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SUGGESTIONS AS TO THE ÆTIOLOGY OF SOME OF THE SO-CALLED SYSTEM-DISEASES OF THE SPINAL CORD.

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AMONG the most salient facts in clinical medicine and pathology that recent investigation has brought to light are certainly those relating to the differential diagnosis of some diseases of the spinal cord. There is no doubt that clinical study, stimulated and enlightened by anatomical research, has enabled us to discriminate affections which really can lay claim to individuality, and are found to be associated with a definite lesion when examined *post mortem*. Till quite lately, as is well known, many of these affections were classed together under the general term of paraplegia, or myelitis. Though doubtless existing for an indefinite time they have remained unnoticed in a confused mass of morbid symptoms, till, like the statue set free by the sculptor from the marble block, they have come into being through the labour of the clinician and the pathologist. Much more can now be learnt than heretofore of the life-history of this class of affections, and an improved prognosis ought necessarily to follow in the wake of this diagnostic advance.

Yet as we contemplate the progress we have made, we cannot but be struck with our abiding ignorance concerning the origin of these diseases, not to mention the absence of any line of treatment, which, of course, to be rational, must largely depend on ætiology. On every ground this question is of commanding interest to the pathologist and the physician. The post-mortem study of such cases of spinal cord disease as end in death, and can be thoroughly examined, has at present given us but little direct help towards tracing their causation from the beginning. When we thus examine the body the mischief is already wrought; destructive lesions at most are demonstrated to us; though we may be led by a kind of natural inference to suppose some change in the cord, antecedent to the symptoms and to the structural lesion before our eyes.

But in face of these facts, and though the morbid anatomist has made no general claim to teach us anything regarding the ætiology of the diseases to which we refer, it would seem that many are led to regard too exclusively the lesion found in the cord as the primary source of the malady in question, and so incidentally to limit the field of inquiry as to how these special diseases may be brought about. But little attention is paid to the possible ætiological antecedence of peripheral disturbance, or what may be called abnormal function, which, by its continuance, may set up the ultimate change in the structure of the cord, entailing all the morbid symptoms of the established disease.

In speaking of the possibility of functional disorder producing structural change, it must be acknowledged that at first sight the natural order of things is apparently reversed, and the behests of a truly materialistic pathology seem to be too lightly regarded. But it must be remembered that the word "functional," when applied to disease, does not in any sense exclude the notion of a material cause. We mean by the term such disorder as is not preceded by any structural change detectable by our present means of research, and further, because in many cases such disorder tends to recovery, in all probability not often followed by any permanent alteration. In such a case the word "functional," though theoretically pro-

visional in character, has a practical value, and denominates a real class of affections. Such diseases as chorea and the many disorders of function shown by the various forms of hysteria are instances in point. There are, as Dr. Wilks has said, but few forms of organic nervous disease (i.e. nervous disease demonstrably associated with structural change) that do not find their copy among the hysterical. Does not this close correspondence of so-called hysterical or functional disease with disease that is called "organic"—a correspondence sometimes so close that almost the only differentia seems to be that the one tends to recovery, the other to permanent disablement or death—give us at least a hint of some possible common element at work which may underlie in some cases the beginnings of both affections? In other words, may we not conceive of the more central nervous structures suffering from a continuance of undue excitation from the periphery, so that what at first may have been, for instance, a temporary derangement of the circulation, may come to be a permanent alteration in structure due to chronic congestion or inflammation?

In applying this view of a functional origin to some of the system-diseases of the cord, we must bear in mind that it can only be regarded as a suggestion, to be used or not as it is found to be fruitful, and not contradictory to established facts. Even if it be looked upon as a "*vera causa*," it could with difficulty be demonstrated, the morbid procedure being ex hypothesi in progress during life, and causing thereby an impossibility of tracing continuously the working of the primary peripheral disturbance towards the ultimate structural change. But it may be of some use to consider the subject for a while from this standpoint, and to inquire whether this view derives any support from analogical instances, or falls in with established facts or received theories regarding the physiology of the nervous system.

The study of the working of the nervous system, both in health and disease, is confused and hampered at the outset by an enormous difficulty. We have to deal with the functional aspect of the nervous system; with nervous action, or, as we often term it, nervous force—a force of whose nature

we are ignorant, uncorrelated as it is at present, but which nevertheless we must recognise and provisionally describe. The functional activity of nervous structures is therefore quite a different thing to discuss from that of other organs. Dilate as we may upon the action of the nervous system, whether normal or abnormal, we can as yet do but little towards explaining the phenomena we are grappling with. The words we use, such as "nervous force" and, more notably, "discharge"—of late so frequently occurring in writings on nerve-pathology—are at best but analogical and indefinite in character, in no degree explanatory in the true sense of tending towards simplification, or of expression in terms of the known. We are indeed almost obliged to call in the aid of non-physical terms when speaking of some nervous phenomena, so intimately are they blended with factors which, for the sake of clearness, must be called mental. The unknown element in this field of study is too great for us to institute as yet a scientifically consistent nomenclature. We cannot intelligibly express in definite terms the simplest voluntary movements of our muscles, for we know not the nature of the functional relation subsisting between the brain-cells and the periphery; and we can hardly do more than attempt to give a metaphorical account of the higher workings of the cerebrum, or of the well-known processes whereby a so-called mental impression, though involuntarily arising, is followed by an obvious physical result. We are, in fact, constantly confronted in our study of nervous phenomena with the deepest problems of life, the relation of brain and mind, and we must, in our ignorance, of necessity confuse (albeit with conscious acknowledgment) the terms of psychology and physiology. This unexpressed vital factor is an ever-present "crux" in our endeavours at explanation, and a defiant enemy of post-mortem discovery.

It is necessary to bear in mind this difficulty and source of inaccuracy in our terminology, when listening to any ætiological speculation in nerve-disease. If physiology gives an uncertain sound, pathology may be expected to do the same, or, at least, must not be forced, if it be heard at all, into narrower and stricter limits of diction than those granted to physiology. It may be legitimate and even useful to assume

an undemonstrated pathological process as a tentative explanation of any given phenomena not otherwise understood, provided such a process be not contradicted by accredited knowledge, and be in nowise deemed to be established just because, if assumed, it appears to be adequate to the production of its alleged result. This justification there seems to be for inquiring as to how far a functional origin might serve to account for certain diseases of the cord (as, for instance, locomotor ataxy or spastic paraplegia), without contravening undoubted facts. For there is an especial difficulty while considering these diseases from the standpoint of the *structural* lesions observed, in the view of the *primary* occurrence of anatomical change in certain *functional* or *physiological* paths.

The pathological aspect of these affections to be discussed, while appearing at first sight out of harmony with the ordinary method of study, seems more reasonable when the complexity and peculiarity of the nervous system is borne in mind. The arrangements and mutual relation of the elements of the nervous system, the elaborate structure of the nervous centres, as well as the question of the action and reaction of the individual and the environment, in the development of the higher centres and functions, may all, to some extent, bear on the pathological study of nerve-disease. Perhaps we are all too apt, especially in medical practice, to regard the human individual in his aspect of independence rather than as an integral part of the universe, and to forget that the manifestations of both normal and abnormal action in man may be partially explained by reference to how and whence he came, and to the influence on his organism, both through heredity and present action, of impulses from the external world.

The current manner of speaking of nervous phenomena tends to present a one-sided, or rather, probably, an incorrect view of the mutual relations in structure and function of the several parts of the nervous system. When we speak of the nervous centres, meaning more especially those in the brain and cord, we are apt to regard them in our minds as in every sense antecedent to and causative of, both retrospectively and actually, the rest of the nervous system and its phenomena. We use, for instance, the term "nerve-force" (though more as an exigency of nomenclature than as conveying a very definite

idea), and almost irresistibly regard the nervous centres as the "fons et origo" of this force. We tend to represent the completely developed nervous system, as we see it, as antecedent to the display of nervous action. Generally speaking, in relation to the individual, it is sufficient for all practical purposes to describe the nexus between structure and function in the ordinary manner, and to regard all function or activity as dependent for its existence and character on the structure of its proper organ, without regard to the evolutionary history of the organ itself. For this mode of regarding it is, as a rule, scientifically accurate *as regards the individual*. We may thus rightly infer a primary alteration of structure in various organs of the body from an ascertained abnormality of their function, and we find that definite lesions are constantly followed by definite sets of symptoms. Ample instances of this are afforded us by the study of visceral pathology. But when we come to the nervous system, the matter and manner of investigation are not so simple. Instead of a comparative homogeneity and simplicity of structure and function, we find the greatest complexity and diversity. The differentiation of structure is here so great, and the nature of the functions so various and, as it were, unique, their study deriving so little proportionate help from direct experiment, that we must carefully consider the subject in all possible aspects before using a terminology and method of description which cannot be without its influence on future work, and may possibly obscure, instead of tending to advance, the true understanding of the problem.

It is the developmental aspect, then, that we cannot afford to ignore in relation to the physiological and pathological working of the nervous system. For here especially does there seem to be some indication of certain practical bearings on the individual of developmental influences, or, in other words, a different ratio of value to obtain between the hereditary and acquired elements of the organism. Though the hereditary factor may still be quantitatively preponderant, the acquired factor asserts itself visibly, and is obviously of great and characteristic importance.¹

¹ This is clearly seen of course in those so-called psychical functions which are more especially characteristic of the human species. The notable modification of an individual by circumstances, or the irresistible effect of what Spencer calls the

But little is known by direct observation of the details of the development of the nervous system in man. Certain main points, however, regarding the evolution of nervous structures generally should be borne in mind. And chiefly does it concern us here to remember that "embryological evidence shows that the ganglion cells of the central part of the nervous system are originally derived from the simple indifferentiated epithelial cells of the surface of the body; while the central nervous system itself has arisen from the concentration of such cells in special tracts."¹ Complexity of the nervous system is a characteristic of later evolution in the animal series, and some of the structural arrangements of the nervous centres existing in the higher animals are not found in the embryo, or even soon after birth, but increase in a demonstrable manner during the life of the individual. Apart from the probable fact that the sharp distinction between the white and grey matter is a feature acquired in the higher vertebrata, we know that the grey matter of the cerebrum does not exist in the human embryo, and that its thickness continues to increase through a long period of years. There is then an increase of structural complexity during the period of active cerebral life. Remembering, then, the admitted influence of external stimuli on the elaboration of the nervous apparatus, and its probable effect on the individual, we cannot but think that some different consequences from what we see in the case of other organs may be entailed by this view on nervous pathology. The lung of the new-born infant is essentially similar to that of the adult; and preparations of its liver or its kidney present no structural difference from those of the man. But when we consider that some of the most pre-eminent functions of the human individual—the functions of the nervous system which

super-organic environment, is a striking example. In illustration of this it may be remembered, as quoted in another context by Mr. Henry George in his recent work on 'Progress and Poverty,' that the special form of articulate speech with which we express our ideas (i.e. our national language) is entirely the result of impressions made on our individual brains, though the hereditary capacity for articulate speech is one of the most important *differentiæ* of our species. A child of any country could learn with equal ease the language of all, if placed in similar native surroundings.

¹ See Balfour's 'Comparative Embryology,' vol. ii. ch. xv.

tends so largely to the differentiation and predominance of man in the animal series—are correlated with structural arrangements which are not complete at the beginning of life, it appears most probable not only that modifications and interferences with the developing organism may take place, but also that their significance, occurring as they do in such important material, will be proportionately great. If nervous structure becomes organised abnormally, we may then expect in turn a more or less permanent aberration of function as a result. The structure of the nervous centres, in fact, is to be contrasted, rather than compared, with that of other organs. The fact, again, of the least stable part of the organism, or that part which is most lately developed, being the first to undergo dissolution or degeneration, is exemplified in the study of the nervous system both in health and disease. The grey matter of the brain in old age tends to waste, and its functions to approximate to those of early life; and the tremors of senility are comparable to symptoms which are referable, before the period of natural decay, to cerebral degeneration.

Similar influences, then, to those which in the normal course of things tend towards the development and complexity of the nervous centres, such as an increasing variety of impulses and stimuli from without, or the multiform contact of the environment with the organism, acting through the peripheric nerves, may in altered conditions produce pathological results. The normal complex nervous centres may be best regarded not as primary originators of "nerve force" and antecedent to its display, but rather as automatic registers of those actions which, at first vaguely exerted, were by frequent repetition conveyed along the "lines of least resistance," and have at last come to be organically marked in definite structure. From this point of view we may roughly liken the nervous centres rather to a calculating machine which, with great complexity of structure, can automatically produce elaborate results, though set in action for every process at a given moment by the simplest means, than, as is so commonly done, to the electric battery of the telegraph system, which is the source of telegraphic phenomena. The calculating machine represents, or, it may be metaphorically said, contains stored up in

its structure the brain-work of the inventor. So the nerve centres are the permanent and complex registers of often-repeated peripheric impressions from the external world, and we may conveniently regard the whole nervous system of man as a reflex apparatus derived from and dependent upon sensory impulses.

It is easy to understand, when these nervous paths are once formed, and closely associated by degrees with their increasingly elaborated central organs, that nervous action may proceed along these paths alone, and that the activity of the central cells in the matured organism is involved in every expression of so-called nervous force. It is as if a central government devised and brought into existence by a community had received in complete surrender, for the purpose of the work being carried on with the greatest efficiency and the least friction, the entire control of the action of that community. Such a government, though really the consolidated expression of many individual wills, would appear at first sight to be the mainspring of the action of all those who had so completely given themselves up to its authority.

On the pathological side, then, this method of presenting the genesis of the nervous centres has its application. As certain peripheric stimuli have gradually contributed in the healthy body (regarding both the hereditary and acquired elements) to a normal organisation of the nervous system, so certain stimuli, not so favourable in their nature, and acting moreover perhaps on an organism with hereditarily weak points, or "unstable" in character, may result in the production of a badly organised nervous structure. And when the nerve-centres become thus mis-organised, their indissolubly associated functions must be abnormal too. Thus, for instance, while we rightly attribute the morbid symptoms which we call a disease of the spinal cord to the structural change observed therein, as proximately causing the symptoms, we may consistently regard the structural derangement of the cord as resulting from primary untoward action from the periphery, preventing or marring the normal course of development. And we may compare the diseased cord which spoils the working of its associated nerves to the calculating machine which is out of

order, and whose maker is not to be found. The complicated nerve-structure, once formed, is absolutely necessary to all action; the contributory constituents have long ago surrendered their initiatory powers. But by a reference to the maker of the machine the cause, at least, of its disorder may be manifested; and thus may the consideration of the development of the nervous centres tend possibly to throw some light on the processes of their derangement.

It is obvious that the more stable or earlier-organised parts of the nervous system, being hereditarily comparatively perfect, may (relatively to the individual) be regarded, both in physiology and pathology, as antecedent to functional action. But the more unstable parts irresistibly remind us of how and whence they came, and to duly understand some aberrations from the normal in the case of nervous disease, we should perhaps have this ever before our eyes, regarding some parts of the nervous centres as dependent for their characters on functional actions in the past, and capable of being modified by its continued influence. For this view is probably true of the whole nervous system when taken in relation with the biological series.

It does not concern us here to trace the first appearance of a nervous system in the animal world, any more than it does to know what order is taken by the nervous link in the vital chain of each individual. But given in man a highly differentiated nervous system, it is impossible but that he will suffer materially from its derangement in any degree. The complexity of its arrangement and the importance of its manifold functions are sufficient evidence of this. By it man is brought into relation with the external world. It is at once the highest in function, and the most liable to derangement of all the systems. Nature's "last work which seems so fair" is open to the evil influences of what seem to be ordinary causes. The highest functions of all may be irreparably damaged by influences which are traceable by inference or conjecture alone, and which reveal no morbid stamp when their associated structures are examined in the light of our present knowledge. But we are none the less sure that, though it eludes our search, some "havoc in the tender cells" of the

brain has yet been wrought. And coming to subordinate departments of the nervous system, less subtly constituted than those whose structure is involved in the manifestations of insanity, we should not be surprised to meet with cases of disease in which some peripheral disturbance, owing to interference with the nervous paths, had caused an alteration, either temporary or permanent, of the central nervous structures—an alteration sometimes to be detected and demonstrated by comparison with the normal structure of healthy development.

Again; as in the individual the most recently differentiated and most complex structures are the most likely to suffer from undue stimulation, so too we might expect to find certain cases where the organism had a special liability to such derangement, or an hereditary tendency to fail on the side of the nervous system, even on the provocation of such slightly undue stimuli as might be difficult to detect or impossible to demonstrate. We may have to deal with an inherited "nervous constitution," the vagueness of which much-used and much-abused term may be to some extent diminished by the present point of view. That a certain so-called "neurotic" tendency is found in certain families is admitted by all clinical observers, and is in accordance with what we think we know of the physiology of the nervous system. There is a further point, again, which these considerations illustrate. Although the neurotic tendency is often inherited, it is not a matter of common observation that this is the case with special neuroses or definite "nervous" diseases. We may have many different morbid expressions of nervous action occurring in the same family, such as epilepsy, or chorea, or perhaps various forms of paralysis. And hysteria in its multiform aspect (though no more multiform than might well be expected if we duly regard it as want of proper systemic nervous control) is often a notable mark of many closely-related persons; but the form which the hysterical display shall take is largely determined by the physical and psychical surroundings of each individual case, or, in other words, by the nature of the untoward stimulus.

It is of course obvious that the application of this point of view to the ætiology of any of the so-called "system diseases"

of the cord is beyond the region of demonstration. We are at once confronted with the difficulty or impossibility of showing how such post-mortem lesions as are observed in the cord can have resulted in the manner suggested; and with the further obstacle, that in many cases we cannot prove the existence of any obviously undue functional action at all. The hypothesis must be regarded as merely tentative, perhaps to be completely discredited by future knowledge, and must be granted the full measure, due to all hypotheses, of a *à priori* assumption. Thus, in the case of any given specialised disease of the cord, although in some instances obvious functional disturbance, whether in a presumably predisposed individual or not, may irresistibly direct our attention to its probable ætiological importance, yet in many others we can demonstrate no probable exciting cause. Then we may call to mind the inference from other considerations which teaches us the presumable existence of hereditarily unstable nervous structure, and the probable fact of this instability being transmitted more as a tendency than in any specialised form or expression. The patient whose case puzzles us may have been the inheritor of a nervous system thus characterised, and hence requires a much slighter exciting cause (though that cause, be it remembered, must be of a special nature) to bring about an ultimate central change similar to that which, with an obviously great exciting cause, may be presumably found in a better constituted individual. It is because the so-called system diseases, especially those connected with changes in the white columns of the cord called respectively *posterior* and *lateral sclerosis*, or "locomotor ataxy" and "spastic paraplegia," seem so difficult to explain ætiologically from direct observation, and yet from their peculiar course are so interesting and important from this point of view, that some hypothetical discussion of their cause seems excusable. As careful anatomical examination has progressed, so has it come to light that in certain diseases, clinically well marked, there are constant demonstrable post-mortem changes, and, moreover, that these changes take a *physiological* direction, or follow a tract of *special function*. It is the question why this change should start into being in a definite functional region that arrests the attention and de-

mands inquiry; the progress of the change once so started, mainly along the same tract, presenting no great difficulty.

The disease clinically known as *locomotor ataxy* is characterised by the well-known sclerotic change in the posterior root-zones of the cord on both sides; and more recently it has been shown with great probability that a definite set of symptoms, now best characterised by the name of *spastic paraplegia*, but commonly called "lateral sclerosis," is associated with a similar morbid process, symmetrically affecting a part of the lateral columns of the cord known as the crossed pyramidal tract. The anatomical recognition of the latter disease does not rest upon a large number of facts; but the clinical picture is very complete. The post-mortem change cannot be often seen in the unmixed cases, as they run a long and indefinite course; but there is at least one case on record¹ which has almost all the value of an "experimentum crucis."

It is this physiological or functional direction taken by the morbid process that suggests some explanation other than the haphazard occurrence there of a primary inflammatory or degenerative change. The peculiarity of the localisation of the mischief would meet with some explanation if it could be admitted as possible that some morbid influence, or irritation, —call it by what name, or express it how we will—working molecularly, or otherwise (for in our ignorance it matters but little what metaphorical term we use), can be transmitted from the periphery to the centres, and bring about the ultimate changes which are now recognised. We could then conceive with some consistency that an over-excitation of motor or sensory functions of certain parts might account for observed changes in the tract or centres in immediate connection therewith. In such a tract or centre the motor apparatus is gathered, as it were, into a focus, and might represent in its morbid state the result of untoward stimulation from the periphery, just as in its healthy state it is the result of a series of normally conveyed impulses.

Have we, then, any justification in fact or analogy for the

¹ A case brought by Dr. Dreschfeld, of Manchester, before the Pathological Section of the recent International Medical Congress. See 'Transactions of Congress,' vol. i. p. 407.

hypothesis of a functional origin for these definitely marked diseases? Considering the peculiarities which specially mark the nervous system, it cannot be expected that we shall find any closely-related facts in illustration of this hypothesis, which for its chance of life must mainly rest its defence on the absence of facts in positive opposition to it; but the following considerations appear to have some relevancy.

The clinical study of nervous diseases suggests several examples of more or less permanent disorder, which may find at least a not unlikely origin in functional disturbance. Leaving out of the question for the moment the existence or not of structural change demonstrable *post mortem*, we can call to mind in this context the innumerable examples of confessedly physical states, brought about and modified by influences acting on some part of the nervous system; these results being tangible and demonstrable, though the so-called central organ whose action takes part in the phenomenon must certainly be looked upon as being affected in a very different way from what we are in the habit of calling "structural" change. The varied field of the so-called action of the mind on the body is an illustration of this — where often a stimulus to some of the peripheral nerve organs, applied either directly or indirectly through the medium of memory of analogical states in the past, or *mental action*, causes an altered physical state easily recognisable by the observer. Thus a word, or a thought, will cause a blush; the blush being proximately dependent on disturbed vaso-motor mechanism. The same condition may be brought about by the purely physical experiment of dividing a nerve. It is not stretching a point too far to surmise that a constantly repeated stimulus of the first order, could it be attained, would bring about a state of more or less permanent congestion of capillaries.

The influence of various sights, smells, and even thoughts, on appetite and digestion is well known. Undoubtedly, a change in the physical state of the organs concerned causes the nausea and indigestion that are so well known, in certain kinds of temperaments, to result from the untoward stimuli alluded to. Atrophy of organs, of course, is obviously due in many cases to their functional disuse; while hypertrophy is a

consequence of their over-use. Hypertrophy of the heart is known to result from an undue strain upon this organ, dilatation being probably the first stage in the process. But as a further result we often find fatty degeneration, accompanied by an increase of the connective tissue. Here we have an example of structural change occurring in ascertained lineal descent from functional over-work. In some cases, as in pregnancy, the cause of hypertrophy of the heart being not continuous, or, in other words, the functional demand being intermittent, the structural change is not permanent, and does not go on to degeneration; the whole process here may be called physiological. But in most other cases the cause is more continuous, and the morbid result is permanent. Thus we see how narrow a line may divide a physiological phenomenon, demonstrable by even structural change, from a pathological process which we call disease.

To come now to what is more proper to the subject in hand—the diseases referable to the changes in the spinal cord. The most common and best-described example of the system-diseases of the cord is the malady known as locomotor ataxy, or *tabes dorsalis*, which, by ample post-mortem observation, has been connected with sclerosis of the postero-external columns of the cord. As the observation of this affection has been made with increasing attention, so the manifold nature of its symptoms has become more and more obvious. The symptom of ataxy which, when the disease was first described, occupied such a prominent position, is now rivalled or out-rivalled in importance by a host of sensory and even of cerebral disturbances which must necessarily be taken into account when considering the probable origin of the affection. Some authors, as is well known, regard the disease as essentially one of the sensory system, including the symptom of inco-ordination. Doubtless in most cases sensory symptoms of a pronounced character mark its onset. The prominence of the sensory symptoms in locomotor ataxy has lately been especially insisted on by MM. Pierret and Rougier of Lyons, whose writings on this subject are very suggestive.

Various exciting causes of this disease have, from time to time, been put forward. Before the post-mortem condition of

the cord had been so carefully studied as it now is, a theory of the functional origin of tabes enjoyed a much larger share of attention than at present. It was a wide-spread opinion that excessive sexual intercourse was its frequent or general antecedent. This opinion, not traceable to any theoretical bias, but presumably to some extent founded on a basis of observation, has almost disappeared from the schools, owing to the proved connection of the symptoms with structural disease of the cord. This definite sclerotic change, whether primarily inflammatory or not, and wherever the exact histological seat of its origin may be, now alone arrests the attention; and every known cause of such a change is called into requisition to explain it. It is not here my intention to discuss this matter in detail; but this much may fairly be said, that no reasonable theory of the origin of this definite and localised change in the cord has as yet been started. Syphilis, that inexhaustible cruse of ætiological oil, may be regarded as having been weighed in the balance of causation and found wanting. Recourse was doubtless had to it, with that natural longing for finality and logical rest which characterises the minds of most of us; but both on account of the wide prevalence of syphilis, and the known habit of its morbid deposits, *which do not make choice of functional tracts*, its supposed causative rôle in locomotor ataxy cannot be at all satisfactory to the serious inquirer. The suggestion, in order to remove a difficulty from the syphilitic theory, that the morbid process may have been first diffused over the cord, and afterwards become localised in the posterior columns, is somewhat gratuitous, and certainly quite out of accord with the clinical history of most cases of ataxy.

But though the origin of this disease is confessedly obscure, there would appear to be some points of probability in the older notion that it may lie in what is to the affected individual relatively excessive sexual excitement. The far greater frequency of tabes in men than in women agrees with this view; and a very strong impression prevails both among practical medical men and others that its causation is of this nature.

I am unable to give adequate details in support of this

position, having been in the habit, in common probably with many others, of to a great extent ignoring this point in cases which have from time to time come before me, owing to the prevailing influence of modern teaching. But as I deprecate these remarks being regarded as claiming any demonstrative value, or as being anything else than barely suggestive, and as I am not attempting here to discuss fully the pathology of tabes, this omission is less inexcusable than it may seem. Long before I was led to theorise at all on the subject, I had difficulty in rejecting from my mind the probable influence of sexual intercourse in the production of this malady. Some cases that I have in my memory strongly impressed me at the time of seeing them; the patients themselves, without previous knowledge of the name or nature of their disease, being confident of its cause, and stating their opinion unasked. These patients' accounts of their excesses were not equivocal; one man especially, with a previously clean bill of health, giving a history where, during two or three years, increasing indulgence had been accompanied by weakness and darting pains in the legs, these symptoms having been relieved by occasional short periods of abstinence. But a typical and rapidly progressive case of ataxy soon developed itself as a sequence, if not as a result, of what all would admit to be a course of extraordinary venereal excitement.

A case is also within my knowledge of a young man of unrestrained desires, who says that after frequent and excessive sexual intercourse he often suffers from symptoms which appear to be temporary attacks of inco-ordination. A commoner complaint, however, after undue indulgence, is that of neuralgic pains in the extremities, more especially in the legs.

It may be said here that Romberg, one of the earliest students of this disease, taught that loss of semen, when combined with that hyper-stimulation of the nerves to which sexual abuses give rise (i. e. to the exclusion of mere spermatorrhœa), not unfrequently favours the origin and encourages the development of tabes dorsalis.¹

Now if this malady be regarded as one especially affecting

¹ See Dr. Sieveking's Translation (Sydenham Society) of 'Romberg on Nervous Diseases,' s. v.

the sensory system, may not some little light be thrown upon it by supposing the first step in the morbid process to be due to abnormal stimulation of sensory organs; and, assuming for the sake of argument the frequency of relatively excessive sexual intercourse as a forerunner of tabes, may we not reasonably consider the intense and widely diffused sensations accompanying the sexual act as an adequate source of disturbance of an hereditarily unstable, or in some cases even of an approximately normal sensory apparatus, provided that it be admitted that structural alterations in the cord may result from prolonged peripheral irritation? And from this point of view the greater frequency of tabes than of a system-disease of the *motor* apparatus proper derives some little explanation. The possible cause that will be presently suggested for the disease called spastic paraplegia, or lateral sclerosis, viz over-work of the muscular system, is one that might be expected on this hypothesis to be less frequently active. For the natural demands for action on the motor apparatus are more constant than those on the sexual organs, and in a normally constituted cord we should look for a greater margin of functional activity to be allowed to the motor tracts without risk of over-stimulation taking place.

It may at least be said that this view of the possibility of a peripheral origin of the disease known as tabes seems to harmonise to a considerable extent with all the characteristic clinical facts. I am fully aware that the opinion of many experienced, and deservedly high, authorities on diseases of the nervous system is opposed to any close connection between tabes and the hypothetical cause under discussion. And were I defending a thesis, instead of inquiring, I should not venture to criticise existing views without bringing at least an equal amount of experience and labour towards the solution of this question. But it may be pointed out that just as in chorea, as ably argued by Dr. Sturges in his recent works, the apparently ordinary emotional causes which may produce it are necessarily often overlooked, so, in this instance, what is really relative excess of sexual indulgence might well be passed unnoticed, quite apart from the number of cases where it might be suspected, but purposely denied. Of course if this sequence

of events could be satisfactorily substantiated, the question of the causation of the disease would not be set at rest entirely. But on the principle, "*Causæ non sunt multiplicandæ præter necessitatem*," this hypothesis, serving to cover a large number of cases, would deserve at least a front rank in the investigation of the matter.

A few words may be said here on the arguments used by those who, partly from certain clinical data, partly from theoretical reasoning, deny the sensory factor in the production of inco-ordination, and whose position would, by implication, be opposed to the probability of the origin of the entire disease in disturbance of the sensory apparatus by peripheral irritation. It would seem that the use of the term co-ordination often causes and implies considerable mental confusion. Because a certain tract of the cord is found to be diseased in cases characterised by a want of co-ordination, the minds of many are satisfied with the conclusion that this tract is in some special way the seat or the conductor of "co-ordinating impulses." Now apart from the distraction which has been caused by the pathological study of locomotor ataxy, the question of a special seat or tract for co-ordination in the cord would never have struck the philosophic neurologist. Physiology tells him nothing of it; but, rather than suggest a search for it, would lead him to regard co-ordination as only a functional aspect of the nervous system. Co-ordination simply means the harmonious working of the nervous system as expressed in muscular action, whether considered as a whole or in its parts; and is implied in the very notion of a nervous *system* at all. That this harmonious working is dependent on the proper conduction of afferent and efferent impulses in connection with organised centres, or in other words implies a certain relation between the sensory and motor apparatus of the nervous system, would appear from a physiological point of view fairly obvious; and an impairment of co-ordination, or want of harmonious action in any part, would be referred to derangement of part of the apparatus involved, causing a want of proper conduction of either sensory or motor impulses. A special tract for the conduction or localisation of "co-ordinating" impulses is as little wanted in

pathology as it is hinted at by physiology. But those who argue against the influence of the afferent factor on the production of the *ataxia* of tabes, regarding it purely as a motor morbid symptom, seem at least unconsciously prepossessed with the idea that some special seat must exist for what they appear to treat as the co-ordinating "function," omitting in thought to analyse it into its simpler elements. Forgetting moreover, apparently, the fact that such a localisation has been suggested only by the observed connection between *ataxia* and disease of the posterior columns, and bearing in mind that physiology has as yet given only sensory functions to those parts of the cord, some have endeavoured to find in the lateral column the true seat of what they regard as co-ordination, believing it *must* have a seat somewhere; so that, on this showing, the well-known and accurately defined lesion in the posterior root-zones of the cord does not, after all, account for one of the most important symptoms of the disease in question.

The clinical evidence called in to support this view is based on the quotation of cases where inco-ordination is observed without loss of sensation; or where anæsthesia takes place without ataxia. Quite apart from the fact that the cases are, to say the least of it, very rare and questionable where some degree of anæsthesia does not obtain in connection with ataxia, the confessedly difficult subject of the due investigation and valuation of the symptom of anæsthesia should prevent us drawing any very important conclusion from the little we know of the matter. We learn but very little from direct physiological experiment on sensations, and especially of sensory conduction in the cord. The subject is beset with obvious difficulties; and though investigators speak with almost one voice regarding the motor functions, there is palpable discord among them respecting sensory phenomena. But when we remember (to quote from Foster's work on Physiology) "that an afferent impulse passing along an afferent nerve may in certain cases, simply produce a change in our consciousness, unaccompanied by any visible movements, and in other cases may give rise to reflex movements, or modify existing reflex or automatic action without causing any change

in our consciousness," we shall at least see that it is premature to deny the afferent or sensory factor in the production of inco-ordination, on the strength of such cases as we have alluded to. This question is argued at some length by Professor Erb, in his article on "Tabes," in Ziemssen's 'Cyclopædia,' where I think, in his advocacy of the exclusively *motor* nature of the ataxic symptoms, there is betrayed the confusion I have referred to concerning the *word* co-ordination, and this partly gives a bias to his arguments and quotation of cases against the possible part played by the sensory side of the nervous system in the production of inco-ordination. The consideration of this question illustrates the danger there is in attempting to make physiology too soon—to build up a physiological theory on pathological facts or clinical observations alone. The lines of physiological and clinical investigation, though often giving mutual help, must be kept distinct; the inferences of the latter being never looked upon as the dicta of the former. In the case we have been glancing at we have scientific warrant in maintaining only the *concomitance* of certain symptoms with a certain observed change. This fact is established. But we may not thereupon invent a function to plausibly occupy an insufficiently explained structural locality. This fallacy and confusion, although confessedly not put forward as a theory, or baldly stated by any writer, nevertheless seems to lurk deeply in many discussions on the subject.

To take now another example of the system-diseases of the cord, which may serve on the motor side as a counterpart to tabes on the sensory side. The disease now known as *primary lateral sclerosis*, or *spastic paraplegia*, must be regarded, owing to the admirable work of Professor Erb, as a clinical entity; and, though in all probability less common than tabes, is being recognised with increasing frequency. It is true that the evidence of the associated cord-change rests upon very few observations; but taking into consideration what has been indubitably made out, and what is known, moreover, regarding other forms of sclerosis in the lateral columns, in conjunction with the concomitant symptoms observed—this disease must challenge the attention of those interested in the ætiology of

the system-diseases of the cord. It is believed that certain tracts symmetrically placed in the lateral columns of the cord are especially occupied in the conduction of motor impulses towards the periphery; the normal conduction of these impulses implying, too, the control, or exhibition, of reflex action. Difficulty of motion, stiffness of the legs, and an exaggeration of the reflexes, are the prominent clinical symptoms connected with this form of sclerosis. Is there any clinical justification for supposing that the cord-change existing here may be secondary to undue stimulation of the motor apparatus: granting, for the moment, the possibility of a pathological effect being wrought on the concentrated nerve masses of the motor tract in the cord by undue irritation of the motor nerves of the periphery? Certain points, both negative and positive, seem to favour such a supposition, though they may be not sufficiently obvious to claim recognition. There is no theory, even vaguely accepted, as to the ætiology of this affection, definite as it seems to be. The syphilitic explanation has not been seriously broached, and by most authors is rejected, to fall back on "cold," possible injuries, and such-like general hypotheses—so general, as almost to defy either proof or disproof. One positive point, however, is noticed by those who have written on this disease: it occurs comparatively frequently in men—and in muscular men. It is perhaps in this class that we may expect to find the material for a disease hypothetically producible by excessive demands made on a possibly unstable nervous apparatus: for here it is more likely that muscular overwork might take place, and the possessor of the strongest muscles is not necessarily the subject always of the most perfect nervous control. It must be remembered, too, that this disease, anatomically characterised by sclerosis, most marked in the lower part of the cord, begins generally in the lower extremities, which are more liable to continuous overwork than other parts of the body. In some cases of this disease, moreover, excessive action of the muscles can be clearly established. One case especially,¹ which has lately been under my care, may, I think, be taken as supporting this view. It is that of a young acrobat or contortionist,

¹ See 'Brit. Med. Journal,' Dec. 9, 1882.

aged twenty-one, who about eighteen months ago began to complain of weakness and tremulousness of his legs. When he first came under my observation he had not developed ankle clonus or the spastic gait, and the knee-phenomenon was not markedly exaggerated. With rest he made some marked improvement for a time; but he again took to his work, and gradually became, what he is now, a typical specimen, as far as clinical observation alone can demonstrate, of a "spastic" paralytic. For six months previously to June in the present year he had been performing, against my advice, literally nightly in Berlin and Paris, being obliged more and more to favour his legs by confining most of his "business" to his arms. This youth had been an acrobat ever since four years old. With the exception of having been a "delicate" child, his health appears to have been very good up to the time of his legs beginning to fail. The rest of his family were always strong, and are following the same occupation as himself. I satisfied myself without doubt that he had not had syphilis.

A case of this kind irresistibly turns our attention to the possibility of its functional origin, though of course much more observation is wanted. Taking into consideration the history of the case, the mode of its onset, the amelioration of the early symptoms by rest, and the extreme difficulty of suggesting any other plausible explanation for this typical case, it may be said that the hypothesis of its functional origin covers the facts before us; and, provided it be not shown to be contrary to others, may possibly, if tentatively held, with full readiness to drop it in the light of further knowledge, be found of some service in the study and treatment of paraplegic symptoms.

Other affections ranked under the head of system-diseases of the cord I do not propose to discuss here. Progressive muscular atrophy, and the disease long known under the name (now proved inaccurate) of infantile paralysis, whose morbid anatomical concomitants are localised in the grey anterior cornua of the spinal marrow, present some differences in character from the affections connected with lesion of the white matter that we have been discussing. The lesions here

do not take a physiological course in the disease known as polio-myelitis anterior; and the existence of a primary inflammation, especially affecting the grey matter of the cord, and occurring, as it is believed, in the cervical and lumbar enlargements, is not out of harmony with the clinical facts or the anatomical conditions of the case. The chronic change in the anterior cornua observed in progressive muscular atrophy, corresponding as it does in position to the entry of the nerves supplying the affected muscles, is not contradictory to the theory of the functional origin of the disease. That the mischief begins in the periphery is held by several observers, both clinical and anatomical, and many facts in the history of these cases seem to favour this point of view.

Certain affections of more or less permanence, characterised by paralysis or spasm, but which are not as yet connected with any observed central change, may be here alluded to, as apparently of functional origin, and illustrative of the point under discussion. The frequent great chronicity, for instance, especially when not sufficiently regarded at the outset, of scrivener's palsy, or writer's cramp, would lead us to suppose that some central change had taken place. The marked failure of co-ordination seen in this disease seems to imply some kind of disorganisation of the central structures immediately concerned in the previously harmonious action of the muscles. Yet the apparently functional origin of such an affection as this is very striking, and widely believed in. There is, moreover, reason to believe that it is most often seen in members of "neurotic" families.

The prognostic and, possibly, the therapeutic aspect of this method of regarding some of the system-diseases of the cord deserves perhaps a few words in conclusion. The obvious physical result of overwork (whether the overwork be regarded as absolutely primary in causation or not) in such a disease as scrivener's palsy calls loudly on Rest for its cure or relief; and if taken in time, the disease frequently yields to this rational treatment.

With respect to the prognosis, and perhaps the prophylactic treatment, of spastic paralysis and tabes, a possible hint may hence be derived. The established disease of course implies

the structural central change; and treatment here can do little or nothing. But the study, when possible, of early untoward symptoms which, though known often to precede the well-recognised disease, yet, according to the hypothesis of functional disturbance, may not portend the necessary sequel of the full-blown malady, might help in some cases to prevent the hopeless and dogmatic diagnosis that is in danger now of being given, even on the strength of a single, and especially a newly-observed, symptom; and perhaps in some few cases might hinder the occurrence of the incurable malady. It is possible that in these days of minute and microscopical research we may sometimes fail to hold ourselves off, as it were, and take a look at our facts from a little distance. To get a true notion of our object we must vary our focus and our points of view. And just as the clinics and pathologies of pulmonary consumption are in some quarters regarded, with apparent satisfaction to the mind, as already entirely revolutionised by the discovery of the tubercle *bacillus*, and the accumulated experience of past observers in the field seems to be overlightly, or at least prematurely, set at nought, so in neurology the recently-studied changes in the cord may tend to engross the attention of observers too entirely as ultimate facts, and thus perhaps prevent us from reaping the advantages that sometimes result from merely changing our point of view.