury. The extirpation in these cases was not in the posterior limb of the sigmoid gyrus, but between the sulcus cruciatus and the frontal (supra-orbital) lobe. Without entering here into particulars, it is evident that such cases offer new proofs, that the functional and the nutritive disturbances are independent one of the other.

<sup>1</sup> The extension of the degenerations and the number of preserved nerve-fibres have been determined with the polarising apparatus. Two determinations were made for each section, one simply with crossed Nicol's prisms and one with the aid of a selenite film.

*Functions of the Posterior Columns.*

The preceding exposition shows, that the opinion of Prof. Horsley is not admissible, when he says that all we have hitherto attributed to the posterior columns may be due to a concomitant lesion of the lateral columns. He states, that in the cases of section of the posterior columns in which only a very slight lesion was found in the lateral, he could not verify the tactile insensibility found by me, and he did not investigate the insensibility for cold found by Herzen. That he could not verify the loss of tactile sensibility in all cases may depend either on the same causes which hindered Ferrier—to whom Prof. Horsley refers—from finding the insensibility after an injury of the excitable area of the brain, or it may depend on the method of dividing the columns. This division, as I have still to show, was imperfect in the beginning of his observations, and very slowly spread by traumatic inflammation to a perfect destruction of the column. If it is so, an examination during the first, the second, even the third day, might have given him an imperfect result.

It is probable that if Prof. Horsley had examined the dogs of this series as we examine the animals after decortication of the excitable area of the brain, he would have convinced himself that all local disturbances after these two operations are absolutely the same. I say *local* disturbances, and I should have added persistent, because the first traumatic disturbances are somewhat different. Besides, the brain is in relation with the entire half of the body (though I have hitherto been unable to produce a cerebral insensibility of the inner surface of the ear). The posterior column acts only on the parts below the operation.

Instead of this, Prof. Horsley contents himself with three methods of examination, which are very fallacious. Fallacious in the sense that their application leads into error, when they are not exactly limited and graduated as to act only on tactile sensibility and not on the other sensibilities. It is so, for instance, with reference to the "clip" Prof. Horsley mentions (*l.c.*, p. 49). It is true that very often I make use of a clip, as Goltz did before me, but I employ it rather for

demonstration than for researches. I have three pairs of clips of different strength and resistance. First, the strongest is applied successively on both sides of the body. Generally sensation is indicated on both sides. But I am not yet warranted thereby to conclude, that tactile sensibility equally exists on both sides, because the clip may act by pressure. I take a weaker one, and often a weaker still, and so I become enabled to conclude, that there exists a certain degree of contact well perceived on one side and not on the other side of the body.

The same precaution must be applied, in a still higher degree, when you touch the skin with a needle, or when you allow an object to fall on a sensitive surface, even when this object is simple water (see *l. c.*, p. 49). So it is evident that the mere enumeration of the three methods of testing used by Prof. Horsley do not warrant the exactness of his result.

These methods are not sufficient for making me believe, that Prof. Horsley has found three exceptions to a general rule, which have held good till now for many years, in very numerous experiments, without a single exception, and which has been confirmed in many species of mammals.

The ascending degeneration after resection of the posterior columns must go in some manner as far as the cortex, but I never pretended, as Prof. Horsley would have his readers believe, that it goes as far as the ganglion cells producing some visible alteration of these parts. I can add, after Löwenthal's examination of the brain of some of my dogs, that the visible degeneration does not reach beyond the pons Varolii, but it can be partly followed as far as the cerebellum.

If I have several times insisted on the fact, that I make not a section, but a *resection* of a piece of the posterior columns, I do not imply that this difference is of any importance for the result, but the difference is capital for the method. We can never make a complete section, and be sure of sparing the neighbouring parts; nay, we may be certain of damaging them.

*How Professor Horsley's Results were obtained.*

If it is allowable to put forth any such supposition, I think that these results are due in part to the method of operating, and partly to the mode of the excitation. Prof. Horsley makes (*l. c.*, p. 48) in a posterior column a small punctured incision, with a sharp-pointed tenotomy knife. It can scarcely be supposed that there exists a form of tenotomy knife which, with a simple puncture, divides all the posterior column, and nothing but this column. And though Horsley's figures generally show the lesion to be very exactly limited to the column, after their inspection I must assume that he made a puncture which did divide only a part, a stratum of the column, and that the rest was compressed and did disappear successively during the five days the animal was living. So a large part of the column found damaged at death may have been destroyed only three, or even two days previously. My experiments<sup>1</sup> have proved that one or two days after the resection of the posterior columns the brain remains excitable. In a more recent experiment, which will be communicated by Schopen, it remained still excitable four days after the resection of one posterior column. It must be partially excitable when a part of the posterior column has been divided only for two or three days.<sup>2</sup> So the imperfect movements perceived by Horsley after his excitation of the brain can be understood.

So we can understand how the fissure of the wound contained débris of nerve-fibres which are not present in a sharp, well-cut wound. The sides of the wound-cavity, says Prof. Horsley, "although so extremely small, showed clearly that there was some degree of tension in it." This can be understood to mean, that the walls of the fissure showed some irregularity. This must be so, even in the cases when an incomplete fissure increases by parietal inflammation. Had Prof. Horsley kept his dogs some days more, his results probably would have been more complete.

I have already spoken of the method of excitation. The

<sup>1</sup> See Pflüger's 'Archiv,' vol. xxx. p. 260.

<sup>2</sup> See the experiment of Herzen and Löwenthal on a cat. 'Archives de Physiologie,' April 1886.

use of induction currents has produced so many errors in the physiology of the brain, and more than elsewhere in the hands of English physiologists, that it is high time to give up the use of the induction coil and to return to the galvanic battery with a rheocord, which allows us to regulate the necessary variations in the strength, and in the duration of the current.

In writing the foregoing pages, it has been my aim to present the English reader with a sketch of the actual position of the question, and to give him some indications which he will seek in vain in the annual reports on the progress of physiology. I must return my best thanks to Professor Horsley for his zeal, and the interest he has shown with reference to our recent researches. I hope that this paper will engage him to undertake, on a new basis, a fresh series of experimental researches, on a subject which fully deserves the attention of English physiologists and neurologists.