Proprioceptors and somatic dysfunction*

IRVIN M. KORR, PH.D.
Kirksville, Missouri

Functional properties of osteopathic lesions, as clinically described, have been reviewed in relation to the physiology of proprioceptors. It is shown that muscle spindles in which the "gain" has been turned up by intensified activity in their gamma motor innervation may, together with other sensory inputs, account for many of the motion characteristics and palpatory features of the osteopathic lesion. "Turning down" of the gain seems to be a common denominator in a variety of osteopathic manipulative procedures. Possible origin of the high gain is discussed also.

The musculoskeletal system is the most massive system of the body, yet in the performance of its infinite repertoire of motions and postures, it is the most delicately controlled and coordinated. Accordingly, the musculoskeletal system is the recipient of most of the efferent outflow from the central nervous system (CNS), with the largest portion by far going via the ventral roots of the spinal cord to the muscles, which carry out the motor commands of the CNS.

It is less well appreciated, however, that for related reasons the musculoskeletal system is also the *source* of the preponderant sensory input to the CNS, an input that is also the most widespread, the most continuous, and the most variable. This sensory feedback, from countless thousands of reporting stations in myofascial and articular components, entering the cord via the dorsal roots, is essential to the moment-to-moment control and fine adjustment of posture and locomotion.

In addition to this influence on the motor pathways, the sensory reporting is selectively routed to various other centers throughout the nervous system, including, of course, the cerebral cortex, where it enters into consciousness and the ordering of volitional motor activity. Relevant portions of the reports also reach and are utilized by the autonomic nervous system in the tuning of visceral, circulatory, and metabolic activity to musculoskeletal demand. Indeed, the sensory input from the musculoskeletal system is so extensive, intensive, and unceasing as to be a dominant influence on the CNS and therefore the person as a whole.

It may be expected, therefore, that disturbances in the sensory input from the musculoskeletal system, whether generally or locally, would significantly impair not only motor function, but also other functions — and that of the person himself. For those engaged in the study of the neural and reflex mechanisms, that premise is at the heart of the clinical significance of the osteopathic lesion — now modishly and euphemistically designated as "somatic dysfunction." One of the first products of experimental research into those mechanisms, pioneered by Denslow,[2-4] was the concept of chronic segmental facilitation. In 1947, the hypothesis was stated[5] as follows:

> (An) osteopathic lesion represents a facilitiated 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.

In speculating further about the site of the "endogenous origin," the author suggested that the proprioceptors, particularly the muscle spindles, were the most likely candidates because: 1) they would be sensitive to musculoskeletal stresses; 2) they are nonadapting receptors, sustaining streams

*Based on a lecture given at the 71st Clinical Conference of the New York Academy of Osteopathy, in conjunction with the Postgraduate Institute of Osteopathic Medicine and Surgery, January 26, 1974. An earlier version appeared in *Osteopathic Annals*[1] in August 1974.

of impulses for as long as they are mechanically stimulated; and 3) their influence is highly specific to the muscles acting on the affected joints and the corresponding spinal segments.

In the intervening 28 years, research in many neurophysiologic laboratories has immensely increased our understanding of the proprioceptors. Concurrently, research under osteopathic auspices (reviewed by various authors[5-11]) has substantially increased our understanding of the mechanisms involved in somatic dysfunction. This paper is an effort to determine what importance may still be ascribed to the proprioceptors in the origin of segmental facilitation (the clinical significance of which has also been explored [5-11]). It is shown that there is now even stronger reason to view the proprioceptors, and most particularly muscle spindles, as key elements in the "neural basis of the osteopathic lesion." A new theory is offered about the neural mechanisms operating in the osteopathic lesion and about their relation to osteopathic manipulative therapy.

The palpatory criteria for identifying and evaluating the musculoskeletal disorders that are designated "osteopathic lesions" have been described and taught in many different ways. Physicians differ in the ways that they use these criteria in diagnosis and as guides to therapy. However, there seems to be general agreement on the importance of at least one feature, decreased mobility — reduced range or ease of joint motion in one or more planes — and on the importance of restoring mobility. It also seems to be generally assumed that the resistance to motion is within the joint itself, ascribable to articular friction or to the visco-elastic properties of ligamentous structures. This assumption needs to be reexamined. It has, however, been so deeply implicit in osteopathic thinking that it is seldom verbalized, much less questioned, for several reasons. First, the view of the *osteo*pathic lesion as a "bony," "structural," intervertebral, or articular derangement has such venerated origins as to border on dogma. This traditional view is reinforced daily by anatomically-worded descriptions that imply displacements and altered interosseous relationships, even when such descriptions are accompanied by protestations that the osteopathic lesion is, of course, a *functional* disturbance, and not a "bone out of place." Second, the resistance to motion and reduced range of motion, whatever their origin, *are* manifest in reduced joint mobility. Third, in manipulation the vertebrae or other bones are commonly the levers to which the manual forces are applied, and effectiveness of treatment is reflected in their improved mobility.

The braking power of muscle

To a physiologist, it seems much more reasonable that the limitation and resistance to motion of a joint that characterize an osteopathic lesion do not ordinarily arise within the joint, but are imposed by one or more of the muscles that traverse and move the joint. Of all the somatic tissues (for example, vertebral and paravertebral), muscle is the only active one, the one capable of self-energized, independent motion and of developing great, widely variable, and rapidly changing forces. The other tissues are passively moved, immobilized, pushed, pulled, compressed, and altered in shape by forces external to themselves — those of muscular origin and those external to the body, such as gravity.

While usually thinking of muscles as the motors of the body, producing motion by their contraction, it is important to remember that the same contractile forces are also utilized to *oppose* motion. By the application of controlled counteracting forces, contracting muscle absorbs momentum (for example, of a swinging limb) and regulates, resists, retards, and arrests motion. Indeed, the energy-*absorbing* function of skeletal muscle is no less important to the control of motion than its energy-*imparting* function. Both are based on the same cellular mechanisms — those involved in contraction.

Valuable and quantitative insights into this aspect

of muscular function, as it relates to the "behavior" of lesioned segments, have come from observations reported by osteopathic physicians who are skilled in so-called "functional technique." While, as in other manipulative approaches, mobility is to them an important criterion, the emphasis in this approach is not on *range* of externally imposed ("passive") motion, but on *ease* of initiation of active, patient-engendered motion. In this form of manipulation, the fingers of the palpating hand are placed on tissues of the segment under examination, while the other hand signals and guides the patient through various motions in which that vertebral segment participates.

Using finger-tip criteria of "ease" and "bind," the physicians describe what seems like exponentially changing resistance (bind) to motion around one axis or another in the lesioned segment. That is, on initiation of motion, there is a rapid rise in resistance in one direction and accelerating collapse along the opposite. In contrast, the nonlesioned segment moves relatively freely in all directions anatomically appropriate to the joint or joints, offering only linearly changing resistance. Hoover,[12] Bowles,[13] and Johnston[14-16] have developed cybernetic approaches to osteopathic palpatory diagnosis and manipulative therapy that have opened new lines of inquiry, including the present one. I am grateful to them for their many provocative "personal communications."

Although these authors have cautiously avoided ascribing the changing resistance in the lesioned segments to any particular tissue, I am convinced that their reports are consistent with the hypothesis that "bind" is the active opposition or physiologic "protest" of muscle to the motion in a particular direction, and "ease" is increased cooperation and compliance in the other. Indeed, the development of resistance is accompanied by a sensation of "bunching-up" under the palpating fingers similar to that when muscles in the extremities are voluntarily contracted.

It is therefore proposed as a premise for this paper that it is in its capacity as a brake that a muscle may become the major, and highly variable, impediment to mobility of the lesioned "joint," whether the motion is produced by external forces or by other muscles. Muscular resistance is not based on inextensibility, as might be expected of tough connective tissues, but on changes in the degree of activation and deactivation of the contractile mechanism.

What would cause a muscle to behave in that manner — increasing or decreasing its contraction (and braking power) according to direction of motion of the joint? First, the amount of contraction from moment to moment is controlled by variations in impulse traffic along the motor axons supplying the muscle. Second, the impulse traffic varies with changing levels of excitation of the anterior horn cells, which, third, are in accordance with changing afferent input during the joint motion. What are the sources of the changing afferent bombardment during the joint motion?

Proprioceptors

The proprioceptors are the sensory end organs to look to for an answer to this question, since it is they that signal physical changes in the musculoskeletal tissues. The three main categories of proprioceptors are those related to joint position and motion, to tendon tension, and to muscle length.

Joint receptors

Endings located in and around joints (for example, in capsules and ligaments) report joint motion, position, and, possibly, force. The Ruffini endings, especially, strategically distributed in the capsules, report direction and velocity of motion and position very accurately. There is little or no evidence, however, that these joint receptors have any direct influence on motor activity through segmental pathways. They certainly do not exert a dominant, selective influence on individual muscles. Their collective influence is on postural and locomotor patterns

through the higher centers, including the cerebellum and the cerebral cortex.

Attention is directed, therefore, to receptors more directly related to muscular contraction: those in the tendons reporting changes in tension and those in muscle itself reporting changes in length. Our current knowledge of the structure and function of these receptors has been admirably reviewed by Houk and Henneman.[17,18]

Golgi tendon receptors

The Golgi endings are located in tendons close to the musculotendinous junction (Fig. 1, tendon and afferent pathway T). Pulling on the tendon distorts these endings, causing discharge of impulses into the spinal cord via afferent fibers. The tension to which these endings are sensitive is under physiologic conditions usually exerted by active contraction of the muscle itself. Lying in tough tendon, which is in series with muscle and relatively unyielding, the tendon endings are responsive, not to changes in length but to changes in force. Change in length occurs mainly in the much more compliant and actively shortening muscle. The tendon endings remain silent or nearly silent when a muscle shortens without developing much tension, but when the muscle contracts against a load or fixed object or against the contraction of antagonistic muscles, the discharge of the tendon endings is in proportion to the tension that is developed. That is, the afferent input varies with the tension exerted by the muscle on the tendon, regardless of the muscle length.

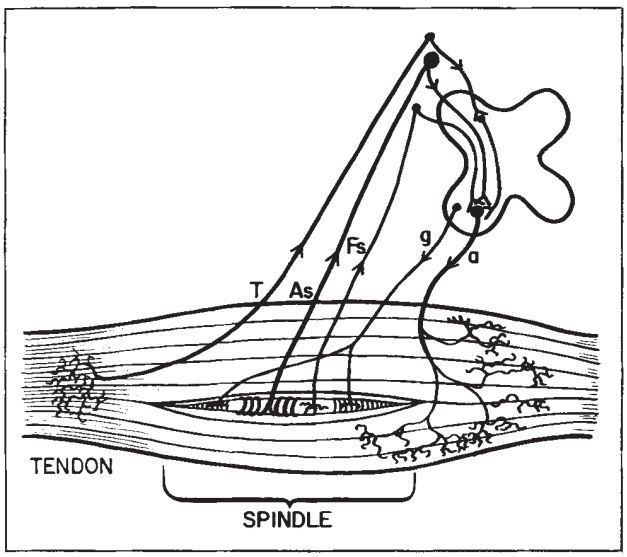

Fig. 1. *Innervation of skeletal muscle.*
Motor: a – alpha motoneuron to main (extrafusal) muscle fibers; g – gamma motoneuron to intrafusal muscle fibers.
Sensory: T – neuron conveying impulses from Golgi endings in tendon; As – neuron conveying impulses from annulospiral (primary) ending in spindle; Fs – neuron conveying impulses from flower-spray (secondary) endings in spindle.
(Adapted from Buzzell.[7])

The discharges of the tendon endings are conveyed to the spinal cord by dorsal root fibers (Fig. 1,T), where they excite *inhibitory* interneurons that synapse with motoneurons controlling the same muscle. The effect of their discharge therefore is inhibitory, tending to oppose the further development of tension by the muscle, that is, to produce relaxation. This is quite opposite to the "behavior" at the lesioned segment described above.

Muscle spindles

Muscle spindles are much more complex than the tendon receptors. Each spindle has two kinds of sensory endings, each with different central connections and reflex influences, and a muscular component with its own motor innervation. Spindles are scattered throughout each muscle in numbers that vary with the function of the muscle and the delicacy of its control. The greater the spindle density, the finer the control.

Only those details of structure and function that are essential to the purpose of this essay will be reviewed. The reader is referred to other sources for further details, such as Houk and Henneman[17,18] or recent editions of other textbooks in medical physiology.

Structure-function of the muscle spindle

Unlike the tendon endings, spindles are within the muscle itself and surrounded by muscle fibers, arranged in parallel with them and attached to them at both ends. The essential features are diagrammed in Fig. 1. Clearly, stretching the muscle also stretches the spindle, and shortening of the muscle slackens the spindle.

Each spindle, enclosed in a connective tissue sheath, and about 3 mm. long, has several thin muscle fibers. They are identified as intrafusal fibers to distinguish them from the much larger and more powerful (extrafusal) fibers that comprise the bulk of the muscle. The intrafusal fibers are attached to the sheath at each end. They pass through an expanded lymph space in the middle of the spindle. This portion of each fiber is rather densely nucleated and is only feebly contractile, if at all.

The intrafusal muscle fibers are innervated by gamma motor fibers originating in the ventral horn and passing through the ventral root (Fig. 1,g). In contrast to the alpha motor neurons supplying the extrafusal muscle fibers (Fig. 1,a), the gamma, also known as "fusimotor," neurons are small in size and their axons quite thin. The importance of the fusimotor innervation is indicated by the fact that the gamma fibers comprise one-third of the ventral root outflow.

The sensory endings of the spindle are in close relation to the equatorial (nucleated, noncontractile) portion of the intrafusal fibers. The so-called primary ending is wound around the fibers and is described as the annulospiral ending (Fig. 1,As). Secondary, flower-spray endings occur on either side of the primary ending and are connected to thinner myelinated axons (Fig. 1,Fs). Both are sensitive to stretch of the central portion of the spindle.

Sensory endings

Figs. 2 A-C illustrate diagrammatically how the primary endings respond to change in muscle length. When the muscle is stretched (Fig. 2B) beyond its resting length (2A), the spindle is also stretched, causing the primary and secondary endings to fire at increased frequencies in proportion to the degree of stretch. Shortening of the muscle (Fig. 2C), whether by its own contraction or by passive approximation of its attachments, slows the discharge proportionately, and may even silence it. For the purpose of this paper, discussion will be limited to the primary ending, about which much more is known than about the secondary ending.†

The spindle is an essential feedback mechanism by which the system that is controlled, in this case skeletal muscle, continually reports back to the controller, the central nervous system (CNS). The feed-

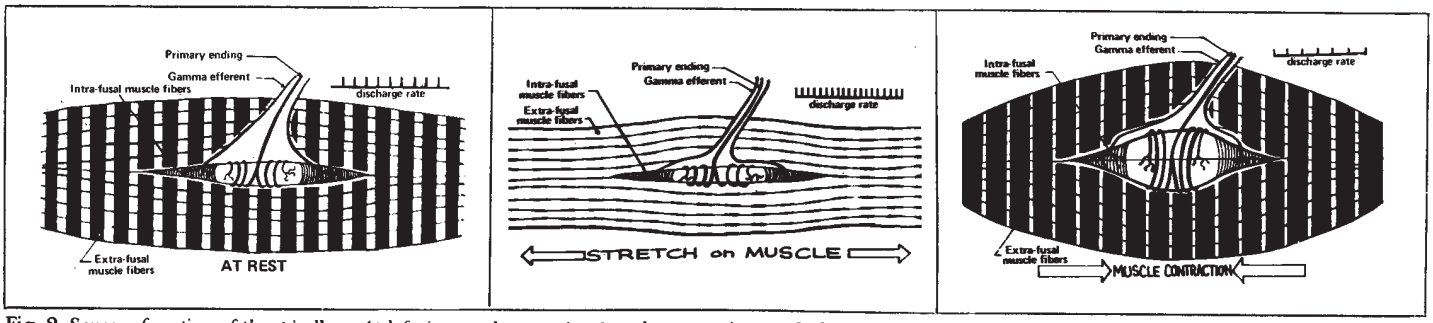

Fig. 2. *Sensory function of the spindle. At left, in muscle at resting length; center, in stretched muscle; at right, in contracting muscle. Relative impulse frequency is shown in the upper right-hand corner of each diagram.* (From Buzzell.[7])

back from the primary endings of each spindle is conveyed by dorsal root fiber directly, that is, monosynaptically, to the motoneurons of the same muscle. There is considerable evidence that the feedback may be even more precisely localized than that — to the motoneurons controlling the muscle fibers in the immediate vicinity of the spindle. This would provide for a high degree of precision and specificity of reflex regulation.

The influence of the afferent discharge of the spindle on the motoneurons of the same muscle is excitatory. That is, when a muscle is stretched it is reflexly stimulated by its spindles to contract, and thereby to resist stretching. Conversely, shortening of the muscle decreases the afferent discharge, reduces the excitation of the motoneurons, thus favoring relaxation (that is, lengthening) of the muscle. The influence of the muscle spindle, therefore, is to cause the muscle to resist change in length in either direction.

The spindle is thus the sensory component of the familiar stretch, or myotatic, reflex. It is an extremely important mechanism in the maintenance of posture, since it causes the extensor and elevator muscles, which tend to be stretched under gravitational influence, to contract against the force of gravity in a smoothly regulated manner. The same mechanism operates in the misnamed "tendon reflexes" of clinical practice. The tap on the tendon momentarily stretches the muscle, exciting the spindles, which in turn excite a contractile response.

Through collaterals and interneurons spindles also influence the activity of muscles other than those in which they are located, such as antagonists and synergists, but these influences and the polyneuronal pathways that are involved are not essential to this discussion.

Intrafusal muscle fibers

How do the intrafusal muscle fibers influence spindle discharge? Since their ends are firmly anchored, contraction of these fibers stretches the middle portion in which the sensory endings are situated, increasing their discharge. The effect of intrafusal contraction on the endings — and their response — is indistinguishable from that produced by stretch of the extrafusal fibers, and the two effects are additive. That is, at any muscle length, intrafusal contraction would increase the spindle discharge, as would an increase in muscle length; stretch of the muscle while the intrafusal fibers are contracted produces a more intense spindle discharge than when the intrafusal fibers are at rest or less contracted.

Gamma motoneurons

The function of the gamma neurons, in turn, is to control contraction of the intrafusal fibers, and, through them, the frequency of the spindle discharge at a given muscle length, and the change in that frequency per millimeter change in length (sensitivity). The higher the gamma activity, the larger the spindle response. Fig. 3 shows the relationship of afferent impulse frequency to muscle length at different levels of gamma neuron activity. Thus, the higher the gamma neuron activity, the higher the spindle discharge at a given muscle length (vertical dashed line) and the shorter the length of muscle at which a given impulse frequency is generated (horizontal dashed line).

Relation of alpha-to-gamma and extrafusal-to-intrafusal

The importance of the foregoing information, at least for the purposes of this paper, is in relation to the regulation of the activity of skeletal muscles. The key fact is that the higher the spindle discharge, the

†One distinction may be worth mentioning. Although discharges of both types of endings are more or less proportional to length, the primary (annulospiral) ending has the additional feature that its frequency of firing *during* a stretch is in proportion to the *rate* of change. That is, the secondary ending apparently reports length at any moment, but the primary ending reports both velocity of stretch (and hence of joint motion) and length (hence joint position). The primary ending, thereby, provides a predictive or anticipatory input to the nervous system. This refinement will not, however, be included in the discussion.

greater the reflex contraction of the muscle. What that contraction accomplishes depends on the other forces acting on the joints crossed by that muscle. But, as a generalization, the greater the contraction, the more the muscle tends to shorten and move the joint, and the more it resists being stretched by movement of the joint in the opposite direction.

Under normal resting conditions, the gamma activity is apparently such as to sustain a tonic afferent discharge from the spindle. This maintains the alpha motoneurons in a moderately facilitated state — a state of readiness — and the muscles in low-grade tonic contraction at their resting lengths. Gamma activity may be turned up or down from this basal level. The higher the gamma activity, because of its influence on the excitatory spindle discharge, the more forceful the muscle's contraction and the greater its resistance to being lengthened. During high gamma activity, the spindle may, in effect, be calling for contraction when the muscle is already shorter than its resting length.

It may be helpful to view spindle function in relation to muscular activity in still another way. Since the sensory endings of the spindle are stimulated by mechanical distortion, whether caused by contraction of the intrafusal fibers or by stretch of the main muscle (or both), the spindle in effect reports not absolute length of extrafusal fibers, but length *relative* to that of the intrafusal fibers. The greater the disparity, however it is produced, the greater the discharge and the greater the contraction of the muscle. In other words, the very small intrafusal fibers, inside the scattered spindles, seem to serve as variable standards of comparison against which the main muscle is continually measured and adjusted; variations of the standard are under gamma control.

An increase in intrafusal-extrafusal disparity increases the afferent discharge, which elicits a contractile response of the extrafusal fibers, which in turn tends to nullify the disparity and to silence the spindle. The greater the gamma activity, the more the muscle must shorten before the spindle is turned back down to tonic, resting discharge. Thus, the CNS can elicit, and precisely control, the contraction and relaxation of (alpha-innervated) muscle through its gamma-mediated control of the several muscle fibers in each spindle. That is, the gamma neurons, in controlling the milligram forces and micron contractions of a few minute fibers, indirectly regulate the kilogram forces and centimeter contractions of massive skeletal muscle. From this viewpoint it becomes clear why the "gamma loop" is often viewed as a high-gain servomechanism, and the gamma neurons as the gain-control components of the system.

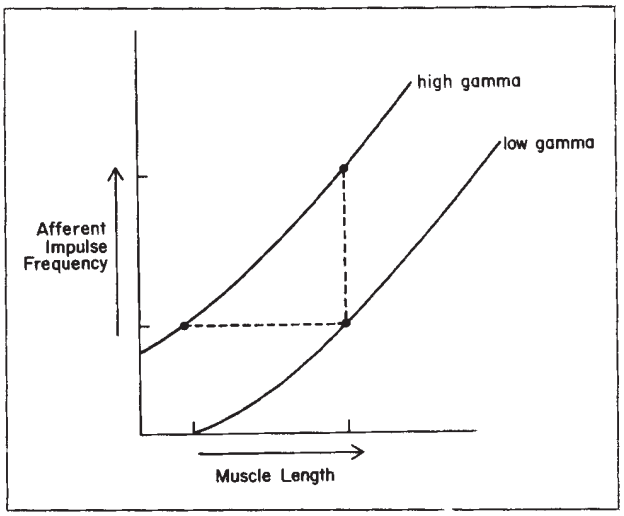

Fig. 3. *Influence of muscle length on spindle impulse frequency at two different levels of gamma motoneuron activity. See text.*

The gamma system in normal life

It is convenient to begin discussion of the role of the gamma system in locomotion and posture by describing what happens when a muscle supporting a moderate load is stimulated to contract briefly. As described earlier, the spindle is slackened during the shortening of the muscle, and the spindle discharge is reduced — even silenced. This is diagrammatically

represented in Fig. 4 (M and S). For contrast, the simultaneous change in the (inhibitory) feedback from the tendon (T), reporting the tension developed by the muscle during its shortening, is also shown. The combined reports from these two sources keep the CNS continually apprised of tension-length, that is, load-motion changes.

When the spindle is silent, the CNS is of course deprived of important information. This may not be a serious loss for a brief twitch, such as that indicated in Fig. 4. When, however, the muscle is called on to carry out well-controlled motion while it remains in a shortened state (as, for example, the biceps brachialis with the elbow sharply bent), then the loss of that information could be a serious one, and could even be disabling. As a matter of fact, there would be a serious impairment *in the course* of shortening, since spindle sensitivity to length-change would progressively decrease as the spindle slackens.

What device does the CNS have for ensuring that the spindle remains reliably operative throughout all length changes of the muscle? A clue is offered by Fig. 5, which shows, in a hypothetical experiment, spindle discharge in several sets of circumstances: at resting length and at two degrees of stretch (X and 2X), at rest and during a twitch of the muscle, and each of these with and without gamma discharge. The discharge is increased by stretch and by gamma activity; it is reduced by shortening of the muscle, for which compensation can be made by appropriate intrafusal contraction under gamma control.

This is precisely the mechanism that the CNS uses: It calls for adjustive tightening and slackening of the spindle by changing the gamma discharges to the intrafusal fibers. Evidently, as a muscle shortens in response to impulses in the alpha motoneurons, parallel volleys of impulses may be dispatched through the gamma neurons to stimulate the intrafusal fibers to contract and take up the slack as it develops. Conversely, the intrafusal fibers are permitted to lengthen as the extrafusal muscle fibers relax and lengthen under alpha direction. In this way, by appropriately varying the intrafusal length-standard, the response of the spindle to each millimeter change in length, and, therefore, the "gain" of the entire mechanism, can be kept relatively constant as a muscle lengthens and shortens.

Through the same mechanism, the CNS retains the option, however, to *vary* spindle sensitivity and gain in accordance with the kinds of motion that are being called for, and in accordance with other circumstances. Gamma activity, and hence gain, may even be preset according to the length changes that are called for or anticipated in a given motion. Through its control of gamma activity, the CNS can set the limits — the maximum lengthening that will be acceptable and the degree of shortening at which the spindle would, so to speak, be unloaded and its discharge turned back to basal tone.

This continual setting, resetting, and presetting of intrafusal fiber-length through the gamma neurons may be viewed as "automatic gain control" of the length-regulating mechanism for each muscle. Gain is continually being adjusted in accordance with the motions and the positions that are being volitionally and reflexly called for. For example, when a tennis player prepares to return a ball with a fast forehand stroke, the gamma activity is turned down (low gain), thus permitting large changes in muscle length during the preparatory backswing and the forward swing. On the other hand, when the player is at the net and wishes minimum motion of his racket for a short volley shot, the gamma activity would be turned up (high gain) to narrow the range of length changes that would be reflexly permitted. Similarly with the violinist, who uses the full length of the bow in a legato passage, and then uses but a half-inch or so for a staccato passage. Or the golfer trying for a long drive, and then a short chip-shot. Of course, gain-settings are subject to continual changes *in the course* of each motion. Also, gain control is individually exercised for each participating muscle according to its role from moment to moment.

The cerebral influences illustrated above in vari-

ous voluntary activities may, however, also be maladaptive, setting the spindle sensitivity and gain inappropriately for regulation of muscular activity. In tension and anxiety states or in situations that are (or are perceived to be) threatening, gamma activity may be set too high for efficient, smoothly coordinated motion. In these states the muscles are tense, stiff, resistant to change in length. The individual is said to be "jumpy" and "spastic," and he tends to move in a staccato manner. His "tendon" reflexes are proportionately exaggerated. Grainger[19] has ingeniously shown how incorrect anticipation of the muscular effort required, for example, to lift an object, may cause the gain to be set too high, with serious and painful consequences. His illustrated article offers many valuable insights into the origin of back problems, and to the mechanisms of their manipulative amelioration.

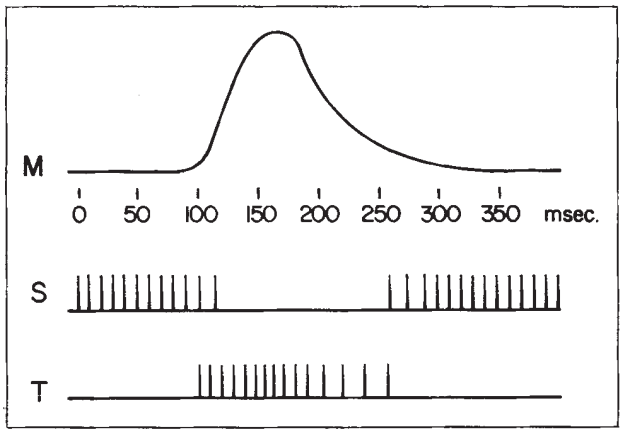

Fig. 4. *Changes in spindle discharge (S) and tendon ending discharge (T) during a brief contraction of the muscle (M).*

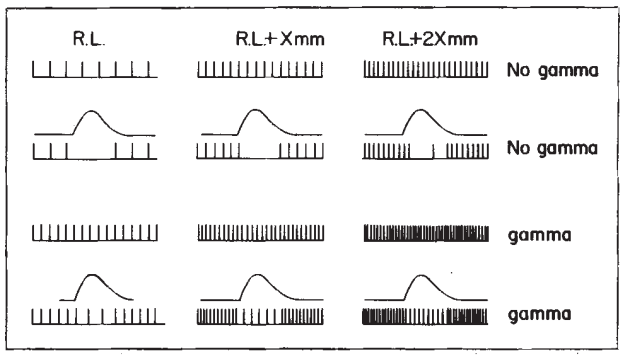

Fig. 5. *Individual and combined influences of muscle length (resting length [R.L.] and two degrees of stretch), gamma neuron activity, and muscle contraction on spindle discharge.*

The spindle and somatic dysfunction

How may this information relate to musculoskeletal disturbances designated as osteopathic lesions? I propose as a hypothesis that in the lesioned area the "gain" has been turned up in the spindles of one or more of the muscles. In other words, according to this concept the discharges of the gamma motoneurons of the related spinal segments are sustained at high frequencies, keeping the intrafusal fibers in a chronically shortened state in which the discharge frequencies of the spindles, and in which their frequency change per millimeter, are exaggerated.

How could this have been brought about? The concurrence of the following two circumstances could, I believe, instigate the high-frequency gamma firing: 1) strong centrally ordered contraction during a moment 2) when the muscular attachments (for example, on two vertebrae) have been closely and abruptly approximated by forces or factors that have *not* been centrally ordered. The abrupt approximation of the attachments, with equally abrupt and unanticipated slackening of the muscle, could be brought about either by an external force or impact or by unanticipated yielding of a load or force opposing a strong isometric contraction. In the suddenly slackened state the spindles would be silenced equally suddenly. In calling (or continuing to call) on the slackened, silent muscle for strong contraction via the alpha motoneurons, the CNS, receiving no feedback, would also greatly increase the gamma discharges to the intrafusal fi-

bers until the spindles resumed their reporting.

On recoil (or reflex recovery) of the body from the forced motion, return of the attachments (for example, the vertebrae) to their resting relationship would be opposed — but not necessarily prevented — by the now (reflexly) resistant muscle. Under the influence of gravitational forces, antagonists, and postural reflexes, which would be tending to stretch the muscle back toward resting length, the spindle would be continually discharging and, through the CNS, ordering the muscle to resist. The more the stretch, the *much* more the resistance. It should be remembered that the more the resistance, that is, contractile tension, the more the joint surfaces would be pressed together and their frictional resistance increased.

There would be little value, at this stage of development of the hypothesis, in cataloguing the many kinds of circumstances, minor "accidents," and microtraumas of daily life in which these two factors — strong contraction and slack-produced spindle-silence — could conspire to turn up the spindle gain. The thoughtful reader can doubtless envisage or reconstruct, from personal and clinical experience, many such circumstances. The hypothesis that is proposed about the osteopathic lesion is primarily concerned with the high spindle gain. Further speculation as to how it may come about is therefore deferred until the primary hypothesis has proved viable.

I turn, therefore, to a few illustrations of the manner in which high-gain spindle function may help explain: a) some functional characteristics of osteopathic lesions and b) the efficacy of certain manipulative procedures. These illustrations are offered as guidelines for testing the theory in practice.

a) How the spindle may be related to certain lesion characteristics.

1. The high-gain hypothesis is consistent with, and offers an explanation for, the steeply rising resistance to motion ("bind") in one direction and the equally precipitous collapse of resistance (increasing "ease") in the opposite direction.[12-16] Since, for reasons given above, the affected muscles would even in resting posture be under some degree of stretch, they would be on the tense side of "easy neutral," that is, in continual active contraction and palpably hard and unyielding. They would also be provoked into stronger and stronger contraction by the exaggerated spindle discharges as motions that tend to lengthen the affected muscles occur.

2. To the degree that spindles reflexly regulate the contraction of muscle fibers in their immediate vicinity, the hypothesis would also explain the "ropiness" often found in muscles in stressed areas. High sensitivity of selected spindles in a muscle would produce spasm in correspondingly selected fascicles, which would feel like tight cords in the muscle.

3. Since the joints that are crossed by the affected muscle are compressed, with their surfaces tightly apposed, they too would appear "stiff" and difficult to "gap." It would be expected that such joints would be much more likely to "pop" when forcefully gapped than those not compressed by muscular forces.

4. The high-gain spindle may also contribute to the "catch" that is sometimes encountered when muscles have been caused (or permitted) to shorten far below their natural resting length. However, in the markedly shortened state, the sensory and reflex mechanism may be complicated by a change in the contractile state — one in which the muscle may have lost the ability to relax, as in contracture.

5. Bailey[20] has suggested in response to this hypothesis that failure by the CNS to turn up the gamma discharges when the spindle has been silenced by marked shortening of the muscle may result in nonreporting spindles. This, she suggests, may account for "dead" segments in which muscles have become markedly *un*responsive to changes in length.

b) Is spindle-gain re-set by effective manipulative procedures?

Since, according to the hypothesis that has been proposed, certain functional aberrations of lesioned segments, notably impairment of mobility, may be ascribable to gamma-induced intrafusal contraction, it is proposed as a corollary that reduction of gamma discharge may be a key element in the restoration of mobility and therefore in the efficacy of manipulative therpay. Following are a few "tests" of this suggestion.

1. According to the hypothesis, motions that in functionally oriented manipulative technic tend to favor "ease" are those that approximate the attachments of the affected muscles, reducing their tension and permitting them to shorten. As the motion continues in the direction of ease, intrafusal-extrafusal disparity narrows. That is, the relative length of the muscle once again begins to correspond more closely to that of the intrafusal fibers. The shortened spindle nevertheless continues to fire, despite the slackening of the main muscle, and the CNS is gradually enabled to turn down the gamma discharge, and, in turn, enables the muscle to return to "easy neutral" at its resting length. In effect, the physician has led the patient through a repetition of the lesioning process, with, however, two essential differences: first, it is done in slow motion with gentle muscular forces, and, second, there have been no "surprises" for the CNS; the spindle has continued to report throughout.

Presumably, the compression of the affected joints would also be relieved by such procedures.

2. A variety of manipulative procedures involve, or seem to have as one of their implicit objectives, the stretching of the hypertonic muscles in the lesioned segments. It is proposed that two mechanisms, operating either individually or conjointly in these procedures, may contribute to the return of the spindles to more normal gain-settings, with resultant relaxation of the muscles: i) Stretch of the intrafusal fibers. Forceful stretch of the muscle against its spindle-maintained resistance is, of course, mechanically transmitted to the spindle. This would produce a barrage of afferent impulses of such high frequency as, conceivably, to signal the CNS to turn down the gamma discharge. ii) Forced stretch of the muscle would, of course, also be transmitted to its tendon, causing intense discharge by the Golgi endings. It is thought that the inhibitory influence of this afferent input extends to the gamma, as well as the alpha, motoneurons, contributing to relaxation of both the intrafusal and extrafusal fibers.

These mechanisms would seem to operate in such muscle-stretching procedures as "taking joints through their full range of motion" in "springing," and in slowly applying, slowly releasing manual pressure transversely to the long axis of a muscle, for example, the spinal extensors. They may also be implicated in the manipulative procedures involving high-velocity, short-amplitude forces. In these procedures, the affected muscles are stretched against their resistance, by appropriate positioning of the patient, before the thrust is applied (often accompanied by a popping sound as the seal of the compressed joint is broken). The gapping of the joint has probably added a further increment of length to the already stretched muscle (and contained spindles) and of tension to the tendon. "Release" may also be obtained, without abruptly applied forces, by maintaining tension on the hypertonic muscles while they "let go," presumably in response to subsiding spindle bombardment.

3. The same mechanisms (stretch of the intrafusal fibers and Golgi discharge) seem to be operating in those manipulative technics in which, under the physician's guidance, the *patient* applies the "corrective force" by active muscle contraction. For example, the patient may be instructed to push or pull against opposing force applied by the physician. In this procedure, the patient is in effect contracting the tense muscles isometrically, the procedure being repeated at progressively increasing lengths. With each isometric contraction, high tension is developed in the tendons, and at each new

muscle length the spindle is also stretched, both factors contributing to resetting of spindle-gain to normal levels.

Apparently similar results may be obtained by eliciting isometric contractions, not of the hypertonic muscles, but of their antagonists. In this procedure, the physician is, of course, utilizing the principle of reciprocal innervation. The inhibitory influence of this mechanism, like that of the tendon receptors, may also be expected to affect the gamma as well as the alpha motoneurons.

Concluding comments

In proposing this rather large place for the muscle spindle in the "neural basis of the osteopathic lesion,"[5] there is no intention of excluding or underemphasizing the roles of other sensory inputs or reflex mechanisms. Reciprocal innervation and the Golgi tendon endings have already been implicated in conjunction with the spindle. Almost certainly involved under various circumstances are impulses from a variety of receptors and pain endings in and around joint structures, ligaments, tendons, fascia, skin, and viscera; muscle receptors other than the spindle may also be involved. It should be pointed out that, whatever their other influences, any of these may directly or indirectly, excite or inhibit gamma motoneuron activity.

Nor is it intended to imply that the spindle is *the* source of facilitation of segments of the spinal cord associated with osteopathic lesions. On the contrary, it is becoming increasingly evident that the spinal cord does not individually "read" and respond to discrete reports from this or that set of receptors. The cord seems, rather, to deal with total *patterns* collectively presented to it by inputs from many reporting stations.

The spinal cord seems to become agitated (facilitated?) when the reports from two or more stations are conflicting and the patterns, therefore, unintelligible (as also happens to the higher centers, with distressing effects, in motion sickness). Spindles to which gamma discharges have been increased may significantly contribute to "jamming" and "garbling" of the input patterns. The high-gain spindles would, for example, falsely report to the spinal cord that their muscle, actually in a shortened contracted state, was stretched to nearly its maximum length. This would signify to the cord that the muscle's vertebral attachments were widely separated, when actually, as *correctly* reported by the joint receptors, they are closely approximated. There obviously can be no appropriate reflex response to irreconcilable reports signifying that the joint is, say, simultaneously flexed and extended. Segmental "facilitation" is beginning to appear more like a state of segmental "consternation." A segment in such a state is to some degree disruptive to every total activity pattern in which it participates. Effective manipulation is that which restores it to concordant function — to "tracking" with its neighbors.[21]

Mention should be made of a factor that would contribute to *sustaining* the exaggerated spindle discharges in muscles of the lesioned segments. One feature of somatic dysfunction that has received emphasis in recent years, as a result of research in the Kirksville laboratories, is local sympathetic hyperactivity (summarized by Korr[6] and Korr, Buzzell, and Hix[7]). One of the effects of sympathetic hyperactivity is increased afferent discharge from the spindles.[22] Insofar as spindle input influences sympathetic preganglionic neurons in the spinal cord, a self-sustaining vicious circle may be envisaged.

The hypothesis says only that the "lesioned" segment *behaves* as though gamma motoneuron activity ("gain") in that segment has been turned up. In presenting this hypothesis, I hope, whether or not it turns out to be valid, that it stimulates testing and inquiry in clinical practice and in the laboratory, leading to new insights, sounder theory, and more efficacious practice.

1. Korr, I.M.: Proprioceptors and the behavior of lesioned segments. Osteopathic Annals 2:12-32, Aug 74

2. Denslow, J.S.: An analysis of the variability of spinal reflex thresholds. J Neurophysiol 7:207-15, Jul 44

3. Denslow, J.S.: An analysis of the irritability of spinal reflex arcs. JAOA 44:357-62, Apr 45

4. Denslow, J.S., Korr, I.M., and Krems, A.D.: Quantitative studies of chronic facilitiation in human motoneuron pools. Amer J Physiol 150:229-38, Aug 47

5. Korr, I.M.: The neural basis of the osteopathic lesion. JAOA 47:191-8, Dec 47

6. Korr, I.M., Thomas, P.E., and Wright, H.M.: Symposium on the functional implications of segmental facilitation. JAOA 54:265-82, Jan 55

7. Korr, I.M., Buzzell, K.A., and Hix, E.L.: Symposium on the physiological basis of osteopathic medicine. G.W. Northup, Moderator. Postgraduate Institute of Osteopathic Medicine and Surgery, New York, 1970

8. Denslow, J.S.: Neural basis of the somatic component in health and disease and its clinical management. JAOA 72:149-56, Oct 72

9. Patterson, M.M.: Spinal responses: Static or dynamic? JAOA 72:156-63, Oct 72

10. Korr, I.M.: The trophic functions of nerves and their mechanisms. JAOA 72:163-71, Oct 72

11. Korr, I.M.: Andrew Taylor Still memorial lecture: Research and practice — a century later. JAOA 73:362-70, Jan 74

12. Hoover, H.V.: A hopeful road ahead for osteopathy. Parts I and II. JAOA 62:485-98, Feb 63; Part III. JAOA 62:608-16, Mar 63

13. Bowles, C.H. A functional orientation for technic. Academy of Applied Osteopathy Year Book. Part I: 177-91, 1955; Part II: 107-14, 1956; Part III: 53-8, 1957

14. Johnston, W.L.: Manipulative specifics. JAOA 61:535-9, Mar 62

15. Johnston, W.L.: Manipulative skills. JAOA 66:389-407, Dec 66

16. Johnston, W.L.: Segmental behavior during motion: I. A palpatory study of somatic relations. JAOA 72:352-61, Dec 72; II. Somatic dysfunction — the clinical distortion. JAOA 72:361-73, Dec 72; III. Extending behavioral boundaries. JAOA 72:462-75, Jan 73

17. Houk, J., and Henneman, E.: Feedback control of muscle: Introductory concepts. In Medical physiology. Edited by V. Mountcastle. Ed. 13. C.V. Mosby, St. Louis, 1974

18. Henneman, E.: Peripheral mechanisms involved in the control of muscle. In Medical physiology. Edited by V. Mountcastle. Ed. 13. C.V. Mosby, St. Louis, 1974

19. Grainger, H.G.: Basic mechanism of the common "crick." THE D.O. 92-8, Jun 69

20. Bailey, H.W.: Personal communication

21. Bowles, C.H.: Personal communication

22. Koizumi, K., and Brooks, C.: The automatic nervous sytem and its role in controlling visceral activities. In Medical physiology. Edited by V. Mountcastle. Ed. 13. C.V. Mosby, St. Louis, 1974, p. 806

Dr. Korr is Distinguished Professor of Physiology at Kirksville College of Osteopathic Medicine, and is director of the college's program in neurobiology.

Dr. Korr, Kirksville College of Osteopathic Medicine, Kirksville, Missouri 63501.