

THE NEURAL BASIS OF THE OSTEOPATHIC LESION¹

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INTRODUCTION

FOUR of the main principles in osteopathy appear to be:

1. Joints and their supports are subject to anatomic and functional derangements.
2. These derangements have distant as well as local effects.
3. They are related, directly or indirectly, to other pathologic influences.
4. They may be recognized, and their local and distant effects influenced favorably by manipulation.

Accepting the existence of joint derangements (osteopathic lesions), it is our purpose in this paper to examine not the mechanical and etiological factors involved, but rather the fundamental basis for principles 2 and 3 and to a small extent principle 4 and report progress in our understanding thereof.

The osteopathic lesion has many aspects which are partly revealed in the local and distant effects referred to as principle 2. Included among these are:

1. Hyperesthesia, especially of the muscles and vertebrae
2. Hyperirritability, reflected in altered muscular activity and in altered states of muscular contraction.
3. Changes in the tissue texture of the muscle, connective tissue, and skin
4. Changes in local circulation and in the exchange between blood and tissues.
5. Altered visceral and other autonomic functions.

How are these effects produced? What are the central factors responsible for these manifestations of structural and postural abnormalities? What in the intrinsic nature of the osteopathic lesion is the basis for the peripheral, palpable, and clinical effects? What fundamental changes take place as a result of effective

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manipulative theory?

The detailed answers to the questions are, of course, not yet available, but reliably indicated are the general nature of the final answer and the direction in which we must proceed in order to obtain it. The research program of the Kirksville laboratories is designed to procure some of these answers through exploration of the intimate mechanisms involved in the osteopathic lesion. We believe that the answers are obtainable only through fundamental experimental research and that the emerging concept of the lesion and its implications will have considerable impact on the practice of osteopathy.

In this paper will be presented some of our current views, some of the practical implications, and some speculations. The details of the experimental methods and the raw data, available in earlier publications, (1,2)) will not be presented, but rather the general experimental approach and the immediate conclusions therefrom. From these, in turn, will be drawn some generalizations.

THE NEURAL BASIS OF THE OSTEOPATHIC LESION

Within the nervous system, in the phenomena of excitation and inhibition of nerve cells, and in the synaptic and myoneural transmission, lie the answers to some of the most important theoretical and practical osteopathic problems. The existence of a neural basis for the lesion has been known, of course, for a long time. The segmental relation of the osteopathic lesion to its somatic and visceral effects is explainable in no other way.

The activity and condition of the tissues and organs are directly influenced, through excitation and inhibition, by the efferent nerves which emerge from the central nervous system and which conduct impulses to these tissues and organs (Fig.1). For example:

Muscle

- A) Anterior horn cells (Motoneurons) - muscular contraction
- B) Lateral horn cells (Sympathetic preganglionic neurons through postganglionic neurons) - vasomotor activity

Skin

- C) Lateral horn cells - vasomotor activity

- D) Lateral horn cells- sweat gland secretion
- E) Lateral horn cells - piloerection

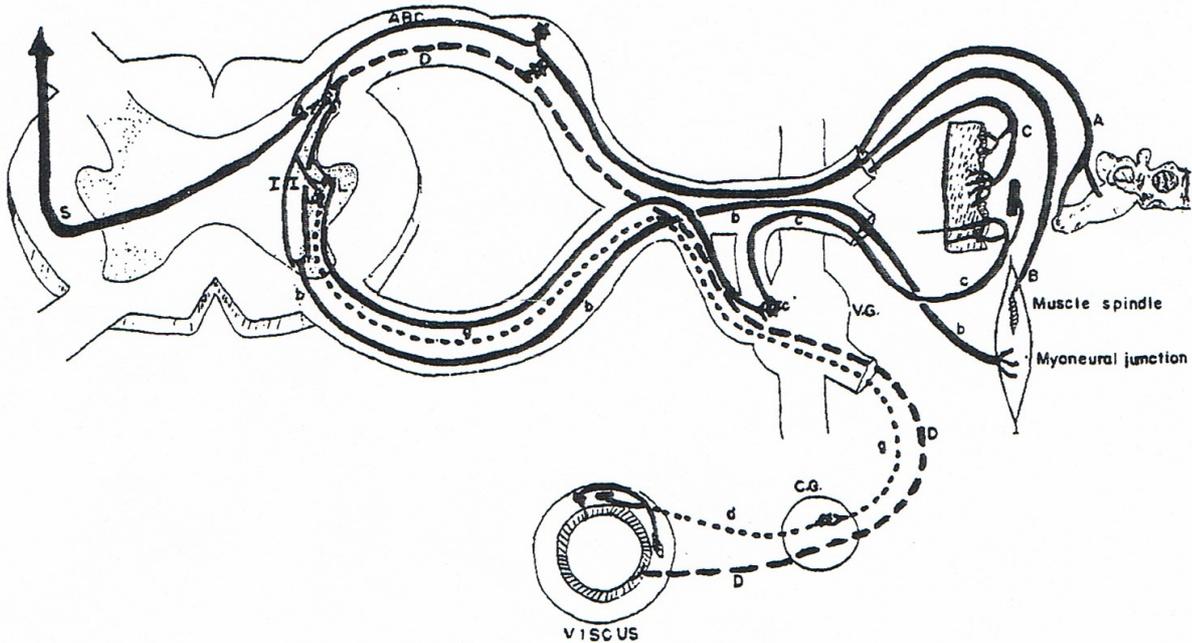


Fig. 1.-Diagrammatic representation of segmental reflex pathways among somatic and visceral afferents and efferents.

Afferents (Dorsal root neurons):

- A-From spinous process, joints
- B-From stretch and tension receptors (proprioceptors) in muscles and tendons
- C-From touch, pressure and pain endings in skin
- D-From viscera
- ABC-Somatic afferents

Efferents:

- b-motoneurons to skeletal muscle
- c-sympathetic postganglionic neurons

- to blood vessels of skin and muscle; to sweat glands and pilomotor muscles of skin,
- d-sympathetic postganglionic neurons to visceral smooth muscle, blood vessels and glands.
- S-Spinothalamic fibers
- I-Interneurons
- L-Lateral horn cells (sympathetic pre-ganglionic neurons)
- V.G. - Vertebral ganglion
- C.G. - Collateral ganglion

Viscera

- F) Lateral horn cells - smooth muscle contractions

G) Lateral horn cells -glandular secretion

H) Lateral horn cells - vasomotor activity

The activity of these organs and cells is directly determined by the activity of their motor nerves. This nerve activity is measured in terms of: (a) The number of impulses conducted by each efferent nerve fiber, and (b) the number of fibers involved.

Thus, in the absence of impulses in the corresponding motor nerve, a muscle is completely at rest.

The amount of contraction (tension produced or degree of shortening) at any moment is in proportion to the number of motoneurons which are conducting impulses at that moment and the average number of impulses per second which is conducting to the muscle. With certain modifications this principle also applies to organs other than the muscle. Abnormalities in these organs are produced by overactivity or underactivity of the efferent nerves.

SECONDARY EFFECTS OF NEURAL IMBALANCE

It is important to emphasize, however, that not all the effects of overactivity or underactivity of the efferent neurons are direct and immediate. Secondary effects often assume predominate influence.

Thus, a muscle's overactivity, over a long period of time, may result in fibrosis and major chemical and metabolic changes; underactivity in atrophy. Overactivity of sympathetic fibers which control arterioles may result in local anoxemia, inflammation, altered capillary permeability, edema, etc. Imbalance in the efferent neurons controlling the smooth musculature of the gastrointestinal tract may result in flaccidity or spasm with serious effects on digestion and absorption and, therefore, on the entire body economy. Overactivity or underactivity of the neurons controlling glands may result in disastrous shifts in acid-base, fluid, and electrolyte balance and in such conditions as peptic ulcers. If the gland happens to be one of the endocrines, the effects may be especially serious and extensive. We may for the present purpose include the spinothalamic fibers among the "efferent" neurons. These convey pain sensations to the brain and, when

overactive, produce not only physical but also important psychological changes with potentially serious and extensive changes in motor and visceral activity. With the crucial importance of the efferent neurons in mind, more precise formulation of the problem is possible. There are three main questions:

1. What factors control the activity, i.e., the number of impulses, in the efferent nerve fibers?
2. How does structural abnormality, i.e., the osteopathic lesion, play upon these factors to produce overactivity or underactivity of these fibers, and, therefore, of the organs which they innervate?
3. How does manipulative therapy play upon these factors to restore balance and cause regression of signs and symptoms?

FACTORS CONTROLLING EFFERENT ACTIVITY

Let us proceed to the first question. What factors has physiological research demonstrated to be primary in the control of activity of the efferent neurons? Two main principles have special pertinence here.

A) The principle of reciprocity states that through the network of interneurons (also known as internuncial neurons, intercalated neurons, and connector neurons), which is situated within the central nervous system, every neuron potentially influences, and is influenced by, almost every other neuron in the body. (3)

B) The principle of convergence states that many nerve fibers converge upon, and synapse with, each motoneuron. These presynaptic fibers convey impulses from a large variety of sources to the efferent neuron, which, therefore, represents a final common path. (4)

Let us examine how these principles operate with respect to the anterior horn cells, keeping in mind that probably operate in a similar fashion upon the other efferent neurons (Fig.1).

1. Each anterior horn cell receives impulses from a large number of sources through the presynaptic fibers which converge upon and synapse with it. All the descending tracts in the spinal cord, conveying impulses from such sources as the

cerebral cortex, red nucleus, medulla oblongata, the vestibular nuclei, cerebellum, the pons, superior colliculi, and other higher centers, establish synaptic connections with the anterior horn cell for the mediation of voluntary motion, equilibrium, postural reflexes, visuspinal reflexes, and others. The proprioceptors, stretch and tension receptors situated in the tendons and in the muscles themselves, are a steady and continuous source of impulses.

They exert their influence directly through the dorsal root fibers into which they discharge their impulses or, indirectly, through the higher postural and equilibrium centers. Afferent fibers from the viscera may also play an important role. In fact, every afferent nerve fiber, whether it mediates touch, pain, pressure, temperature, sight, or any other sense modality, exerts influence upon the final common path represented by the motor nerves.

2. Some of the converging fibers exert an excitatory influence, others an inhibitory influence on the same motoneurons.

3. The activity of the motoneuron at any moment, that is, the frequency with which it delivers impulses to the muscle fibers, represents a dynamic balance among all the excitatory and inhibitory influences being exerted by the many neurons which converge upon it. The proprioceptors and some of the higher centers, through their steady, tonic control, act as governors or buffers. The balance, however, is shifted from moment to moment in accordance with changes in the internal and external environment and in response to volition. As previously stated, pathology results when the balance is shifted too far in one direction or the other (excitation and inhibition) for too long.

4. The collective action of the presynaptic nerve fibers upon the final common path is further reflected in the phenomena known to physiologists as reinforcement and facilitation. Before the anterior horn cell can discharge impulses into the muscle fibers, it must itself receive excitatory impulses simultaneously from a number of presynaptic fibers. (5)

Stated another way: Before a given stimulus (e.g., to the skin) can produce a reflex muscular response, the anterior horn cell must first be “warmed up” or “put on edge” (facilitated) by impulses from other (excitatory) fibers which synapse with it. The efferent neuron must already be in a state of sub threshold or subliminal excitation. In other words, the various fibers converging upon a given group of

motoneurons must cooperate (reinforce each other) in order to open the final common path leading to the muscle. In a whole nerve it has been demonstrated that a considerable portion of the nerve fibers must be in a state of subliminal excitation before any of them fire and cause muscular contraction. (6)

5. This requirement serves as a margin of safety or an insulation, preventing muscles from responding to every impulse which reaches the anterior horn cell.

6. When a significant percentage of the anterior horn cells in a given segment of the spinal cord is maintained in a state of subliminal excitation, they require a little additional stimulus to produce a reflex response. This is reflected in our frequent use of the terms "on edge", "jumpy", "tense", which imply motor aspects of physic imbalance. In individuals thus characterized the anterior horn cells are maintained close to, or at, threshold, even during rest.

THE OSTEOPATHIC LESION AND THE FACTORS CONTROLLING EFFERENT ACTIVITY

The second question in our series of three was "What is the relation of the osteopathic lesion to the above factors?" How do anatomic and functional derangements of the joints and their supports operate on these factors to produce seriously altered activity of the efferent neurons? Considerable light is being thrown upon this problem by the research in progress at Kirksville College Of Osteopathy and Surgery under the directorship of Dr. J. S. Denslow. The research has revealed close relations between lesion mechanisms and certain well-established physiological principles. The general experimental approaches and the major conclusions from each are presented in the following section.

EXPERIMENTAL

Reflex Threshold - Denslow, proceeding from the observation made by all osteopathic physicians that pressure to the spinous processes of lesioned segments produces much more contraction in the spinal extensor muscles, and with less pressure, than is true at nonlesioned segments, set out to determine in a precise, objective manner how much pressure is required at each spinous process

to elicit reflex contraction of the spinal extensor at the same level. In other words, his object was to determine whether, and to what degree, lesioned segments were distinguished from the normal by differences in reflex threshold.

Muscular activity was determined electromyographically, that is, by recording the electrical signs of muscular activity. Measured pressure stimuli were applied to the spinous processes by means of a calibrated pressure meter which stimulated the action of the osteopathic thumb. At each segment gradually increasing pressure stimuli were applied to the spinous process until just detectable activity in the erector spinae mass was obtained; this represented the reflex threshold for that segment. The reflex arc under investigation might be said to consist of these parts: spinous process, dorsal root fiber, inter-neuron, anterior horn cell, and muscle fibers. (See Fig.2, disregarding segmental designations and intersegmental connections).

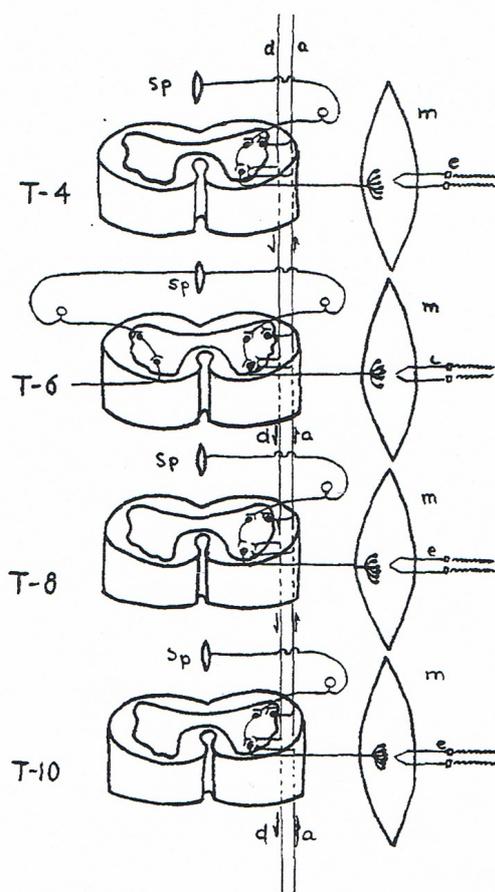


Fig. 2. -Diagram of reflex pathways involved in experimental measurement of segmental reflex thresholds and of intersegmental spread of excitation. (See text.)

sp - sensory endings in spinous process
a,d - ascending and descending intersegmental neurons
m-erector spinae mass
e-recording electrodes

This pioneer study upon a large number of human subjects resulted in the demonstration that the reflex thresholds in lesioned segments were much lower than in normal segments. The more severe the lesion, as determined by palpation, the lower the threshold. The thresholds may be constant over periods of months.

What is the basis for the lowered reflex threshold of the lesioned segment? There were two obvious alternatives. (1) The sore spines. It was reasonable to suppose that the pressure receptors and nerve endings in the tender spinous process were hypersensitive and that, per gram of pressure, they fired more impulses at the anterior horn cells than did the corresponding endings in the normal spinous process.

(2) Hyperirritable motoneurons. It appeared equally reasonable to suppose that for some reason or other the anterior horn cells of the spinal extensor muscle in the lesioned segments were maintained at a higher level of excitability. In order to decide which was the more likely alternative, the following experimental approaches were undertaken. (2)

Intersegmental Spread of Excitation. - A lead to the answer was given in experiments in which spread of excitation from segment to segment was examined in relation to their respective thresholds, to the distance between them, and to other factors, The experiments were conducted as follows (Fig.2) The four thoracic segments, designated as T4, T6, T8, and T10 were selected for this series of experiments on 30 subjects. Needle electrodes were inserted into the spinae erector mass 5cm, to the left of the spinous processes in each of the four segments, for the detection and recording of muscular activity. Pressure stimuli were applied to the spinous processes with the pressure meter.

The minimum pressure stimuli (threshold) required at each of the four spinous processes to elicit activity from each of the four muscle segments was then determined in turn. Thus, the pressure required upon the spine at T4 to elicit activity in the muscle at T4, in the muscle at T6, in the muscle at T8 and in the muscle at T10 was determined. This was then repeated at the other spinous processes, giving four thresholds at each spinous process, sixteen in all, in each

experiment. The results will be summarized by illustrating with one hypothetical subject, eliminating some qualifications for the sake of brevity.

It was found that there was much more spread to lesioned segments than from lesioned segments.

Thus, if T6 were a severely lesioned segment (very low threshold) while T8 and T10 (neglecting T4 for the moment) were normal or high threshold segments, the following obtained. It required very little pressure to the spinous process of T6 to elicit activity of the muscle in the same segment; but even very strong pressure stimuli to the same spinous process failed to produce any signs of activity in the muscles at T8 or T10. Conversely, although even very high pressures to the spinous processes of the latter two segments produced no activity in either of these segments, relatively slight pressures upon the spinous processes of each of them did stimulate reflex contraction at T6. Thus, excitatory impulses entering the cord at T10 "bypassed" the motoneurons of the same segment and those of a neighboring high threshold segment, to emerge or produce effect at a more remote lesioned segment.

If T4 were moderately lesioned (as was often the case if there was a lesion at T6), it participated in exchange of excitation with T6, but usually only received excitation from T8 and T10.

Our conclusion from this series of experiments can be simply stated in an analogy. The anterior horn cell of the lesioned segment represents a bell easily rung from a number of push buttons, while the spinous process or push button of the lesioned segment does not easily ring bells other than its own.

The hyperexcitability of the lesioned segment (that is, the relatively low reflex threshold) is demonstrable without applying pressure to the corresponding spinous process.

Procaine Studies. - It was of interest in this connection to determine whether and how the excitability of the lesioned segment was affected by desensitization of the spinous process with procaine. Infiltration of the periosteum around the spinous process raised the local threshold to that of a normal segment, that is, it was no longer possible to produce reflex response of the muscle of that segment with slight, moderate, or even heavy pressure stimuli to spinous process of that segment.

In contrast, however, spread of excitation to that segment from other segments remained unimpaired; although pressure to the procainized spinous process of T6 no longer elicited reflex contraction at T6, it was still possible to elicit contraction at T6 by pressure upon spines T8 and T10. Thus, the hyperexcitability of the lesioned segment was again demonstrated independently of the spinous process, it exists even when the spinous process is desensitized.

Bilateral Differences. - On a few subjects the reflex responses on both sides of the same segment were simultaneously observed (Fig.2, T6). It was found that the spinal extensors on one side of the segment may respond reflexly to very slight pressure upon the spinous process while the other side requires much higher pressure to the same spinous process. In other words, low or high thresholds are not determined by the spinous process. The neurons in the left and right horns of the same segment may be maintained in different degrees of excitability. The left and right “bells” are rung with different degrees of facility from the same “push button”.

Rest Activity. - The differential excitability of anterior horns in lesioned and nonlesioned segments was further and clearly shown in experiments in which the anterior horn cells were exposed, not to impulses from spinous processes but to generalized stimuli coming from within and without the body.

When a patient is prone and completely relaxed there is usually no activity in the spinal muscles; there is not even tone, as indicated by the absence of action potentials in those muscles, This is true, usually, even of segments in lesion.

Occasionally, however, it was found that muscular activity persisted in the absence of external stimulation. The subjects had to be carefully positioned and repositioned to eliminate as far as possible the afferent bombardment from the proprioceptors. It was found that when the rest activity did occur, it was almost invariably in the lesioned, low thresholds segments.

Thus, the segment in lesion is the most sensitive to positional stress, conveyed by the proprioceptors in the muscles and tendons.

Mental factors may also be responsible for rest activity. Subjects who are apprehensive, anxious, or emotionally upset often show persistent rest activity. Such activity was always most marked in the lesioned segment; often it occurred only in the lesioned segments. The lesioned segment is thus hyper-responsive to

impulses descending from the cerebrum.

Tactile stimuli also demonstrated the hyperexcitability of the anterior horn cells in the segment of lesion. If the back was slightly scratched or tickled with a pin, as over the shoulder or scapular area, continuing activity in the spinal extensors at the lesioned segment was often precipitated, but very rarely in the nonlesioned segment of the same subject. Thus impulses from touch and light pressure receptors in skin also find the most responsive anterior horn cells in the segments of lesion.

Occasionally we found a motor unit which fired in synchrony with inspiration and expiration; such a unit was invariably situated in the lesioned segments.

Apparently such segments are hyper-responsive also to impulses from bulbar and pontine centers.

Interpretation of Experiments. - The following general conclusions may be drawn from these experiments as regards to motor activity in lesioned segments.

1. An osteopathic lesion is associated with a segment of the spinal cord which has a low motor reflex threshold, i.e., it represents a hyper-excitable segment of the cord. At least in the lesioned segments studied by us it may be said that the balance has been shifted too far too long toward the excitatory side.

2. The lowered reflex thresholds are demonstrable independently of the related spinous process.

Even though changes in the palpable characteristics and in pain sensitivity of the spines are important diagnostic features, they are apparently secondary to other, more fundamental alterations in the cord.

This aspect will be discussed later.

3. The lesion represents an anterior root at least some of whose motoneurons are maintained in a state in which they are relatively hyper-excitable to all impulses which reach them. In a severe lesion many of the motoneurons are so close to the threshold, even when the subject is at rest and reclining comfortably, that it requires very few additional impulses from the neurons which synapse with them to trigger those motoneurons into overt activity. Those additional impulses may come apparently from almost any source; the spinous process is but one such source.

4. The lesion, therefore, is to be conceived, not as a radiating center of

irritation, spreading excitation to other segments, but rather as a segment upon which irritation is focused. It represents a place in the cord where barriers to motoneuron excitation have been lowered and which, therefore, channelizes impulses into muscles receiving motor innervation from that segment.

Basis For Segmental Hyperexcitability. -What is the basis for this segmental hyperexcitability? What keeps the motoneurons of the lesioned segment “on edge”, that is, at a high level of subliminal excitation? The anterior horn cells can be maintained in this facilitated state by continuous and excessive bombardment from some untiring source or sources.

The Source of Impulses. -What are the untiring sources of impulses with which the anterior horn cells in the lesioned segments are continuously and excessively bombarded? First, their excessive activity is apparently associated with postural, mechanical, and articular derangements. Second, it must be recognized that they are apparently a highly stabilized and chronic source. Reflex thresholds in segments of lesion have been found to be very constant over periods of months and even years. Third, it must be recognized that they are probably highly localized, often restricting their facilitating effect to only one or two segments.

The sources which, in our opinion, most closely fulfill these qualifications are the proprioceptors, i.e., the stretch, tension, and pressure receptors in the muscles and connective tissues.

First, postural, mechanical and articular derangements unquestionably cause increased fiber-length or tension in the muscles and tendons on at least one side of the articulation in question. The proprioceptors are highly sensitive to changes in fiber-length or tensions.

Second, they are the nonadapting type of receptor. They keep firing impulses into the cord via the dorsal root fibers as long as they are under tension and at frequencies which are proportional to the tension.

The higher the tension, the higher the afferent bombardment for as long as the tension is maintained.

Third, the afferents from proprioceptors not only have segmental distribution, but they specifically influence the activity of the muscles to which they are most closely related or in which they are situated.

This specificity extends not only to the muscles themselves, but to specific

muscle heads. It is thought that the muscle spindle cells reflexly influence only the muscle fibers in their immediate vicinity. In this way, highly localized, vicious cycles of irritation may be set up.

We, therefore, believe that these receptors play a prominent role in maintaining segmental hyperexcitability in areas of lesion. As a result of the continuous barrage of impulses which they fire into the cord at their level, the anterior horn cells of the corresponding segment are maintained in a state of chronic facilitation at a high level of subliminal excitation, even during rest.

Effects of Chronic Facilitation. -In these segments, therefore, it may be said that the normal "insulation" which keeps the efferent neurons from firing in response to every impinging impulse has become worn. Since under normal conditions of life, requiring constant adjustment to the external and internal environment, requiring motion and the maintenance of the erect posture, so many impulses from so many sources are constantly impinging on the motoneurons, in all segments of the cord, that those which are already facilitated, as in the lesioned segment, will inevitably be more active than the other. The muscle fibers to which they are connected will then be excessively high in tone. If maintained for sufficient periods of time this hypertonus would lead to textural, morphological, chemical and metabolic changes which may, in turn, become secondary and chronic sources of irritation.

Other Neurons. -We have thus far discussed only the motoneurons and alterations in motor reflex activity in areas of lesion. What of the other efferent nerve fibers and the organs and tissues which they innervate?

Our experimental studies have demonstrated that closely and quantitatively correlated with lowered motor reflex thresholds are three other features of the lesion: (1) Alteration in the texture of the tissue overlying the spinous process, (2) lowered pain threshold, and (3) increased susceptibility to trauma.

1. Tissue texture: As is well known to osteopathic physicians, there are striking changes in the texture of the tissues over the spines of lesioned segments. It was found that the degree of change in tissue texture was so closely related to the degree of lowering of motor reflex threshold from the normal that Denslow, through palpation of subjects prior to each electromyographic determination of reflex threshold, was able to predict with remarkable accuracy

the reflex threshold of each segment.

It is probable that these changes in texture are due to local changes in vasomotor activity, fluid balance, capillary permeability, trophic factors, and other features which are directly or indirectly under the influence of the lateral horn cells of the sympathetic nervous system.

2. Pain Threshold: A direct correlation was found between motor reflex threshold and segmental sensitivity to pain. As is well known, the spines in lesioned segments are much more tender than those in normal segments. In other words, it is easier to reach the "consciousness" of the patient, i.e., the cerebral cortex, through the lesioned segment than through the nonlesioned. This, we interpret as signifying a facilitation of the spinothalamic fibers.

3. Susceptibility to trauma: It was found that if one applies equal mechanical irritation, (measured impacts) to the spinous processes of lesioned and nonlesioned segments, the former may remain painful for several days, whereas the subject soon forgets which of his normal spines received the pounding.

We may conclude from these correlations with motor reflex threshold that neurons other than the anterior horn cells may also be facilitated and maintained in a state of hyperexcitability in the lesioned segment. This appears to be true, at any rate, of certain of the preganglionic fibers of the sympathetic nervous system and of the spinothalamic fibers conveying pain sensation to the higher centers.

Experiments are now in progress to establish the degree of involvement of neurons of the sympathetic nervous system. Dermatomal alterations in sweat gland activity are being determined by measuring the electrical conductivity of the skin in lesioned and nonlesioned segments. We are measuring alteration in the activity of sympathetic fibers controlling vasomotor activity by electrical measurements of skin and deep muscle temperature. Although these studies are still in a preliminary stage, it has become evident that sympathetic activity in the skin is altered in lesioned areas and that instruments used for the measurement of these peripheral changes, and others which are contemplated, will in one form or another become valuable diagnostic aids. They are much more sensitive than fingertips and certainly easier to standardize.

There is no reason to doubt that lateral horn cells which influence specific visceral functions are fundamentally similar to those controlling the sweat glands.

A project, in collaboration with Dr. D.E. Drucker of the Department of Physiology is under way for the objective and precise determination of alterations in various visceral functions resulting from acute experimental spinal lesions in animals. The effects on visceral functions, in normal unanesthetized dogs, of lesions in segments related to the viscus under examination will be compared with the effects of similar lesions elsewhere. At present, these investigations are limited to to a study of renal blood flow, glomerular filtration, and tubular secretion. The kidney was selected for the first such investigation because methods of quantitative study in normal animals and humans are highly developed and because of the clinical significance of renal blood flow and renal metabolism in connection with such entities as hypertension. Similar studies upon other visceral organs are projected for the near future. It is hoped that from these studies will emerge a clearer understanding of the relations of the osteopathic lesion to visceral disease.

CHARACTERIZATION OF THE LESION

On the basis of the experimental studies so far, I believe we are ready to attempt a characterization of the osteopathic lesion in terms of basic neural mechanisms. Let us first summarize our general conclusions.

1. Normally, efferent neurons are kept from firing in response to every impulse that reaches them by the fact that a relatively high level of subliminal excitation or facilitation must be established, by other impulses converging upon them, before the firing point is reached. This requirement serves as a sort of insulation.

2. In the lesioned segment this insulation has been weakened. A large portion of the efferent neurons are kept near the firing point (facilitated), even under conditions of rest, by chronic afferent bombardment from segmentally related structures.

3. Proprioceptors are undoubtedly an important source of this bombardment, but any segmentally, related structure may be such a source. A pathological viscus, or a cutaneous trigger spot, or any other inflamed or irritated structure which concentrates its afferents in one or a few dorsal roots may be responsible

for more or less tonic facilitation. (The close relation of the osteopathic lesion to referred pain mechanisms is clear, but space does not permit a discussion of this most important aspect.)

4. The firing process in the facilitated efferent neurons may be completed by any impulses impinging on those neurons, whether the source of those impulses be the cerebral cortex, postural and equilibrium centers, bulbar centers, cutaneous receptors, or others. Should this superimposed bombardment be sufficient and enduring, the facilitated neurons (and the organs that they innervate) may be maintained in a continuous state of excessive activity.

5. The state of facilitation may extend to all neurons having their cell bodies in the segment of the cord related to the lesion, including the anterior horn cells, preganglionic fibers of the sympathetic nervous system, and apparently the spinothalamic fibers.

6. Because a structural defect, an osteopathic lesion, sensitizes a segment to impulses from all sources, and for reasons previously given, the lesioned segment is to be considered not a radiating center of irritation, but rather a neurological lens which focuses irritation upon that segment. Because of the lowered barriers in the lesioned segment, excitation is channelized into the nervous outflow from that segment.

7. It is a truism in neurophysiology that when something is excited, something functionally related is simultaneously inhibited. Although in our studies we have not yet directed attention to this aspect, it cannot be doubted that facilitation in the segment of lesion also extends to neurons exerting inhibitory influences upon other neurons or organs.

It may be concluded that: an osteopathic lesion represents a facilitated segment of the spinal cord maintained in that state by impulses of endogenous origin entering the corresponding dorsal root. All structures receiving efferent nerve fibers from that segment are, therefore, potentially exposed to excessive excitation or inhibition.

MANIPULATIVE THERAPY

We come now to the last question: What, basically, does manipulative therapy

do? Here, we can only guess, but at least our guesses are based on sound, experimentally established hypotheses.

Manipulative procedures applied by osteopathic physicians induce relaxation of muscle which has been maintained in a continuously contracted state and which, as a matter of fact, may not be able to relax spontaneously, even when excitation is removed (contracture). The increase in muscle-fiber length eases the tension on the proprioceptors in the muscles and tendons, bringing about at least a temporary diminution in afferent bombardment of that segment of the cord.

Since the excessive tendinous and muscular tension produced around a joint, let us say, by some bony displacement tends reflexly to produce more tension, the manipulative easing of tension breaks a vicious cycle.

Still another type of vicious cycle may be in operation and be broken by manipulative therapy (Fig.1). Through overexcitation of sympathetic fibers in the segment of lesion, visceral pathology may be established. The anterior horn cells may then be subjected to additional bombardment with impulses conveyed by visceral afferents, thus causing exaggeration of the somatic lesion, which, in turn, further irritates the viscus. Manipulative relaxation of the muscles may break this cycle too, through diminution of proprioceptor discharge into the cord. Even a brief respite from this irritation allows better opportunity for the natural healing processes to operate.

By manipulative rearrangement of the skeleton and through postural adjustments, the original cause of the strain, that is the excessive tension on muscles, tendons, and ligaments, may be eliminated and the beneficial results made more lasting.

This is unquestionably an oversimplified version of the basic effects of manipulation, but it certainly will serve as a working hypothesis, as a guide to further experimental investigation.

SPECULATIONS

Assuming the importance of the proprioceptors in the lesion mechanism, it must be kept in mind that any segmentally related structure which sends afferents to the spinal cord may be an important participant in the establishment

or maintenance of the lesion. In fact, through the network of interneurons, practically any efferent, segmentally related or not, may exert some influence.

To all these sources of impulses must be added the suprasegmental sources all the higher centers, from medulla to cerebral cortex which contribute to the descending spinal tracts. Many of these are continuous and highly variable sources of impulses. They exert their influence excitatory or inhibitory upon efferent neurons at every level of the spinal cord.

It is, indeed, most important to keep in mind that the efferent neurons do represent final common paths shared by a host of impulse sources, in addition to those associated with joint and supporting tissues. In this light, it is apparent that the articular derangement or the osteopathic lesion cannot be conceived as the cause of disease; rather it is one of many factors simultaneously operating.² The lesion is a most important factor, it is a sensitizing factor, a predisposing factor, a localizing factor, a channelizing factor. The lesion sensitizes a segment of the cord, lowers the barrier, facilitates without necessarily producing symptoms, although signs of its presence may be demonstrated by the osteopathic physician. Sufficient additional excitation has to be superimposed upon that from articular and periar titular origins. This is not to minimize the importance of the osteopathic lesion. On the contrary it is to widen the concept. For one thing, it certainly points up the tremendous contribution that osteopathic diagnosis and therapy can make to preventive medicine.

To osteopathic physicians there is, of course, nothing unfamiliar in the practical aspects of this concept. One patient has relatively severe lesions, yet is symptom-free, pain-free, and not readily subject to fatigue, etc.; another patient with very similar lesions, on the other hand, may be subject to serious disturbances directly related to those lesions. Further, the lesions of the first patient may “act up” under certain circumstances, and then subside into “quiescence” again. Why? What accounts for the difference between such patients, and between the “acting up” and “quiescence” periods in the same patient? In our opinion and, I believe, implicit in the osteopathic concept, one important basis for the difference lies in differences in the amount of nervous excitation continuously

² In fact, it is doubtful whether there is ever a single cause of any effect, whether there is ever an isolated etiological factor in any clinical entity. Each factor operates in the context of many factors and produces certain effects only in a certain combination of factors.

impinging on the efferent neurons of the lesioned segments, over and above that from the muscles and joints. The lesion operates in different contexts with different effects.

Given an articular disturbance which, through the mechanisms discussed above, determines the location of the low threshold segments, then the severity of the lesion, the associated pathology, and the response to treatment will be to a great extent, often to a decisive extent, determined by how much additional neuron pressure from other sources is chronically present. Such pressure may be present upon all the segments, but because of lowered synaptic barriers, the effects will be exaggerated at the lesioned segment. The lesion not only focuses; it magnifies.

This superimposed excitation may come from any of the sources previously enumerated, and others, which converge upon the anterior horn cells and the other efferent neurons; the cortex, the basal ganglia, cerebellum, vestibular nuclei, bulbar center, the eyes (via the tectospinal tracts), or any steady, tonic source of impulses.

Since all these sources may directly affect, favorably or unfavorably, the lesion and its associated phenomena, they are all properly within the province of the osteopathic physician. All of them may contribute to the lesion and to its effects on total body economy. Important as is the structural factor, treatment of it alone is not to treat the patient as a whole and would be, if I interpret it correctly, a corruption of the osteopathic concept.

I shall try to illustrate with one source of bombardment which is very of general significance, namely, the cerebral cortex. As previously indicated, the words "tense" "high-strung", "jumpy", "keyed up", "on edge" are more than figures of speech.

They bespeak the well-recognized fact that psychic stress, emotional imbalance, environmental strains, etc., influence and are reflected in motor activity, an increased muscular tone and hyper-responsiveness, in generally lowered reflex thresholds. A familiar illustration is the exaggerated knee jerk of a tense individual. (Other types of imbalance may, of course, have depressing or inhibitory effects, resulting in fatigue, hyperreflexia, inertia and other symptoms).

These are obviously due to impulses passing down the descending spinal tracts

and impinging, directly or through interneurons, on the anterior horn cells and increasing their excitability and activity.

In a segment already sensitized by an osteopathic lesion the effects will be especially severe. Just as important is the fact that descending impulses may exacerbate the lesion and produce increased effects on segmentally related organs, may cause or intensify pain, and make the lesion less responsive to manipulative therapy. To treat only the structural source of bombardment is only to half treat and to neglect a most important part of the lesion mechanism, and to take the lesion out of context. This does not mean, of course, that every osteopathic physician should become a psychiatrist, but he certainly must take into consideration the home factors, family relations, emotional adjustments, tensions, etc.

In this light the as yet unexplored relations of osteopathy to psychosomatic medicine become obvious. It is now well established that many organic ills, including angina pectoris, gastric and duodenal ulcer, gallbladder disease, mucous colitis, asthma and others, may be directly related to psychoneuroses, emotional imbalances, and anxieties. What factors determine the organic manifestation of the neuroses? The autonomic nervous system, of course, has regional representation in the cerebral cortex and the hypothalamus is under cortical influence. It has been thought that the unconscious may select the organ or organs to be affected. Without prejudging these and other theories, it would seem most profitable, clinically and experimentally, to explore the probability that the incidence and location of the osteopathic lesions may be an important factor in determining the incidence of nature of psychosomatic ills. Certain aspects of this hypothesis are under experimental investigation in the Kirksville laboratories.

SUMMARY

1. Some of the neural mechanisms involved in the osteopathic lesion, in its local and distant effects, and in manipulative therapy are examined.
2. The results of experimental investigations are presented which indicate that the lesion is associated with a segment of the spinal cord which is hyper-excitable to all impulses which reach it, and that the hyperexcitability may extend to any

neurons having their cell bodies in that segment.

3. It is concluded that osteopathic lesion represents a facilitated segment of the spinal cord maintained in that state by impulses of endogenous origin entering the corresponding dorsal root. All structures receiving efferent nerve fibers from that segment are, therefore, potentially exposed to excessive excitation or inhibition.

4. Evidence is presented that the stretch and tension end-organs (proprioceptors) in the muscles and tendons are the most important source of afferent impulses which produce the changes in the cord that are associated with the osteopathic lesion.

5. The effect of the lesion as a predisposing and localizing factor is emphasized and discussed in relation to certain aspects of osteopathic practice.

REFERENCES

1. Denslow, J.S.: An analysis of the variability of spinal reflex thresholds. *J. Neurophysiol.* 7:207-215, July 1944.
2. Denslow, J.S.; Korr, I.M. and Kreams, A.D.: Quantitative studies of chronic facilitation in human motoneuron pools. *Am. J. Physiol.* 105:229-238, Aug. 1947.
3. Lloyd, D.P.C.: Intercellular transmission in Howell's Textbook of physiology, edited by J. F. Fulton. Ed. 15. W. B. Saunders Co., Philadelphia, 1946, p. 134.
4. Sherrington, C.S.: Correlation of reflexes and the principle of the common path. *Brit. Assoc. Rep.* 74:728-741, Aug.18, 1904; abstr., *Brit. M. J.* 2:443 Aug. 27, 1904.
5. Lorente de Nó, R.: Synaptic stimulation of motoneurons as a local process. *J. Neurophysiol.* 1:195-206, May 1938.
6. Lloyd, D.P.C.: Reflex action in relation to the pattern and peripheral source of afferent stimulation. *J. Neurophysiol.* 6:111-119, March 1943.