Balneological and Climatological Section.

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A Discussion on Fibrositis.

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As you are all aware, it was originally hoped that Sir William Gowers would have been present with us to-night. Unfortunately, a regrettable state of ill-health has deprived us of that added distinction which his presence and support would have conferred on our discussion. It would, I need hardly say, have been singularly appropriate that the subject, chronic rheumatism, should have been introduced by this eminent physician; for it was he who first gave to this morbid condition, if not a local habitation, at any rate the name "fibrositis," the term now generally in use to denote this troublesome affection. Albeit the conception that chronic rheumatism, pathologically speaking, is represented by an inflammatory change in the fibrous tissues, is perhaps one of the oldest in the history of morbid anatomy. Foreshadowed in the first instance by Bichat in his celebrated work on "Anatomy," it fell to the lot of a distinguished Scottish physician, Dr. Craigie, to lay down the dictum that whatever the clinical manifestation of chronic rheumatism, it was in all instances referable to an inflammatory change in the fibrous tissues. But without in any way detracting from the pioneer work achieved by Craigie, Scudamore, Fuller, and others, all will readily admit the pre-eminent part played by Professor Stockman in proving beyond cavil the nature of the anatomical lesions typical of chronic rheumatism. Armed with this pathological fact, we are in a position to unify all the varied clinical manifestations of the affection. For whether it affect the fibrous sheath of a muscle, the fibrous investments of a joint, or the similar coverings of a nerve-trunk, each and all are but variations of one and the same morbid process. Thus

apprehended, we see that the differences which obtain between the various clinical syndromes, lumbago, sciatica and so forth, are due, not to any variations in the nature of the morbid process, but are determined solely by the structure predominantly affected, whether muscle, nerve, or joint. Therefore, having regard to the protean aspects presented by the disease, it would be idle for me to attempt an adequate description of all the clinical varieties. Consequently, I shall confine my attention to one group, muscular rheumatism, or muscular fibrositis, as I prefer to call it.

Firstly, in regard to the vexed question of the nomenclature, I should suggest that the term "myalgia" be abandoned, inasmuch as it predicates the absence of any anatomical lesion, and in addition assumes that the pain in muscular fibrositis is spontaneous, whereas it is always evoked by motion. Again, the term "myositis" is misleading, for the parenchyma of the muscle is not affected, save secondarily, the morbid process being primarily located in the interstitial fibrous tissues of the muscle. The term "myofibrositis," or "muscular fibrositis," seems most appropriate, inasmuch as it indicates clearly the site of the lesion, while it commits us to no definite view as to its pathogeny.

With regard to the ætiology of muscular fibrositis, I would only lay stress upon the great frequency with which its subjects are the victims of ancestral or acquired gout. Indeed, this ætiological factor, in my experience, figures more definitely than any other in the genesis of fibrositis of all kinds. Out of a series of 1,250 cases, no less than 28 per cent. showed definite stigmata in the shape of tophi, or a classical arthritic outbreak in the great toe. In this respect they contrasted very strongly with the records of previous attacks of acute articular rheumatism, as forthcoming in the same series, their incidence only amounting to 8 per cent. This incidence of gout would have been appreciably higher had we included all that motley group of disorders comprised under the gouty diathesis. Other factors assigned a prominent ætiological rôle by many are influenza and sore throat, but in the majority of instances we have to rest on an assumption of auto-toxæmia of varied origin as the responsible *fons et origo mali*. Of exciting causes, the chief are atmospheric changes, gastro-intestinal derangements, and prolonged muscular exertion. Nor should we overlook in this connexion the marked liability to the affection that characterizes certain callings.

With regard to muscular exertion, it is a curious fact that, while inordinate exercise is prejudicial, yet, paradoxical as it may seem, rest of the affected muscle is equally detrimental. On the other hand, moderate

exercise is beneficial, a peculiarity in reaction which we have largely utilized in the therapeutics of the disorder, as shown by the various systematic exercises very generally adopted for its amelioration. The predisposing effect of certain occupations is strikingly exhibited by the fact that out of a series of 1,000 hospital male examples no fewer than 600 were miners, labourers, gardeners, or coachmen. In this connexion, did time permit, many interesting facts could be adduced regarding the marked influence that the special character of the occupation has in determining the localization of the morbid process.

Passing now to discuss the clinical features, time will preclude my doing anything more than referring to the alterations which ensue in the functioning and general reactions of muscles, when attacked by acute or chronic fibrositis. But firstly a few words may be profitably said with regard to the mode of onset of the acute varieties. This, in its classical form, is dramatically sudden, and usually occurs during some slight muscular effort. This abruptness, we believe, is more apparent than real, for the muscular effort impeached is often so trivial that it can hardly be regarded as the cause of the malady, but rather as the occasion of its appearance. In other words, we believe that, despite the startling suddenness of the pain, the responsible morbid process has been slowly, though silently, maturing. This supposition is favoured by the fact that inquiry in many instances elicits the statement that for some days previously the patient had been conscious of stiffness or lack of accustomed suppleness in his affected muscles. That an absolutely healthy muscle is ever suddenly attacked by acute muscular fibrositis is, we believe, highly improbable, the pain being simply an abrupt manifestation of a morbid process which has been developing insidiously in a latent form.

Of constitutional symptoms we need only lay stress, firstly, on the fact that a rise in temperature is far more common than is supposed, even in the milder forms of lumbago, torticollis, and so forth; and secondly, that even in these localized varieties endocardial changes are by no means common.

From the subjective side, the salient and often the sole symptom of acute muscular fibrositis is pain, often of excruciating intensity. Its distressing character lies in the fact that it follows not only volitional contraction but also passive movement, not only of the affected muscles themselves but in addition of those functionally allied to them. Nor does this exhaust the sources of induced pain in the affected muscles, for they are, in addition, exquisitely sensitive to the slightest variation in tension following contraction of their antagonists.

On the objective side, a muscle attacked by acute fibrositis undergoes three main changes:—

(1) An increase in volume as betrayed by swelling.

(2) Increased local reaction as shown by undue warmth of the affected area, presumably due to increased heat production.

(3) An alteration in the tone of the muscle.

With regard to the first two conditions, swelling and heat, these have long been recognized, but we do not think that sufficient stress has been laid on the most important factor, namely, an exaggeration of the tone of the affected muscle. A few words are demanded on this, which we consider to be the fundamental change that occurs in muscles as the result of acute or chronic muscular fibrositis. All skeletal muscles, as we know, are in a state of steady slight contraction, constituting what is known as their normal tone. This tonicity may be augmented by physiological conditions, as during physical effort, but this, of course, is a physiological hypertonus, in other words, an increase of a normal state, due to an increase in the intensity of normal stimuli. Now, from time immemorial physicians have noted that rheumatic muscles as a result of the morbid process become excessively irritable, and in consequence readily pass into a state of peculiar spasmodic contraction. The degree of their contraction, though variable, always exceeds the limits of normal variation; in other words, rheumatic muscles, as a result of the morbid process, are constantly maintained in a condition of excessive contraction or hypertonus. That this is so is easily ascertainable by palpation of any superficially situated muscle the seat of an acute attack and comparing it in this respect with the corresponding unaffected muscle on the opposite side. Let us take, for example, the clavicular bundle of the trapezius, very commonly at fault in rheumatic torticollis. On palpating it transversely to the general trend of its muscular fibres the muscle will be found unduly tense. This unusual tenseness is immediately increased if any pressure be made on the affected muscle owing to induced secondary contraction. The healthy muscle on the opposite side communicates an entirely different sensation, merely a feeling of softness, and, moreover, no contraction ensues in the muscle when pressed upon, as in the hypertonic muscle on the other side.

Now, in regard to the course of the disease, the important point to grasp is, that the objective changes outlast the subjective symptoms. Thus pain, the first symptom to appear, is also the first to disappear, this being closely followed by the decline of local heat and swelling.

But the marked hypertonus of the muscle present during the acute stage, while it diminishes with the decline of the subjective symptoms does not wholly disappear for some considerable period after their cessation, as may be ascertained by palpation. Clinically, this persistent hypertonus finds expression in the fact that long after the pain has passed away the patient is conscious of a feeling of stiffness. Moreover, the abnormal swiftness and certainty with which it reacts to the exciting causes, exposure to damp, draughts, and so forth, plainly indicate that the condition has merely passed into a latent stage.

Passing now to the chronic form, on the subjective side the intense pain of the acute variety is transmuted into a more or less persistent aching and stiffness, which from time to time lights up during periods of exacerbation. Indeed, the clinical aspect presented by the chronic form is that of a series of acute attacks linked together by more or less aching and stiffness. The hypertonus, so marked a feature of the acute form, is present in the chronic, though maintained at a lower level save during relapses. Its intensity is an index to the degree of irritability of the affected muscle. We have already pictured its slow decline during the acute attack and its persistence during the latent period, and we are inclined to find in it the clue to the chronicity of the affection, forming as it does a link between successive exacerbations. But we have another factor in the chronic forms which makes for the maintenance of this tonicity, the development of nodules in the belly of muscle or more commonly in the neighbourhood of its tendinous attachments. Of these two varieties of fibrous overgrowths, those situated in the tendons, to our minds, are the most important. Tendinous structures, as we know, are richly supplied with peripheral sense-organs, the organs of Ruffini and Golgi, which are keenly sensitive to the slightest variations in tension. This has an intimate bearing on the clinical features of the disease. Infinitely more hyperæsthetic than muscle nodules, the slightest touch immediately determines contraction and augmented tension in the affected muscle. Lying, as they do, close to the bone, these nodules are therefore extremely difficult of detection, for if perchance one be palpated it is immediately masked by the contraction of the overlying fibres, which instantly assume a felt-like rigidity. Herein lies their importance, in that they, by their very presence, perpetuate the irritability of the muscle and the resultant hypertonus. For a muscle the seat of such nodules is in an allied condition to one disabled by trauma, in that it displays a marked tendency to pass into a state of continued spasm. This is easily

understood when we have regard to the fact that the pathological end-products are the same in both cases—sclerosis, or an overgrowth of scarlike fibrous tissue. Once established, both muscle and tendon nodules constitute a flaw in the continuity of the muscle, a constant menace to its functional integrity.

These fibrous overgrowths are, we think, the objective expression of the rheumatic predisposition. That vague and nebulous entity, the rheumatic diathesis, as it were, crystallizes itself and becomes incarnate in the shape of these fibrous hyperplasias, which confer upon any individual unfortunate enough to develop them a potential capacity to suffer from rheumatic troubles under the most trivial provocation.

We have before stated the belief that the fundamental change in the muscle is the increase of tone that develops as the result of the morbid process. In regard to this point, Sherrington's brilliant researches have, we think, furnished us with a clue to the manner in which this hypertonic state is probably induced, and a brief digression on this point will, we think, be not out of place. All familiar with his work on the physiology of muscles will recall to mind that his researches prove that the maintenance of tone in skeletal muscles is entirely dependent on the integrity of the reflex arc. Now all the muscular actions in their primitive form may be regarded as examples of reflex action, which, as Sherrington has shown, are associated and regulated by afferent impressions, of which two main groups may be recognized—the proprioceptive and the exteroceptive. Under the former are comprised those secondary afferent impulses arising in the deep tissues—viz., muscles, joints, and ligaments—which, as we have seen, are furnished with special sensorial endings, capable of excitation by any mechanical changes of tension or pressure set up by movement. Now, in health, these afferent impulses are continually passing from the muscles to act upon the related spinal centres, but the bulk of such impressions do not pass the threshold of consciousness, and consequently pass unperceived. Should these impulses for any reason become intensified, we are instantly made aware of their existence. Thus, perchance, if any group of muscles, say the lumbar, becomes attacked by acute fibrositis, we are painfully alive to the slightest variation in their contraction or tension. Moreover, not only is their response exaggerated and painful, but unduly prolonged, persisting long after the exciting cause has ceased to act.

The excessive irritability of rheumatic muscles is, we think, capable of the following explanation: The basal pathological change is an inflammatory process in the interstitial fibrous tissues of muscles, of

toxic or infective origin. In this tissue lie embedded the afferent sense-organs, the muscle-spindles, and the organs of Ruffini and Golgi. Presumably, either through actual invasion or mere contiguity, these sensorial end-organs become hypersensitive, and the impulses transmitted by them correspondingly intensified. Now Sherrington has demonstrated that the normal tone of skeletal muscles is dependent on the integrity of the proprioceptive system, of which these afferent sense-organs are essential components. These sensorial endings in rheumatic muscles being the seat of an irritative lesion, the impulses transmitted by them are exaggerated, and therefore an exaltation of muscle tone, or hypertonus, ensues. We see, therefore, that the augmented excitability of these end-organs accounts not only for the pain but also for the spasmodic contraction of rheumatic muscles. To this condition of hypertonus is to be ascribed not only the distress aroused by volitional effort, but also that following both direct and indirect passive movement of the affected muscles. The same pain accompanies the performance of any complex movement in which they are synergically concerned.

A few words, however, are necessary to explain the reason why painful sensations are aroused in the diseased muscles when their antagonists undergo contraction. As we are all aware the contraction or shortening of one set of muscles involves the relaxation or elongation of its opponents. Now all skeletal muscles being in a state of tone varying their extension, it is obvious that in the absence of any special arrangement, every contracting muscle, by stretching its antagonist, would *ipso facto* raise its tone and thus automatically engender resistance to its own effective action. In short, under such conditions every muscular contraction would be foiled of its endeavour by the resistance of its opponents, and thus the subject would be "muscle-bound.". But, as Sherrington's brilliant researches have shown, the necessary inhibition of the tonus of a voluntary muscle is automatically ensured by the excitation of its antagonist. It appears that normally the elongation of a muscle which follows the shortening of its opponent stimulates mechanically the sensorial end-organs of the muscle undergoing stretching or extension, and thus abolishes its tone. Now the phenomena exhibited in the more acute form of muscular rheumatism seem to indicate that this automatic inhibition of tone is in some way interfered with. Thus all must have noted that in acute lumbago the subject, in the attempt to flex his trunk forward, is brought up sharp, held in a vice as it were, and is for the nonce "muscle-bound."

Now in the normal state, when flexion occurs through contraction of the ventral spinal muscles their opponents, the dorsal extensors, undergo simultaneous elongation, this taking place easily and painlessly owing to the automatic abolition of their tone. Presumably in acute lumbago the flaw in the mechanism would appear to be that, owing to the diseased condition of the lumbar extensor muscles, the usual inhibition of their tonus does not ensue, hence the sudden painful check. We would suggest in explanation that the muscle-spindles, situated as they are in the inflamed fibrous tissues of the dorsal spinal muscles, become exquisitely sensitive. Their response to excitation, therefore, is correspondingly excessive and painful, consequently the tone of the lumbar muscles instead of being inhibited is increased. Now we know that muscle tonus may be abolished by destructive lesions of the proprioceptive or secondary afferent nerves, as is shown by the inco-ordination of movements which follow peripheral neuritis, when it affects the sensory nerves of muscles. This being so, it seems reasonable to expect that irritated lesions involving or influencing these same structures would augment muscular tone.

While the foregoing remarks may serve to explain the disorders of movement that result from the rheumatic process, we have now to account for the development of those objective changes characteristic of the affection. In the acute forms of the malady swelling and heat production are the chief phenomena, while in the chronic these are replaced by the formation of nodules. Now, it cannot be doubted that these are the outcome primarily of circulatory disturbances, which ensue in any muscle the seat of hypertonic contraction. To appreciate this sequence we must recall to mind the fact that the circulation through a muscle varies, according as to whether it undergoes the normal rhythmic contraction and relaxation, or is in a state of tonic spasm. Thus, in the former instance, every muscular contraction, while it has little effect on the muscular arteries, drives blood out of the veins which, during the period of relaxation, rapidly refill from the dilated arteries. As a result the flow of lymph derived from the capillaries is greatly augmented. On the other hand, in a muscle the seat of tonic spasm, while blood pours in through the dilated arteries, the muscle veins throughout the same period are being steadily compressed. Thus it comes to pass that much less blood flows from a tonically contracted muscle than one undergoing rhythmic contraction.

In the light of these facts we arrive at the following conclusions: Rheumatic muscles, as we have seen, both in the acute and chronic stage,

are in a state of tonic contraction, or hypertonus. In the acute variety the hypertonus is of high grade, but of comparatively brief duration. But even so the circulation through the muscle is impeded, and becoming surcharged with blood, an increase in volume or swelling of the muscle takes place. This is, however, temporary, and subsides with the lessening of the hypertonus. The local reaction, as shown by increased heat, is attributable to this same tonic contraction of the affected muscle, for increased tension augments all the processes of muscle, including chemical changes and heat production. Where the pathological state is prolonged, as in chronic forms, the hypertonus, though slighter in degree, is correspondingly persistent. This being so, the rhythmic working of the muscle pump is permanently disabled, and a state of chronic venous stasis ensues in the affected structures. When such a state of venous stagnation occurs in any organ or tissue, sooner or later proliferative changes take place in the involved structures—the so-called congestive induration—with which we are all familiar.

In conclusion, while it may be doubted whether these mechanical results of hypertonus are adequate of themselves to produce the condition of fibrositis, they may reasonably be regarded as strong contributory factors. Such a conception does not necessarily conflict with the infective or toxic theory of the origin of fibrositis. For such disorders of circulation, by depreciating the nutrition of muscle and lowering its resistance, must pave the way for the effective action of microbic or toxic agencies.

RHEUMATIC AFFECTIONS OF THE MYOCARDIUM.

By JAMES MACKENZIE, M.D.

I HAVE been struck with the occurrence of heart complaints of such obscurity that it was impossible to be certain of their nature. Among them were cases where the condition was associated with attacks of muscular rheumatism, and it was reasonable to infer that the heart muscle was affected in a manner similar to that affecting the skeletal muscles. Hitherto the evidences of the nature of heart affections have been too slight to enable one to recognize its nature. The symptoms have been for the most part subjective, such as distressing action of the heart on exertion, or attacks of palpitation coming on at uncertain intervals. Usually the heart-rate is increased, while at other times the pulse becomes abnormally slow, regular, or with intermissions,