in the muscles of the calves, in which the A.C.C. was obtained more readily than the K.C.C.

Sensibility in the lower limbs was everywhere normal. No pain or subjective sensation was complained of, but there was some pain in the back. The deep reflexes could not be anywhere obtained. There was no affection of the sphincters. A curious symptom was the excessive amount of perspiration, both on the face and limbs. Apart from exertion the sweat would often stand in beads on the surface.

That was her state on admission. Unfortunately the affection had then reached an advanced and chronic state. The inflammatory process in the muscles had attained its cicatricial stage, in which the newly-formed connective tissue, almost universally distributed, had already contracted. We know, from varied experience, in many places, and in many forms, how strong is the contracting tendency of this abnormal structure. In this patient we have striven to overcome the contraction by passive extension and by massage, and have endeavoured to increase the strength of the muscular fibres by movements and electricity as well as by therapeutic agents. I cannot say that much change has been produced in the arms. But if our success in improving the condition of the arms has been small, we have at least the satisfaction of having done good to the legs and back. Their condition is far better than when she came under our care. We have succeeded in overcoming the contraction of the hip and thigh muscles by passive extension, and so have enabled her to use the limbs, with the result that there has been a great increase of their power. The inability to stand has been exchanged for the ability not only to stand, but also to walk. There is still some contraction in the calf muscles, preventing full passive flexion of the ankle joint, but this residual amount will soon yield under perseverance in standing and walking, the most potent of all agents for the purpose. The state of the arms you can observe. In spite of variously contrived efforts to obtain the extension of the elbows, as you see, the contraction of the biceps prevents the extension of the forearm. That of the flexors of the wrist prevents the full extension of this joint, and the abduction of the upper arm is hindered by the contraction of the adductors, and especially of the pectoralis major. Note how the rigidity of this muscle prevents the movement of the arm far from the side. The deltoid, the chief abductor, is rigid and firm, and presents much less response to the faradic stimulus than does the pectoralis. You may observe the same relation between the voluntary power and the degree of shortening in other opposing muscles, in the biceps and triceps, and also in the forearm muscles. She can produce only very slight contraction by the will in the extensors of the wrist and fingers, while there is considerable voluntary power in the flexors. The electrical irritability agrees in the degree of response to the voluntary power. Accordingly, though both sets of muscles are small and hard, those which are least paralysed are most contracted. We may see in this the correspondence of this affection and polyneuritis. There is the same flexor contraction and extensor weakening in both sets of muscles, although all are involved in the cicatricial inflammatory changes. We must therefore infer that there is polyneuritis as well as polymyositis, that both nerves and muscles are involved in the morbid process; but the affection of the muscles is far more extensive than it is in the pure affection of the nerve. How widely and profoundly the former have been involved is shown not only by the degree of hardening and contraction, but by its extensive distribution. The affection of the facial muscles still exists; you may note not only its stiff aspect, but the small amount of movement which results from voluntary or emotional influence, for still more evident is the contraction of the masseters. As you can see, she cannot open her mouth to much more than half the normal extent, and if you presently feel the muscles as thus extended you will note how hard they are and how distinct it is that their contraction is the agency which prevents a greater depression of the jaw.

I do not doubt that her power of walking will improve to a useful degree. Its influence on the contraction of the calf muscles is, as I have said, steady and maintained. It is surprising how much contracture may be thus overcome in the course of time, even a degree that, at first, seems to permit the expectation of release only from tenotomy. But we have

no means to effect the extension of the arms of similar potency. I fear we shall not be able to secure her ability to use effectively what power she possesses in the hands without having recourse to division of the tendons of the biceps. Once developed, such contraction of the flexors of the elbow is a most formidable condition. The extensors act at great disadvantage when the joint is flexed, while the influence of the flexors is then at its maximum, while we have no habitual movement of which to avail ourselves, comparable to that of the weight of the body on the contraction of the calf muscles.

This therapeutic inability brings me to the last point which I desire to impress on you, and like many last words it is the most important. The scope for treatment, at this stage of the affection, is small. If you picture to yourselves the pathological condition, you must conceive the muscles as now containing only a small proportion of normal fibres, and that these are separated by fibrous tissue which has undergone cicatricial contraction. Many muscular fibres have perished in consequence of the acute inflammation and of the degeneration of their nerve fibres. The primary disease had already ceased when she came here, and only its effects remained. You know, in all diseases, what a different task it is to treat the primary affection and to treat its late results when these consist of grave structural damage. So far as recovery of the partially damaged muscles is concerned, these have probably regained as nearly a normal condition of the remaining fibres as they can reach. Some increase of power, however, may result from their voluntary use, if this can be increased by getting rid of the disabling shortening which interferes with all effective employment of the arms. But in this, as in so many diseases, the only opportunity for really effective treatment is that which is presented in the early period of the case, when the malady is in the stage of development. I cannot doubt that, in this patient, rest, diaphoretics, and salicylates, with perhaps small doses of mercury, would then have controlled the inflammation, and lessened very much the subsequent mischief which it has induced. Every degree of diminution of the intensity and duration of the active processes of disease is multiplied many times in the ultimate diminution which is thus caused in the later and permanent condition. I emphasise the fact, because to some of you may come the opportunity of treating such a case in the only stage in which treatment can be really effectual, and because the general lesson is applicable to many maladies of many kinds.

A CLINICAL LECTURE

ON

A CASE OF UNILATERAL (HEMIPLEGIC) SPASM AND CONTRACTURE WITHOUT PARALYSIS.

Delivered in the University of Edinburgh,

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Professor of the Practice of Physic, Physician-in-Ordinary to the Queen for Scotland, and President of the British Medical Association.

(Concluded from page 5.)

THE outstanding features of the case as explained in the part of the lecture already published are:

A soldier, aged 25, had severe typhoid in India; on recovering from coma, found himself unable to use his left hand on account of stiffness of the fingers. Gradually they became flexed, and the arm, foot, leg, and left side of face became rigid with constant hypertonicity and frequent tonic spasms. This condition makes him resemble a case of hemiplegia with descending sclerosis, but there is no paralysis; and all the muscular disability is due to antagonistic action of opposing muscles. Otherwise at present no abnormality of any system or of the nervous system in any respect, except the unilateral spasm. The locomotory system is interfered with only in so far as the spasm, the contracture, and the consequent changes in joints and bones are concerned. He has, since his illness began, had two attacks of mania.

It should be particularly noticed, as to the state of the muscles, that the deep reflexes are exaggerated, and that, although the electric relations differ on the two sides of the body, there is no trace of the reaction of degeneration.

The condition thus described is, so far as my own observation goes, unique, and I have not been able to find an exactly corresponding case recorded in any books to which I have had access. Neither have any of the eminent authorities in neurology, to whom I have shown or described the case, met with one precisely similar.

It is a difficult task to attempt to settle the probable nature of the process, and in considering such a question I like to inquire, first, what is the seat of the disease, and, secondly, what is the nature of the morbid process.

I.—AS TO THE SEAT OF THE MORBID ACTION.

The fault might lie (1) in the muscles themselves, (2) in the lower neuron, either in the nerve fibres supplying the muscles or in the white columns or the cells of the spinal cord, (3) in the upper neuron, either in its fibres or in the motor area of the cortex or possibly in one of the basal ganglia.

1. *Is it in the Muscle?*

A. *Myositis*.—It is scarcely necessary to argue against this suggestion for the unilateral distribution of the symptoms, the examination of the muscles themselves, and the condition under chloroform conclusively exclude it.

B. *Hypertonicity of Muscular Origin*.—No such condition has been recognised, and the history of the case as well as the unilateral distribution lend no support to such an explanation.

2. *Is it in the Lower Neuron?*

A study of the case warrants the exclusion of the lower neuron as a possible seat of disease (a) because there is no paralysis, (b) because there is no trophic change, (c) because there is no reaction of degeneration, (d) because the reflexes, superficial and deep, are exaggerated.

3. *Is it in the Upper Neuron?*

It is thus clear that the lesion must be proper to the upper neuron, and must be referred (a) to the fibres, or (b) to the cellular structures of the grey matter.

A. It cannot be referred to the fibres, for no change in them could suffice to produce such a group of symptoms.

B. It must, therefore, be referred to the cellular structures of the grey matter. There are two areas of grey matter which might be suspected, namely, the Cortex and the Optic Thalamus. It is obvious enough that an irritation of the cortical cells might conceivably give rise to such irritative phenomena of muscle, but there are several difficulties in the way of accepting such an explanation. We would have to assume:

1. That the whole of the right motor cortex is affected with this irritation, all the muscles of the left half of the body being involved.

2. That the irritation has lasted for three and a-half years without undergoing any material change in character or degree.

3. That the irritation has never in any muscle or group of muscles passed, as irritations so often do, into other conditions, such as paralysis.

4. The fact that sudden voluntary action is better performed than slower movement seems to imply that the fault does not lie in the psycho-motor areas or the tracts leading down from them, but rather in some structure affecting the movements in a less direct way.

These considerations seem to me to throw considerable doubt upon, though not to disprove, the view that the cortical cells are at fault. I think that they pretty definitely exclude a diagnosis of organic cortical lesion, although not necessarily a functional affection of that area.

Turning now to the *Optic Thalamus* as a possible source of such irritation, the first point in its favour is that other irritative phenomena, such as Athetosis and Hemichorea, occurring in old Hemiplegias are, to say the least, often associated with lesion of the Thalamus, and that presumably in such cases that lesion accounts for the spasmodic contractions, while lesion of the internal capsule or of the cortex accounts for the paralysis. One must admit that we have as yet no adequate explanation of the way in which these lesions produce the movements, although it seems probable that it is by influencing the cortex through fibres afferent thereto, rather than the periphery directly; but the coincidence of the lesion and the symptoms in so many cases cannot be overlooked. It must also be

frankly admitted that no case is on record in which lesion of the Optic Thalamus produced irritation phenomena precisely corresponding to that of our patient. Secondly, the fact as to sudden movement, just insisted upon, might perhaps be explained on the supposition that a side nucleus such as the Optic Thalamus came into play and modified the motor action.

4. *Is it in the Cerebellum?*

5. It might be suggested that the cerebellum, or possibly some part of the cerebrum other than those mentioned, might be the seat of morbid action, but I have not been able to lay hold of any facts that would justify or even render probable such a suggestion.

Conclusion.

Thus while I should incline to the Optic Thalamus as a probable seat of disease, I should not venture to give it as a positive diagnosis, but fully admit that the symptoms may originate in the Cortex.

II.—THE NATURE OF THE PROCESS.

Turning now to the question of the nature of the process, the first point I like to settle always is

A. THE QUESTION OF MALINGERING.

Such a diagnosis seems to me entirely shut out by the facts

1. That the most careful watching of the patient while under my care has failed to discover absence of spasm at any time.

2. That the patient has during the past three years and a half been from time to time under the observation of a number of different members of the profession, and has always exhibited essentially the same symptoms, and never been seen without spasm unless on one doubtful occasion.

3. That a measure of spasm persists during sleep.

4. That the unilateral distribution affecting all the muscles of one-half of the body was in all probability beyond the knowledge or imagination of such a patient.

5. That actual changes in the joints have occurred and are revealed by the skiagraphs.

6. That he was once seen in a state of profound intoxication, and that the spasmodic contraction remained unchanged.

These considerations satisfy me that he is not malingering.

B.—IS IT FUNCTIONAL?

The second question is whether we are to regard it as functional, toxic, or organic. We must consider first whether it is of that kind of functional disease which may properly be called hysterical.

1. *Is it Hysterical-Functional?*—The fact that he has had two attacks of insanity manifestly implies instability of the nervous system, and his constitution strong and vigorous as it is, seems not incompatible with hysteria. But while these considerations deserve a certain amount of weight they are by no means conclusive, and we ought not to yield to the temptation to call a thing hysterical simply because it presents out-of-the-way nervous features which we find it difficult to explain.

The complete disappearance under chloroform and the partial disappearance during sleep might be regarded by some as strongly favouring such a diagnosis. But I have found that in Spastic Paraplegia and in post-hemiplegic spasm, which are undoubtedly due to organic lesion, that when the patient is brought fully under the influence of chloroform the muscles become lax, and the deep reflexes disappear. I cannot, therefore, accept the chloroform or sleep argument as conclusive.

In regard to the electrical reactions, while it cannot be denied that they are compatible with a diagnosis of hysteria, no one could assert that they are characteristic of it, and for myself I should incline to regard the evidence as negative, unless I had demonstration, which I do not possess at present, of such electrical aberrations occurring in cases of indubitable Hysteria.

Perhaps the strongest point in favour of a diagnosis of Hysteria is the character of the ankle clonus. It is not of the ordinary kind occurring in organic disease, beginning with depression of the toes, but of the rarer variety seen in hysterical conditions, when the first movement

is made by the anterior muscles of the leg. But the symptom is only rarely to be elicited, and is even when present far from being well pronounced; and I think that altogether it does not constitute a strong piece of evidence as to the nature of the case.

Then, against the hysterical hypothesis, there are the very important facts that the patient has remained with one symptom only for three years and a-half, that it took origin during typhoid, and that there never has been the slightest trace of other hysterical symptom, or, indeed, of any nervous symptom in any part of his body except the attacks of insanity, and that these attacks did not in any way modify the motor phenomena which we are studying.

2. *Is it Non-hysterical Functional?*—As an example of such a malady, I quote the case of a patient whom I saw frequently, a boy, aged 15, of a nervous and rheumatic constitution, who had suffered a good deal from Asthma as well as Pneumonia and Acute Rheumatism. After one of his rheumatic attacks he was taken with rather severe chorea, and as the chorea passed off it was noticed that he held his head in a peculiar position, twisted and partly to one side. It was feared by the surgeons in attendance that there might be disease of one of the upper cervical vertebræ. But there was no definite evidence of this. He was carefully treated by constant extension with weight and pulley, but the muscle, on the whole, held its own and resumed the abnormal attitude as soon as set free. The boy never showed a trace of hysterical tendency. At last it was suggested by Professor Ferrier, after the condition had persisted many months, that profound narcosis with chloroform should be induced. As soon as he was fairly under, the spasm relaxed, and the boy on awaking was free from it, and never had the slightest recurrence. That is a typical instance of a non-hysterical, but still functional, disease. If our patient's case is functional I should refer it to this class. But its behaviour under chloroform and other modes of treatment is very different from that seen in the case of the boy just quoted.

C. TOXÆMIA.

It might be suggested that the symptoms are caused by some toxic material acting upon the brain. This suggestion seems to me to have practically nothing in its favour. It is impossible to conceive that a poison should be generated continuously for three and a-half years, that a poison should induce spasm in one-half of the body and in one-half only. The mens medica seems to revolt against such a hypothesis.

D. ORGANIC.

It may be best to review the series of possible organic changes.

1. *Congestion*.—It might be a result of congestion; but a limited congestion of such a kind persisting through so long a period does not correspond to the known facts of medicine.

2. *Inflammation*.—The same argument holds good, with the addition that spasm persisting, as this has done, is not the kind of result that flows therefrom.

3. *Softening*, whether due to a pre-existing inflammation, or to a thrombotic or embolic process, could not remain stationary and active through so long a time, even if it could be supposed that mere softening would induce such symptoms.

4. *Sclerosis*.—If there be organic disease this seems to me the most probable. It is quite conceivable that during the typhoid a degree of thrombosis occurred, and that sclerosis followed in the affected area. Such a change would be necessarily permanent, and might be arrested and remain limited. I fail to discover anything justifying the absolute exclusion of this suggestion, although it could not apply to a lesion of the cortex, and could not be more than suggested as a possible lesion of the Optic Thalamus.

5. *Tumour*.—There can be little reason for suggesting a lesion of this kind in the case; for there are neither eye changes, headache, or other symptoms pointing to a lesion of the kind, and a tumour remaining practically stationary for three and a-half years, producing the same symptoms all the time, is not to be conceived.

Conclusion.—On the whole, then, without in the least desiring to dogmatise on the matter, and sitting quite loose to the hypothesis, I would suggest as the most probable diagnosis either sclerosis following upon thrombosis affecting the right

optic thalamus, or a non-hysterical functional condition of the whole right psycho-motor cortex, or of the grey matter of the right Optic Thalamus.

TREATMENT.

Meanwhile, whatever the true pathology of the case may be, we must do what we can in the way of treatment.

It may be well, although there is no history of syphilis, to give potassium iodide a thorough trial, knowing, as we do, that it sometimes proves of unexpected value in obscure cases.

I cannot but hope that as we possess in chloroform a remedy capable of controlling spasm, while at the same time widely affecting the nervous system, we may one day discover a drug or drugs which shall be able to influence spasm without involving the nervous system in other important particulars. Unfortunately we are not yet acquainted with any remedy of such a kind, but I purpose to try any medicines which promise an approach to a fulfilment of the indication.

Lastly, although I very rarely have recourse to treatment by hypnotic suggestion, I have considered myself justified in trying it in this case. My friend Dr. George Kerr has undertaken to apply the method. I am not able as yet to speak definitely of its results. They certainly are not as marked as they generally are in purely functional cases.

CLINICAL MICROSCOPY AT JOHNS HOPKINS MEDICAL SCHOOL, BALTIMORE, UNITED STATES OF AMERICA.

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THREE years ago, in the second year of the organisation of the medical school, the clinical laboratory was built, in part by a bequest of \$10,000 from friends interested in medical education. It consists of two storeys, each of which has a main laboratory floor 64 feet by 20 feet, with windows on either side, and accommodation in each for 60 students. There are six smaller rooms for the assistants and for private work. One laboratory is exclusively for the third-year, the other for the fourth-year, students. Before proceeding to speak of the classes, I may refer to the organisation of the medical service of the hospital. There are four more or less permanent officers: a first assistant or resident physician, with two juniors, one of whom takes charge of the private ward, the other of the bacteriology of the service; and a physician in charge of the clinical laboratory. The permanency of these officers is of the greatest advantage to the service. Since the opening of the hospital nine years ago I have had only two first assistants, Dr. Lafleur, now of McGill University, Montréal, and Dr. Thayer, who has been with us seven years. The first assistant gets his quarters and \$500 a year from the hospital, and \$500 from the medical school for helping with the teaching. He is also allowed the privilege of outside consultation work. These are appointed by the trustees on the nomination of the Medical Board. There are four house-physicians selected each year from the graduating class. The general microscopical and chemical work of the wards is done in three separate rooms under Ward G, the equipment and current expenses of which are paid for by the hospital. The expenses of the clinical laboratory are met by the medical school.

The work of the third year in medicine consists of (1) an out-patient class from 12 to 1 three times weekly. Adjoining the medical dispensary is a class room accommodating 50 students. I take charge of this class, in which are considered the objective features of disease as illustrated in select cases. (2) On Wednesday, at 12, I hold a set clinic in the amphitheatre, at which both third and fourth-year students attend. (3) Twice a week for two hours Dr. Thayer takes the class in physical diagnosis. (4) On Monday and Friday, from 2 to 4.30 P.M., Dr. Fletcher meets the class in clinical microscopy. It may be well to state here that our classes are as yet small, and as the students enter the school with laboratory training in both biology and chemistry, they are able to take pathology and bacteriology in the second year, so that they have an excellent preparation for the clinical microscopy.

The microscopes and reagents are provided, each student paying a rental of \$5 a year for the microscope.