ON CERTAIN RELATIONS OF THE CEREBRUM AND CEREBELLUM (ON RIGIDITY OF HEMI- PLEGIA AND ON PARALYSIS AGITANS).

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As the subject of the relations of the cerebellum and the cerebrum to the spinal motor centres, and by them to the muscles, is—in consequence of Dr. Charlton Bastian's important researches on the effects of total transverse lesions of the supra-lumbar cord—now receiving considerable attention, I have asked permission of the Editor of *BRAIN* to republish in its pages two articles I wrote a little more than twenty years (1877 and 1878) ago, one on "Rigidity in Hemiplegia," and one on "Paralysis Agitans" (Parkinson's Disease).

REMARKS ON RIGIDITY¹ OF HEMIPLEGIA.

(*The Medical Examiner*, April 5, 1877.)

With reference to several cases of hemiplegia, Dr. Hughlings Jackson expressed the opinion that the rigidity in hemiplegia is not owing to the cerebral lesion nor to the lateral sclerosis. There is evidently a duplex symptomatic condition, negative and positive; with loss of power over the muscles there is tonic action of them. Whilst the primary cerebral lesion can account for the paralytic element—the negative condition—it cannot (nor can the sclerosis in the lateral column) account for the tonic condition of the muscles, the positive element. Negative states of the nervous centres cannot *cause* positive states of muscles, they may *permit* them. His speculation is that the rigidity is owing to unantagonised influence of the cerebellum. He believes that in health the

¹ 1899. In some cases of hemiplegia there is a kind of pseudo-rigidity; I mean a rigidity from joint disease ("locking of the joints"), with no or very little muscular rigidity. I think this pseudo-rigidity occurs far oftener with left than with right hemiplegia.

whole of the muscles of the body are doubly innervated—innervated both by the cerebrum and cerebellum; there being a co-operation of antagonism betwixt the two great centres. Whilst the cerebrum innervates the muscles in the order of their action from the most voluntary movements (limbs) to the most automatic (trunk), the cerebellum innervates them in the opposite order. This also is supposed to be the order of their degree of influence on parts; and is equivalent to saying that the cerebellum is the centre for continuous movements and the cerebrum for changing movements. Borrowing medical terms to describe normal physiological conditions, the former are tonic the latter clonic. The muscles which on the above suggestions, are chiefly innervated by the cerebellum, are those which have the fewest different, the most tonic, the most “confluent” movements; the reverse for those which the cerebrum chiefly innervates. Thus, in walking, the cerebellum preserves the equilibrium of the body, tends to “stiffen” all the muscles; the changing movements of walking are the result of cerebral discharges overcoming in a particular and orderly way the otherwise continuous cerebellar influence. This is what is meant by a “co-operation of antagonism.” The cerebrum does most “work,” the cerebellum does little (if any) “work”; from continuous action of muscles balancing one another there is dissipation of energy (heat); from cerebral action there are molar movements (work).

When, however, the influence of the cerebrum is permanently taken off by disease of the cerebrum, as in hemiplegia, from the parts which it most specially governs [arm and leg], the cerebellar influence is no longer antagonised; there is unimpeded cerebellar influx and hence rigidity of the muscles which in health the cerebrum chiefly innervates. The principle, he thinks, accounts for the duplex, negative and positive, conditions in the contractures of hemiplegic children, in the athetosis of Hammond, and accounts for the rigidity and “propulsive” walk in paralysis agitans; temporary removal of cerebral influx also for tetany and certain hysterical attacks, and also for some post-epileptic conditions of movements. Physiological experiments were adduced in favour of the doctrine that two impulses arriving at a centre interfere with one another; the opposite effects of ligature in epileptiform seizures and in tetany were spoken of.

The above hypothesis is, Dr. Hughlings Jackson considers, only an application on a large scale of Thompson Dickson's¹

¹ 1899. The “Principle of Loss of Control” was, I believe, first stated by Bernard and afterwards by Anstie, and then by Thompson Dickson.

Principle of Loss of Control, a principle applicable to all nervous centres, as is, Dr. Jackson thinks, the physiological Principle of Antagonism. In healthy operations the two principles are displayed; did not centres "resist" one another there could be no independence of operations; differentiation would be practically annulled. On the other hand, if some centres could not overcome the resistance of others—discharge them by their discharges—there would be no association of operations, differentiation would be useless; and if there were not some order of "resistances" and of "dischargeabilities" there would be no orderly co-operation.

ON CEREBRAL PARESIS OR PARALYSIS, WITH CEREBELLAR TREMOR OR RIGIDITY.

(*The Medical Examiner*, March 28, 1898.)

In the *Examiner*, April 5, 1877, are reported some remarks by me on the interpretation of the rigidity in hemiplegia. I then suggested that the condition is duplex, that there is a negative condition, "cerebral paralysis," due to the destructive lesion in the corpus striatum, and a positive condition "cerebellar rigidity," due to increased cerebellar influx. The hypothesis starts with the assumption that the spinal centres receive impulses from both the cerebrum and cerebellum, which impulses in health interfere with one another (inhibit one another). If so, we should not say that the cerebral lesion, nor the wasting in certain spinal tracts consecutive upon it, *causes* rigidity; negative lesions can only cause negative conditions in muscles; in this case the paralysis. But on the hypothesis the cerebral lesion, in other words, loss of cerebral influence on the spinal centres, may *permit* the rigidity, for then the cerebellar influence is no longer interfered with, and, metaphorically speaking, "flows into the parts deserted by the cerebral influence." Hence it is better to say "unantagonised cerebellar influx" than "increased cerebellar influx." The cerebellum is healthy, or if over-active in effect,¹ it

¹ 1899. I have been supposed to hold that there is disease of the cerebellum in cases of paralysis agitans. On the contrary, as will be seen later in the text, my hypothesis implies that in this malady there is integrity of the cerebellum; the tremor and rigidity of paralysis agitans are, I suppose, the outcome of activities of perfectly healthy structures, cerebellum and anterior horns.

I do not believe that any *continuous* and *positive persisting* symptoms—positive symptoms so continuous and *persistent* as the tremor and rigidity of paralysis agitans—are ever the *direct* result of disease—of disease in the proper sense of pathological process. I think that, like those of paralysis agitans, they are always the *indirect* result of disease, and that they are directly the result of activity or of over-activity of healthy structures, that they are problems in the domain of physiology, not of pathology.

is not so because its cells have become morbidly over-unstable, but because there is less or no resistance to overcome, or, as we say, less or no cerebral influence to antagonise.

Mutatis mutandis, the same explanation applies to rigidity of the spinal and other muscles in cases of destroying lesions of the middle lobe of the cerebellum; there is then "cerebellar paralysis" and "cerebral rigidity."

I wish now to show how the hypothesis applies to the interpretation of several symptoms of paralysis agitans.

The regional distribution of this motor affection points unmistakably to the cerebrum (whether of the motor tract or cerebrum proper is not said) as the seat of the morbid anatomy. It is essentially hemiplegic, although very commonly it *becomes* two-sided. I believe that we have here, too, a minus cerebral and a plus cerebellar condition. It is sometimes asserted that there is no paralysis in what is called *paralysis agitans*; but some of those who say so add that the movements are slow, and slowness of grasp is as much evidence of paralysis as weak grasp is. To ignore the element of rapidity in estimating the "momentum" of a mobile part would be like ignoring the velocity of a cannon-ball in estimating its momentum. It is certain that we have tremor and rigidity.¹

On the supposition that the cerebral influence is diminished, we may explain the tremor—negative clonic cerebral spasm (most of the spasm in ordinary epileptiform seizures being positive clonic cerebral spasm). On the supposition that the cerebral influence is taken away altogether, we may explain the rigidity. Tremor, at least rhythmical tremor, is a minor degree of tonic spasm. If, experimentally, shocks at comparatively long intervals are sent into a muscle, there is a sequence of contractions, the analogue of tremor; but if they be sent in at exceedingly short intervals, there is tetanic contraction, the analogue of rigidity. Thus the tremor of paralysis agitans differs from the rigidity of hemiplegia, not fundamentally, but in degree; in the former the cerebral influx is diminished; in the latter it is taken off altogether. And as the disease progresses, or, as the hypothesis has it, when the cerebral influx ceases entirely, then rigidity of the parts *does* ensue, which in tremor were approaching rigidity.

(1) In health the cerebellar influx is fully antagonised; in (2) the early stages of paralysis agitans it is intermittently antagonised—the movement constituting each single tremor occurring

¹ In a few cases there is no tremor, or scarcely any; there is rigidity only.

betwixt the cerebral impulses ; in (3) late stages it is not antagonised at all, and there is such a stream of cerebellar impulses that rigidity occurs. We have cerebral paresis with cerebellar tremor ; later, cerebral paralysis with cerebellar rigidity.

So much for the parts which the cerebrum *chiefly* innervates in health, and which it slowly "abandons," and which, in consequence, the cerebellar influx gradually "invades."

In paralysis agitans there is "loss of expression" of the face, but this so-called "loss" of expression of the face is one *fixed* (starched) expression. An unvarying expression, however caused, is in effect a loss of expression. The hypothesis is that there is some facial paralysis "caused" by the cerebral disease, and some facial rigidity, owing to "permitted cerebellar influx."

On the hypothesis (see *Examiner*, April 5, 1877) the spinal muscles are those which the cerebrum influences least and the cerebellum most ; thus there is rigidity without prior tremor. This is manifested by the well-known attitude, and indirectly by the propulsive gait. The rigid state called "attitude" of the body, face, and limbs is more characteristic of the disease than the tremor, in spite of the name "paralysis agitans."

This is a mere outline.¹ It may be remarked that the general attitude often seen in cases of paralysis agitans (face, arms, and legs) is like that seen in cases of rapid locomotion ; it is at least a good caricature of the position, supposing locomotion to be suddenly arrested, and parts of the body remaining fixed. In running, the body is bent forward, the arms are flexed, &c., and the facial muscles come into vigorous action.²

As before said (*op. cit.*) the hypothesis is applied to cases of athetosis, and to the mixed condition of paralysis with twisting movements in persons hemiplegic from early life.

For *all* morbid over-mobile conditions of one side I have suggested the name hemikinesis, although I have been supposed to use it only for the athetosis of Hammond. But we have two different sub-classes ; the hemikinesis due to cerebral discharges (unilateral chorea and unilateral epileptiform seizures), and the hemikinesis owing negatively to cerebral destroying lesions with (positively) permitted cerebellar influx.

¹ With the assistance of my clinical clerk, Mr. Whitford, I tried, a few days ago, the effects of ligature on a patient, the subject of paralysis agitans, the tremor being on both sides. At first trials we obtained arrest of the tremor of the arms, even when the ligature was applied round one calf ; but at the second *séance* we got no such results. The plan is worthy of trial in slighter cases.

² 1899. The attitude in an advanced case of paralysis agitans is something like that of a feeble old man when he is going upstairs.

REMARKS, 1899.

I have written on this subject several times since 1878. (*Med. Times and Gazette*, February 12, 1881; BRAIN, January, 1888, p. 312 *et seq.*; *Brit. Med. Journal*, March 5, 1892; *Lancet*, December 12, 1896, and January 8, 1898.) In these papers I have acknowledged all the difficulties I knew of.

To the hypothesis propounded in the two papers reproduced from the *Medical Examiner*, it was objected that the "fact" that upon complete transverse lesion of the cord above the lumbar enlargement (whereby both cerebral and cerebellar influences are cut off from motor centres below that transverse lesion) the knee-jerks are exalted and that the legs are, or soon become, rigid—that these super-positive conditions exist when the cerebellum is altogether excluded—disprove the hypothesis.

I was so impressed by this "fact," as I then took it to be, that¹ I went a long way towards abandoning the doctrine of cerebellar influx, if not all the way. I then wrote: "I admit . . . that since after section of the cord right across, destroying communication with both cerebrum and cerebellum, there is increased reflex activity of the parts below, we cannot suppose that unantagonised cerebellar influx is essential for the conditions mentioned. I make this admission in consequence of some verbal criticisms by Ferrier."

Since I wrote that, Dr. Charlton Bastian (Quain's "Dictionary of Medicine," p. 1480 (1882), "Paralyses, Cerebral, Bulbar and Spinal," 1886,² and also "On the Symptomatology of Total Transverse Lesions of the Cord," *Med. Chir. Trans.*, 1890), has done far more important work on the matter than I have. He has shown that upon a complete transverse lesion of the cord above the lumbar enlargement there is, contrary to what, heretofore, had been universal physiological

¹ "On a case of Temporary Left Hemiplegia with Foot Clonus and Exaggerated Knee Phenomenon, after an Epileptiform Seizure beginning in the Left Foot," *Medical Times and Gazette*, Feb. 12, 1881.

² In this work, "Paralyses, &c.," 1886, p. 538, Bastian writes, ". . . with a total transverse lesion in the lower cervical region, nearly all reflexes dependent upon lower portions of the cord are abolished rather than exaggerated."

and medical doctrine, flaccidity of the legs and loss of the knee-jerks.

Bowlby (*Med. Chir. Trans.*, 1890), Thorburn (*Med. Chronicle*, May, 1890), and Bruns, of Hanover (*Archiv für Psychiatrie*, Band xxv., pp. 1-72), have confirmed Bastian's conclusions.¹ I do not imply that the writers just mentioned accept my hypothesis of the nature of the dynamical relations of the cerebrum and cerebellum. My hypothesis is rejected by Bruns, also by Gerhardt, and by Ferrier and other eminent neurologists.

Dr. Charlton Bastian's conclusions as to the state of the muscles and tendon reactions of parts below a *complete* supra-lumbar transverse lesion of the cord, are now admitted by nearly all medical neurologists to be correct.² The real "facts" of cases of the complete transverse lesions mentioned are not against the hypothesis I put forward in 1877-8. Bastian wrote (*Med. Chir. Trans.*, vol. lxxiii., 1890, p. 208):—"I came to the conclusion that a notion closely akin to that of Dr. Hughlings Jackson was most capable of explaining all the facts." After considering the then (1890) current views as to the causation of rigidity and exaggerated tendon reactions in hemiplegia, Bastian wrote (p. 209):—"Great difficulties formerly stood in the way of explaining many of the facts without the aid of some such views (although grave objections could always be alleged against them); hence the few adherents which the counter-explanation of Dr. Hughlings Jackson has hitherto been able to command. Now, however, it seems to me that the new facts established in this paper will be found to be

¹ See a most able paper, "On the Conditions of the Reflexes in Total Transverse Division of the Spinal Cord," by Ernest S. Reynolds, *BRAIN*, vol. xviii., p. 148 *et seq.*, 1895.

² Sherrington writes in a very remarkable and most valuable paper (*Trans. Royal Society*, vol. cxc., B., 1898, pp. 144-145):—"My results on Monkeys bear out strikingly and fully what Bastian describes as the typical condition in Man after complete transverse destruction across the cord. The chief difference is that in the Monkey in most cases—partly, perhaps, because the lesion is more localised by experimental infliction than by accidental—the depression is not so severe. For instance, the knee-jerk, which disappears almost immediately after the transection, returns usually in a week or ten days (Sherrington, *Journ. of Physiol.*, 13, 1892; A. S. F. Grünbaum, *Journ. of Physiol.*, 16, p. 368); often, however, not for three weeks; occasionally, on the other hand, in ten minutes."

altogether opposed to the fashionable views above cited, and to be just as much in favour of some modification of the doctrine of Hughlings Jackson."

In the second reprint (*Medical Examiner*, 1878) I spoke of localisation of the disease in paralysis agitans, suggesting that the regional distribution of this motor affection points to the cerebrum. I have, since 1878, submitted the hypothesis that in this malady there is slowly-spreading atrophy of cells of the Rolandic region of the cortex in the order of their size, from smallest towards largest. Presumably these cells, in their order from smallest to largest, represent muscles,¹ in their order from smallest towards largest. In paralysis agitans the interossei² are the muscles of the hand which are preponderatingly affected, and the set of the hand resembles that of the tetany of children. In most cases of ordinary hemiplegia with rigidity there is destruction of fibres of some part of the course of the pyramidal tract (internal capsule), a comparatively random and brutal lesion (clot or local softening). In most cases of this hemiplegia with rigidity the set of the hand is not interosseal, but is due especially to contraction of the large flexors and extensors; this set of the hand a little resembles that of the hand in carpo-pedal contractions of children. I believe that both tetany and carpo-pedal contractions are owing to loss of cerebral control permitting greater cerebellar influx, and surmise that, in children, the differences in the disposition of the thumbs and fingers and of the hands in carpo-pedal contractions and tetany, depend, at least partly, on differences in the degree of development (or of perfection) of the pyramidal tract and cortex; the nervous arrangements for the intrinsic muscles will, I imagine, be later in their development or perfection than those for the large flexors and extensors of the hand.

A case, very important in many ways, has been published by Strümpel (*Deutsche Zeitschr. f. Nervenheilk.*, B. v., 1894) of a woman whose attitude was very like that of a subject of advanced paralysis agitans. Brissaud (*Leçons sur les Maladies Nerveuses*,

¹ Properly "small and large movements." See my second Lumleian Lecture, *Brit. Med. Journ.*, vol. i., 1890. For important remarks on the significance of sizes of nerve-cells see Bevan Lewis "On Mental Diseases," 2nd ed., 1899.

² In some cases of paralysis agitans there is over-contraction of the interossei, and thus hyper-flexion of the first phalanges and hyper-extension of the other phalanges of the fingers. See a very interesting paper, *Des Déformations de la Main*, by J. E. Siotis. I need scarcely remind the reader of Charcot's important researches on paralysis agitans, in which, among many other things, he considers the set of the hands in cases of paralysis agitans, likening the disposition of the fingers in some cases to that in some cases of chronic arthritis.

Salpêtrière, 1893-4) reproduces (p. 45 *op. cit.*) a portrait of this patient, and (p. 486 *op. cit.*) says of it:—"Est-il possible d'imaginer une attitude et une physionomie plus *parkinsoniennes*? C'était une pseudo-bulbaire, puisqu'elle avait le syndrome d'une paralysie labio-glosso-laryngée spasmodique, causée par une double lésion systématique du faisceau paramidal." (See also Brissaud, *op. cit.*, pp. 41 *et seq.*) There was no tremor; there was rigidity of face, tongue, neck, arms and legs. There was difficulty in turning the eyes to the left, and ultimately all ocular movements were lost. Strümpell writes (*op. cit.*):—"On being asked, the patient is unable to carry out the smallest movement in any part of the body or with the eyes. But of her own accord she occasionally makes a slight movement with the eyes or with the eyelids." No morbid changes were found in the cells of the cortex cerebri. Degeneration was traced upwards beyond the upper end of the internal capsule, but could not be followed to the cortex cerebri. The lesion in Strümpell's patient was a slowly-developed one, whereby cerebral control would be slowly taken off anterior horns, and consequently, as I suppose, there would be permitted gradually greater cerebellar influx on those parts (anterior horns) from which the cerebrum was being, by the lateral sclerosis, slowly cut off. I do not say that this case was one of paralysis agitans, but I think it illustrates, if it does not confirm, the *principle* of the hypothesis I have put forward as to the pathology and physiology of that malady.

In cases of paralysis agitans, as in so many other nervous maladies, we have to bear well in mind Broadbent's hypothesis,¹ 1866. Bearing it in mind, I should say that double hemiplegia is more than the double of hemiplegia. In hemiplegia the bilaterally-acting muscles of both sides escape, or suffer very slightly, whereas in double hemiplegia the bilaterally-acting muscles of both sides suffer as well as do the unilaterally-acting muscles of both sides. Hence, in Strümpell's patient (symptomatically a case of double hemiplegia with rigidity), there was, as Brissaud puts it, pseudo-bulbar paralysis; there was involvement of very important bilaterally-acting muscles of both sides.

¹ I have profited very greatly by the application of Broadbent's hypothesis to the explanation of complex neurological problems. Thirty years ago (*Med. Times and Gazette*, Aug. 15, 1868) I wrote, under the heading "Remarks on Broadbent's Hypothesis," to urge those who observe defective bilateral movements with hemiplegia—particularly difficulty in swallowing—to enquire carefully if the patient has not been previously paralysed on the other side. Again, at an autopsy on a patient known to have been paralysed of *but one side*, and who had had difficulty in swallowing, we should search both sides of the brain for evidences of disease.

An advanced case of paralysis agitans is double hemiplegia with rigidity (and some tremor, which is an approach to rigidity). It will be found in some cases of this malady that, if there be not bulbar (pseudo) paralysis, there is involvement of other bilaterally-acting or alternately-acting muscles—rigidity of those of the face, neck, back, and abdominal wall. The hypothesis may be considered from another standpoint.

In an early stage of etherisation for surgical purposes there is muscular rigidity, and the knee-jerks are exalted. I think it possible that this occurs when the small cells of the cerebral cortex have succumbed to the ether, and when the large cells of the cerebellum have not been rendered functionless by it. In this stage, according to my hypothesis, there is negatively loss of some degree of cerebral control, and positively there is permission of a greater degree of cerebellar influx. In later stages, when I suppose the large cerebellar cells also have been rendered functionless, there is flaccidity of muscles and loss of the knee-jerks.

The situation, however, is complicated. The ether has, so to say, no malice against any particular part of the nervous system, but will put *hors de combat* small nerve-cells, wherever they are (cerebral, spinal, or cerebellar), sooner than it will large cells, wherever they are.

Dr. Hewitt, in his remarkably interesting work, "Anæsthetics and their Mode of Administration," p. 154, says of etherisation:—"In exceptional cases a fine tremor, known as 'ether tremor,' may occur." Perhaps the annulling of function of cells of the cortex is not solely by ether or chloroform, but both by the anæsthetic used and by supervenous blood (deficiency of oxygen). I say this in consequence of what Dr. Hewitt writes, p. 281 *op. cit.*:—"Rigidity will often disappear when more air is allowed with the anæsthetic, a fact which leads one to suppose that the condition may sometimes depend upon intercurrent asphyxiation." If so, there may be removal of cerebral control, partly by the action of the anæsthetic and partly by that of supervenous blood. But there is a difficulty to my hypothesis, so far as supervenosity is concerned. Risien Russell has found that the knee-jerks in dogs are exaggerated in early stages of artificial asphyxia, even when the supra-lumbar cord has been divided. It may be that in rigidity under anæsthetics, &c., super-venous blood, which annuls the function of cortical cells (of the Rolandic region at least) and which stimulates some medulla centres (respiratory, &c.), may stimulate some of the cells of anterior horns, and thus produce the rigidity or part of it.