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BY

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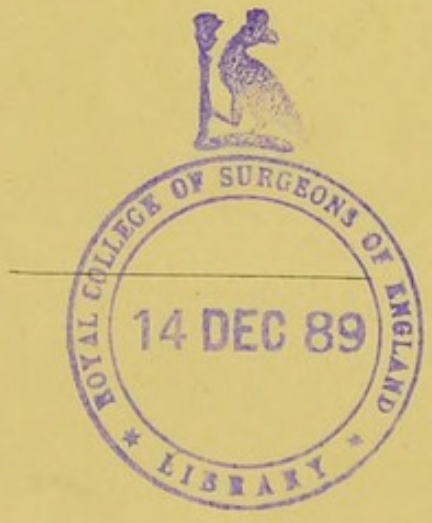
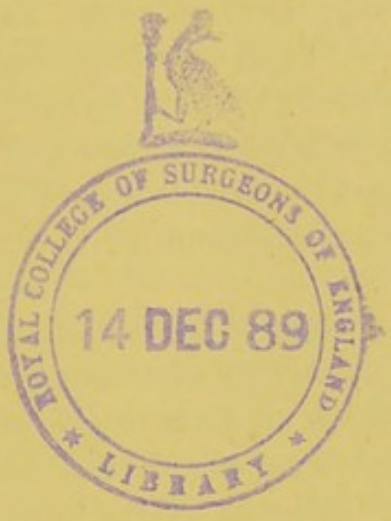


LONDON:
HARRISON AND SONS, ST. MARTIN'S LANE,
Printers in Ordinary to Her Majesty.

1886.

[*Reprinted from the 'Medical Society's Proceedings,' Vol. IX.*]



SPASTIC PARALYSIS.

By A. Hughes Bennett, M.D., F.R.C.P.

Mr. President,—At your invitation I attempt to bring before the Society a subject of great importance. It concerns a morbid state of widely-spread occurrence, and one which must therefore frequently come under the notice of every medical man. It is, notwithstanding, a condition very obscure in nature, causation, prognosis, and treatment. In consenting to open a conference on spastic paralysis, I do not anticipate that the darkness which surrounds the question will be illuminated by any original revelations of my own. I am, however, encouraged to venture the undertaking by the belief that directing attention to the leading problems to be solved will, without effort on my part, supply a text which the learned may expound, and thus initiate a discussion which may prove of scientific and practical value.

Spastic or spasmodic paralysis is the name which has been given by Professors Erb and Charcot, not to a disease, but to a group of symptoms—to a clinical picture—which must be familiar to everyone. It essentially consists of a morbid condition in which motor weakness, exaggeration of the tendon phenomena, and rigidity of muscles are the prominent and characteristic features. Owing to the existence of these physical states the patient afflicted suffers from certain symptoms which as a consequence ensue—namely,

deficient voluntary activity, tremors, peculiarities in gait, and distortions of the limbs. This combination of circumstances is, moreover, not of necessity associated with any interference with the general heath or the functions of the body, and the intellectual, sensory, and nutritive organs may be preserved intact. In short, this train of abnormal symptoms, to which the term "spastic" has been applied (owing to muscular spasm being the leading feature of the disorder), is strictly confined to the motor elements of the nervous system.

The *distribution* of this spastic condition varies. It may be confined to a single limb, it may assume a hemiplegic or a paraplegic form, or it may involve all four extremities and every district of the body. As regards *causation*, it may be associated with degeneration of the lateral columns of the cord, secondary to disease of the brain or pyramidal tract, as in cerebral hæmorrhage, myelitis, &c.; or the lesion may be of primary origin in the same regions, thus constituting a disease *per se*, as in so-called primary lateral sclerosis. Also it is met with as a functional disorder from unknown causes, as in hysteria; and it may be the result of certain definite blood-poisons, such as strychnia and lathyrus cicera. Further, as to *pathology*, it is frequently accompanied with demonstrable structural alteration of the nervous system, which appears to be its direct cause, but as often all traces of discoverable tissue change are absent. Again, the *progress* of the disorder is not uniform: at one time it invades rapidly, is acute, and of short duration; and at another is insidious in origin, chronic, and of long persistence. Finally, its *termination* varies, in some instances the disease being hopelessly and permanently incurable, while in others the patient rapidly and completely recovers. This varied distribution, origin, pathology, progress, and termination of the same group of symptoms furnishes a mystery to unravel, and serves to show how perplexing are the difficulties of the subject under notice.

Various theories have been propounded to explain the occurrence of spastic paralysis, the essential features of which, it may be repeated, are motor weakness, exaggeration of the tendon phenomena, and muscular rigidity. Professor Charcot maintains that the symptoms are due to irritation of the ganglion cells of the grey matter of the cord by degenerated contiguous fibres of the lateral columns. It is now well known that a destructive lesion of the

motor cortical area in the cerebral hemispheres, or in any portion of the pyramidal tract, induces secondary degeneration of the efferent fibres below, which are thus cut off from the trophic influences of the cells in the grey matter of the brain. This sclerosis extends downwards as far as the multipolar ganglia in the anterior cornua of the cord, and the hypothesis suggested is that the morbid process on reaching the cells causes their irritation, and as a consequence the phenomena of spastic paralysis. That this theory is plausible cannot be denied, and it is possible that it may be a factor in the production of the clinical symptoms in question. It is at best, however, only an imperfect explanation, and not the essential cause, as spastic paralysis may exist in its most typical form when no degeneration of nerve fibres can be demonstrated, as in various functional complaints such as hysteria. Moreover, it may occur under circumstances where it is impossible to conceive the presence of any such permanent or gross structural alteration, as in strychnine poisoning or during the hypnotic state.

Another theory, and perhaps the most popular, asserts that the symptoms of spastic paralysis are due to the simple severance of the spinal from the cerebral centres. In this way it is supposed the inhibitory action of the higher is removed from the lower ganglia. These last are, as it were, thus permitted, from want of control, to run riot, and as a consequence to manifest hyperexcitability of reflex acts, in the shape of increased tendon phenomena and muscular rigidity. It is admitted that during the absence of brain influences the reflex excitability of the cord is augmented, but the train of symptoms included under the term spastic are not produced. Moreover, in sudden destructive lesions of the brain, such as hæmorrhage into the pyramidal tract, although spastic symptoms subsequently ensue, they do not appear until a considerable period of time has elapsed. Now, if simple inhibition were the sole cause of the phenomena, we should expect the effects to be most marked at the moment of interruption, and not gradually acquired, as is the case.

A third theory, which is a modification of the last, maintains that when cerebral influences are suppressed, the uncontrolled action of the cerebellum as a stimulator of muscular tonus becomes prominent, and hence the stiffness which takes place. The same arguments which can be urged against the last hypothesis seem to me to equally hold good against this. A further proof of its

inability to account for the position is, that when the cord is completely divided, as in transverse myelitis, spastic symptoms subsequently ensue, although all communication between the cerebellum and cord has been severed.

Such are the leading theories which have been advanced to explain the phenomena of spastic paralysis, as well as some of the reasons to show that no one of them by itself is adequate to account for their production. While it is not difficult thus to take a negative view of the position and criticise existing doctrines, it is not so easy to successfully assume a positive and constructive attitude, and to replace received opinions by others more worthy of acceptance. In attempting to frame a hypothesis which will harmonise with all the facts of the case, it seems to me that the entire question must be viewed from a more comprehensive standpoint than heretofore. The present tendency of neurological science is towards specialisation, and doubtless the great advance it has made of late years is mainly due to success in that direction. At the same time, in order that the accumulation of isolated facts may be utilised to the fullest extent, the laborious process of individual inquiry must be supplemented by generalisation. In the disorder under consideration, as well as in many other morbid states, by taking a more extended view of the situation, it may be found that the supposed classical lesion of a disease is only one factor in its etiology ; that, indeed, it may be a result rather than a cause, and that a variety of circumstances from different directions may all equally tend to produce the same effects. Looking, then, at the clinical picture of spastic paralysis, as it were, from a distance, and taking into consideration the manifold conditions of its causation, progress, and morbid anatomy, the following conclusions seem to me to be those which best accord with known facts.

From whatever source the disorder may arise, the symptoms are essentially the same, varying only in distribution and degree. The characteristic motor weakness, the exaggerated tendon reflexes, and the muscular rigidity probably belong to the same series of phenomena, and have the same physiological signification. The common factor in the production of this series of conditions is an abormally increased irritability of the motor ganglion cells in the anterior cornua of the cord. Physiological knowledge has taught us that one of the functions of these centres is to maintain a certain degree of permanent tonus of muscular fibre. For this purpose integrity of

the reflex arc is necessary. Hence if either the ganglion itself, or its afferent or efferent elements are destroyed, the muscle relaxes, and its response to mechanical percussion (in other words, its tendon phenomenon) is lost. If, on the other hand, the ganglion is from any cause in a state of hyper-excitability, the opposite conditions ensue—namely, hyper-tonicity or rigidity of muscle and spastic symptoms. The order and extent in which the different symptoms appear to develop seem to bear some relation to the amount of the excitability of the cells ; hence the first manifestation of their irritable weakness is motor paresis, and then in order appear, according to its degree of severity, increased tendon reflexes, clonic spasms, muscular rigidity, and permanent contracture.

If, then, a morbid irritability of motor ganglia is the common property of all forms of spastic paralysis, we have next to determine how this in turn is produced. I believe that this is not to be found in any single explanation, but that a variety of circumstances may act as exciting agents. These circumstances may be mainly divided into three categories: (1) primary irritation of the ganglion cells themselves; (2) their irritation through the afferent and efferent paths from below; and (3) their irritation through the pyramidal tract from above.

The ganglion cells may be primarily affected in many ways. Strychnia and other poisons, for example, may directly derange their nutrition and produce temporary spastic symptoms. Changes in the quality of the blood, such as anæmia, fevers, &c., may in a lesser degree be followed by the same results, and it is common in such circumstances to meet with muscular weakness, increased tendon phenomena, and a tendency to cramp and stiffness of muscle. The same irritable weakness is found in most exhausting ailments, such as phthisis and convalescence from acute diseases, in which the grey matter of the cord participates with the other tissues of the body in an abnormal readiness to respond on stimulation. It is still further developed in those states of nervous instability characterised as neurotic, such as hysteria, in which spastic symptoms often reach a very high degree.

In addition to direct irritation of the ganglion cells themselves, these elements may be influenced by irritation from below. The well-known observations of Brown-Séquard demonstrated that injury to afferent fibres may cause so-called spinal epilepsy—in

other words, extreme hyper-excitability of the grey matter of the cord. Again, Dr. Weir Mitchell and my colleague, Dr. Donkin, have advanced clinical reasons to show that over-stimulation of the efferent tracts may produce the same results. Such evidence tends to indicate that peripheral irritation of both afferent and efferent fibres may alike lead to hyper-activity of the ganglia to which they are attached.

Influences from above may in like manner be followed by similar results. Experimental irritation of the motor cortical area induces muscular spasm. In disease the same occurs. It has been observed that during an epileptiform attack—that is, during a period of cortical irritation—there is both contraction of muscle and exaggeration of the tendon phenomena, showing that the excitement of nerve tissue has extended to the cord. Even after voluntary motion has been abolished, owing to the temporary exhaustion which follows an explosion of the cortical motor centres, the exalted functional activity of the lower ganglia may remain, and consequently the spastic symptoms may continue. In a short time, however, when the secondary irritation of the cord subsides, the symptoms disappear. In like manner, irritation from inflammation or otherwise in any portion of the pyramidal tract may be followed by similar results, and if the cause be chronic the symptoms may be permanent.

Again, it has been found that not only an irritative lesion, but destruction of the cortical motor area subsequently, but not immediately, leads to muscular hyper-tonicity. The same takes place from disease of any part of the pyramidal tract by which the distal portion is separated from the cerebral trophic elements. This may possibly be due to direct irritation caused by the secondary descending sclerosis, as suggested by Charcot. But prior to demonstrable degenerative change in the lateral columns there are evidences of motor instability, as a few hours after an apoplexy exaggerated tendon phenomena gradually develop. It is therefore obvious that nutrition changes, as a result of removal of cerebral trophic influences, must exist in the motor tract before structural alteration can be appreciated. The nature of this change, whether vascular, molecular, or dynamic, we do not know, but that it is sufficient to produce objective functional symptomatic disorder is certain. A plausible explanation of this fact would be that anæmia or other nutritive modification of the pyramidal tract

was the almost immediate consequence of the disorganised trophic centres above, and that this change in the circulation or molecular stability led to irritable weakness of the adjacent grey matter of the cord. The same effects might supervene upon conditions other than those of actual gross cortical disease, as when the higher nervous elements, without demonstrable anatomical change, are functionally disordered. In such a case the actions of the parts below which are dependent for their constancy on healthy influences from above, would be deranged. Thus from functional irritability or depression of the cerebral cortex there might arise instability of the pyramidal tracts and the motor apparatus connected with them, and, as a consequence, a train of typical spastic symptoms. Such excitants would be all the more potent in a predisposed subject in whom the spinal ganglion cells were already in an abnormally exalted condition, as in such the slightest physical injury readily provokes muscular spasm. Under the same or similar conditions it is but a step further to realise that psychical influences may act as exciting agents for the production of like results. Thus, misdirected volition, emotion, or imagination acting as afferent stimuli might be the originators of abnormal motor phenomena, even when the nervous system itself is in other respects healthy. Again, even normal mental impulses may cause undue motorial demonstrations if the spinal centres are hyper-excitable, as is seen in strychnine poisoning, hysteria, &c. It is therefore obvious that if both these abnormal conditions exist together—that is, increased power of the higher centres to receive or originate mental impressions, and augmented capacity of the lower centres to respond to psychical stimuli—the effects will be the more violent in degree and permanent in duration, and serves to explain the severity and intractability of many functional disorders. It also gives an explanation of the fact that under these circumstances the physical motor symptoms are sometimes such as could not voluntarily be produced in health, as instanced by the phenomena of hypnotism and hysterical malingering.

In connection with these reflexions it is a question of the greatest practical importance to know whether repeated and permanent functional over-activity or depression can lead to demonstrable structural changes in the tissues affected. *A priori* reasoning would favour such a hypothesis, although there is not a sufficient series of recorded facts to demonstrate it. At present brevity com-

pels me to forego speculation on this interesting subject. Everything, however, seems to indicate that the causes which lead to hyper-excitability of the ganglion motor cells, and consequently to the production of spastic paralysis, are of an active or irritating rather than of a passive or inhibitory nature. This is supported by many facts; among others, that when one side of the body is primarily affected, there is invariably a tendency for the other gradually to become involved; also, that all circumstances which allay irritability, such as rest, sleep, tranquillity, and sedatives, relieve the symptoms, while the reverse conditions of emotion, exertion, injury, and stimulants augment them.

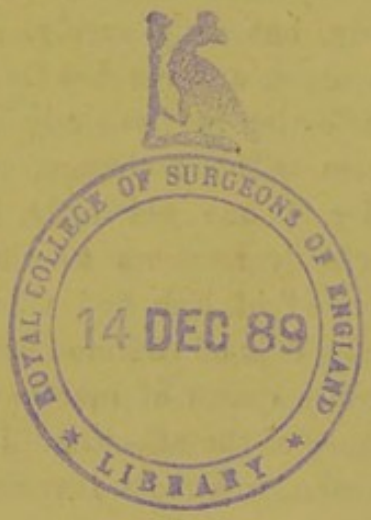
To sum up, it is suggested that spastic symptoms, from whatever cause arising, are essentially due to abnormal functional hyper-excitability of the motor ganglion cells of the anterior cornua of the cord. The hyper-excitability may for various reasons be inherent in the cells themselves, or may be induced therein by influences emanating from the brain, the cord, or the peripheral nerves. These influences are probably of an irritating nature. The irritation, when it exists in the pyramidal tract, may be excited there either directly by organic disease or functional excitement, or indirectly by changes in its nutrition as a result of severance from its trophic centres. These changes in nutrition may be either gross or dynamic. The gross changes usually follow gross disease of the cortical centres, while the dynamic changes may either follow their structural alterations or their functional disorder. Finally, this functional disorder of the motor tract may have its origin in psychical as well as in physical activity. These theoretical considerations concerning the pathology of spastic paralysis, like other hypotheses, are open to criticism. I admit they are somewhat involved, but this is scarcely to be avoided in attempting to account for the complicated circumstances connected with this group of symptoms.

If, gentlemen, the doctrinal speculations on the subject we are discussing are uncertain, the practical difficulties met with at the bedside are none the less perplexing. As their clinical features are now well recognised, I pass over those forms of spastic paralysis associated on the one hand with secondary changes from gross disease of the nerve centres, and on the other with obvious functional disorder. I limit myself to directing attention to a condition in connection with this subject which has

always seemed to me most obscure, and which I cannot find anywhere accurately differentiated or satisfactorily explained. It is a group of symptoms which everyone must have met with, and of which there are in the other room several illustrative examples. In my own limited experience it is of frequent occurrence; and I find in my note-books of the last two or three years the detailed account of twenty-four typical examples. An individual, either male or female, in otherwise good health, without obvious cause, gradually experiences slight weakness in a limb—say the leg. This, as a rule, very insidiously increases, and, after a year or more, extends to the arm of the same side, sometimes to the opposite leg, and subsequently to all the extremities. On examination motor weakness is detected without incoördination; there are clumsy and feeble actions of the upper, and a weak, shuffling, stiff gait with the lower extremities. At an early stage there is great exaggeration of the tendon phenomena, the cutaneous reflexes remaining normal. Afterwards ankle-clonus is developed, and later slight stiffness of the muscles and trepidation of the limbs. The general health remains robust, and all the organs and functions of the body are normal. Sensibility and nutrition continue unimpaired. Occasionally, but very rarely, in my experience this abnormal condition is acute, and reaches its full development with great rapidity. The affection may entirely recover, but as often it persists for many years without change, or slowly increases in severity. The disorder is one evidently limited to the motor apparatus. It imperfectly answers to the description of the "primary lateral sclerosis" of Erb and Charcot, but differs from that disease, inasmuch as it is of common occurrence, it frequently recovers, and also because spasm of muscle with its consequences is not the leading feature of the affection. It is a condition which, when occurring in women, is almost invariably pronounced to be hysterical; when in men, it is vaguely called paraplegia or myelitis. For convenience I describe the group of symptoms under the term "hyper-tonic paresis," a symptomatic nomenclature which commits to no theory. This in every respect answers to the hypothesis I have already advanced concerning the nature of spastic paralysis.

In conclusion, I venture to suggest the following points for discussion:—1. Is the organic degeneration of the lateral columns of the cord, so frequently associated with spastic symptoms, essential

for their production, or may they be accounted for by functional derangement of the same regions, or of the psychical or motor centres? 2. Whether organic or functional, is the primary seat of disease in the cells of the brain or cord, or in the conducting fibres, central or peripheral? Are the symptoms the result of irritation or inhibition? 3. May primary functional disorder eventually lead to secondary organic degeneration? 4. Regarding the cases described under the term hypertonic paresis.—What is the anatomical substratum of the affection? Where and in what manner does it originate? Is the condition one of organic or functional disease, and by what clinical criterion is the point to be determined during the life of the patient? What is the cause, prognosis, and treatment of the disorder?



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