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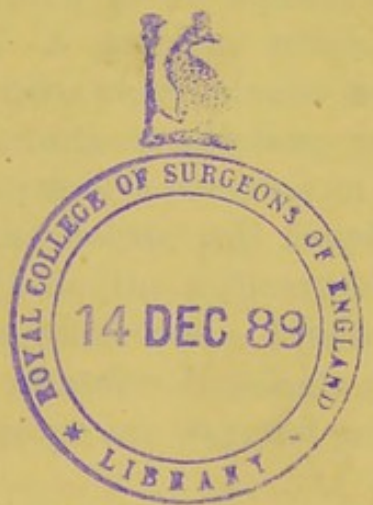
BY

A. HUGHES BENNETT, M.D.



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MUSCULAR HYPERTONICITY IN PARALYSIS.

BY A. HUGHES BENNETT, M.D.

HAVING been requested by the Council of the Neurological Society to provide a subject for consideration at the present meeting, I have selected one, which will require no advocacy on my part to ensure an interesting and profitable discussion. The morbid condition which will form the basis of this communication is of sufficiently frequent occurrence to render its study instructive to the practical physician, and the explanations of its phenomena are so contradictory and obscure, that their investigation will prove equally attractive to the speculative pathologist. A problem which is at the same time practical and scientific, which almost daily comes before us only to baffle our efforts at solution, is obviously well-adapted for the deliberation of a congress of medical experts. My duty in opening the debate will simply be to lay the case before you, to indicate the difficulties of the position, and to invite discussion thereupon. I trust that those who follow me will in some measure illuminate the darkness into which I am about to grope my way, not without feelings of considerable insecurity.

The general subject which it has been suggested I should lay before you is the apparently paradoxical condition of increased muscular tonicity as associated with motor weakness —a question in its entirety so vast, that it would be hopeless for me to attempt to cover the whole ground in the time at

my disposal. I will therefore limit my observations to certain special points, that will serve both to direct attention into channels convenient for discussion, and to provide a text for those desirous of dealing with the subject in a more comprehensive manner.

I propose first, to illustrate the association of muscular hypertonicity with motor weakness, by the brief description of a morbid state, which although commonly met with in practice, is apparently neither generally recognised nor understood, and which moreover is not formulated in any of our text-books on medicine; and secondly, to hazard such explanations of its phenomena as seem warranted by the present state of scientific knowledge.

An individual in his or her usual health and without discoverable cause, becomes aware of a feeling of weakness in one or both lower extremities. This at first is so slight as to attract little attention. As months pass by, very insidiously the debility becomes more marked. This condition may continue with little change for many months, or even years, nevertheless it very slowly increases. The sufferers consult many doctors, from whom they derive little benefit or consolation. If a woman, she is probably pronounced to be hysterical; if a man, he is hypochondriacal: or an explanation in his case is abandoned in despair. In both instances a fertile opening for plunder is provided for the bone-setter and charlatan. When seen at this stage, that is from a few months to even several years after the first discovery of the weakness, the patient still may preserve robust general health. The most careful and exhaustive physical examination fails to discover anything objectively abnormal, except evidence, 1st, of motor weakness, and 2nd, of excessive contraction of the muscles to percussion of their mass or tendons.

On investigating the first point, movement is seldom found to be completely lost, the actions of the affected parts can usually be performed in a natural manner, but are simply deficient in power. There is no special peculiarity of gait, it is only weak, shuffling, and wanting in vigour; the limbs feel heavy, and the patient is easily fatigued by efforts at walking. There is special difficulty in ascending stairs, and

even on level ground the feet are liable to trip over any irregularities on the floor. The points and inside of the boots are worn. The same condition may exist in the upper extremities, the movements being natural in kind, but deficient in quantity. The patient soon becomes tired when writing, sewing, and in the performance of accustomed acts. All the finer actions are slow and clumsy. The grasp of the hand, as determined by the dynamometer, is weak. Movement whenever modified is simply defective in vigour, without cramp, spasm, pain, inco-ordination, or other qualitative peculiarities.

The second prominent feature in these cases is the behaviour of the muscles of the affected parts when percussed. The phenomena which in health are observed to follow the usual test of a blow on their substance or tendon, are exalted in degree, although apparently remaining the same in kind. Expressed clinically, the response of the muscle to percussion is increased, and the tendon phenomena are exaggerated. Every degree of exalted action may exist, from the slightest elevation, impossible to differentiate from health, to the most extreme demonstrations of muscular excitability. In advanced cases trepidations and cloni may be produced in various situations, especially at the ankle and knee. These, however, are usually absent or only imperfectly developed. Tapping the bone in its neighbourhood is often sufficient to cause contraction of a muscle.

With the exception of these two particulars, namely, motor weakness and muscular irritability to percussion, nothing further abnormal is to be found in this class of case. There is no marked rigidity or spasm, although sometimes there seems to be a feeling of somewhat increased muscular tension to passive movement. The superficial reflexes are usually normal, if anything, slightly increased. Sensibility is everywhere unimpaired, and no pain accompanies the complaint. Occasionally the patients assert they experience a feeling of numbness or formication; but this is entirely subjective, and the most careful testing fails to determine any alteration of sensibility. The so-called muscular sense is generally intact, though in some instances I have found

slight loss of capacity to localise movements with the eyes shut. In these cases, however, some doubt existed as to the uncomplicated nature of the disorder. There are nowhere primary trophic changes of any kind. In very chronic cases the affected limbs become somewhat smaller than the others, evidently due to physiological atrophy from diminished functional activity, as the decrease in size is universal, and the electrical reactions remain normal. The affected parts are cold, and sometimes slightly cyanotic, but not to any great extent. Intellect and the various organs and functions of the body are normal, the bladder, rectum, and sexual apparatus remaining as in health. Occasionally, but rarely, the patient complains of some difficulty in retaining urine for long periods, otherwise the bladder and its accessories are intact.

The foregoing brief account may be looked upon as typical of the group of symptoms under consideration, and serve to describe the majority of cases, as they have come under my observation. The only complaint made by the patient is motor weakness, and the only objective sign discoverable by the physician is excessive muscular contraction on percussion. As in other morbid conditions, there are varieties in severity, course, complications, termination, and so on, which deviate from the more common forms. For example, as to distribution, the most ordinary seat of the affection is the lower extremities. Usually it begins in one leg, and sometimes but rarely remains limited to it. A young lady, aged 26, and otherwise in good health, suffered from the characteristic symptoms of this complaint, and limited to the left lower extremity. This appeared very gradually, remained for three years, and slowly disappeared. She is now perfectly well. As a rule, however, the morbid state after a shorter or longer interval extends to the other leg, thus constituting the paraplegic form of the disease, one extremity remaining more advanced than the other. This is by far the most common variety. Beginning in the leg, the malady not infrequently spreads to the arm of the same side, producing the hemiplegic type. Occasionally the weakness and muscular irritability to percussion is limited to one side of the body, more frequently

however, the other soon also becomes affected, even if to a lesser extent. Less commonly all four limbs are equally involved, and still more rarely every muscle in the body shows signs of instability. For example, in 'BRAIN,' for July 1886, I described a case of a young woman, aged 26, who for two years suffered from general motor weakness. All her muscles displayed extreme irritability to percussion, and clonus could be produced by tapping the tendon of any of them, including those of the face, neck, and trunk. This girl subsequently recovered.

The condition I am describing is met with in every degree of severity. From the slightest diminution of physical power and exaltation of muscular irritability to percussion, to complete paralysis with extreme excitability—as evidenced by the production of cramps, spasms, trepidations, and cloni on the slightest provocation—every possible variety and degree may exist. In the cases under the first category it may be difficult to determine if the actual condition found is morbid or not, as the muscular response to percussion in health greatly varies. In those under the last there is always a doubt, unless the case has been followed from the beginning, whether the symptoms are not the result of secondary degenerations from a gross lesion higher up in the pyramidal tract.

Again, the subjects of this disorder may be liable to various symptoms not of necessity associated with, or essential to it, and which may sometimes complicate the diagnosis. Aching in the back and limbs, subjective sensations of numbness, headache, depression of spirits, and so on, may be present, as in any other disease. As a rule, all these symptoms are conspicuous by their absence.

In most cases the onset of the complaint is very insidious, and the progress is very slow and chronic. For example, I have a man under observation who has suffered from this affection for 8 or 10 years, and without any perceptible change either for better or worse. In others, however, with apparently identical symptoms the onset has been very sudden, and after a rapid course the symptoms have disappeared with equal promptness. Thus a young man, aged 27, while in his usual good health, with no apparent cause became weak in his legs,

This increased so rapidly that in a week he could scarcely walk; the debility extended to his arms. On examination, the fact of the motor weakness was established; there was, in addition, extreme readiness of the muscles of all four extremities to contract on percussion, and clonus, more especially of the ankle and knee, could easily be provoked. Beyond this absolutely nothing abnormal could be detected, and there was no rigidity of the muscles. This man completely recovered in a few weeks, with rest and large doses of chloral hydrate, and he has remained quite well ever since. Between these two extremes, namely, those who seem to remain for years in statu quo, and those whose illness is completed in a few weeks, are many who run a medium course of varying periods and duration, ending in more or less complete recovery.

In my experience the two sexes are about equally subject to this disorder. Of the last twenty uncomplicated cases, twelve were males and eight females. The ages in my cases have ranged between twenty and forty years. I have not been able to determine any cause for the complaint, either predisposing or exciting. In one instance only, two brothers were affected; in none of the others was there any special evidence of hereditary predisposition to this or other nervous disease. Some of the patients were subject to exposure and hard physical work, but quite as many had favourable surroundings and were of sedentary habits. No special association with syphilis or other constitutional peculiarity could be determined.

The final evolution of this group of symptoms is difficult to ascertain in a sufficiently large number of cases to enable me to speak with certainty on the subject. Owing to the very chronic nature of their complaint, many patients are lost sight of. At the same time the cases which I have had opportunities of observing for some years have enabled me to formulate the following conclusions: 1st, some of the patients completely recover; 2nd, others continue for many years without apparent change; and 3rd, others slowly progress, the disease extending in distribution from below upwards, and increasing in severity. On each of these heads a few details may be given. At the onset it may be observed that in the cases composing these three groups the symptoms are apparently identical, and

there is nothing to give any indication for making a prognosis.

Complete recovery may take place, sometimes rapidly, but more commonly very slowly. The young man cured by rest and chloral, already referred to, is an instance of the former. The girl already mentioned, who had been ill for three years, is another example. Improvement takes place, as a rule, more slowly. Thus the young lady, of whom I have before spoken as having been affected in one leg only, began to recover six months after she came under observation. Slow improvement continued for three years, after which she expressed herself as quite well. No special treatment had been employed, and the favourable termination has since been maintained. Again, an omnibus conductor, aged 25, had the typical paraplegic variety of the affection. Treatment of various kinds for many months had no appreciable results. For two or three years his condition remained unchanged. Recently, however, he presented himself perfectly well, having gradually improved during the last six months, and without treatment of any kind except that of rest. I could cite several other instances of the same kind. Not unfrequently partial recovery only takes place. In these cases considerable power returns, but not to the full extent. Although the motor activity improves, the muscular irritability to percussion does not diminish in a corresponding ratio, but usually still remains abnormally exalted.

The second class is that of persons who, while apparently in exactly similar circumstances as those just considered, remain for years without undergoing perceptible change in their condition. I have watched several of these for between five and six years. The weakness and muscular irritability to percussion continue as before, but there is no tendency to the development of new complications, or even to an increase of the existing state. The patients have for the most part abandoned medical treatment, and have so far become reconciled to their infirmity as to do the best they can under the circumstances. I recently saw a man whose case I carefully investigated, and minutely recorded just five years ago. He stated that his general health was excellent, and that he was

not conscious of the slightest change in his condition having taken place since he was first examined. On going over my notes I was unable to discover anything which deviated in the smallest particular from my former report.

The third group of patients are those who seem gradually to get worse. The advance is, as a rule, very slow in progress. The morbid condition extends from one limb to the other, till at last the entire body is affected. The severity of the symptoms also increases, but in my experience not to any great extent. It is probable, however, that we lose sight of the worst cases among our hospital patients, who usually gravitate into the infirmaries. I have myself never seen a case such as has just been described, afterwards pass into one of typical spastic paralysis with its characteristic rigidity of muscle. I am not prepared to affirm that this does not occur. I can only assert that I have never seen it in any of those cases which I have had an opportunity of watching for five or six years.

On the subject of treatment I have little to say. Rest is undoubtedly an important element, as exertion or fatigue increase the severity of the symptoms. Hydrate of chloral in large doses temporarily depresses the hyper-excitability of the muscles, but whether it induces any permanent beneficial result I do not know. In one case, and the worst I ever saw, the patient after having been afflicted for three years with this illness, recovered in a fortnight after an attack of acute pleurisy. All the usual therapeutic measures, massage, electricity, blisters, actual cautery, setons, &c., have in my experience been followed by no appreciable results. Those who have recovered have done so in the progress of time, and I have been unable to give the credit to any particular remedy.

The group of symptoms which I have described is, as has been already stated, commonly met with in every rank and condition of life. Its existence is frequently overlooked, its importance underrated, and its nature misunderstood by the practitioner. Its consequences, however, very seriously impair the comfort and professional capacity of the patient. It is therefore highly important, not only that the morbid symptoms be recognised and studied with the attention their gravity

merits, but that if possible some estimate be arrived at as to their true significance. Recognising the facts, and admitting the existence of a malady in which motor weakness and abnormal irritability of muscle to percussion are the characteristic and only discoverable features, we have to enquire what are the causes, pathology, prognosis, and treatment of this clinical condition. In attempting to solve these difficult problems, the ground may at once be cleared by the admission that the manifestations of this disorder are strictly limited to the muscles, and motor elements of the nervous system. Every other tissue and function of the body appears to be unaffected. There is no evidence that the muscular substance itself is the source of the disease, as its structure and nutrition are apparently normal, and its alteration in function is not of that nature which would occur from primary changes in its tissues. The seat of the morbid process therefore narrows itself to the nervous apparatus that presides over muscular contractility. Of the morbid anatomy we know nothing. In seeking an explanation of the phenomena we are therefore compelled to enter into the field of speculation, and to utilise collateral physiological and pathological knowledge in attempts to throw light on the subject. Hence there is ample scope for exercising the imagination and ingenuity of the scientific pathologist, and a wide field for divergence of opinion and argument. Discussion will be facilitated by the acceptance of certain elementary physiological premises. Before the audience I now address, it is unnecessary to enumerate these in detail. I therefore confine myself to briefly formulating such general propositions connected with the matter in hand, as in my judgment our present state of knowledge warrants, and concerning which there is a tolerable consensus of opinion.

I. Healthy muscle contracts when its fibres are subjected to sudden stretching (extensile stimulus). This may be induced by percussion on its substance, and still more effectively by a blow on its tendon, the last constituting the so-called tendon phenomenon. The mechanical condition necessary for the production of the response following percussion, is sufficient passive tension of the muscular fibre.

II. This property is intimately associated with and obeys the same laws as what has been termed reflex muscular tonicity, or that state of slight constant tension which is the characteristic of healthy living muscle.

A. Both are *abolished* when there is interruption at any portion of the reflex arc of which muscle is the terminus.

(*a.*) At the posterior spinal roots or sensory nerves, as in locomotor ataxy or neuritis.

(*b.*) At the ganglion motor cells, as in poliomyelitis.

(*c.*) At the anterior spinal roots or motor nerves, as by experiment or injury to nerve.

B. Both are *increased* by causes which augment the excitability of the same reflex mechanism. The circumstances under which this takes places are—

(*a.*) Idiopathic excitability of reflex centres of cord.

1. Isolated, as in strychnine poisoning.
2. Associated, as in neurasthenia.

(*b.*) Excitability of reflex centres, secondary to

1. Direct influence from afferent and efferent elements.

(*a.*) Afferent (reflex), as from cutaneous or visceral irritation.

(*b.*) Efferent (exhaustion), as from fatigue or over-exhaustion.

2. Indirect influence, as result of severance from pyramidal tract. This is variously explained by—

(*a.*) Irritation of sclerosed lateral columns.

(*b.*) Removal of cerebral inhibition.

(*c.*) Unbalanced cerebellar action.

(*d.*) Nutritive change in ganglia due to severance from trophic centres of cortex.

III. The preceding considerations show that integrity of the reflex arc is essential for the maintenance of muscular tonicity, and the response of muscle to extensile stimulus, or percussion. It is probable that both these phenomena have the same physiological significance, and that the one is dependent on, if it is not an identical process with, the other.

Throughout this paper, in using the terms muscular irritability or excitability, it is to be understood that those expressions are employed only in reference to this tonicity, or property of contracting under the stimulus of percussion.

Admitting the soundness of the preceding general propositions, assuming that increased response of muscle to percussion or extensile stimulus is due to hypertonicity, and that the essential factor for its production is an exaggerated excitability of the ganglion cells of the cord, it has next to be determined more particularly under what circumstances the latter change takes place. For descriptive purposes these may be considered under three heads. 1st, irritation arising primarily in the ganglion cells themselves. 2nd, their irritation due to changes in their afferent and efferent elements. And 3rd, their irritation secondary to influences acting through the pyramidal tracts.

The spinal centres may be primarily rendered unstable in many ways. Strychnine, alcohol, and other poisons, for example, may directly damage their nutrition and produce temporary hyper-excitability of their function. Changes in the quality of the blood, such as anæmia, fevers, acute disorders, &c., may in a lesser degree be followed by the same results; and it is common in such circumstances to find muscular weakness, increased tendon phenomena, and a tendency to cramp and stiffness in the muscles. The same irritability is present in circumstances in which the general system is in a state of subnormal nutrition, as it seems to be a neurological law that nervous depression is associated with instability of function, excessive motor display, and an abnormal readiness to respond to stimulation. Hence in childhood, where the nervous system is as yet imperfectly developed, the characteristic restlessness and proneness to muscular demonstration on the slightest provocation. The diseases at this period of life are also of the same type, as seen in chorea, tetany, convulsions, &c.; and these, as is well known, are usually induced by debilitating conditions, such as syphilis, rickets, fright, and so on. In the adult the same holds good, if to a lesser extent, and loss of blood, fatigue, diarrhœa, cholera, sexual excess, excessive discharges, and other ex-

hausting maladies, may cause temporary cramps, spasms, and other evidence of muscular hyper-excitability. In chronic diseases such as phthisis, it is quite common to find patients in whom the muscles react with abnormal activity to percussion, and in whom the tendon phenomena are exaggerated. In patients convalescent from various diseases I have often found the same, which symptoms disappeared when health was completely restored. In all of these cases the grey matter of the cord participates in the general debility and malnutrition of the tissues of the body, and as a consequence it is in a state of morbid excitability, which manifests itself by abnormal muscular hypertonicity. The same condition is still further, and permanently developed, in those states of nervous habit characterised as the neurotic diathesis, in some of the varieties of which, such as hysteria, this muscular peculiarity often reaches a high degree.

In addition to direct primary irritation of the reflex centres themselves, these ganglia may be thrown into a state of over-activity as a result of morbid impressions derived from their afferent or efferent elements. The well-known experiments of Brown-Sequard demonstrate that injury to *sensory* nerves may induce so-called spinal epilepsy : in other words, a chronic state of extreme hyper-excitability of the grey matter of the cord.

The same has been abundantly proved to be the result of various clinical conditions, and may follow wounds, and accompanying cutaneous, visceral, joint, and other affections, constituting the so-called reflex hyper-excitabilty of the cord. Whether the stimulation of the peripheral afferent elements induce central changes by direct irritation, by reflex circulatory alterations, by simple functional exhaustion, or by an ascending neuritis, need not now be discussed. The fact remains that irritation of sensory nerves in any part of the body may conduce to over-activity of the motor cells in the cord to which they are attached. The permanence and degree of the peripheral stimulus appears to bear some relation to the amount and distribution of the reflex effects produced. These in consequence may be local or general. If the excitant be moderate, the result, by irritation, is over-action; if excessive, by exhaustion, is abolition of function. Hence

hyper-excitability of motion on the one hand, and paralysis on the other. Thus in the same nervous mechanism, and from the same cause, may arise both irritability and incapacity of muscular action, or as it has been called, a state of "irritable weakness," the two phenomena being essentially the same in kind, only differing in degree. In this way irritations originating in the uterus, kidneys, bladder, prostate, teeth, and other localities may, according to their permanence and severity, cause either spasm or paralysis, or both. In connection with this subject one point may be noted, that a peripheral irritation on one side of the body may not only induce reflex motor responses on the same side, and on the same segment of the cord, but on the other side and in various other regions of the body. This shows that the excitation induced by the afferent stimulus has extended through the grey matter of the cord itself, and is therefore the result of an active irritation process propagated from cell to cell. The effects produced will vary in intensity and distribution according to the character of the excitement, and the original stability of the central nerve elements. Thus the effects of a decayed tooth in one man is slight and local, in another is severe and general. In some constitutions a wound is innocuous, in others it is followed by tetanus.

With regard to the influence of the *motor* elements on the central reflex centre, clinical evidence appears to show that fatigue, muscular exertion, and over-work, lead to instability of the ganglionic cells. This is notably instanced in the case of professional hyperkinesis, or so-called cramp. Excessive physical labour has for long been supposed to have originated various forms of spinal disorder. Dr. Weir Mitchell and Dr. Donkin have recently advanced reasons to indicate that over-fatigue is the chief exciting cause of spastic paralysis, with its well-marked hypertonicity of muscle.

The preceding considerations would seem to indicate that peripheral irritation or exhaustion of both afferent and efferent fibres may alike lead to secondary hyper-activity of the central ganglia with which they are connected, although the last may have originally been in a healthy condition.

Besides the artificial or constitutional circumstances which

directly bring about primary instability of the spinal nerve centres, and the irritation of their ingoing or outgoing paths which produce the same as a secondary consequence, a like pathological condition may follow a totally different source of origin, which brings us to the consideration of the third series of conditions under which hyper-excitability of ganglion cells may be produced, namely, through the influence of the pyramidal tract and cortex of the brain. Although the reflex mechanism already described is the essential factor in the production and maintenance of muscular tonicity, its action is presided over and controlled by influences from above. In health there is a state of equilibrium between the higher and lower centres which permits the normal amount of muscular tone, but if the former are in any way interfered with, the latter become deranged in a corresponding manner. By means of the pyramidal tract, the spinal centres are in direct communication with certain areas in the cortex cerebri. Experimental irritation of the Rolandic regions causes muscular spasm, showing that a stimulus applied to the cortex is by the pyramidal tract conveyed to the spinal ganglia, and through them to the muscles. Irritation by disease of the same regions produces the same effects, and during the paroxysms of muscular convulsion, which as a consequence of the cortical excitement ensues, I have often satisfied myself that in addition to the spasm, the tonicity of muscle was augmented as evidenced by a increase of the knee-jerk. In such cases the exalted functional activity of the lower ganglia may remain, even after voluntary motion has been abolished, owing to the temporary exhaustion which follows an explosion of the cortical centres. When the secondary irritation of the cord subsides, this symptom quickly disappears. The same condition is met with in the so-called "late rigidity" following certain brain lesions. Irritation of any portion of the pyramidal tract may in a like manner induce tonic spasm, and if the cause be chronic the effect may be persistent. The excitability is directly conveyed to the ganglion cells of the cord, and induces in them that exaltation of function which causes excess of muscular tonicity.

A remarkable and seemingly paradoxical fact is that

destruction of the same regions, namely, the motor cortical centres and the pyramidal tract, is gradually followed, not immediately but after an interval of time, by the same hyper-excitability of the spinal centres. This is notably seen in the late rigidity succeeding brain lesions and in primary lateral sclerosis. The explanation of this phenomenon will be afterwards discussed ; it is sufficient at present, in connection with this fact, to state that the cells in the cerebral cortex are the trophic centres of the fibres in the pyramidal tract, and that when the former are destroyed the latter in consequence undergo degeneration as far as the ganglia in the cord, which, although left structurally intact, develop as a result hyper-excitability of function.

To repeat and sum up, exaggerated activity of the ganglionic cells in the grey matter of the cord which results in hypertonicity of muscle may result from—1st, primary irritability of the cells themselves (strychnine, &c.) ; 2nd, irritability of the entire reflex arc in common with the whole nervous system (neurasthenia, &c.) ; 3rd, irritability of cells secondary to abnormal state of their afferent and efferent elements (injuries to sensory or motor nerves, &c.) ; 4th, direct irritation of cortex cerebri or pyramidal tract (tumour of brain, early stages of lateral sclerosis, &c.) ; and 5th, destruction of cortex or pyramidal tract (cerebral softening, and late stages of lateral sclerosis, &c.). The essential result from all these causes seems to be the same, varying in intensity and distribution according to the severity and extent of the existing irritation or destruction. An important distinction as to the *time* of production between the two last is, that in the former the effects are immediate, and within certain limits are proportionate to the amount of irritation ; in the latter the effects are gradually developed, and depend upon the situation rather than the severity of the irritation.

These, as far as we know them, are the chief phenomena concerned in the production of hypertonicity of muscle. There remains to be enumerated the hypotheses which have been advanced to explain the facts. The more important only of them will be referred to.

The theory of the French school, as especially represented

by Bouchard, Charcot, and Brissaud, maintains that the symptoms of hypertonicity present in spastic paralysis, are due to an irritative process produced in the ganglion cells by the degenerated contiguous fibres of the lateral columns. That this hypothesis is plausible cannot be denied, and it is probable that it is a factor in the production of the clinical symptoms in question. It is, however, as thus expressed, an incomplete explanation, and not the essential cause of the phenomena, as hypertonicity of muscle may exist in its most typical form and in an extreme degree when no degeneration of nerve fibres can be demonstrated, as in various so-called functional complaints, such as hysteria. Moreover, it may occur with such rapidity and under such circumstances as to preclude the existence of gross structural alteration of tissue, as in strychnine poisoning and the hypnotic state.

Another theory, and perhaps the most popular one, assumes that the increased excitability of the cord centres which leads to hypertonicity of muscle is the result of simple severance of the cerebral from the spinal centres, by which the controlling influence of the former is removed from the latter. By some it is supposed that the cortex cerebri actively inhibits the lower ganglia, while others consider that the actions of the last are only as it were "let go" by the removal of a higher passively controlling force. It may be admitted that in the absence of brain influence the reflex excitability of the cord is augmented, and, on the other hand, that volitional efforts in a certain measure modify its manifestations, but whether the above explanations are sufficient to fully account for these facts is open to doubt. If the cerebral centres, by an active process, inhibited, in the strict sense of the term, the centres concerned with muscular tonicity, we should expect to find that when these were irritated, the muscle would relax. Irritation of the vagus directly controls, and if persisted in arrests, the action of the heart; hence it may justly be called an inhibitory nerve. No such effect follows stimulation of the cerebral centres; on the contrary, as has already been stated, during excitement or explosive discharge from the motor cortex, as takes place in those cases of epileptiform convulsion without paralysis, which points to irritability and not de-

struction of nervous tissue, the tonicity of muscle is not diminished, but is actually increased. Experimental stimulation of the optic lobes in frogs controlling reflex acts is probably only another form of peripheral sensory irritation, and does not prove the existence of a special inhibitory centre. That mental effort modifies the effects of muscular tonicity is mainly due to the putting in action of antagonistic muscles. It does not control the organic reflexes, but only those in which voluntary muscles play a part. The "let go" theory, on the other hand, does not account for the fact that in sudden destructive lesions of the brain, or in experimental decapitation, hypertonicity of muscle does not appear suddenly, as might be expected on this hypothesis, but supervenes gradually and after a certain lapse of time; and although the original lesion which causes it may remain in statu quo and even decrease in energy, the muscular tonicity nevertheless continues slowly to increase. This condition, moreover, if developed, relaxes during sleep or chloroform narcosis when cerebral influences are temporarily in abeyance, a fact which is also opposed to this view. Again, if the cord is cut across, the sudden "let go" of energy ought to cause immediate tension of the muscles, which it does not at first (the contrary, in fact, takes place), and this only occurs after a considerable interval, and in a slowly progressive manner. How also on this hypothesis could the fact be explained that when one side of the body has been affected by a lesion on the other side of the brain, generally before long the other side, though to a lesser extent, shows signs of augmented muscular tonicity? These, among other circumstances, would serve to indicate that for the production of hypertonicity of muscle some other agent is necessary than the mere release of physiological action by interruption of controlling influences from above. They would seem to show that in the causation of the phenomena in question some new and morbid active process in the cell elements is necessary in addition to the mere freeing of restrained natural impulses.

A third theory, which more or less modified receives the support of Hughlings-Jackson, Bastian, and others, assumes that when cerebral influence is suppressed the uncontrolled action of the cerebellum (which is believed to be the excitor

of tonic muscular action) becomes predominant, and so produces the exaggeration of muscular tonicity. In health, it is supposed these two centres of innervation counterbalance one another. If the one is enfeebled, the other by retaining its natural vigour causes a excess of action in that direction. The same arguments which can be urged against the last, seem to me to tell equally against this theory. Is there any positive evidence that the cerebellum plays any part in the production of muscular tonicity? Electrical stimulation of its substance certainly is not followed by any visible phenomena. How on this hypothesis can be explained the fact that in complete division of the cord, when all connection with the cerebellum is severed, that hypertonicity of muscle subsequently becomes developed?

Such are the leading theories which have been advanced to explain the phenomenon of muscular hypertonicity, as well as some of the reasons to show that no one of them by itself is adequate to account for its production. While it is not difficult thus to take a negative view of this complicated position, and to criticise existing doctrines, it is not so easy to successfully assume a constructive attitude, and to replace doubtful opinions by others more worthy of acceptance. One of the main impediments to arriving at sound conclusions, is the conflicting and contradictory statements regarding elementary physiological and pathological facts. But when the delicate and complex conditions which surround the nervous system are considered, it is not surprising that the results of experiment and disease should prove so difficult of exact definition. Reasons having been given for not admitting the Inhibition and Cerebellar theories, I am disposed to think that the irritation hypothesis of the French school, with certain modifications, offers the fewest objections to any yet advanced, and best explains all the facts so far as we yet know them. In the production of spastic paralysis, it is assumed by those holding this view that the hyper-excitability of the ganglion cells is caused by the irritation of the contiguous sclerotic fibres of the lateral columns. Now it has been seen that muscular hypertonicity occurs too late after the central lesion to permit of its being explained by the Inhibition and Cerebellar theories;

on the other hand it takes place too soon to be satisfactorily accounted for by this one, namely irritation by secondary sclerotic processes. For, 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 some change (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 symptoms in the shape of muscular hypertonicity is certain. A plausible explanation of this fact would be that vascular 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. We know that cortical destruction subsequently leads to positive degenerative changes in the lateral columns; it is therefore not difficult to believe that long before this is capable of demonstration, molecular or nutritional alterations may be produced by the same cause in the same parts, which if not capable of appreciation by our senses is sufficient to induce functional derangement of the regions with which they are connected. The same effects might supervene upon conditions other than those of actual gross cortical or pyramidal 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 fuuctional 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 hypertonic symptoms. Such excitants would be all the more potent in predisposed subjects whose spinal ganglion cells are already in an abnormally exalted condition, and whose muscles readily react to slight physical injuries. Under the same or similar condtiions it is but a step further to realise that psychical

influences might 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 was in other respects healthy. Again, even normal mental impulses might cause undue motorial demonstrations if the spinal centres were hyper-excitable, as is seen in strychnine poisoning, hysteria, &c. It is therefore obvious that if both these abnormal conditions existed together—that is, increased power of the higher centres to receive and reflect impressions, and augmented capacity of the lower centres to respond to psychical stimuli—the effects would be the more violent in degree and permanent in duration, and serves to explain the severity and intractability of many functional disorders. This would also account for 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 mimicry. It would also offer an explanation of the sudden onset, of the rapid and complete recovery which so often takes place in such cases.

Taking all these possibilities into consideration, I am inclined to think, that the hyper-excitability of the reflex spinal centres which is the fundamental element in the causation of hypertonicity of muscle, from whatever cause it arises, is an active rather than a passive process; that it is due to a new and abnormal irritation, the result of nutritional change, either directly and primarily affecting the cells themselves, or induced in them indirectly and secondarily by irritation of their afferent or efferent elements, or by interruption from the trophic centres with which they are associated. The whole progress of the condition seems to favour this view; its comparatively gradual onset, the fact of its being favoured rather than arrested by cerebral irritation and mental activity, its tendency to extend from one side of the body to the other when originally caused by an unilateral cerebral lesion, its aggravation under such circumstances as produce excitability, and its amelioration under opposite conditions, all point in the same direction. This irritative theory is substantially that of

the French school; but instead of speaking of degenerative processes as the cause, I would replace the term by that of nutritive alterations, an expression of wider scope, and better adapted to account for the varied forms of the disease.

In connection with these reflections it is a question of the greatest practical importance to know whether repeated and permanent functional over-activity is capable of subsequently inducing demonstrable structural changes in the tissues affected. *A priori* reasoning would favour such an hypothesis, although there is not a sufficient series of recorded facts to make its demonstration certain.

To sum up the heads of this complicated enquiry: it is suggested that increased muscular tonicity, from whatever cause arising, is essentially due to abnormal hyper-excitability of the motor ganglion cells of the anterior cornua of the cord. The hyper-excitability may for various reasons be due to idiopathic alterations of 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 irritative nature, and may therefore be considered as a new morbid process. This 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 the activity of psychical as well as of motor centres. These theoretical considerations concerning the pathology of hypertonicity of muscle are, like other hypotheses, open to criticism. I admit them to be somewhat involved, but this is scarcely to be avoided in attempting to account for the complicated circumstances connected with this group of symptoms.

If such doctrinal speculations on the subject we are discussing are uncertain, the practical difficulties met with in actual clinical observation are none the less perplexing. As their features are now well recognised, I have passed over those

forms of muscular hypertonicity associated on the one hand with secondary changes from gross disease of the nerve centres, and on the other with obvious functional disorder. I have limited the practical question to the uncomplicated form of the affection described in the first part of this paper. Now, I would ask, what is the anatomical substratum of this condition? Is it a so-called functional disorder—that is, one unaccompanied with discoverable tissue change—or is it the result of gross organic disease; and how is this to be determined during the life of the patient? Where, and in what manner, does the affection originate? Is it a primary disease of the brain, cord, or nerves? What are its causes, by what criteria are we to be guided in framing a prognosis, and what is the suitable treatment? Although I have seen many such cases and have followed the progress of some of them for years, I confess I am unable to give a satisfactory reply to the above enquiries.

The classical morbid type which this condition most nearly resembles is the primary spastic or spasmodic paralysis of Erb and Charcot, which is asserted to be caused by primary sclerosis of the lateral columns of the cord. There are, however, certain considerations which render it impossible for the two to be considered as identical. The essential elements of spasm and rigidity of muscle in the one are, as we have seen, not necessary features of the other. It is of such frequent occurrence, it so often ends in recovery, and its course and termination are sometimes so rapid, and influenced by such circumstances, as to render its explanation by permanent organic degeneration of the pyramidal tract impossible. That a close resemblance exists between these two conditions cannot be doubted, and it is probable that the essential phenomena of both have the same pathological significance. It may even be asserted that the one is merely an initial form of the other; but this is just the point which yet remains to be proved, and which constitutes the real question at issue. We find, however, that the one may exist independently of the other, and may therefore be said to represent a separate clinical state. Again, it might be maintained that the condition is an incipient stage of some gross form of disease, such as disseminated sclerosis, or the secondary results of primary lesion elsewhere. It can only

be replied that in typical cases no evidence can be traced of any such connection.

Finally, to place such cases under the category of Hysterical or Functional, is simply to shelve the difficulty, and brings us no nearer the true explanation. For convenience of recognition the clinical group of symptoms might be named "*Hypertonic paresis or paralysis.*" It is not pretended that this term is a perfect one, or that it represents a true disease. It professes only to be a convenient symbol expressive of a group of symptoms, frequently met with in practice, the causes of which are probably various, and of the nature of which we are ignorant.

In conclusion, I trust that this communication will serve the purpose for which it was undertaken, namely, by indicating certain obscure problems of practical and scientific interest, to elicit the opinions and experience of the members of the Neurological Society. As a guide to the debate, I beg to suggest the following propositions for special consideration, which provide a wide and varied field for discussion on the subject, from every point of view.

I. With regard to the clinical condition of so-called Hypertonic Paralysis.

1st. Is the existence of the clinical type admitted? If so, what is its anatomical substratum? Where, and in what manner, does the affection originate? Is the primary disturbance in the brain, cord, or nerves? Is it due to gross organic lesion, or the result of so-called functional disorder?

2nd. What are its causes?

3rd. What is its prognosis? By what criteria are we to foretell in individual instances whether the affection will be temporary and comparatively innocuous, or whether it is permanent and incurable?

II. *With regard to the Physiological and Pathological problems in connection with muscular hypertonicity.*

1st. What hypothesis best accounts for hypertonicity of muscle?

2nd. Admitting that visible organic degeneration of the lateral columns is frequently associated with the production of hypertonicity of muscle, is this an essential element for its

production? May a like condition result from undemonstrable nutritive modifications in the same regions or in the psychical or motor centres.

3rd. May a primarily so-called functional disorder, consisting of definite symptoms without appreciable tissue change in the nervous system, eventually lead to demonstrable structural degeneration?