Electromyographic Studies of Structural Abnormalities*

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The osteopathic lesion, since its discovery by Dr. A. T. Still, has been thought to be characterized by muscle contraction or muscle tension.

The experiments that we will report have been done in an attempt to secure objective findings concerning the osteopathic lesion. Because electromyography makes possible a determination of the presence and character of muscle activity, this field was selected for a beginning study.

In 1903 the development of the string galvanometer by Einthoven made possible studies in electrocardiography, in electromyography, and in electroencephalography. The discovery of the vacuum tube brought about amplification with which exceedingly small currents (fractions of one-millionth of a volt) could be recorded.

Electromyography is defined as a method of recording somatic electric currents induced by muscular action. Carlson and Johnson describe this activity as follows: "During activity a muscle fiber becomes a minute battery, with positive and negative poles, the latter at the active region, the former at any inactive region. The electrical potentials (called action potentials) developed are physically identical with the potentials developed in any battery. If the poles of such a living battery are connected by a conductor, a current (called an action current) flows. The current can be led through a suitable recording instrument which indicates the passage of the current and measures its magnitude."

Adrian and Bronk demonstrated muscle action potentials from single motor units. A motor unit is described as being one motor neuron together with 100 to 200 muscle fibers which it supplies. Each muscle is composed of thousands of these motor units. When an impulse is passed over the motor neuron, all of the muscle fibers, which it supplies, contract practically simultaneously at a rate, usually, of 5 to 7 times a second. However, frequencies as low as 4 and as high as 40 have been seen.

Muscle contraction is a result of voluntary effort, of reflex and of artificial stimulation. At this point, the question of the presence of residual muscle contraction in a normal resting muscle is of importance. Creed et al. state: "In the unstrained muscle no centrifugal impulses are set up by the tension receptors. . . . If at first the muscle is at rest there will be no action currents. On passive extension of the muscle whereby the stretch reflex is elicited, small action currents begin at a rate of 10 to 25 per second." In the literature concerning experimental physiology as far back as 1925 there is an inference that in a normal resting muscle there is no muscle contraction. In that year Adrian, discussing the interpretation of the electromyogram, pointed out the absence of muscle action currents as long as the muscle is completely relaxed. Adrian and Bronk in reporting the isolation of single motor units, discuss the electromyogram which characterizes muscle contraction. Their comments refer only to activity during contraction and leave the implication that there is no electromyographic evidence of contraction when the muscle is in the resting state. Lindsey stated, "No electrical activity has been demonstrated at any part of the

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relaxed muscle." Smith's made a similar observation. Both sampled numerous areas of normal relaxed muscle and found no evidence of contraction. We have had the same experience in our laboratory. Repeatedly electrodes have been inserted in various parts of normal relaxed muscle. Except for occasional momentary contraction, following the introduction of the electrodes, no contraction has been found (See Fig. 1A).

The success of these experiments hinges on the absence of muscle contraction in a normal resting muscle. However, as this has been demonstrated repeatedly, we can assume that when contraction is seen in a resting muscle it must be due to reflex activity.

There have been numerous reports in the literature concerning action potentials accompanying artificially stimulated muscle, voluntarily contracted muscle, muscle tension in neuromuscular diseases, and muscle tension occurring in the “imaginary” performance of tasks. In addition, Jacobson has reported extensive studies of muscle action currents in nervous and neurotic patients. Except for Jacobson’s studies of neuromuscular hypertension, and the material which deals with organic neuromuscular disease, observations of muscle action currents have dealt primarily with artificially stimulated or voluntarily contracted muscle. There have been no reports of muscle contraction except for changes noted by palpation.

The osteopathic lesion is characterized clinically by an increase in resistance to pressure deformation of the periarticular tissues of spinal, sacroiliac, and appendicular joints in certain cases which may have neither obvious organic pathology nor voluntary muscle contraction.

In the present study an attempt has been made by electromyographic findings to determine whether or not muscle activity underlies in part these clinical findings.

METHODS

Electrodes were placed on normal and lesion areas. Currents from these electrodes were led to dual high gain, 4 stage, resistance coupled, balanced amplifiers which drove either loud speakers for listening to the currents, a Sanborn cardioscope for visualization, or Westinghouse sensitive bifilar oscillographs for recording on electrocardiograph paper.

The electrodes were soder skin pads, hypodermic needles, or concentric needle electrodes. A concentric electrode consists of a hypodermic needle with a fine insulated copper wire placed in the lumen. The tip of the copper wire is bare at the bevel of the needle. Thus the tip of the wire and the shaft of the needle become two separate contact points. This permits a fine localization of pick up.

The concentric needle electrodes were used almost exclusively. In voluntary contraction a large number of motor units are picked up by these electrodes. However, in reflex contraction, such as is found in a lesion area, their pickup is usually limited to one or two units. Thus, it is possible to study the amplitude and frequency of a single motor unit in an area of reflex contraction.

The subject, in a copper screen room to shield out 60 cycle and other interference, was placed prone on an upholstered table which had a padded slot 3 inches wide and 6 inches long for the face. This permitted the head to be in the mid-line position.

Areas of lesion and normal control areas were selected by palpation. Electrodes were inserted into these areas in the erector spinae mass. The electrodes and skin were treated with 70 per cent alcohol and the skin was anesthetized by intradermal injection of 1 to 2 cc. of 2 per cent procaine hydrochloride.

Recordings from these electrodes were made with the patient completely relaxed, in the fraction of a second between exhalation and inhalation and with the patient’s head in the mid-line position. This was necessary because voluntary movement or even involuntary “tension”, respiration, or an asymmetrical position of the head all result in muscle contraction. As has been seen, the base line of a recording from a normal resting muscle is “quiet.” As the subject uses the muscles for any purpose, electromyographic evidence appears in the record (See Fig. 2). In this figure the electrode leading to the upper record is in an area of lesion and shows continuous activity. The electrode leading to the lower record is in normal area, and the base line, when the patient is inhaling or exhaling, is quiet. As the patient breathes, the activity in the spinal extensors appears in the record.
A simultaneous recording was made from a lesion area and from an adjacent normal area in each instance. The recording from the normal area served as a control to demonstrate that the activity of the lesion is local and not just part of a generalized muscular tension.

Twenty-five records were taken on 16 students and instructors in whom areas of lesion were found by palpation. All were free from gross disease except for one with mild attacks of asthma. All were carrying a full program of work. Each one, however, had a postural error in the form of either a slight lateral curvature or a lessening or an exaggeration of the normal anatomical curve. Some subjects gave a history of having had discomfort in the lesion area, others did not.

RESULTS

Localized motor unit activity was seen in a lesion area in 21 of the 25 experiments in this series. This activity or contraction is called a lesion reflex. That this activity was local and not merely a part of a generalized neuromuscular hypertonicity is shown by the absence of activity in the control, an adjacent normal area observed simultaneously.†

The reflex activity in the lesion area varied in degree during each experiment. It ranged from occasional quiet periods through the activity of single units to that of many (See Fig. 3). There was no definite pattern to the increase and decrease of activity except that the quiet periods were more frequent when the subject first reined, activity increasing as the subject became fatigued from being in the same position for 30 minutes or more. Both the number of active units and the frequency of single units were increased by respiration of the thoracic type, by mild voluntary contraction, and by involuntary tension. Involuntary tension was frequently a disturbing factor. This could be eliminated by talking to the subject, by telling him to take two or three breaths or to move a bit, or to relax until the activity heard in the speakers was reduced to a minimum.

The frequency of a majority of the single motor units in an area of lesion was from 6 to 10 a second. Single active units were seen at frequencies as low as three a second and as high as twenty-four a second. There was often a change in the frequency of the active unit in an area of lesion without apparent cause. However, the frequency of a single unit was invariably increased by one of the factors which have been mentioned.

INHIBITION

In individual instances a single active motor unit in a lesion area could be inhibited. The result of this inhibition might be in the form of a decrease in frequency of contraction of the active unit or the activity might stop entirely. This finding was discovered accidentally. As the rhythmic staccato firing of a single motor unit in an area of lesion was being followed in the loud speaker, the subject asked a question. As he talked the contractions slowed and stopped. When he ceased talking they immediately started again. To check this we had him count from 1 to 10. When he reached 2 or 3 the contractions had stopped and they picked up 2 or 3 seconds after he ceased counting.

Further experimentation revealed that a similar inhibition could be brought about by extremely slight voluntary contraction of the abdominal wall, by shallow respiration of the abdominal type, and by swallowing (See Fig. 4).

Several factors are necessary for the successful inhibition of the reflex activity of a single motor unit in an area of lesion. The most important is the strength or persistence of the reflex. In some instances this seemed to be borderline and the activity of a single motor unit came in and faded out. Slight changes in the position of the subject and momentary periods of voluntary contraction might result in a cessation of activity. Or the area might be quiet until activity was initiated by a slight movement of the skin about the electrode.‡

Inhibition produced by one of the factors mentioned, that is by counting aloud, swallowing, or contracting the abdominal wall occurred only in these areas where the activity was borderline.

Where the lesion reflex persisted without periods of inactivity, it could not be inhibited. Additional factors which mitigate against demonstrating inhibition are the presence of more than one or two motor

† McKeeley,19 at the University of Minnesota stated, "As to the movement of electrodes producing action currents, that happens, and yet we have been able to place our electrodes in the muscles and then move those muscles in rather lively fashions without any action potentials being developed whatsoever."

‡ We have had the same experience. When the electrodes are in normal muscle the skin about them may be moved to a considerable degree without initiating muscle contraction.
units in the lesion areas and the inability of the subject to limit voluntary contraction to the flexor groups exclusively. Such subjects became tense when trying to follow instructions. In one subject, a single unit was firing in the lesion area. He swallowed, and the activity stopped for a few moments. He swallowed again and the activity stopped again. But when we attempted to photograph the finding he became tense. The single unit we had been following was smothered by the bursts accompanying the tension.

Because all of the factors which inhibit the lesion reflex produced mild flexion, it is felt that the reflexion itself created an inhibitory factor. Whether the flexion was a result of contraction of the muscles of the anterior part of the neck or of the anterior abdominal wall seemed immaterial.

**ABSENCE OF ACTIVITY IN LESION AREA**

In only four experiments no activity was observed in the lesion area. In each of these the observations were made over 30 to 60 minutes and the electrodes were slowly removed from the muscle and reinserted at least 5 times to insure against missing a small localized area.

**POSTURAL REFLEXES**

In the first experiments the subject was placed prone on the table with the head turned to one side. Inconstantly, and never in some subjects, varying degrees of activity were seen in the thoracic extensors on the jaw side while the vertex side was quiet (See Fig. 5). This might have been predicted as it is well known that through the cutaneous reflexes of Magnus and DeKleyn the extensors of the extremities are contracted on the side toward which the head is turned and relaxed on the opposite. We might deviate here a moment to recall that osteopathic physicians have automatically turned the patient’s head toward them while they treated the side of the spinal column which is away from them.

**DISCUSSION**

The motor unit activity demonstrated in lesion areas is local and is reflex in character. It is similar to the stretch reflex as described by Greel, et al. with exception of the finding of low frequency rates in 10 records. There is also similarity with the extensor response as first described by Magnus.

The action potential findings in a lesion area have characteristics identical with those of the stretch reflex in its response to tensions and head positions. However, instead of being due to a stretch or to a turning of the head they were observed when the patient was completely relaxed, in the resting period of the respiratory cycle and with the head in the mid-line. The activity occurred in local areas where palpation revealed findings characteristic of osteopathic lesion. Hence, they represent a muscle contraction which could be designated as an abnormal reflex or, as we have called it, a lesion reflex. The difference between the stretch and postural reflexes and the lesion reflex lies in the cause of its development, there being no stretch or positional asymmetry in the lesion reflex. The cause is either a stimulation of tension receptors.
within the muscle itself or stimulation of other receptors which are segmentally related.

Because the subjects did not have visceral disease and as palpable joint abnormality was present in the area examined, we feel justified in the view that the cause of the lesion reflex is the abnormal pressures and tensions developed in the functionally pathologic joints.

Joints which show some arthritic changes become stiff and uncomfortable when the patient remains for an hour or more. They improve when he moves about. This is probably a parallel with our observation that at times more activity in a lesion area is not seen until the spinal articulations have been relatively immobile for some time.

In some experiments slight movement in the skin around the electrode was followed by motor unit activity while the control areas similarly treated remained quiet. Here the lesion reflex perhaps created an enduring subliminal central excitatory state in a motorneuron pool. The nerve cells in this area were stimulated by the impulses coming from the joint. The stimulation, however, was not great enough to reach a threshold level and, consequently, no motor unit activity ensued. However, when additional stimuli from an electrode movement reached the motorneuron pool which was established by the impulses from the lesion the combined effect of the impulses from the lesion and impulses from the irritation of the electrode were great enough to reach threshold level and motor activity was the result. Conversely, activity of the lesion reflex was temporarily terminated by flexor activity, only when the nerves bombardecd from the lesion area were apparently just above the threshold level. Not being strong they could be inhibited by the flexor reflex, which, all other things being equal, is stronger than that from the extensors.

There are several possible reasons why in several experiments no motor unit activity was found in lesion areas. The most likely was that there was none present at that time and that the apparent tense ness in the muscle was due to some other factor rather than muscle contraction. The scope of this paper does not permit a discussion of what that other factor might be.

**SUMMARY**

1. Reflex muscle activity, similar to stretch or postural reflexes, is seen in areas of lesion in the erector spine muscles when the subject is relaxed, the extremities in symmetrical position, and the head and face in the mid-line. Adjacent normal areas do not show motor unit activity. One or more motor units may be active, sometimes after a delay of 5 to 45 minutes after inserting the electrodes.

2. Single units discharge at rates of 2 to 24 per second.

3. A single active unit firing intermittently in the lesion area could at times be stopped by flexor contraction.

4. In a few instances motor unit activity was not seen in an area of lesion.

5. In some normal subjects muscle activity appeared in the spinal extensors on the "jaw" side when the head was turned.

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**REFERENCES**