On the study of muscular atrophy as an aid to the physiological investigation of the spinal cord / by William Allen Sturge.

Contributors

Sturge, William Allen, 1850-1919.
Doran, Alban H. G. 1849-1927
Royal College of Surgeons of England

Publication/Creation

[London] : Printed by J.E. Adlard, [1881]

Persistent URL

https://wellcomecollection.org/works/uh28azuj

Provider

Royal College of Surgeons

License and attribution

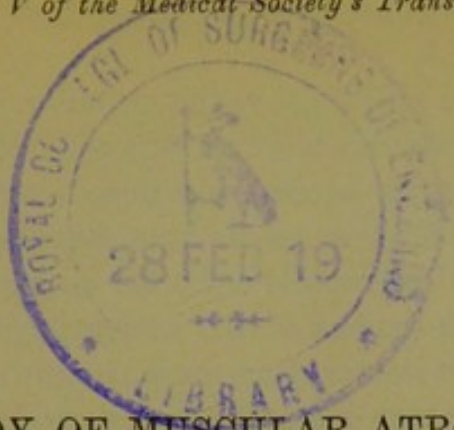
This material has been provided by This material has been provided by The Royal College of Surgeons of England. The original may be consulted at The Royal College of Surgeons of England. where the originals may be consulted. This work has been identified as being free of known restrictions under copyright law, including all related and neighbouring rights and is being made available under the Creative Commons, Public Domain Mark.

You can copy, modify, distribute and perform the work, even for commercial purposes, without asking permission.

wellcome collection

Wellcome Collection
183 Euston Road
London NW1 2BE UK
T +44 (0)20 7611 8722
E library@wellcomecollection.org
https://wellcomecollection.org

Reprinted from Vol. V of the Medical Society's Transactions.]



35

ON THE STUDY OF MUSCULAR ATROPHY AS AN AID TO THE PHYSIOLOGICAL INVESTIGATION OF THE SPINAL CORD.

By William Allen Sturge, M.D.

ASSISTANT PHYSICIAN TO THE ROYAL FREE HOSPITAL.

Notwithstanding the attention which has been paid of late years to the subject of muscular atrophy, and the great advances that have been made in our clinical and pathological knowledge of the various diseases of which it is the most important symptom, there yet remains much to be learned about it, not only as regards the exact nature of the lesions giving rise to some of its forms, but also in respect to some of the functional modifications to which the lesions give rise By a careful study of these functional modifications, we may, I believe, help to clear up certain problems connected with the anatomy and physiology of the spinal cord.

In order to prepare the way for what I shall have to say, I will give a brief account of the condition leading to muscular atrophy. You are doubtless all aware that the nutrition of the muscles is intimately dependent upon the anterior horns of grey matter in the spinal cord, and more particularly that it is to certain large multipolar cells lying in, and forming part of the grey matter of these horns that this special function has been attributed. I am not now going to stay to consider what is the nature of the connection between the large multipolar cells and muscular nutrition; for my present purpose the nature of this connection is immaterial. The point of importance is that all voluntary muscles are connected with the anterior horns of grey matter in the spinal cord, or, in the case of muscles supplied by the cranial nerves with nuclei of grey matter in the medulla oblongata or pons Varolii. So long as the connection between the muscles and the anterior horns or medullary and pons nuclei is uninterrupted; and so long as the

anterior horns or nuclei are healthy, the nutrition of the muscles is carried on properly. Should, however, the connection between the muscles and these centres become interfered with or broken, or should these special centres themselves be diseased, then the muscles waste more or less rapidly. So far as we know at present it is only the particular regions of the nervous system that I have indicated that have this influence on muscular nutrition.

Paralysis of muscles may result from lesions in a great variety of situations. A leg, for instance, may be paralysed by a lesion of a particular portion of the cerebral cortex; by disease of the corpus striatum; by destruction or pressure upon certain parts of the internal capsule; by morbid conditions of the pons Varolii or medulla oblongata; by various diseased states of the cervical or dorsal regions of the spinal cord; by softening, sclerosis, or other lesions of the antero-lateral columns of the lumbar enlargement of the cord; by disease of the anterior horns of grey matter in this lumbar enlargement; and lastly, by disease of the anterior roots of the nerves going to supply the leg, or of the trunks of these same nerves. Of all these various ways in which a leg may become paralysed, it is only when the cause is in the lumbar enlargement, or in the roots or trunks of the nerves, that the nutrition of the muscles is affected; and unless the lesions of the lumbar enlargement involves the anterior horns of grey matter, disease even of this part of the cord, although it may cause paralysis of the leg, will be unattended by change in the nutrition of the muscles.

Muscular atrophy, then, when it results from a nerve lesion is due to a limited number of causes which may be summed up as follows:

I. Disease of the anterior horns, or of their representatives in the medulla or pons Varolii.

1. Acute.
2. Subacute.
3 Chronic..

II. Disease of the anterior roots of the nerves.

III. Disease of the trunks of the nerves.

In addition to these nervous causes for muscular atrophy, there are probably one or more varieties of the disease which are dependent upon a primary modification in the nutrition of the muscles themselves, independent of any nervous influence. The muscle ceases to grow, not because there is a change in the influence exerted over it by the nerve centre, but because there is some change

in the muscular tissue itself, so that, although the nerve stimulus remains the same, it is no longer able to bring about the due maintenance of the muscle. That such cases exist there can be little doubt, since the researches of Friedreich, Debove, and others; but it is little less certain that they are rare and form a small proportion only of the cases of musclar atrophy.

Having thus stated in general terms the causes of muscular atrophy, I pass on to consider these various lesions in detail.

We will begin with acute disease of the anterior horns of the spinal cord. The lesion in this case is usually an acute inflammation. It may occur at any period of life, short of extreme old age; but the fact that it occurs more often in children than in adults has given rise to the well-known name "infantile paralysis." It has now been abundantly shown that the form of paralysis so commonly met with in children, characterised by the peculiarities I shall presently mention, is also met with in adults, and now that attention has been called to this fact, it will, I believe, be found that the disease in adults is by no means so rare as is generally thought. To this disease, whether occurring in infants or adults, have been more recently applied the names "acute anterior polio-myelitis" or acute anterior cornual myelitis, either of which names implies an acute inflammation of the anterior horns.

Now, the well-known characteristics of this disease are a feverish attack accompanied by severe pains in one or more limbs, which, when the disease occurs in an adult, are not unfrequently mistaken for rheumatism. This is shortly followed by paralysis of the corresponding limbs. The patient quickly recovers from the general illness, and the paralysis may pass away from certain of the affected parts, but one or more of the limbs remain permanently affected, and the affection is in this wise. Instead of the limb being affected *in toto*, all parts of the limb being equally involved, as is generally the case in the other forms of paraplegia, the muscles become affected by groups. In every limb there are several such groups; for instance, in the leg there is the group of muscles that extend the knee, the group that flexes the knee, the group that extends the ankle and flexes the toes, the group that turns the foot in, the group that turns the foot out, and so on. Now, in ordinary paraplegia all the muscles of a limb are as a rule affected together and in about equal proportion—the paralysis has the whole limb for its boundary. In paralysis due to acute cornual disease, on the contrary, it is

groups of muscles which are affected instead of a whole limb. It may happen that all the groups of one limb are affected, so that the whole limb is paralysed, but far more often one or two groups are picked out, the others being left untouched; or one group is entirely paralysed, one or two or more groups partially so, and the rest only slightly or not at all. I wish to lay great stress upon the tendency in this disease to paralyse by groups of muscles only, and to contrast it with the tendency of other forms of paralysis to paralyse by limbs.

The next point of importance is this, that the groups of muscles which remain permanently paralysed rapidly waste, the wasting being as marked a symptom as the paralysis. The wasting is accompanied by extreme flaccidity of the muscle, a flaccidity much more marked than it is in any case of ordinary paralysis unaccompanied by wasting. There is a complete loss of muscular tone. A further point is that the wasting and flaccidity are associated with a marked change in the electric irritability of the muscles. The well-known degeneration-reaction becomes developed; that is to say, the muscles lose their power of responding to faradism, whether the current be passed through the nerve supplying the muscles or be applied to the muscles themselves; they lose the power of responding to Voltaic electricity when the current is passed through the nerve, but their irritability to this current when passed through the muscles themselves is increased for a time, although ultimately as the muscles continue to waste even this property is lost.

As regards the other symptoms of this disease there is little to be said. It is generally stated that sensation is unaffected, that in fact the lesion is entirely confined to the motor portions of the grey matter. This, however, is not strictly correct. When we have an opportunity for examining the disease in adults, from whom we can obtain a more satisfactory account of subjective phenomena than we are able to do from children, we find that sensation is almost invariably more or less affected; that in fact, as might be expected, an acute inflammation, occurring in an organ of restricted area like the spinal cord, where parts representing widely different functions are crowded together into a small space, is not strictly confined to one small portion of the cord. Although it is the anterior horns which form the point of attack for the disease, and which receive the brunt of the morbid invasion, yet surrounding districts suffer more or less from the increased vascularisation, and

hence result temporary or permanent modifications in sensation over limited areas. In common with other observers I have noted this in a considerable number of cases, and clinical experience has been borne out by pathological investigation.

The disease has the general characteristic of being unilateral, and in the majority of cases it is one limb only of one side which is involved. It may happen that the limbs of opposite sides are affected, just as it may happen that all the groups of muscles in one limb are affected. This is, however, merely a question of the extent of the lesion. The inflammation may be so widespread and so intense as to involve and to destroy the anterior horns on both sides, and throughout the whole lumbar enlargement, leading to paralysis and wasting of both legs, but, as I have before said, the unit in these cases is a muscle-group, and not a limb, and such extensive disease as I have just mentioned results from an aggregation of such units.

I will now pass on to the chronic form of muscular atrophy depending upon lesion of the anterior horns. This is the disease known as progressive muscular atrophy. There are probably several varieties of this disease. There are certainly, from the clinical point of view, several distinct groups of cases. Some cases are exceedingly chronic from the outset, running a very gradual but slowly progressive course, extending over fifteen or twenty years; others kill the patient in as many months. Some cases, after a comparatively rapid onset, leading to the atrophy of a larger or smaller number of muscles within a few months or a year or two, seem to pull up, and for a series of years the patient remains in a stationary condition, and perhaps is never again troubled by a recurrence of the disease. In some cases the atrophy affects all the limbs more or less, in others one or two limbs only. Some attempt has already been made to show that these various groups of clinical cases depend upon correspondingly various lesions of the anterior horns; that in the very chronic cases we have to deal with a primary fibrosis of the anterior horns leading to slow degeneration of the large multipolar cells; and that in others the lesion of the anterior horns is secondary to a subacute inflammation of the white columns which surround the horns. I have not sufficient pathological material at my disposal to enable me to say anything definite in these difficult questions. I wish merely to call your attention to the existence of these clinical varieties.

Now, just as in the case of acute disease of the anterior horns, the chronic disease tends to affect groups of muscles rather than a whole limb, and this is the place to speak at greater length of this grouping.

It may be said roughly that there are two ways in which muscles are grouped :—anatomically and physiologically. There are two ways in which muscles may be said to be grouped anatomically. (A) According to their relative position in a limb, as, for instance, the muscles in the calf of the leg. (B) According to their nerve-supply, all these muscles being grouped together which are supplied, for instance, by the musculo-spinal nerve. These two varieties of anatomical grouping frequently coincide, because, as a rule, muscles are supplied by the nerve the trunk of which runs in their immediate neighbourhood, and thus all the muscles lying in one particular part of a limb tend to be supplied by the same nerve At the same time, it must not be forgotten that a large nerve may supply several groups of muscles on its way from the proximal end of a limb to its distal extremity, and hence a single group of muscles, from the point of view of nerve supply, may be composed of several groups from the point of view of position in the limb.

The other great method of grouping muscles together, the physiological, is based upon the association of muscles in accomplishing certain functions. This is a much more complex matter than the method of grouping just described, for muscles may be associated together functionally in a great variety of ways; and, on the other hand, certain functions involve a large number of muscles in different parts of a limb. The inquiry into this functional grouping is a very large one and replete with difficulty, but it is one of very great interest and importance. Confining myself, however, to some of the more simple examples of functional grouping, and avoiding, for the present occasion, those which are more complex and difficult to follow, I hope to be able to make clear the points to which I call attention.

I will begin with two of the simplest and most evident motor functions in the body. The one is the action of bending the elbow, the other is that of extending the wrist and fingers. In the former action three muscles take a chief part, the biceps, the brachialis anticus, and the supinator longus. Speaking generally, these three muscles are always employed in the production of this action,

which is one of the most common actions of daily life. On the other hand, these three muscles are employed for little or nothing else, the supinator longus having really very little to do with supination. Now, what is the fact with regard to their anatomical grouping? The biceps and brachialis anticus form a sharply-defined anatomical group, both as regards their position in the arm and as regards their nerve supply, being supplied by the musculo-cutaneous nerve, which, with the exception of the coraco-brachialis, supplies no other muscle. The supinator longus, on the other hand, both from its position and from its nerve supply, is grouped anatomically with the chief supinator muscle of the forearm, the supinator brevis, and with the extensors of the wrist and of the fingers, all of them uniting to form the great muscular mass on the outer side of the elbow-joint and the back of the forearm, and all of them being supplied by the musculo-spiral nerve.

Now, what I have already said of the causes of muscular atrophy generally applies to atrophy of these muscles. They may atrophy from acute disease of the anterior horns in the spinal cord, from chronic disease of these horns, from disease of the anterior roots or of the trunks of the nerves, or they may become atrophied from primary disease in the muscles themselves. It can easily be seen that if the atrophy results from disease or section of the nerve trunks, it will be confined strictly within the limit of the group supplied by the nerve trunk, and will involve all muscles within these limits. For example, if the musculo-cutaneous nerve be involved, the biceps, brachial anticus, and coraco-brachialis muscles will be affected; if, on the other hand, it be the musculo-spiral that is injured or diseased, the atrophy will affect the biceps, the supinator longus, the supinator brevis, and the extensors of the wrist and fingers. In other words, the grouping of the atrophied muscles is anatomical and not functional. Let us now see what happens in the case of atrophy of the same regions from disease of the anterior horns in the spinal cord.

A careful examination of cases of progressive muscular atrophy will, I think, show that the grouping of the atrophied muscles tends to be physiological rather than anatomical; for instance, when the biceps and brachialis anticus are involved it will also be found that the supinator longus is diseased, and probably that the coraco-brachialis is scarcely if at all affected. On the other hand,

if the extensors of the wrist and of the fingers be involved, the supinator longus and biceps will, in the majority of the cases, be free from disease.

In proof of this assertion I show you the following cases. The first is a man, aged 32, who has been suffering from progressive muscular atrophy for ten years, dating from a severe chill which he received at that time. Without going into the details of the history of his disease or of its other manifestations, I will pass on at once to speak of the condition of his arms. The muscles about his shoulder-joints are very slightly if at all involved, but the biceps, the brachialis anticus, and the supinator longus—the three muscles, in fact, associated with flexion of the elbow—have almost entirely disappeared. On the other hand the triceps, the muscles of the forearm, and those of the hand are scarcely affected. The condition is precisely symmetrical on the two sides. This case is a remarkably clear instance of the fact that in disease of the anterior cornua the muscles tend to be affected in physiological rather than in anatomical groups.

The second case is also one of long-standing progressive muscular atrophy, and it is in many ways the complement of the last case. This patient, a man, aged about 45, has atrophy of the muscles of several limbs, but it is to the condition of his left arm that I wish to call attention. The deltoid is almost completely atrophied, as are also most of the other muscles in the neighbourhood of the shoulder-joint; the triceps is altogether atrophied, and so also are all the muscles on the front of the forearm, in the hand, and nearly all of those on the back of the forearm. In striking contrast are the biceps, the brachialis anticus, and the supinator longus—the three muscles associated together in producing flexion of the elbow-joint —these are very little affected, and he retains the power of bending his elbow with only slightly diminished force.

The third case is a typical instance of the ordinary form of lead palsy. Objection may be taken to my placing lead palsy in the same category with the diseases due to lesion of the anterior horns of the spinal cord. It is stated in some books that lead palsy is due to the action of the lead upon the nerves themselves, and in the most common form of this paralysis upon the musculo-spiral nerve. From what I have already said it will be evident that if the lead acts upon the musculo-spiral nerve it would cause paralysis of muscles according to their anatomical rather than according to their

functional grouping, and hence we ought to have the supinator longus, the supinator brevis, the extensors of the wrist, and those of the fingers, all implicated in about the same degree and at about the same time, and the triceps would also become implicated if the lesion of the nerve passed upwards. As a matter of fact, however, the triceps is very rarely much affected in lead palsy, except in those cases which we occasionally meet with, where there is extensive paralysis and wasting of muscles all over the body. It is rare to have much affection of the supinator longus, and the supinator brevis also is generally only slightly affected. Even the other muscles supplied by the musculo-spiral nerve are affected in different degrees, and, what is more, different parts of the same muscle are affected differently, for instance, those portions of the extensor communis digitorum, which go to the ring and little fingers, are liable to be affected more severely than the portion going to the middle finger, and much more so than the extensor muscles of the thumb, or than the extensor indicis. On the other hand, in a certain number of cases the interossei and muscles forming the ball of the thumb are affected in common with the extensors of the wrist and fingers—muscles with which the musculo-spiral nerve has nothing to do, and here again we find different degrees of affection, for in the slighter cases the interossei of the ring and little fingers are involved alone, the other interossei and the short muscles of the thumb being involved only in severe cases. Now the interossei are most intimately connected with the extensor communis digitorum in their action, complete extension of all the phalanges of the fingers being produced by the combined action of the extensor communis digitorum and of the interossei. It is, therefore, very significant to find that it is those parts of the extensor communis digitorum going to the ring and little fingers which are the first to be involved in lead palsy, and that when the paralysis extends to the interossei it is the interossei connected with these two fingers which are most liable to be involved—a fact which I could prove by reference to cases, if the time at my disposal allowed me to do so.

I think I shall have shown to your satisfaction that lead palsy affects muscles according to their physiological rather than according to their anatomical grouping. This points to the existence of a lesion in the spinal cord rather than in the nerve-trunks, and the phenomena of lead palsy give additional support to my proposition, that lesions of the anterior horns of the spinal cord tend to affect

simultaneously muscles that are commonly associated together in action rather than those which are supplied by the same nerve or which are placed together in the same part of a limb.

I have taken these three cases as examples, because they are comparatively simple and more easily analysed than many of the more advanced cases of disease of the anterior horns; but I believe that a careful examination of any such cases with reference to the distribution of the affected muscles would tend to confirm my statement. The more extensive the disease the more difficult does it become to perform an exact analysis of this kind, because to do so requires an accurate knowledge of the various muscular combinations involved in the production of co-ordinated actions. In every case, however, I believe a certain number of points of this kind can be made out, if sufficient care be exercised.

If it be a fact that disease of the anterior horns affects muscles in the manner I have stated, what inference is to be drawn from it? It suggests that to a certain degree muscular action is co-ordinated in the spinal cord. As I said, early in my paper, paralysis in a limb may be due to a lesion of grey matter in at least three different situations. It may result from destruction of a certain portion of the cerebral cortex; from a lesion of the corpus striatum; or from disease of the anterior horns of grey matter in the spinal cord. This triple connection of a limb with grey matter at very different parts of the cerebro-spinal axis suggests that the different portions of grey matter have different functions in connection with the limb. It would be too large a question to go fully into the matter now, but I will throw out the following hints:

1. In the first place certain muscles can, under the influence of the will, be moved independently or nearly independently of all other muscles; for instance, I can flex my forefinger without putting any other muscle into action. Certain other muscles, on the contrary, cannot thus be isolated in their action; for instance, I cannot dissociate the action of the different muscles with which I flex my elbow-joint or of those involved in inspiration.

2. Certain muscular co-ordinations are performed as well in infancy as they are in adult life, whilst others require education before they are properly performed. Some of these acquired co-ordinations come easily and are mastered by every one. Others are acquired with difficulty, and only after elaborate education and long practice.

3. It will, I think, be found that it is just these muscles which it is impossible to dissociate from one another in action that are employed chiefly in actions performed from birth and without education of any kind.

We thus see that anatomically all muscles are connected with several different grey centres, and that physiologically there are several types of movement. Is there not some connection between these two facts? Is it not probable that the higher muscular functions are guided by the centres higher in the cerebro-spinal axis, and that the lower muscular functions depend upon less elaborate centres, the lowest of all muscular actions, viz. those co-ordinations which we possess already when we come into the world being dependent upon the lowest grey centres. These we have seen to be the anterior horns of grey matter in the spinal cord, and we should expect that disease of these horns would affect the muscles in groups depending upon their association for the performance of this type of movement, which I believe, upon clinical evidence, to be the case.

If this be so, we have, I think, an explanation of the use of plexuses of nerves, such, for instance, as the brachial or lumbar plexus.

Let me take the simple co-ordination I have already several times mentioned—flexion of the elbow. To produce this action three muscles combine, the biceps, brachialis anticus, and supinator longus. The spinal centre upon which these three immediately depend for their co-ordination will consist of a certain number of nerve cells, grouped together in the anterior horn of the cervical enlargement. The nerve fibres proceeding from this centre would form a bundle passing out by the nearest anterior root to the corresponding spinal nerve. As, however, the muscles are placed in different positions, it is more convenient that they should receive their nerves through different nerve trunks; and hence the biceps and brachialis anticus which occupy one part of the arm, receive their nerves through one trunk, and the supinator longus, which occupies a different part of the arm, receives its nerve through another trunk. The function of the plexus is to accomplish this arrangement. All the nerve fibres going to these three muscles come out together in one bundle from one nerve centre. In the brachial plexus this bundle is split into two parts; one part joins with other nerve fibres to form the musculo-cutaneous nerve, the other part joins with other nerve fibres to form the musculo-spiral nerve. It is

the aggregate of such rearrangement of nerve-bundles which gives rise to the plexus.

Let me recapitulate the points to which I have been trying to call attention. The grey matter forming the anterior horns of the spinal cord is associated first with the lowest and most automatic of the muscular co-ordinations, and, secondly, with the nutrition of the muscles.

Disease of any particular portion of one of these horns produces corresponding disease of the muscles whose centres for co-ordination are in the diseased portion of the horn. Hence, the distribution of the muscular disease will not correspond to what is ordinarily called the nerve supply of the muscles, nor to their geographical grouping in the limb, but to their physiological grouping in the performance of certain functions.

The nature of the disease will be a combination of paralysis with wasting.

I now pass to another part of my subject, viz. the comparison of the acute with the chronic disease of the anterior horns. In speaking of acute anterior polio-myelitis, I said that it affected one or more groups of muscles in one limb, that all the muscles in a given group were affected equally, and, I might have added, that all the muscular bundles of which a muscle is composed appear to be affected about equally, by which I mean that if any part of the muscle is completely paralysed and wasted, all parts of the muscle are so affected, and if the degree of paralysis is less complete, that all parts of the muscle shared in this partial exemption. If more than one limb is affected there is no special tendency to association of limbs. A right arm and left leg may be affected together, or a left arm and left leg, or both legs. The limbs are affected independently, so to speak. There has been at the outset an extensive cornual inflammation, giving rise, perhaps for a time, to complete paralysis of all the limbs. The inflammation passes off for the most part without leaving permanent general damage behind it; but here and there will be a patch of permanent mischief ending in the production of a scar in the cord. There may be one, or there may be several, such patches, and when there are several they may be near one another or far apart from one another, as the case may be; there will be no tendency to symmetry, in fact, the permanent scars will be quite independent of one another.

In the chronic affection it is quite otherwise. Here there is a very

strong tendency to symmetry. If one arm is affected the other arm has a strong tendency to become affected also, or it would be more accurate to say that both arms begin to be affected at about the same time. Moreover, the symmetry is even more striking than is here expressed, for in not a few cases the same groups of muscles tend to be affected in the two arms. The symmetry in this respect is not always exact, the muscles being sometimes affected in different degrees on the two sides, but in other cases, as in the case to which I have already referred, it is very remarkable. There is yet another point in regard to symmetry, in which the chronic offers a wide contrast to the acute affection; viz. that there may be a slight and symmetrical affection of the upper extremities, and, at the same time, an affection of the lower extremities, often in about the same degree, there being no signs that the spinal cord between the cervical and lumbar enlargements is in any way implicated. There is thus evidence of a simultaneous affection of the cord in four different places—on both sides of the cervical enlargement and on both sides of the lumbar enlargement. I believe that careful examination will often show that homologous sets of muscles in the upper and lower limbs tend to be affected; for instance, that if the flexor muscles of the elbow are wasted, the flexor rather than the extensor muscles of the knee will be wasted, and *vice versâ* as is the case with the patient exhibited. There is nothing very unusual in this symmetry of disease, or in a homology of affection in the upper and lower limbs; it is frequently seen in skin diseases—in psoriasis, for example—but in this respect progressive muscular atrophy contrasts sharply with acute anterior polio-myelitis.

There is another point of distinction between the acute and the chronic disease. In the acute disease, as far as we are able to ascertain, all parts of a muscle are affected about equally; that this is so, is shown by the electric reaction. Even though a muscle be only partially paralysed, it shows a very marked modification to the faradic current, its irritability being reduced in direct proportion to the amount of paralysis present. Now, if the partial paralysis of the muscle were due to complete paralysis of certain fibres, other fibres being left untouched, we should find that, although the muscle was weak and partially wasted, yet there would be marked diminution in the sensitiveness of the muscle to faradism, on account of the comparatively healthy fibres that would still be left. We infer from this that when a muscle is completely paralysed and wasted in acute

disease of the anterior horns, all the large multipolar cells with which the main strands of the muscle are connected are destroyed. When the muscle is partially paralysed and wasted, all the large multipolar cells with which it is connected are diseased without being destroyed. The lesion is in fact a molar rather than a molecular one, attacking the tissue in mass rather than in detail. This is only what we should expect when we consider that we are dealing with an acute inflammation.

Progressive muscular atrophy, on the other hand, differs widely from the acute disease in this respect. Charcot, in his chapter on this affection, says, "the muscle, even when the atrophy has reached an advanced degree, retains its normal reaction to faradism. Diminution or abolition of this reaction shows itself only in the last stages of the disease, when the atrophy has gone to the furthest possible degree." My own experience would not entirely confirm this dictum, for I think it is rare to be unable to detect some slight diminution in reaction in muscles which are only partially atrophied, but this diminution in electric irritability is very much less marked than it would be in a corresponding degree of atrophy and wasting due to the acute disease.

What is the interpretation of this absence of marked electrical change? If from acute disease of the large cells of the anterior horns we get paralysis, wasting, and loss of faradic reaction, why should we get from chronic disease of these same cells paralysis and wasting without much modification of electric reaction? The explanation is that, in the chronic disease, all parts of the muscle are not equally affected; some fibres of the muscle are entirely wasted and paralysed; others scarcely at all so. Those fibres which are wasted and paralysed will have lost their irritability to faradism, but those which are not wasted, or are only slightly so, retain their irritability; and hence, when the muscle is tested *en masse*, it is found to respond normally or only with slightly diminished irritability. This picking out of certain fibres or bundles of fibres points to the conclusion that certain cells have been selected in the anterior horns and destroyed before other cells in their immediate neighbourhood are much if at all affected. The disease is in fact molecular rather than molar, attacking individual cells rather than a whole district of the cord containing numbers of cells.

Another fact which brings into prominence this difference between the acute and the chronic disease is the following:—In the acute

disease the attack always leaves behind it a real and evident paralysis and not merely a weakness of muscle due to the wasting, and hence it has received the name infantile paralysis. In the chronic disease, on the contrary, paralysis does not become a marked feature until a late stage. The patient feels his muscles to be weaker than they used to be, and the physician finds a weakness; but so little does this weakness resemble what is commonly known as paralysis, that the name for the disease has always been derived from the atrophic rather than from the paralytic phenomena—*e.g.* progressive muscular atrophy, Cruveilhier, atrophy, &c. The meaning of this difference obviously is that in the acute disease, where the loss of power and the atrophy are partial only, they are about equally great in all the fibres of the muscle. No fibres act properly, and consequently the action of the whole muscle is modified and a real paralysis results. In the chronic disease, on the other hand, some fibres have completely lost their action, but others retain their power in an almost normal degree, and their comparatively healthy fibres are able to carry on the action of the muscle in an almost normal manner, only, being so reduced in number, they are unable to exercise the degree of power that was possible before the muscle was diseased.

In conclusion, then, the gist of the whole matter is this:—That whereas diseases of other parts of the spinal cord produce results affecting limbs as a whole, disease of the anterior horn of grey matter produces results affecting physiological groups of muscles. Of the disease affecting these anterior horns, the acute forms attack, mutilate, and destroy at one fell swoop, the centre for a whole muscle group; the chronic forms select for their first attack single cells, which they slowly disorganise and destroy. In the acute forms a whole district is taken by storm; in the chronic there is a slow process of detailed destruction.

PRINTED BY J. E. ADLARD, BARTHOLOMEW CLOSE.