REMARKS

ON THE

PHENOMENA OF LOCOMOTOR ATAXY:

WITH AN APPENDIX RELATIVE TO DISCUSSION
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AT meetings like these of the British Medical Association, I think we should take the opportunity of bringing questions forward which will lead to a discussion of well-known difficulties. We discuss those subjects best at public meetings, of which we already know the main facts, and we can deal better with new interpretations of things already verified than with new statements which require verification.

Instead, therefore, of laying before you, as I had thought of doing, some observations on the use of the ophthalmoscope in railway accidents, I have determined to ask you to consider whether we rightly interpret those phenomena which are familiar to us in the group called Locomotor Ataxy. The group of facts which we designate under this name, and under other similar names, are known to us all, but I venture to think that their interpretation differs somewhat from that which is usually put forward. I will first enumerate the symptoms of the disease, and I will take the liberty of enumerating them in the words of Dr. Radcliffe in his recent article in Reynolds's *System of Medicine*. There is "a peculiar gait—a gait precipitate and staggering, the legs starting hither and thither in a very disorderly manner, and the heels coming down with a stamp at each step. No true paralysis in the lower extremities or elsewhere. Characteristic neuralgic pains, erratic, paroxysmal in the feet and legs chiefly—pains of a boring, thrilling, shooting character like those caused by a sharp electric shock. More or less numbness, in the feet and legs chiefly, in all forms of sensibility, excepting that by which differences of temperature are recognised. Frequent impairment of sight and hearing, one or both. Frequent transitory and permanent strabismus or ptosis, one or both. No very obvious paralysis of the bladder or lower bowel. No necessary impairment of sexual power. No tingling or kindred phenomena. No marked tremulous, convulsive, or spasmodic phenomena. No marked impairment of muscular nutrition and irritability. No impairment of the mental faculties. Occasional injection of the conjunctivæ with contraction of the pupils."

Now, in Dr. Radcliffe's summary, I have only omitted one sentence, and that one I will now restore, as upon it I have to speak. He describes the peculiar gait as "*arising from want of coordinating motor power in the lower extremities.*" The rest of the summary is confined to the statement of facts; but there is an inference—"the gait is due to loss of coordinating motor power," and Dr. Radcliffe inserts this explanatory clause into what is otherwise only a summary of observations, because, I presume, it appears to him to be quite obvious, as evident as a directly observed fact; because, again, he is expressing not his own opinion only but the general opinion; and, finally, because he is following the name of the disease—"Locomotor Ataxy."

Now, is the peculiar gait due to "loss of coordinating motor power"? To inquire into this properly, we must first ask what this expression, *coordinating motor power* means; secondly, whether the loss of such a power is the cause of the symptoms named together locomotor ataxy; and if so, then, thirdly, how this power comes to be lost. Let us take into our best consideration, first of all, this expression, "*coordinating motor power.*" I am afraid that there is still some disposition to assume the existence of a power or faculty of coordination, governing the parts which cooperate. Now, I need not argue at length against the assumption of such an essential faculty; it is clear that such assumptions belong to a metaphysical habit of thought and can only blind us to physiological facts. Let us at once admit that coordination is not a governing faculty resident here or there, but that it is a result of the accurate adaptation of parts. The coordination of the parts of the body is no other in kind than the coordination of the parts of a watch, in which the harmonious cooperation of the parts is the result of their perfect adaptation. Disturb or break through this adaptation at any point, and coordinate movement is impeded or ceases. Coordination, then, does not exist here or dwell there, but is found everywhere where two parts are so connected, directly or indirectly, as to act together for a certain

end. If, on the other hand, we are led to assume that coordination is the function of a particular organ superadded to preexisting parts, which preexisting parts, though complete, have not coordination without the superadded organ, then, I think, the sooner we banish the word the better, the more clearly to see that coordination is only the establishment of such relations between the parts—such a connexion of them in pairs and in groups—as to ensure their combined action for some uniform purpose. Coordination of function, then, is the correlation of parts, and nothing more than this. In the lowest animals coordination is attained by the direct attachment of part to part, as in the simplest machines. In more complicated machines, however, the direct union of parts is impossible; and indirect or mediate union is obtained by means of gearing. The gearing of higher animals is the nervous system. In those animals which still retain much simplicity the nervous gearing is also simple; as parts however become numerous, as they become separated by varying and considerable distances, and as they have to be combined in more complicated operations the unifying apparatus—the nervous system—becomes more elaborate.

To apply this to the human limbs, we have a coordination of receptive surfaces* with instruments of motion, the coordination being obtained by means of afferent or efferent nerves meeting or coordinated in ganglia. This coordination takes place in certain of the spinal ganglia—for the legs it takes place in the lumbar enlargement. Such is the coordination in one leg, a coordination between the medium and the organism. But we have two legs, and these two legs must work together; they must work together for such simple efforts as walking or standing, and again for such complex efforts as dancing, skating, gymnastics, and so on. Now, simple efforts, such as standing or ordinary walking are mainly performed, we admit, by a reflex action, and interest but little the rest of the body; so far, that is, as the harmonious working of the legs is concerned, apart from the balancing of the body, this harmony depends upon a commissural connexion between the two lateral ganglia, which, by this blending or coalescence, form the lumbar enlargement. So far, then, as the "coordination of the movements of the legs" is concerned, we have to deal, first, with coordination of the receptive surfaces with the instruments of motion in each leg; secondly, with the blending or commissural connexion between the two legs. To proceed; if we wish to co-ordinate the movements of all four limbs, as in fencing for instance, we must have first a coordination of the receptive surfaces with its instruments of motion in each limb; secondly, a commissural blending of each pair of limbs; and thirdly, a commissural blending of the upper pair with the lower pair. In addition to this, in fencing, a farther coordination by means of commissural fibres must bring into gearing the muscles of the trunk; and finally other receptive surfaces also, such as the eyes, must be coordinated with the instruments of motion of the trunk, of the legs, of the arms, and of both pairs together. In such elaborate exercises as fencing, therefore, there probably exists some centre, such as the cerebellum, which serves as a common meeting ground or "clearing house" for emissary fibres communicating with each and every separate part, thus annihilating distance and making the separate parts one whole. Now let us apply this reasoning. We shall see, in the first place, that, literally speaking almost every structural disease results in more or less loss of coordination. Coordination of the eyes, for instance, is destroyed by fatty degeneration of the outer rectus of either orbit; by disease dividing one sixth nerve; or, again, by disease dividing the commissural connexion between the deep origins of that and of any other nerve. Myelitis, again, dividing the cord in the dorsal region, destroys the coordination, between the pair of legs and the rest of the body above the seat of the injury, myelitic paraplegia being a loss of coordination between the upper and lower halves of the body. Now, in the disease called locomotor ataxy, it is commonly said that there is a loss of coordinating power, not between the upper and lower halves of the body, but between the legs as a pair.† Those two which were joined as one have now been put asunder. That is to say, coordination is not lost between the upper and lower halves of the body, as in paraplegia; nor is it lost between the various motor instruments forming groups in each limb; nor, again, between the receptive surfaces and the instruments of motion in each limb; but, while each limb is coordinate within itself, and each is separately coordinate with the body, yet they are incoordinate with one another. Such I suppose to be the current opinion. Now that each limb though not coordinate with its fellow, is still coordinate within itself and with the trunk, cannot be the case in the disease before us. This seems quite clear to me from one fact, that, were it so, the ataxic patient ought to be able when one leg is tied up to stand or to hop upon the other,

* Since this was written, Mr. Spencer has introduced the still better term, *receptive motor system*, for the sensitive system.

† Dr. Radcliffe's words are: "What they had lost was that power of coordination by which the two limbs are enabled to act together." (Reynolds's *System of Medicine*, vol. ii, p. 336.)

* Read in the Medical Section at the Annual Meeting of the British Medical Association in Oxford, August 1868.

neither of which actions is possible to him. Each leg singly, then, is either out of coordination with the trunk or is incoordinate within itself. Now, were it coordinate within itself though incoordinate with the trunk, the patient should, when lying down, be able to perform calculated movements with one limb. But this is not so; if an ataxic patient have his eyes closed, he is incapable, according to the degrees of his disease, of any calculated movements even with one leg. We are brought, then, to this conclusion, that the incoordination lies within each limb. Is it, then, an anarchy of the motor instruments which have lost connexion, or is it a want of coordination between the instruments of motion and the receptive surfaces? Now, coordination is not cut off in the muscles themselves; they are fairly nourished and capable of much movement; nor is it in the anterior region of the cord, as we know by pathology; the seat of dissociation may be found, however, in the sensory connections—in the coordination of the receptive surfaces with the ganglia and nerves of the instruments of motion. Coordination, then, is not severed between one set of motor fibres and another, but between the sensation and motion in each limb severally. It has so long been known that loss of sensation is a constant phenomenon in locomotor ataxy, and this loss is clearly so sufficient to account for the disordered actions, that I am astonished to find that the full meaning of this condition has never received the attention which it deserves.

The loss of sensibility noticed in well-marked ataxy is not a mere numbness—not a superficial lesion of the end organs or of the terminal nerve branches, for these are found to be anatomically sound—but a retardation in the transmission of impressions, a slowness of reception. It would seem as if the impression had a difficulty in arriving at the encephalic centres, and we may infer, therefore, that there is also a like retardation in the transmission of the impression into and across the lumbar ganglia. Clarke and Vulpian* have noticed as long a time as five seconds to intervene between the prick or pinch on the foot of an ataxic patient and his perception of it. Mr. Clarke tells me that he has noticed much longer intervals than this, and I have noticed intervals at least as long. I think we may all feel sure that loss of tactile sensibility in the feet, etc., is an invariable symptom in locomotor ataxy.† Out of many cases, I have seen none in which it was absent, and of the multitude of recorded cases I have collected, I find it prominent in almost all which were carefully and intelligently observed. Of the cases in which its absence is recorded, some no doubt belong to other categories, being chronic myelitic or cerebritic processes of other modes, and the rest have been insufficiently investigated, for it is not easy to determine slight degrees of deficiency in sensation.‡ And the appearances after death are as uniform. Invariably the posterior roots are atrophied between their ganglia and the cord, their fibres which penetrate the posterior columns suffering with the latter, and their fibres which enter the posterior horns also suffering atrophy (as Lockhart Clarke has demonstrated), with the lesion of those posterior horns. I do not say that the mischief in the posterior roots is primary; on the contrary, I believe the sclerosis of the posterior columns is primary, and the atrophy of the afferent roots secondary and consequent, but I believe this secondary and consequent process to be the cause of the peculiar gait. I believe that in all locomotor ataxy there is some loss of power—that degree of loss of power which my friend Dr. Dickinson has shown to exist in destruction of the cerebellum;§ and I believe that such loss is due to the lesion of the posterior columns, but the peculiar stumbling, uncertain gait is chiefly due, I am sure, to loss of sensation. The commencement of the implication of the sensory roots we recognise by the violent neuralgic shocks and darting pains which are so characteristic of the disease. At this time there is little more than a certain degree of loss of power. Gradually, however, the patient loses tactile sensation in the feet as the secondary lesion of the sensory roots advances, and at this time we may see distinctly that motor coordination between the two legs remains, while balancing power is failing. Let the patient get a fair start with

his eyes open, let him once get a balance by impetus, and he will manage by means of the coordination in the two limbs to move across the room tolerably well. At this stage of the disease, if the symptoms are well watched, nothing can be clearer than this fact, that the coordination between the two legs as a pair of legs is well enough; that there is, however, a distinct, though not a great, loss of power; and that finally and chiefly, the sensory surfaces are incapable of quickly transmitting to the cord the true relations between the body and surrounding things. Hence the legs act in a blundering way, not because they are very weak, not because they are divorced, but, if I may use the metaphor, because they are blind. I think we do not fully realise how much all our actions depend upon a continuous and rapid perception of our true relations to external surfaces; how much, that is, motor coordination consists in sensory perception. Our education in complex movements depends quite as much, and often depends more, upon education of the sensibilities, and the interpretation of them, than upon an education of our instruments of motion. A man uses the same muscles to walk on board ship as upon dry land; but he has to get his sea-legs by attending to, and learning to interpret, a new set of sensations. An ataxic patient walks on a pavement as a landsman walks the deck in a storm, because, by blunting of the sensations, he is reduced to the same conditions: there is the same lack of ready interpretation of the true relations between his body and its supports. How exquisitely we gauge our relations to our supports in locomotion, we know by the inevitable trip we make when, in running up many stairs, we come to one which is a quarter of an inch higher than the rest. Indeed, in all calculated movements, in skating, in cricketing, in climbing, in conjuring, in playing musical instruments, in all such cases we learn our dexterity—that is, our motor coordination—quite as much by the increased delicacy of our sensitive perception as by the nutrition of our muscles. Any musician will tell us that, if he try to play the pianoforte with benumbed fingers, he is ataxic upon the instrument.* Nor is it a question of tactile sensation only; in cricketing and in Alpine climbing, it is a question of the eye, as much as of the touch of the feet. A man who is unaccustomed to walk along a slippery glacier before sunrise is ataxic till he learns to interpret the new tactile sensations; but, when he has to walk along a narrow ledge, it is a matter of eye as much as of foot; and he knows how much it helps him, if he can add to his sensations by running his fingers along a face of rock beside him, even if he cannot lay hold of it. I have an ataxic patient now under my care, who can walk readily enough if he may run his finger-tips—thus supplementing the faded tactile sensation of his feet by other connexions—along the wall and look at his feet, but who cannot stand for more than a few seconds with his hands before him and his eyes shut. To use his own very appropriate expression, he “loses himself,” and, in fact, he has no bearings. We all know the uncertain gait of a blind man; it much resembles ataxy, though he has the tactile sensations of his feet remaining. If, again, we observe his eyeballs, which have no sensation to fix them, we see they are truly ataxic. To perform all definite movements, these two factors are absolutely necessary, and must be in exact correlation: first, a system of motor instruments which are directly or indirectly combined; secondly, a correlated system of sensibilities, by which alone our movements can be directed and calculated, and the relations of external objects interpreted. These two factors we may separate in reason, but in reality they are inseparable.

In locomotor ataxy, then, we have two things to deal with. First, we see some loss of power, probably due to the degeneration of the posterior columns (this is a most interesting condition, but to discuss it would be to go beyond the limits of my present purpose); secondly, a disorder of movement, due to the atrophy of the roots of the sensory nerves, with consequent inability to take the bearings of external surfaces. “Locomotor ataxy” is not a bad name if we mean no more by it than this, that we have ill-regulated movements as a prominent symptom; but it is a bad name, if we mean that the disorder is due to some break in the gearing of the motor machinery; and a worse name still, if it lead us to conceive of a loss of some supposititious “faculty” of coordination. If we determine to retain the name, we must have a lively knowledge of this, that ataxy of some kind, and due to some break or hitch in machinery, is common to many diseases, or, indeed, in its widest sense, to all diseases; and that, in fact, it simply means *disorder*. But, when given as a name to the special set of symptoms we have been discussing, it is given to it quite arbitrarily, and merely as a ticket to designate a particular and tolerably uniform group. If we forget this, and include with our special group all other groups to which the name could in etymology be applied, we shall fall into great confusion.

* A glass or two of wine will have a similar result; though in this case the failure of perception is at the centre. A like difference lies between a general paralytic or a drunkard and an ataxic patient. The gait of vertigo differs again from each.

* Clarke, *St. George's Hospital Reports*, 1867, p. 77; Vulpian, *Archiv. de Phys. Norm. et Path.*, 1868, p. 150.

† In a lecture recently published in the *Medical Times and Gazette*, Dr. Wilks attributes the disorder of locomotor ataxy to loss of the “muscular sense”. I do not know what Dr. Wilks intends to imply in the expression “muscular sense”. I mean by it, our consciousness of the varying states of neurility in the motor nerves and of tension in the central cells connected with them. Now, in “locomotor ataxy”, these parts and the muscles are singularly free from disease. Dr. Wilks adds that cutaneous insensibility is not a constant member of the group of symptoms named “locomotor ataxy”. I am sorry that there is this discrepancy in the results of our clinical observation.

‡ For instance, my friend Mr. G. H. Lewes was so good as to furnish me with the details of a case of “ataxy” which had been shown to him by Frerichs. The patient, though free from choreism, was utterly unable to determine or to complete any movements with the upper extremities. The case is a most interesting one, and the state may be called ataxy, no doubt; but it presents a different *ensemble* of phenomena from that we have named “locomotor ataxy”.

§ Vide *Med.-Chir. Review*, October 1865.

APPENDIX.

After the foregoing paper had been read at Oxford, a second paper was read on the same subject, by Mr. Lockhart Clarke, and it gave me great pleasure to find that he took a view of the reasons of locomotor ataxy substantially like my own. Dr. Duchenne of Boulogne, who was present, differed from both of us and strongly urged his own original interpretation. Both publicly and privately Dr. Duchenne was most unwilling to allow that there was any loss of motor power in the disease; but of this I have no doubt, and I think that Mr. Clarke agrees with me. I have subsequently found that Oppolzer also enumerates "weakness of the lower extremities" among the early symptoms.* My own opinion on this point was formed independently, as I have repeatedly proved it during the last four years to the Leeds students, and have stated it in clinical lectures. In his paper, in the *St. George's Reports* for 1867, I see that Mr. Clarke does not distinctly lay down weakness of the lower extremities as a symptom of locomotor ataxy; and it is distinctly denied by Dr. Radcliffe.† I attach much importance, however, to this symptom, for, I believe, in opposition to many much better men than myself, that the disease of the posterior columns is chiefly the cause of this weakness, and that it has not much to do directly with the incoordination. Indirectly, it has much to do with it; and that, as I have said, by its encroachment upon the fibres of the posterior roots; for, herein, namely, in our interpretation of the way in which this encroachment causes incoordination, lies the difference between the view I venture still to hold and that of my friend Mr. Lockhart Clarke. My own view is simply this: that the loss of tactile sensibility is sufficient to cause loss of coordination, that the tactile sensibility is lost, and, therefore, that the loss of tactile sensibility is probably the cause of the parallel loss of coordination; that, in short the extravagant gait results from loss of consciousness of the relations in space between the body and the medium.‡ Lockhart Clarke's view differs importantly in detail from this, and his view seemed to me at first so suggestive and so ingenious withal, that, on the reading of his paper, I felt it only remained for me to pass on my own to the tallow-chandler or to the British Museum. On subsequent reflection, however, unfortunately for the reader, I have recanted, and, like other converts, am now more than ever disposed to preach my former doctrine. Clarke's view I take to be this (if I misinterpret him I trust that the publication of his paper will set me right); that the muscle may, with sufficient accuracy for the present discussion, be said to complete the circle formed, partly by a sensitive nerve and partly again by a motor nerve; that the completeness of this circuit is necessary for the activity of its component parts, and, in particular, let us say, of the muscle; that the disease, involving the posterior roots, destroys their nerve-fibres, not all at once, but separately, or by a few at a time; that in consequence there is at any given moment a rupture of a certain number of such circuits corresponding to the number of ruptured fibres, while at the same time there remains as great a number of unbroken circuits as there are fibres as yet uninvaded; that there are, therefore, muscles still forming parts of complete circuits, and, still therefore, quick, and muscles no longer forming parts of such complete circuits, and therefore deadened; that, as the progress of the disease is irregular, there is consequently, in all movements of the limb, an irregular predominance of the quick muscles over those rendered sluggish or inactive, which change of conditions must end in spasm and disorder. This theory, though fascinating and suggestive, does not, I now think, coincide with the facts; and for these reasons: firstly, the spasmodic disorder of the limbs is not an early symptom, coinciding with the early anæsthesia and with the commencement of the attack upon the posterior fibres in detail, but it is a late symptom and increases with the more and more complete disorganisation of the whole roots. This I have verified by the microscope in one case in which death occurred at the height of the "spasmodic" stage, and it is borne out by the far more valuable investigations of Clarke himself, of Vulpian, and of other observers. Were the wild muscles those whose connections remained entire, their wildness should gradually cease as these connections are cut through; whereas the contrary is nearer the truth, that the muscles become wilder and wilder as the roots become structureless. Secondly, there is no uniformity in the abnormal movements in any given

case at a given time. The disease is often of very slow progress, and, as the destruction of sensitive nerve-fibres slowly advances or remains stationary, so we ought to have mutiny in particular muscles or sets of muscles and uniform deviation from normal movements. But the movements, so far as I can make out, and I have looked into this again in the present aspect of the inquiry, are wholly capricious, and cannot even from one minute to another be seen to form any calculable combinations.

Thirdly, and on the other hand, I consider that the tactile insensibility is amply sufficient to explain not only the maladaptations, but the "spasmodic" movements also; and certainly the spasmodic movements do increase as the tactile æsthesis decreases. If, for example, all the members of a crowd, excited to some common purpose, were to become blind, they would first show indecision in their common movement; and secondly, losing relations among themselves, they would, if still excited, lose all combination with each other. The blinder the men, the blinder the fury. That which keeps muscles or crowds in action as groups is their combination for some common purpose. The common purpose, recognised by the æsthesia, and striven after by impulse, is the nexus, not only between the group and the object, but also between the members of the group themselves. If, while the impulse be continued, the perception of a common purpose be dimmed, the combination will be weakened, and the end unattained. Let it be cut off, and not only will the end be wholly lost sight of, but all combination to that end will cease, and individuals will take the uppermost, according to the momentary and varying states of their tension, their innervation, their size, their lever-ages, and other incidental attributes of men or of muscles. Confusion only can be the result of excitement aroused in a multitude, unless an unifying impression from without can make its way through general or special channels to the centre, be that centre a ganglion, a Napier, or a Beales.

CASES OF SALIVARY CALCULUS:
WITH REMARKS.

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THE following cases seem to be tolerably characteristic examples of a disease not very common, and regarding which little specific information is given in the more familiar text-books.

CASE I.—A. B., aged 19, called on me in April 1868, in great mental distress. She was a strong, healthy-looking, and very attractive young lady. For two years she had noticed a tumour under her tongue to the right side of the middle line; but, as it seemed stationary and gave her no pain, she had never mentioned its existence to any one; for some months, however, it had been rapidly increasing, and within the last few weeks still more rapidly, and had given her most acute pain. She was now certain that she was suffering from cancer of the tongue.

Present Condition.—On opening the mouth, there was seen on the right side of the tongue a red, fungating, angry-looking tumour as large as a walnut, which forced the tongue upwards so as to interfere with speech, and certainly had a most malignant-looking aspect. It was of stony hardness, and contained no fluid. Trusting to her appearance of perfect health, I excised the projecting portion of the tumour with probe-pointed scissors. Considerable hæmorrhage followed, and no cavity or calculus was exposed as I had hoped. However, by cautiously continuing my incision into the densely fibrous substance of the tumour, I was at last rewarded by extracting an oval calculus, about three-quarters of an inch in length. The relief to the pain was instantaneous, and in a very few days the tumour had entirely disappeared. There has been no return.

CASE II.—Miss F. M., aged 30, consulted me in December 1868. She stated that for the last eight years she had noticed a tumour under the right side of her tongue, which had been frequently the seat of uneasy sensations; at times it had given acute lancinating pain, and latterly, from its size, had interfered with speech and deglutition. All the symptoms had been increasing during the last twelve months. She had consulted at different times two different surgeons, who had both counselled delay. I found that the tumour was of stony hardness, and that a small portion of it projected forwards into the mouth. A free incision enabled me very easily to remove a calculus of peculiar shape, twisted like a ram's-horn, and about an inch and a half in length, and of a bright orange colour. All the symptoms disappeared along with the tumour.

REMARKS.—Salivary calculi are generally found at the orifices of the Whartonian ducts, or in those of the smaller ducts of the sublingual glands. (Craigie's *Elements of General and Pathological Anatomy*, p. 829). They are very rarely found in the duct of the parotid. In most

* Vide *Wien. Med. Wochens.*, 26-28, 1866; quoted *Virch. Jahresh.*, 1867, Band ii, p. 49.

† As my dissent from some points in Dr. Radcliffe's article called forth my own paper, I have seemed to stand in constant opposition to him. I should much regret it, however, if I thus appeared to speak with anything less than the highest respect for Dr. Radcliffe's writings in general, for few writings have given me more pleasure, and I have found few so fruitful.

‡ I gather from an indirect source that this explanation of the phenomena called "locomotor ataxy" has been already given by Professor Leyden, of Königsberg, in *Virchow's Archiv.*; but, owing to the carelessness of a reference, I have not yet found his paper. What will be the doom of authors who have wasted the time and energy of their fellow-creatures by giving insufficient or inaccurate references?