The role of autogenic inhibition in the reduction of muscle splinting

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Muscle 'splinting' (acute hypertonicity) is the clinical entity commonly featured as a result of somatic or visceral trauma. One of the implicit objectives of manipulative therapy is to stretch hypertonic muscles. The mechanical parameters of the manipulative thrust are reviewed in relation to the physiology of the muscle afferents which are activated by changes in length, tension and momentum. During muscular contraction, autogenic inhibition is initiated in parallel (with the motor excitation), via the interneurons that are activated by the Golgi tendon organs. It appears that when an excessive force is applied to or exerted by a muscle at varying lengths (extensions), the Golgi tendon organ inhibition regulates not only the frequency of discharge, but also the range of firing of the motoneurons. This relaxation (the inhibition of gamma, as well as alpha motoneurons) is one of the desired end results of manual therapy in most conditions. The role of other muscle afferents (groups Ia and II) and the "gamma loop" are also discussed in relation to the muscle 'splinting' phenomenon.

The distinctive focus of manipulative therapy since its inception has been the musculoskeletal system (MSS). Accordingly, the MSS assumes a dominant role as the source of sensory input to the central nervous system (CNS) as well as being the recipient of the preponderance of efferent output from the CNS. Thus, the neuromuscular component is implicated in the maintenance of health, as well as in the transmission of disease processes in the body.

Musculoskeletal pain and decreased mobility affect the majority of patients who are treated by manipulation. It is not surprising then, than one major criticism of manipulative therapy is directed at a lack of agreement and understanding of the underlying pathology involved (1). The problem is further compounded because of the variety of tissues and the multiplicity of locations that receive nervous innervation from the same spinal segment. For instance, non-specific pain that is primarily of muscular etiology may be observed as hyperalgesia of the skin of the same segmental distribution (2). Alternatively, protective muscular spasm may result initially from irritation of another structure, which is also segmentally related, or, if prolonged, become a secondary, self-perpetuating focus of irritation (3). For example, in numerous patients, spasm of paraspinal muscles after whiplash injury may favour ligamentous damage, rather than nerve root impairment as indicated by normal electromyographic (EMG) results in the distribution of the anterior primary rami (4).

Objective, measureable clinical signs are difficult to find in the manipulative context. EMG studies (5,6) would appear to offer the greatest promise for pre- and post-manipulative monitoring. Nachemson (7) suggests, however, that some EMG studies that have been done lack quantitative documentation, a double-blind format and a statistically valid evaluation of the results.

It is one of the peculiarities of neural function that strong stimuli result in very pronounced responses, but that minor irritations, extended over a considerable period of time are much more detrimental to the integrity of the structure. Nerve compression of high intensity produces morphological axonal damage (8) referred to as axonotmesis (e.g. Wallerian degeneration). The distal nerve segment becomes electrically inexcitable, and neuromuscular junctions and sensory end-organs degenerate (9,10). Regeneration is dependent, however, upon the internal architecture being fairly well preserved. Following injury, regenerating axons of nerves of protopathic senses (pain and severe degrees of temperature) return relatively rapidly (approx. 7-10 weeks), whereas epicritic senses (perception of light touch, 2-point discrimination and small differences in temperature) may remain impaired for six months — two years or never return at all.

Finally, the perception of pain is actually a continuum composed of three distinct stages: (A) a sense of touch followed by, (B) well-localized acute pain perception, and (C) then after 2-3 seconds a dull, prolonged, unpleasant burning pain. Furthermore, the painful sensation is completely subjective, and is dependent on many variables that are impossible to assess accurately.

Thus, it appears from the preceding discussion that objective patient criteria, as well as the specific mechanisms which underlie pain for which manipulation is prescribed (11), have not been satisfactorily documented. However, most patients consistently do have some degree of muscle spasm present. Although this finding is not directly quantifiable, it does indicate the presence of a common factor that may be evaluated both before and after manipulation.

In this connection, it is necessary to give some thought to the property of muscles known as "stiffness", which is increased during muscular spasm and is defined as tension/length. Stiffness increases with tension whether active or passive. Golgi tendon organs (GTO's) are functionally known as β-receptors (12) which record active or passive tension and also possess a dynamic phase, i.e. are sensitive

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to the rate of increase of tension. The rate sensitivity of the GTO's will make them respond optimally to fast stretch. Of similar importance is the response of the GTO's to the rising phase of a tetanic contraction (13). The dynamic sensitivity of the GTO's is not dependent on visco-elastic interaction but is an inherent receptor property (13,14).

The evaluation then, of the effects of manipulative therapy in this narrative is based to a great extent on the muscular component, i.e. the reflex relief of muscle spasm that may or has become self-perpetuating, and specifically GTO's and Ib afferent neural input. In spite of this special status of the subject matter, such myo-afferent stimuli may to some extent permit the characterization of the information flow in the nervous system that must underlie the proprioceptive experience; and such stimuli enable the distinction between processes at the receptor level and in the CNS.

The Physiology of Corrective Reduction

(A) Biomechanical considerations

Even a simple movement about an extremity joint in a two-dimensional plane requires a complex pattern of contraction and relaxation in numerous muscles involving multiple degrees of mechanical freedom. At any given time, each moving segment has a certain amount of mechanical energy, part of which may be potential and part of which may be kinetic. Additionally, the kinetic energy may exist in two forms, namely translational and rotational (15). Lowering a body segment (decreasing potential energy) or by reducing its velocity in either the translational or rotational mode (decreasing kinetic energy) (16) is an example of the ready energy reserves (principle of conservation of energy) which are used by the sensorimotor system to induce rapid corrections or adjustments in movement patterns.

Specialized sensory receptors transduce the forces associated with muscular contraction into a biologic signal. During rectilinear motion (from Newton's second principle) force is a product of mass and acceleration, but in muscles it is also intimately connected with length. As a result, force is evaluated by length and tension, and by the velocity at which alterations in these two variables take place. In order for higher levels of the CNS to monitor this displacement and make effective adjustments when necessary, it is essential that both the number of control parameters (principle of least interaction) (17) and the specific afferent input requiring analysis (feature extraction) (18) be limited.

Stiffness in a joint, as opposed to muscular stiffness (tension/length) is defined as the change in moment per change in angle (19). If pain, loss of joint mobility, or sensorimotor loss exist, abnormal muscular forces thus produced, when combined with gravitational forces effect adjustments in the torque-angle relationship, or stiffness about closely approximated joints (20). The result is a diminution of movement, and ultimately the reduction in momentum of a particular segment.

Manipulation is concerned with increasing the range of motion and overcoming resistance in a joint. The specific method of execution induces certain elements of the joint to move beyond its limits seen during voluntary movements (plastic deformation preceding a range which causes luxation), but does not violate anatomical integrity (21). Often, a popping sound accompanies a thrust which is caused by cavitation (under sub-atmospheric pressures, the synovial fluid vaporizes and gas is released from solution; the collapse of the vapor cavities gives rise to the noise (22). A manipulation thus stretches involved structures and produces a sudden traction on contracted muscles and other elements. Thus, the thrust stimulates the corresponding vector induced proprioception which in turn initiates a sequence of inhibitory influences, contributing to the relaxation of both intrafusal and extrafusal muscle fibres (23).

At extreme lengths, total tension ultimately approximates elastic tension and, in effect, corresponds to a functional, muscular de-efferentation (complete inhibition). As a result, the completely inhibited muscle delivers a tension length curve which is that of passive tension (24). One protective mechanism employed when tension on a muscle and therefore on the tendon becomes extreme, is the "Clasp-Knife" or lengthening phenomenon. The inhibitory effect from the tendon organ can be so great that it causes sudden relaxation of the entire muscle. A kinematic justification maintains that during rectilinear motion on the same action line, when the velocity of a body is opposed to the force applied, the body moves more slowly. If the force continues to act, it will ultimately reverse its direction of motion and accelerate in the same direction as the force.

A phenomenon commonly encountered during traumatic insult is that of muscle 'splinting'. Rigidity of muscles is used as a protective mechanism to avoid pain caused by movement of the part. Certain manipulative techniques employ principles which are based on "the reflex amount of static tension in stretch to different extensions" (25,26). Thus, in an extremity, for example, muscular contractions leading to spasm may be remedied by manual, active and/or passive stretching movements designed to oppose the direction of contraction of the affected muscle (27). This corrective strategy may be subdivided further into two additional categories: (a) the muscle is slowly stretched to a point just short of full stretch and isometrically contracted, thus establishing a new functionally increased resting length or, (b) very rapid extension or forced over-extension will ultimately mobilize a powerful response from the GTOs so that the limb goes clasp-knife slack. After stretching (24,28) of the contracted muscle the length of the inhibited, weakened antagonist improves sometimes immediately, or within a few days without any other additional treatment.

(B) Neurological considerations

Three different categories of stretch-sensitive mechanoreceptors are normally distinguished: primaries, secon-

daries and tendon organs. Muscle spindles (primaries and secondaries) are "in parallel" and tendon organs "in series" with the extrafusal musculature.

The "primaries", or Ia spindle afferents, respond to stretch with an initial velocity-sensitive (29) (dynamic) response, subsequently followed by a constant, stationary (static) phase (32). Ia fibres monosynaptically (31) activate motoneurons of the muscle of origin and inhibit the muscles' functional antagonists (32-34). Thus, the primaries signal change of length as well as length itself.

The most striking property of the "secondaries" (group II afferents) is their relatively insignificant dynamic phase. Group II fibres inhibit antigravity muscles and excite their functional antagonists by multisynaptic arcs (35). It is now generally accepted that secondaries monitor instantaneous and maintained length (36-39).

Ib afferent input originates in the GTO's (at the musculotendinous junction) in direct contact with the tendon collagen (40) and is activated by muscle tension, particularly in response to a discreet number of motor units whose muscle fibres insert directly into the tendon organ (14,41,42). Additionally, stretch sensitivity of individual GTO's depends on the muscle in which they are located (43). Ib afferents inhibit the motoneurons of the muscle of origin (44,45), but excite the motoneurons of its functional antagonists with both mechanisms working through a disynaptic reflex.

Thus, spindle primaries and GTO's function as negative feedback mechanisms monitoring length (extension) and tension respectively. Furthermore, the secondaries also monitor length changes and inhibit extensor muscles (46).

Ib fibers from GTO's, like those from the primary receptors of muscle spindles, transmit signals into local areas of the cord (47) as well as into the cerebellum via the spino-cerebellar tract (52,48-50). The implication of he large role played by the GTO in manipulative therapy is in no instance intended to deemphasize or exclude the roles of other sensory inputs or reflex mechanisms. However, the purpose of the present hypothesis is to show that the operational, inhibitory, internuncial system may be selectively activated by Ib afferents during manipulative adjustments. In addition, the inhibitory mechanism is facultative in that it depends on how the interneurons are biased (51,52).

A comparison of antidromic and autogenic inhibition

During the elicitation of a given posture or movement its reciprocal action comes under the influence or reciprocal inhibition. Autogenic inhibition is operant as an additional control mechanism and is activated at a certain level of excitement (simultaneously with the excitatory action) thus functioning as a negative feedback system, i.e.: progressively controlling and slowing down the motor action itself. Deceleration of movement and periods of arrest, on the other hand, necessarily result in a subsequent release of the reciprocally inhibited action. In a functionally related manner, the enhancement or attenuation of the reciprocal interaction of antagonist motoneuron pools may involve the participation of recurrent effects associated with the impulse activity of motoneurons. Central (intraspinal) inhibition of motoneurons is mediated by Renshaw cells (antidromic inhibition) which are activated under natural conditions by impulse activity from the motor nucleus, as well as inhibition via the interneurons that are activated by the Golgi tendon organs during muscular contraction (autogenic inhibition) (53).

The muscle spindle functions as an essential feedback mechanism. The feedback of primary endings of each spindle is excitatory, and is conveyed monosynaptically to the motoneurons of the same muscle. Inhibition of motoneurons produced by group Ia afferents arising from the

Figure 1: Interneuronal inhibitory systems of agonist muscles

DESCENDING MOTOR CONTROL SYSTEMS			
CORTICOSPINAL	RUBROSPINAL	VESTIBULOSPINAL	RETICULOSPINAL

↓

INTERNEURONAL INHIBITORY SYSTEMS			
SPINAL REFLEX PATHWAYS	PRESYNAPTIC INHIBITION	GAMMA MOTONEURONS	RENSHAW CELLS
Activated by primary afferent depolarization and connected to spinal motoneurons	Depolarization of intraspinal primary afferents and supraspinal and intraspinal fibers	Regulate activity of Ia and II afferents of some reflex pathways	Intraspinal antidromic inhibition

annulospinal (primary) endings of muscle spindles in antagonist muscles is mediated predominantly by interneurons located in the ventral horn dorsomedial to the motor nuclei (Rexed's lamina VII). These interneurons are subjected to recurrent inhibition via motor axon collaterals (48) and receive monosynaptic excitatory actions from the vestibulospinal tract (54) (Figure 1). Although the spindle is the sensory component of the stretch reflex, reciprocal inhibition is mediated by interneurons located in the intermediate nucleus of the cord (51) which do not receive these connections, although they are subject to descending facilitation from the pyramidal (55,56) and rubro-spinal tracts (57) as are the 'ventral' Ia interneurons (Figure 2).

Experiments utilizing volleys produced by electrical stimulation, brief stretches (36,58) and longitudinal vibration (59,60) have been designed to activate Ia afferents independently of Ib and II muscle fibres. Brief stretch of muscle has an advantage over electrical nerve stimulation since it allows the separation of the Ia and Ib components of the group I volley. In the confines of these parameters maximal focal synaptic potentials are demonstrable in the intermediate and motor nuclei (61). Results confirm that the majority of group I activated interneurons in the intermediate nucleus lie in the Ib pathway from Golgi tendon organs (62,63). Ia fibres evoke only small, focal, subthreshold potentials in the intermediate nucleus (Figure 3), but do elicit a field potential in the motor nucleus (Figure 4) which is consistent with the low-threshold component of the group I volley (the smallest detectable field occurs with a smaller group I volley for the motoneuron field than for the interneuron field.).

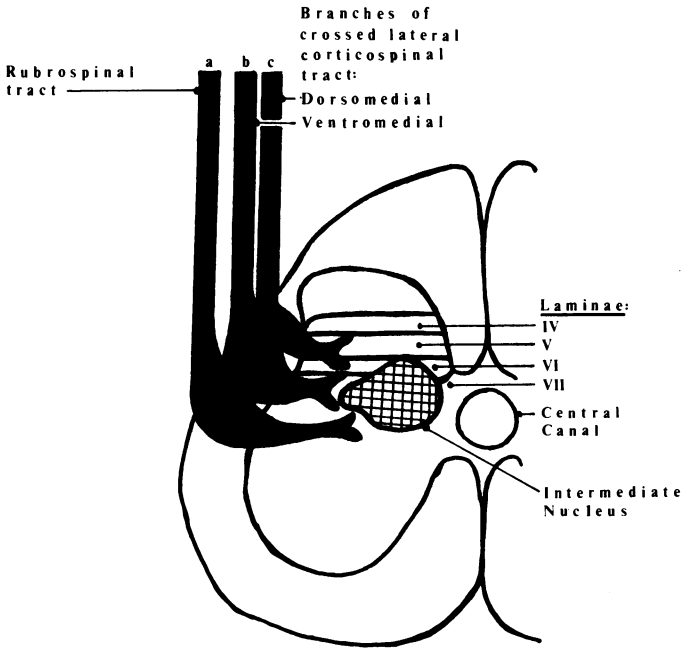

Figure 2: Descending influence on interneurons of the intermediate nucleus.

The regulation of the firing rate by the recurrent collaterals of motoneurons is accomplished mainly by postsynaptic inhibition mediated by the Renshaw cell (64,65), and in some cases also by facilitation of motoneurons (66,67). Of the two processes, inhibition is more common and more powerful than excitation.

Many studies (67-70) have indicated that the excitation of Renshaw cells by volleys in the low threshold (group I) muscle afferent fibres is solely a secondary consequence of reflex excitation of motoneurons. Small motoneurons (71,72) are most readily recruited into polysynaptic reflexes (PSR's). Thus, the excitation of Renshaw cells by motoneurons is relatively weak (67). Other studies (61,73,74) have demonstrated excitation of Renshaw cells in the absence of concomitant motoneuron discharges following flexor reflex afferent (FRA) stimulation. Therefore, Renshaw cell responses are likely to be dependent on the activation of the internuncial pathways which are to a great extent independent of those responsible for the production of PSR's (75).

The amount of recurrent inhibition is also affected by the proximity in the spinal cord of the nuclei of two muscles (76,77) (transverse activation for cells of the same level). It has also been confirmed that small, tonic motoneurons are the ones whose discharge frequency could be most systematically reduced by recurrent inhibition (76-78). Thus, a facultative regulation of firing by the firing rate, should increase the precision of the performance of the muscle's motor units (79), and augment mechanisms essential for postural tone.

Inhibition inherent in central coactivation

The combined control of two feedback mechanisms, one for length, the other for tension, is necessary to make motor adjustments. Length sensitivity is accomplished by two separate, interacting sense organs and three types of fusimotor fibres: dynamic gammas, static gammas and dynamic fusimotor alphas (28). Gamma motoneurons together with muscle spindles (limited to the action of spindle primaries alone) and the monosynaptic reflex arc (stretch reflex) comprise a residual servomechanism (53,80).This mechanism ultimately results in a functional equality of length in the two muscle types thereby silencing the spindles. Thus, a synchronous adjustment of gamma activity with the anticipated speed of contraction (28) can prevent any decrease in spindle discharge occurring during muscular contraction. Extrapolation of this hypothesis to pathological conditions characterized by aberrant muscle contracture has been suggested, when: palpatory examina-

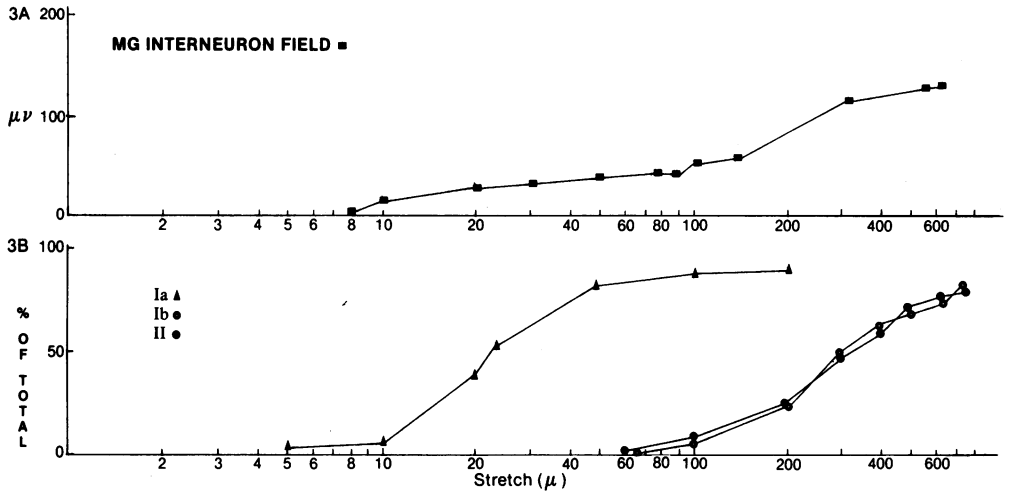

Figure 3: Relationship between the amplitude of focal synaptic potentials, and graded electrical and natural stimulation (laminae VI and VII). The measurements of field potentials were made from signal-averaged records (100 responses). Afferent volleys and stretch amplitudes were made from film records of the Medial Gastrocnemius (MG) muscle nerve at transected lumbar spinal cord of the cat. A: in laminae VI and VII only a small field potential was produced with a stretch of 60μ or less. As brief stretch was increased in amplitude, the focal synaptic potential continued to grow until the maximal stretch was reached. This suggests a small contribution by group Ia fibres and a larger contribution by Ib and/or II fibres to the generation of field potential. B: contributions of Ia, Ib and II fibres to the afferent volley for different stretches. Brief stretches below 60μ at 100 g. initial tension activated just Ia fibres, and brief stretches of 100μ activated essentially all of Ia fibres and very few Ib or II afferents to threshold. (Reproduced with permission of M. E. Lucas and W. D. Willis. Identification of Muscle Afferents Which Activate Interneurons in the Intermediate Nucleus, J Neurophysiol 1974; 37:282-93.)

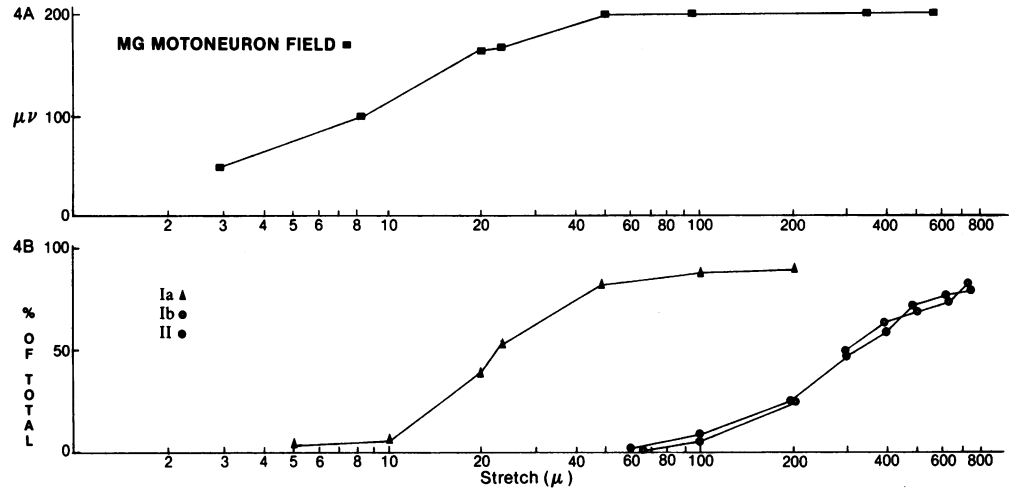

Figure 4. Relationship between the amplitude of focal synaptic potentials and graded electrical and natural stimulation (motor nuclei). The measurements of field potentials were made from signal-averaged records (100 responses). Afferent volleys and stretch amplitudes were made from film records of the Medial Gastrocnemius (MG) muscle nerve at transected lumbar spinal cord of the cat. A: investigations of responses in the motor nucleus to brief stretches showed a small field potential using a stretch of 5μ or less. This potential grew with stretch amplitude until a stretch maximal for Ia afferents was reached. The smallest detectable field occurs with a smaller group I volley for the motoneuron field than for the interneuron field. This suggests that the lowest threshold component of the group I volley contributes more to the development of the field potential in the motor nucleus than to that in the intermediate nucleus. B: contributions of Ia, Ib and II fibres to the afferent volley for different stretches. Brief stretches below 60μ at 100 g. initial tension activated just Ia fibres, and brief stretches of 100μ activated essentially all of Ia fibres and very few Ib or II afferents to threshold. (Reproduced with permission of M. E. Lucas and W. D. Willis. Identification of Muscle Afferents Which Activate Interneurons in the Intermediate Nucleus, J Neurophysiol 1974; 37:282-93.)

tion has disclosed discreet areas of (a) hyperalgesia and (b) inflammation (81). The servo-theory has a somewhat limited applicability in that it only applies to the proprioceptive reflexes of primaries, while not elaborating the effects of tendon organs and secondaries on motoneurons.

Supraspinal control of the "Gamma Loop" is augmented by all the basic descending motor systems that exhibit monosynaptic connections with the fusimotor neurons (82-85).

The presence of two types of functionally opposed gamma motoneurons (28,86,87) suggests that a highly specialized and differentiated control system is necessary for coordination of movement. Dynamic gamma fibres excite the spindle's primary ending during the act of stretching, while static gamma fibres raise the firing level at the particular extension to which the same muscle was subjected, once the final length after stretching had been attained. Stimulation of the red nucleus or the inferior olive (83) selectively induces dynamic gamma fibres to amplify the response of muscle spindle primary endings. Conversely, static gamma fibre activation via Deiter's nucleus can accelerate muscle spindle discharge at constant muscle length, primarily of the extensors (85). Additionally, static gamma fibres depress the fusimotor-amplified discharge of primary endings of muscle spindles during phasic muscular stimulation (83,85).

Research has shown, however, that acceleration of muscle spindle discharge can occur simultaneously with, or subsequent to, the onset of voluntary muscular contractions (88-90). Additionally, disruption of the afferent limb of the gamma loop has no effect on the onset of voluntary impulse activity by spinal alpha motoneurons for many voluntary phasic movements. Delayed muscle contraction, transport lag and the resulting systemic oscillation in the stretch reflex are counteracted by the increased dynamic response (to velocity) of the primaries when assisted by the action of fusimotor alphas and gamma fibres (80). Thus, the greater efficacy of the connection between the motor cortex and fusimotor neurons in comparison with alpha motoneurons (i.e. the latent period of the reciprocal influence on gamma motoneurons is shorter), can compensate for the considerable delay attending the reflex gamma activation of the alpha motoneurons (53,91).

These studies indicate that supraspinal structures exert a controlling influence on the spinal reciprocal inhibitory apparatus. Furthermore, alpha control (fast motor units capable of producing large contractions) may be dominant in many rapid and strong movements. Thus, in normal muscular contractions, for example, the force developed to overcome obstacles to motion may be achieved by pure alpha motoneuron activation (92). Maximal efforts would push the motoneurons into the secondary firing range (a high-frequency response to accelerate the rate of rise of contraction and to potentiate motor endplate action) (93) with a very large increase in gain (increment of current strength per unit increase of firing rate).

Kinematics of the skull and upper cervical spine

Kinematic models of linear and angular acceleration are used to validate the reflex activation of motor control systems concerned with postural and 'righting' reflexes. However, the evaluation becomes increasingly complex when specifically computing the motion of the upper cervical spine, C1 - C3, and the skull.

Motion of the skull is relative to two different reference systems, namely the C1 and C2 vertebrae, which in certain manipulative situations are themselves in motion relative to each other (Galilean-Newtonian transformation). Furthermore, an element of lateral flexion plus pure flexion and/or extension in the sagittal plane may occur simultaneously with angular acceleration. Bending the neck of an animal, for example, leads to ipsilateral extension from the labyrinth reflexes but ipsilateral relaxation from the neck reflexes (94). Slow sinusoidal movements of the skull alone causes the lower limbs to extend and the upper limbs to relax, whereas synchronous reciprocal movements of the C1 vertebra produce opposite results (95).

The major neck afferent input to the vestibular nuclei arises in the paravertebral joints and capsules of the upper cervical vertebrae (96), C1 - C3 dorsal roots and the most cephalad portion of the cervical spinal cord. The vestibulospinal system is an integral regulator of posture and locomotion, including ipsi- and contralateral facilitatory effects (97) of the axial and proximal limb musculature (particularly the extensors).

Angular acceleration can significantly amplify gamma motoneuron activity (98). Fast fibres in the vestibulospinal tract originating in Deiter's nucleus have mainly mono- and disynaptic excitatory effects to extensor alpha and gamma motoneurons (99). The gamma motoneurons have a significantly lower threshold of activation than that for alpha motoneurons. High threshold cutaneous and muscular afferents may also facilitate crossed excitatory and inhibitory reflexes via Deiter's nucleus (100). Monosynaptic excitatory and inhibitory connections to the cervical and thoracic spinal cord originate in the medial vestibular nucleus and descend via the medial vestibulospinal tract (99).

Phasic pyramidal activity initiates at least two major changes: (1) the movements of the body (spatial) and (2) vestibular stimulation. In like manner, the postural changes (temporal) that trigger the vestibular sensory organ reaction must reciprocally evoke correctional changes in the phasic movement. This readjustment can only be accomplished with the participation of the pyramidal system (101). Thus, it appears that every complete motor act is innervated by both systems.

The question that arises from the proposed kinematics of the cervical model is whether multimodal afferent stimulation can coincidentally activate alternate routes of transmission to flexor and extensor motoneurons. It has been suggested that concomitant peripheral inputs such as from the joint, muscle or tendon, or other skin afferents

may inhibit or facilitate transmission at the interneuronal level; the specificity being aided by supraspinal inputs to these interneurons (102). Upper cervical articular receptors are functionally involved in direct, tonic reflex effects (103) as well as multisynaptic, intersegmental muscular corrections via interneurons in the intermediate area of the cord (laminae V-VII). There is an extensive convergence pattern of rubro-, vestibulo- and reticulospinal tracts as well as different types of afferent pathways on Ia interneurons in the cat which elicit inhibition in specific target nuclei (104). Further evidence indicates that there is a common segmental interneuronal pathway for these systems (105).

Some functional implications of autogenic inhibition

The alleviation of muscle 'splinting' (acute hypertonicity) is one of the prime objectives of those engaged in manipulative therapy. However, direct evidence of the effects of manipulation on reflex activity in the CNS has remained somewhat obscure. At this point it should be noted that there are at least three types of stretch reflexes each corresponding to the three types of receptors, primaries, secondaries and GTO's (106). The latter may fail to respond to passive stretch due to their anatomical location in the proximal part of the muscle (13.14). However, the selective sensitivity of individual GTO's is a function of the specific muscle of their origin (107), since many GTO's in other muscles respond similarly to both stretch and contraction (13). Reflex activation of both tendon organs and spindle secondaries is accomplished through polysynaptic circuits. As well, the tonic component of the action of primaries in the stretch reflex is also modulated by interneurons (53). Thus, a multiplicity of factors tend to support the interaction of multisynaptic circuits in mediating supraspinal, intraspinal and segmental inhibitory channels.

Supraspinal centers can act on segmental neurons of presynaptic inhibition. Stimulation of the sensorimotor cortex (109) or the pyramids evokes depolarization of flexor-reflex afferents, 1b muscle afferents and low-threshold skin afferents but is absent in 1a muscle afferents. Facilitation of interneurons in the intermediate nucleus is evoked by stimulation of the red nucleus (110,111). This enhances the inhibitory effect of the 1a and 1b afferents of antagonist muscles as well as the facilitatory and inhibitory effect of the 1a and 1b afferents of antagonist muscles as well as the facilitatory and inhibitory effects on the motoneurons of the low-threshold joint and skin afferents and the flexor-reflex afferents.

Direct reciprocal inhibition is dependent on pyramidal facilitation of the spinal interneurons incorporated into this reflex path (56,112-114). These segmental interneurons in the intermediate nucleus are activated by impulses of the 1a afferents from the antagonists (54). 1b afferents evoke inhibition in the motoneurons of agonists that is mediated by interneurons in the ipsilateral intermediate nucleus (54). The intermediate nucleus is also involved in other segmental reflex pathways of reciprocal interaction of the motoneuron pools of antagonist muscles. The crossed extensor reflex, for example, is accompanied by the facilitation of the reciprocal inhibitory interneurons linked with flexor motoneurons (104,115).

The preceding information in the narrative has attempted to deal with the significance of well-defined physiological mechanisms in an overall biological context. The complexities involved in this approach become readily apparent when trying to apply the knowledge in a clinical setting. Recently, there has been emerging concern that increased clinical expenditure may not represent further progress toward better patient care. In addition, unnecessary treatment may serve to complicate the prognosis and adversely affect the decision-making process. It is clear that any therapeutic program must maximize costs, time and health care resources. Thus, there must be a broadening of perspectives about manipulative therapy in order to keep up with the changing pace of health care, and to satisfy the criteria of the scientific community.

Conclusion

1. Muscle 'splinting' appears to be one of the most consistent and reliable indicators of trauma in patients who are treated by manipulative therapy. To some degree, the etiology of muscle 'splinting' may involve higher order functions such as the control of posture and movement. In this respect, an application of the underlying, well-defined, physiological mechanisms in the manipulative framework, may assist the diagnosis and treatment of the problem.
2. The source of both force and energy for locomotion is derived from muscle. More specifically, force and velocity (of shortening) are the variables which firing motoneurons deal with in order that voluntary movement can take place (116). Additionally, motion is produced by overcoming such restraining forces as friction and viscosity of the surrounding environment.

The function of muscle spindles has generally been considered in relation to the control of muscular tension. Viscosity modifies the firing rates of muscle spindles (28), i.e., dynamic gamma fibres act by increasing the viscosity of the intrafusal muscle (fusimotor damping). The initial increased discharge rates of primary endings of muscle spindles is a function of velocity of stretching (39) and is due to dry friction at the moment of initiation of movement (117). When resistance is encountered during muscular contraction, the spindle's secondary endings counteract the obstacle by load compensation, which also produces velocity compensation (118).

3. The functional significance of GTO's in moment to moment regulatory mechanisms has become increasingly evident. For example, during stepping cycles of the cat hind limbs (119,120), all three endings, Ia, II, and also Ib are activated during both active and passive phases of the stepping cycle. Muscular contraction does elicit autogenic postsynaptic inhibition in both extensors and flexors (121). Furthermore, if a motoneuron is not driven by the primaries and the muscle is under some tension, an

increase in membrane potential may be expected due to autogenic inhibition from the tendon organs (122).

A comparison of many published accounts of the mechanism for increased force during a voluntary contraction of increasing magnitude, i.e., the relation between GTO discharge frequency and tension, indicates that the significant consideration involves the method by which tension was altered. In vitro stimulation of GTO's signifies that their discharge frequency can be increased to progressively higher levels by increasing the number of stimulated motor units (123), i.e., to raise tension at low forces. As well, firing increases with tension over a wide range (123). An alteration of the muscle length creates a highly nonlinear summation relation and suggests the relative independence of impulse generation sites as per the "Probabilistic Mixing Theory" (123-126). Since GTO's have dynamic sensitivity (15) it is likely that CNS monitoring of tendon organs supplied by branches of a single axon, may be a function of the magnitude of asynchronous twitch contractions of individual motor units, whose fibres have a direct insertion into the tendon organ (123,126).

Thus, both the level of force applied to, or exerted by a muscle, as well as its rate of change, determine the discharge frequency of its firing motor units and also that of the GTO's. In addition, the thresholds of the interneurons in the intermediate nucleus to natural (tension and stretch/extension) and electrical stimuli suggest that their excitation is dependent on group Ib afferents, which is an essential function of GTO recruitment.

4. For simple mechanical reasons, the neural response pattern to external stimuli are appropriate during certain phases of motion or lack of it, but not in others. Movements performed at abnormal ranges of speed may be characterized by a lack of alpha-gamma coactivation. Such adjustments could be carried out by an exchange of motor units with individually different tension/length curves (127) while the muscle is being extended. In addition, cutaneous stimulation and conditions of passive mobilization can excite functionally opposed muscles, and can also inhibit or excite the same muscle group in various circumstances.

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