THE NATURE OF FIBROSITIS

I. THE MYALGIC LESION AND ITS SECONDARY EFFECTS: A REFLEX THEORY

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Hench and his collaborators (1941a, 1941b) have complained more than once of the dearth of original work upon fibrositis. Nearly all of those who write upon the subject, they say, merely copy the observations of Stockman (1920); and, of those who have attempted to make original pathological studies, the work has been too inadequate and sketchy to be of much value.

Should Demonstrable Pathological Changes be Expected?

To many writers, the term fibrositis bears a degree of reality corresponding to the extent of the demonstrable pathological change in the tissues. If abnormal thickenings and nodules can be demonstrated in the painful areas, the diagnosis of fibrositis is accepted; but the case is suspect if nothing but tender spots can be found. There has been much debate on the subject, the doubters often demanding organic proof before they will believe, and the believers mostly asserting that such proof could be provided were a biopsy but practicable. It has seemed to the present writer, however, that the case has been argued incorrectly from both sides. The doubters are not entitled to demand demonstrable changes in the tissues as a condition of acceptance; and it must follow therefore that the believers should not find it necessary to assert their faith in the reality of such changes. For the idea of pain unaccompanied by organic change is quite familiar to all, and in many recognized painful syndromes, usually called neuralgias, the idea of trying to find structural alterations has long been abandoned.

THE PAINS OF FIBROSITIS

The pains of fibrositis, however, frequently differ from the more discrete painful states in their irregular distribution and in the generally vague manner in which the patient describes them. Instead of being localized to a well-defined spot or region, they are often spread irregularly over a large area; they may not be made worse by movement; there may be no objective signs or only a few tender spots in the deep tissues. The pains may be intractable, and wearing both to the patient and to the physician; it is small wonder that they so

frequently acquire the label "psychogenic." But those who believe in the existence of fibrositis, as a somatic rather than as a psychic disturbance, would do well to remember that there is no evidence to support their opponents in their demand for objective changes. The "myalgic spots," which have been demonstrated in the majority of cases by a large number of observers, are in themselves enough to explain all the symptoms.

ASCERTAINED PATHOLOGICAL CHANGES

Stockman quotes Balfour, in 1816, as the first to describe definite thickenings in chronic rheumatism; since then their existence has been confirmed many times. Stockman described swelling or necrosis of collagen fibrils, associated with collections of round cells and surrounded by an oedematous or fibrinous reaction. In the more chronic cases fibrosis of the tissues supervened. Collins (1940) removed some subcutaneous thickenings and found only normal fatty tissue with some fibrous encapsulation; he examined Stockman's material and was convinced that many of the sections showed little departure from the normal. Abel, Siebert, and Earp (1939) reported low-grade inflammatory changes with early degeneration of muscle-fibres; in their chronic cases fibrosis was observed, with degenerative changes more advanced. In the cases reported by Jordan (1941) the painful and indurated tissues did not appear abnormal upon microscopy. More recently Copeman and Ackerman (1944) explored the backs of a number of sufferers from lumbago, and found the main lesions to consist of fatty tissue with proliferation of fibrous tissue and some vascular congestion. Travell, Rinzler, and Herman (1942) appeared to strike a true note when they summed up as follows: "It is possible that the fibroblastic proliferation which has been observed is secondary to a functional disturbance and occurs only if the latter persists for a period of time."

Hench and his collaborators have suggested that a useful beginning could be made by an intensive study of the fibrous and muscular tissues at all ages; this would indicate with certainty what are the normal results of ageing and of prolonged use. Until this is done, they say, we have no data upon which to base our ideas of the "abnormalities"

found in fibrositis. In the opinion of the writer even the most comprehensive study would prove inconclusive, and in a proportion of undoubtedly painful areas no histological change would be observed. The changes associated with rheumatic disease are closely related to the changes inherent in normal degeneration, and there always will be some cases in which the observable changes are so early or so mild that they are not easily differentiated from the normal.

The Myalgic Spot and its Effects

The myalgic spot is a small area of tenderness; but it sometimes give rise to a larger area of secondary tenderness of a less acute nature. Emphasis was laid upon this distinction by Kellgren; and in the experience of the writer it is an important distinction. The myalgic lesion is recognized by its more acute tenderness, and by its affinity for certain well-defined "sites of election." The smaller lesion is the cause of the diffuse radiating pain, sometimes accompanied by patches of secondary tenderness. Upon the injection of procaine into the lesion, both the radiating pain and the secondary tenderness will be observed to disappear forthwith. This result is diagnostic; if it is not obtained, the injection has not been correctly placed. The phenomenon suggests a few questions relating to the nature of the secondary tenderness.

ARTIFICIAL MYALGIC LESIONS AND SECONDARY DEEP TENDERNESS

Experimentally produced deep somatic lesions cause not only radiating pain, but "referred" tenderness of the deeper structures in the painful area (Lewis, 1938; Inman and Saunders, 1944; Granado, 1945). All are agreed that the tenderness appears at the same time as the pain, and abates when the pain abates. It must follow that the tenderness is a reflex phenomenon; the time-relations can be explained upon no other basis. When the primary lesion ceases to give off painful impulses, the surrounding deep tissues cease to be tender to pressure. This deep hyperalgesia, therefore, must be the result of reflex nervous impulses, acting in a fashion analogous to the reflex production of cutaneous hyperalgesia (Lewis, 1937, 1942). Lewis demonstrated that cutaneous hyperalgesia could be produced by the anti-dromic stimulation of sensory nerves; its spread in the skin from a point of injury he showed to be brought about by the medium of axon-reflexes. In the efferent component of an axon-reflex, the impulses run antidromically in the nerve-fibre and release a "pain factor" at the nerve-endings.

Arguing from analogy, it seems reasonable to regard the appearance of deep hyperalgesia as the result of a similar mechanism in the deep tissues. The myalgic spot behaves like the artificial lesion, sending off a succession of abnormal impulses which cause reflex effects in the deep tissues. A small amount of procaine solution, correctly placed, cuts off the stream of impulses and causes the imme-

diate abatement of the secondary tenderness. Deep hyperalgesia, which is the result of a neuro-chemical change, should not of necessity be accompanied by recognizable structural alterations in the tissues. It does not seem valid, therefore, to demand structural change as proof that the tissues are tender to pressure.

"SITES OF ELECTION" FOR MYALGIC LESIONS

The causes of the myalgic lesion are numerous, though the mechanism by which it is produced is unknown. It may result from trauma (Good, 1942), from established rheumatic disease (Good, 1941), or it may arise *de novo*. Copeman (1943) noticed that tender spots often appeared in the deep tissues after such febrile diseases as influenza; they remained latent, however, until their activity was aroused by further damage in the shape of an injury, a chill or another febrile illness. That these lesions must have some anatomical significance is suggested by their predilection for certain localities. A myalgic spot may occur in muscular tissue anywhere, but the available evidence suggests that they have a strong affinity for musculo-tendinous junctions. The lesions are found most commonly in the erector spinae in the lumbar region, where muscular and tendinous tissues are so interspersed that it is difficult or impossible to tell by palpation exactly the kind of tissue which is involved. The same remark applies to the intercostal muscles. When lesions occur in the larger muscles of the limbs, however, they are found most commonly at a short distance from the attachment of the muscle, where fleshy fibres and tendon should meet. Thus the commonest site for myalgic spots in the buttock is the gluteus maximus near its attachment to the posterior superior spine or the iliac crest (Kellgren, 1938a, b; Steindler, 1940; etc.).

Pain in the forearm and hand can frequently be attributed to lesions in the extensor group of muscles just below the external epicondyle (Good, 1940; Kelly, 1944b), while lesions in the deltoid muscle nearly always occur near its upper or lower attachment. Pain referred to the shoulder, however, is more often caused by lesions in the region of the supraspinous and infraspinous fossae, in which the trapezius and the spinati are most commonly involved (Kelly, 1942; Travell, Rinzler, and Herman).

When pain in the leg is due to myalgic spots, these often can be found in the region of the knee on the inner side, either just above the internal condyle of the femur, or immediately behind the internal condyle of the tibia (Good, 1942; Kelly, 1944a). In most cases of painful stiffness of the knee of myalgic origin, the injection of procaine into such lesions will completely remove the disability for a few hours, and in an appreciable number the relief is lasting.

After observing many hundreds of cases of fibrositis, the writer has become convinced that these "sites of election" have some significance in the pathology of fibrositis; so frequently do they become involved that it appears as though the

distribution of deep pain is governed by laws hitherto unknown. It can be shown further that the "sites of election" are tender to pressure in the normal subject. Muscles as a rule display greater sensitivity near their musculo-tendinous junctions than over their bellies; anyone can prove the truth of this statement within a very few minutes. Thus it can easily be demonstrated that the following "sites of election" (which are responsible for about 50% of cases of myalgic pain in the head and limbs) are more tender to pressure than the surrounding muscular tissues: (1) neck muscles just below superior nuchal line; (2) trapezius muscle half-way between acromion and neck; (3) extensor muscles in front of the neck of the radius; (4) gluteus maximus in situation described earlier; (5) deep tissues just above internal femoral condyle.

Case 1 is typical of many in which widely distributed tenderness disappeared upon treatment of a myalgic lesion.

FIG. 1.—Case 1. *a*—Lesion in exterior muscles. *b*—Tenderness related to *Brachialis anterior*. *c*—Tenderness in front of wrist.

J. J. B. suffered from pain in the right shoulder for five months, brought on by exposure to cold in Central Australia. After two months the pain began to radiate to the hand; it seemed to spread from the middle of the humerus to the wrist, with numbness in the thumb and tingling in the index and ring-fingers. No alteration of cutaneous sensation was detected, but three main areas of deep tenderness were found: (1) an oval area, 3" long, above the external epicondyle in relation to the brachialis anterior muscle; (2) a circular area, 2" in diameter, in the extensor muscles 3" below the epicondyle; (3) a circular area, 3" in diameter, on

the front of the wrist. Upon the injection of a few c.c.s. of procaine solution into area (2), the tenderness of the other two areas disappeared as though by magic. Though he had suffered considerably for five months, after the injection he remained completely free from pain. (See Fig. 1.)

Pressure—Pain

Whatever value attaches to such shadowy observations as these rests upon the recognition of painful pressure, and especially of the intensification of painful pressure, or deep hyperalgesia. Therefore it becomes necessary at this stage to review the present knowledge of the anatomy and physiology of pressure-pain. It can be demonstrated that deep tenderness consists of an intensification of the well-known pressure-discomfort, which normally increases to pain when pressure rises to the required intensity. A certain degree of pressure with the thumb on subcutaneous periosteum, joint-capsule, or tendon will cause a diffuse pain which is not related to any cutaneous sensation, for it can be produced by applying the same pressure through anaesthetized skin. If the pressure is made over a muscle-belly, the pain is more intense and radiates more widely. Certain spots in the muscle, as already described, are more acutely tender. It is possible, by the use of selective local anaesthesia, to demonstrate that the receptors subserving the sensation of pressure-pain reside in the perimysium in the case of muscles, in the peritenal tissues in the case of tendon, in the periosteum in the case of bone, and so on. That the skin and subcutaneous tissues play no part can be demonstrated with ease; thus the pressure required to elicit pain remains the same whether the skin be anaesthetized or no. Where the skin is loose, large folds of skin and subcutaneous tissues can be picked up between finger and thumb and firmly squeezed. If the skin is not sharply deformed by pinching, the greatest possible pressure can be exerted without pain. If muscle is included, however, the well-known sickening sensation of pain is brought on by comparatively light pressure, while the pressure required to elicit pain from tendon is somewhat greater.

PREVIOUS RESEARCH ON PAINFUL PRESSURE

Pressure-pain, therefore, is purely a function of the deep tissues. It now remains to localize it more accurately. Judging from the results of recent workers, we should normally expect to find that the pain is elicited from the tissues covering them rather than from the substance of the muscles and tendons. Lewis (1938) and Kellgren (1939) found that pain was not normally elicited from muscle-substance by puncture with a needle, whereas puncture of the deep fascia covering the muscle usually gave rise to the familiar sensation of deep pain. In similar experiments upon tendon, Weddell and Harpman (1940) found that pain was elicited on puncturing the peritenal tissues but not by transfixing the tendon itself. An exception to this was

found in the region of musculo-tendinous junctions, where pain was elicited from the tissue substance; this may be of significance in view of the previously mentioned high sensitivity of such regions to pressure. Weddell and Harpman were able to demonstrate in the peritenon a fine plexus of nerve-fibres, similar to that described in the skin and other organs by Woollard, Weddell, and Harpman (1940). This was regarded as the pain-plexus; it was found in all tissues which gave pain upon pure mechanical stimuli, such as puncture with a fine needle; it could not be demonstrated in muscular tissue, though it was found in the intermuscular fibrous layers and in the perimysium. Thus there has been a happy correspondence between the labours of a number of observers, approaching the subject from different aspects. The present writer, however, could find only a few references to research upon pressure-pain, and these were directed towards its pathways of conduction rather than the tissues in which its receptors are located (Head and Sherren, 1905; Stopford, 1923; Duthie, 1926).

Experimental Observations

The following experiment was performed a number of times upon himself by the writer. A fine hypodermic needle was passed down to the surface of the muscle (or tendon). The familiar sickening pain indicated that the objective had been reached; the needle then was withdrawn $\frac{1}{4}$ " and 2 c.c.s. of local anaesthetic solution rapidly injected in contact with the surface of the muscle. The needle was withdrawn immediately, and pressure was made at once upon the anaesthetized spot. In every case an area of muscle about 2" in diameter was found to be completely painless to any pressure which could be applied with the thumb; similarly, when a muscle was pinched between finger and thumb. A small area of deep fascia was anaesthetized on each side of the biceps brachii, or the lower fibres of the gastrocnemius; so long as the tips of finger and thumb were not moved from the anaesthetized deep fascia, no pain was felt. The same results were obtained by compressing the tendo calcaneus after both sides had been anaesthetized.

In every case the skin was normally sensitive, and in every case the pressure was applied so soon after the injection of the procaine that the anaesthetic could not have diffused into the muscle or the tendon. A further experiment was performed upon one of those especially tender spots which are found in the region of the musculo-tendinous junctions. The spot selected was a well-defined one in the inner head of the left gastrocnemius, behind the internal tibial condyle. When the needle reached the sensitive muscle-covering, a peculiarly unpleasant pain was felt, accompanied by twitching of muscle fasciculi. The injection of procaine caused the abolition of pressure-pain; this region was quite as "dead" to pressure as any other area of muscle similarly treated.

DEEP HYPERALGESIA

Though these results do but corroborate the observations of earlier workers, they are conclusive as relating to the sense of painful pressure which can be elicited normally from muscle and tendon. It remains now to decide the nature and location of deep tenderness; is this merely an intensification, of normal pressure-pain, residing in the perimysium, or is it more generalized, with normally insensitive muscle-tissue becoming sensitive to pressure? The following experience suggests that it consists of an accentuation of the physiological sensation.

A severe and painful reaction followed the anaesthetization of the sensitive spot in the head of the gastrocnemius. The site of the injection was painful, the pain radiating down to the ankle and halfway up the thigh. The calf muscles were tender over their whole extent, though the site of the lesion was excessively so. No alteration of cutaneous sensation could be detected. A spot was selected, in the centre of the calf, into which 2 c.c.s. of procaine solution were injected just superficial to the surface of the muscle. Within 5 seconds an area of tissue 3" in diameter was totally insensitive to pressure with the thumb, though no change had taken place in cutaneous sensation (see Fig. 2).

FIG. 2.—*a*—Artificial lesion in inner head of gastrocnemius. Oblique hatching=area of secondary tenderness. *b*—Area in which tenderness was abolished by procaine.

Many similar observations have been made upon the secondary tenderness in patients with fibrositis; in the great majority a small area of tenderness can

be abolished by injecting 1-2 c.c.s. of procaine solution in contact with the surface of the muscle. Pathologically intensified pressure-pain, therefore, consists of increased sensitivity of the fascia overlying the muscle, rather than of abnormal pressure-sensitivity of the muscle itself. This layer contains a plexus of nerve-fibres, with free nerve-endings which are generally acknowledged to be the peripheral receptors of deep pain sensation.

The reflex hypothesis of "referred" deep hyperalgesia is thus supported upon both anatomical and physiological grounds. The picture is singularly complete, and the converging evidence from a number of separate sources cannot be disregarded.

Fibrositis and the Deep Pain System

If the foregoing remarks are true, the question of the nature of fibrositis must resolve itself largely into an inquiry into the anatomy and physiology of the deep pain system. Nor is this surprising, for the majority of patients with fibrositis have pain and nothing else. Few objective signs are available, and often the only available evidence presented is the assertion of the patient that he feels the pain. Therefore it would seem a more rational procedure to assemble all the available facts regarding pain, than to cling to outworn and unproven conceptions of the nature of fibrositis. There is in fact very little evidence in favour of the generally accepted hypothesis which relates fibrositis to disorders of muscular metabolism. According to this theory, the resultant circulatory disorders lead to lymphatic blockage and to consequent indurations, which cause pain by pressure on nerve-fibres. It was well pointed out by Buckley (1940) that this hypothesis corresponds ill with our present knowledge of sensory function. A critical examination of present views upon fibrositis, says Buckley, is long overdue. The deep pain system, according to Lewis and Kellgren (1939), is anatomically and functionally distinct from the cutaneous pain system. Reflex effects may spread widely in the deep system without affecting the superficial component; and, in those cases in which hyperalgesia of the skin accompanies deep tenderness, there is between the two no correspondence, either in distribution or in time-relationships.

An appreciation of the reality and the importance of the deep pain system will help to throw a good deal of light upon many of the problems of somatic pain. The theory of the reflex spread of hyperalgesia was foreshadowed more than 40 years ago by Gowers (1904), who said that the pain of fibrositis was due to "induced excessive sensibility." In opposition to the reflex hypothesis, it may be argued that, though a nervous mechanism may be sufficient to explain such sensory effects as hyperalgesia, it will not adequately account for the physical changes which may be encountered. The fibroblastic hyperplasia, it may be said, with its surrounding tissue reaction and its later fibrosis, demands more tangible causes of a physical or a chemical nature; it is hard at first glance to believe that palpable

indurations in the tissues can result from the effects of disordered nerve-impulses. It should be remembered, however, that very little is known of the relations between the somatic tissues and their nerve-supply, for the reason that it occurs to few people even to think of the possibility of such a relationship. The bulk of the evidence, however, indicates that nervous influences may be responsible for profound tissue changes, both indirectly through alterations in the circulation and directly through effects upon the vital functions of the cells. That acute and careful observer, Sir James Mackenzie (1920, 1926), regarded as the most important of his works the discovery of the part played in the production of symptoms by nervous reflexes. But this, the fruit of the labour of his last few years, went unnoticed by the profession and passed into an oblivion which cannot but be temporary.

Trophic Nervous Effects

When a sensory nerve is cut, the tissues which it had supplied, after displaying swelling for a few days (Sharpey-Schafer, 1927), become atrophied. When a motor nerve is cut, the corresponding motor fibres atrophy. In like manner, denervated fatty and fibrous tissues undergo retrogressive alterations in structure. Oedema is a recognized effect of interstitial neuritis (Jones and Lovett, 1923; etc.); and the general acceptance of the view that pathological nervous impulses may be responsible for tissue changes is shown by the common use of the term "neuro-pathic oedema." That trophic effects are not wholly due to circulatory changes is shown by the fact that they are not seen after sympathectomy. From this point of view, what appears to be a more important circulatory effect is the vasodilatation which results from the antidromic stimulation of sensory nerves.

ANTIDROMIC NERVOUS IMPULSES AND AXON REFLEXES

It appears to be no coincidence that both hyperaemia and hyperalgesia of the skin result from impulses propagated antidromically through sensory nerves (Lewis, 1937). In like manner, the hyperalgesia which surrounds a small crushing injury is accompanied, in suitable subjects, by whealing and hyperaemia, both, according to Lewis, the effects of axon reflexes. The fibres which produce hyperaemia appear to be identical with those which produce hyperalgesia; and in both cases they belong to the posterior root system. Lewis thinks it possible that a hitherto unrecognized system of fibres is involved, but Woollard, Weddell, and Harpman pointed out that the pain nerves possessed all the qualities necessary to produce the observed effects. The weight of evidence, in fact, seems to indicate that the pain fibres are responsible (Walshe, 1942).

An analogy was drawn earlier between the superficial and the deep pain fibres; and the fact that cutaneous hyperalgesia is known to spread by nervous reflexes was used to support the proposal

that deep hyperalgesia spreads in an analogous fashion. In like manner, the knowledge that the pain nervous system mediates the spread of hyperaemia in the skin lends great support to the supposition that the deep pain system could be responsible for the propagation of vasodilatation in the deeper tissues. Here again the picture is singularly complete, and the converging evidence once more focuses upon the important point—the implication of the pain nervous system in the production of oedema and tenderness in the deep tissues.

BI-DIRECTIONAL FUNCTIONS OF SENSORY NERVES

The concept of spread by axon-reflexes involves an idea which is not generally familiar, but which is now well established—the bi-directional propagation of nerve-impulses. The likelihood of bi-directional function in sensory fibres was mentioned by many earlier writers, including Weir Mitchell (1872), Charcot (1877) and Bayliss (1900). The hyperaemia which had been observed to follow antidromic propagation of nerve-impulses was thought by many to be merely a freak effect, with no physiological counterpart. Barron and Matthews (1935*a*, *b*), however, have shown that the branching of sensory axons is much more profuse than had been thought. In the cat and the frog the proximally-directed axon from the root ganglion cell gives off collaterals in the cord, which leave by adjacent posterior roots. In all branches of both the proximal and the distal axons, impulses may run in either direction, so that physiological sensory impulses from one region will give rise to impulses antidromically to sense-organs in another region. The simple mechanism of an axon-reflex, therefore, may be sufficient to explain the reference of pain to regions at a medium distance, but, where a leap of several segments is made, one or more relays must take place in the cord.

ANALOGY PROVIDED BY SOME PAINFUL POST-TRAUMATIC STATES

An interesting example of a similar mechanism is provided by those painful atrophic conditions which sometimes follow injury to an extremity. Sudeck's atrophy, post-traumatic painful osteoporosis (Miller and de Takats, 1942), minor causalgia (Homans, 1943), post-traumatic spreading neuralgia (Leriche, 1939) and post-traumatic pain syndromes (Livingston, 1943) are terms which have been used by various writers to describe a process which is essentially the same in all instances. Several groups of workers in America have brought forward a good deal of evidence to suggest that the pain, dysaesthesiae, oedema and generalized tissue atrophy are reflex effects, the pathological impulses originating in the damaged tissues. "It seems probable," says Livingston, "that the injury to certain tissues starts a cumulative process which is not confined to a single nerve-distribution, and which tends to spread to involve the spinal cord in diffuse reflex phenomena."

Physiology of Musculo-Tendinous Junction

If the hypothesis is accepted that most of the manifestations of fibrositis represent only secondary or reflex effects of a myalgic lesion, it must follow that the functional pathology of the lesion differs fundamentally from that of the bulk of the affected tissues. The lesion may arise anywhere in muscular tissues (and perhaps in fibrous and synovial tissues), but it manifests a predilection for some well-defined situations near the junctions of certain muscles with their tendons. Perhaps some significance can be attached to the observation of Weddell and Harpman that pain could be elicited from the substance of the musculo-tendinous junctions, though not from pure tendinous tissue. It can be no mere coincidence, either, that the special neuro-tendinous receptors, or tendon spindles, are found in no other situation than at the musculo-tendinous junctions (Maximow and Bloom, 1934). In each spindle Weddell and Harpman demonstrated in addition to its special sensory nerve-supply an "accessory" nerve-fibre which divides to form in the capsule of the spindle a plexus of unmyelinated fibres, with free nerve-endings. This plexus is indistinguishable from the pain plexus, and the authors say that the naked receptors are the source of the pain which is felt when the tendon receptor is over-stimulated by stretching the muscle.

In the region of the musculo-tendinous junctions, too, are found the majority of the muscle-spindles. In view of the large concentration of sensory organs in its vicinity, there is little reason for wonder that the junction is normally more tender to pressure than the adjacent muscle fibres; nor is it surprising that painful myalgic lesions are found commonly in its neighbourhood. Considering, too, that the muscle spindles and the tendon spindles are the receptor organs for muscular stretch reflexes, it is not surprising that muscular spasm and stiffness usually result from myalgic lesions.

Many interesting questions here suggest themselves, relating to the formation of myalgic lesions in regions apart from the musculo-tendinous junctions; to the formation of multiple lesions simultaneously or in succession; to the causation of the myalgic lesion itself; and to the radiation of the pain in well-recognized patterns. Some speculations upon these points will be reserved for a future article.

Summary

1. Recent work upon the pathology of fibrositis has added little to the principles laid down by Stockman. Frequently the painful tissues do not present any recognizable histological change.

2. The work of Lewis and his associates upon artificial deep lesions indicates that widespread pain and secondary tenderness may radiate from a localized lesion through the medium of nervous reflexes.

3. The essence of the fibrositic syndrome consists of a localized myalgic lesion together with its more widespread reflex effects.

4. The myalgic lesion shows a predilection for certain localities, mostly in the region of musculo-tendinous junctions, where concentrations of sensory organs are known to reside.

5. Present knowledge of the deep pain system is reviewed, together with experimental observations intended to locate the site of "pressure-pain" receptors.

6. The reflex theory of fibrositis is shown to be consistent with present knowledge of deep sensory function, in which axon-reflexes and antidromic impulses play a large part.

The Director-General of Medical Services, Australian Military Forces, has kindly permitted the publication of this essay.

Addendum.—Since this article was set into type I have had the opportunity of reading in full the excellent contribution by Copeman and Ackerman. The correlation of the "fat pattern" of the back offers a fascinating field for study. Also, perusal of further work by Barron (*J. Neurophysiol.*, 1940, 3, 403) shows that the original findings of Barron and Matthews are not confirmed in all particulars. It now seems unlikely that collaterals leave the cord by adjacent roots; but the reflex spread of sensory impulses, through profuse branchings, has been amply confirmed.

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