# The Golgi Tendon Organ: A Review and Update

(neuromuscular receptors; proprioceptors; sensory receptors)

Josephine C. Moore

*This article presents current information concerning (a) the structural relationships of the Golgi tendon organ (GTO) with different types of extrafusal muscle fibers; (b) the Gp.Ib re-afferent or feedback fibers from the GTO; (c) the way in which these proprioceptors monitor muscle tension and function in parallel processing with other receptors and with the central control mechanisms of the nervous system; and (d) how GTO's are involved in regulating autogenic excitation, autogenic inhibition, or co-contraction of extrafusal muscle fibers.*

Several major sensory systems, the general proprioceptors and exteroceptors and the special receptors of the vestibular, visual, auditory and olfactory systems, are believed to be the principal sensors that enable organisms to have orientation in space and function against the forces of gravity. The Golgi tendon organ (GTO) is one of a number of proprioceptors or mechanoceptors that play an important role in the regulation of muscle tone and movement in relation to spatial orientation and gravity. Discussion of this receptor does not imply that it is more or less important than other receptors involved in spatial perception and movement. Rather, this article attempts to alert therapists to the changing concepts concerning the morphology and physiology of the Golgi tendon organ especially in relation to past and present concepts in rehabilitation.

Prior to discussing the Golgi tendon organ, two basic concepts concerning the nervous system should be reviewed. These con-

*Josephine C. Moore, Ph.D., OTR, DSc (Hon), is Professor and Head of Neuroanatomy Section, Department of Anatomy, University of South Dakota School of Medicine, Vermillion, South Dakota 57069.*

cepts are helpful, not only for understanding receptors, but for comprehending normal and abnormal central nervous system (CNS) function in relation to rehabilitation techniques.
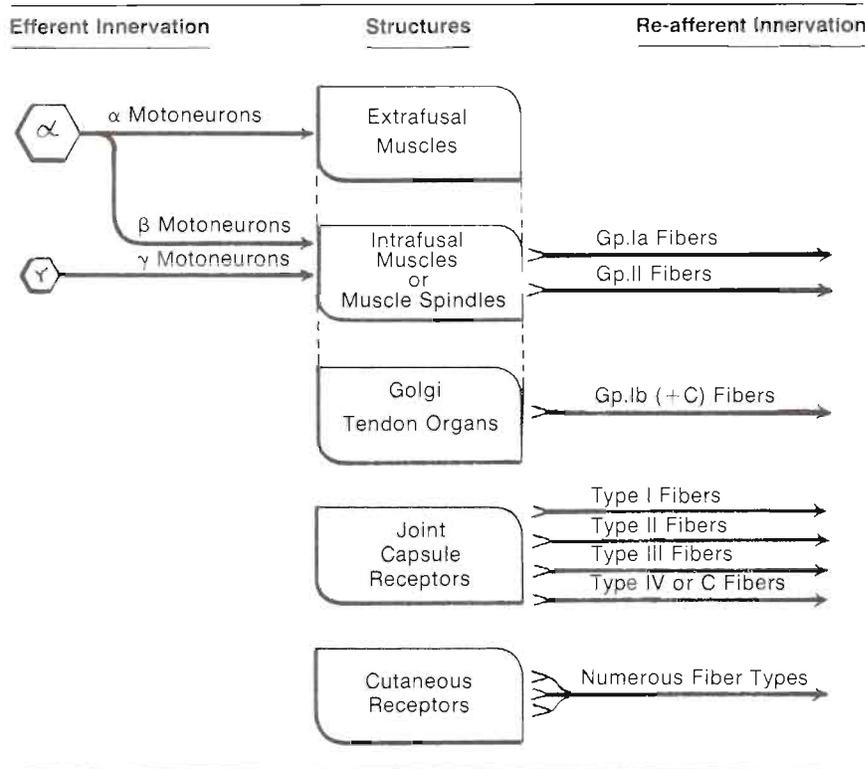
## Feedback Systems

The first concept concerns *feedback systems,* also known as re-afferents or the sensory components of internal loop control systems (1). Simply stated, if a structure is innervated by an efferent (motor) neuron, there must be (a) receptors available for monitoring the effects that the motoneurons have made upon the structure, and (b) re-afferents or "feedback" systems for relaying this information to the CNS (1, 2, 3). Without receptors and feedback neurons, the CNS would have no way of knowing whether a movement had been properly programmed or if the muscle tone had been correctly adjusted so that normal movement could occur. In other words, the CNS has to be continually informed about its motorical programs in order to react appropriately. Lack of feedback, inappropriate feedback, or faulty programming within the CNS, due to pathology, results in varying degrees of abnormal muscle tone, movement, and/or problems associated with gravity and orientation in space (2, 3, 4, 5).

In some structures, like the muscle spindle, both the motor innervation and the re-afferent or "feedback neurons" are closely associated, that is, the motor and sensory neurons are "packaged" together with the receptor organ. In other structures like the extrafusal muscle fibers, there are no "direct" or anatomically related re-afferent fibers (see Figure 1). Examples like this do not dispute the

Figure 1

Schematic illustration of the relationships between muscle fibers innervated by lower motoneurons and the re-afferent (feedback) neurons that, directly or indirectly, monitor muscle tension, length, velocity. position, and movement



concept that there must be feedback systems for monitoring the motor innervation of structures. Rather, this alerts one to the fact that feedback mechanisms need not be directly or anatomically related to a given receptor but can be indirectly related. For example, extrafusal muscle fibers, though lacking direct feedback systems of their own, have a whole host of "indirect" receptors monitoring changes occurring in these fibers: (a) GTOs; (b) joint capsule receptors; (c) muscle spindles; and (d) many kinds of cutaneous receptors. This "indirect" relationship illustrates another important concept concerning nervous system function and dysfunction. This is called "parallel processing" or "comparator mechanisms."

## Parallel Processing

It is well known that the complexity of the nervous system, including structures associated with it, increases as the phylogenetic scale is ascended. In a primitive nervous system only a few receptors are needed to inform the CNS of changes occurring in the organism's environment. In slightly more complex organisms there are hundreds of receptors continually sending signals to the CNS, not only from different kinds of receptors, but also from receptors that are dispersed throughout a variety of locations within the organism. What is the basic reason for this increasing complexity in terms of survival? A simple organism can react to stimuli only with an all-or-none response. This

is not the most efficient means of survival or adaptation to a changing environment. In slightly more complex organisms, stimuli are detected by several receptors located in various positions. These receptors send "parallel" yet different signals to the organism's CNS. These signals are compared, their significance is determined, and graded responses are made. In more complex systems, multiple parallel inputs are received, not only from direct stimuli from the environment, but also from re-afferents or feedback systems within the organism. These are compared with previous experiences, and finally the appropriate graded responses are made. For each graded response new parallel feedback signals are sent to the CNS from the variety of receptors monitoring the graded response, and these in turn are recompared so that fine adjustments can be made (2, 3, 6). Thus parallel processing assures the organism that the most appropriate response will be made in relation to the demands of the environment. Even within the CNS, every nuclear center, from the spinal cord to the cerebral cortex, is known to be involved in parallel processing. Each nuclear area, and even individual "polysensory" neurons, receives multiple parallel inputs from many other centers via feed-forward and feedback circuits. These signals are compared with each other, as well as with stored memories, before appropriate and newly integrated messages are relayed to other centers. Loss of any one nuclear area or depletion or loss of multiple parallel inputs renders the nervous system deficient or incapable of comparing and preparing the organism for normal function. Instead, some

form of dysfunction results (depending on the location and extent of the lesion), such as dystonia, dyskinesia, dyssynergia, affective disorders, cognitive and perceptual changes of higher cortical functions, and/or combinations of these syndromes. In rehabilitation, many different techniques are utilized to treat these disorders. No matter what type of therapy is used, the basic premise of treatment is to dampen or inhibit the abnormal patterns that are interfering with function, and enhance those that are beneficial. In so doing, this enables the abnormal nervous system to "experience" more normalized stimuli, responses, and feedback from these responses. It is theorized that this helps re-establish more appropriate feed-forward and feedback circuits and comparator systems, or alternate methods of parallel processing, thus improving the quality of performance.

In summary, parallel processing or comparator mechanisms are an inherent property of both the CNS and the peripheral nervous system. This concept reiterates the fact that in order for any nervous system to function normally, it must receive multiple afferent and re-afferent parallel inputs, and it must be able to compare these stimuli with one another as well as with previously stored memories (3, 6). Only in this way can the system be prepared for the moment-to-moment future and react appropriately. In rehabilitation, it is important to keep this in mind, for this is the only way in which the nervous system functions. Lack of therapy, use of just one therapeutic technique, or excessive repetition of one method of treatment deprives the system of reaching its full potential and in-

stead may serve to reinforce abnormal muscle tone and behavioral patterns (7). Knowledge of feedback mechanisms, which are an integral part of parallel processing or comparator systems, helps one understand the relationships and interactions of the GTO receptor to be discussed in this article.
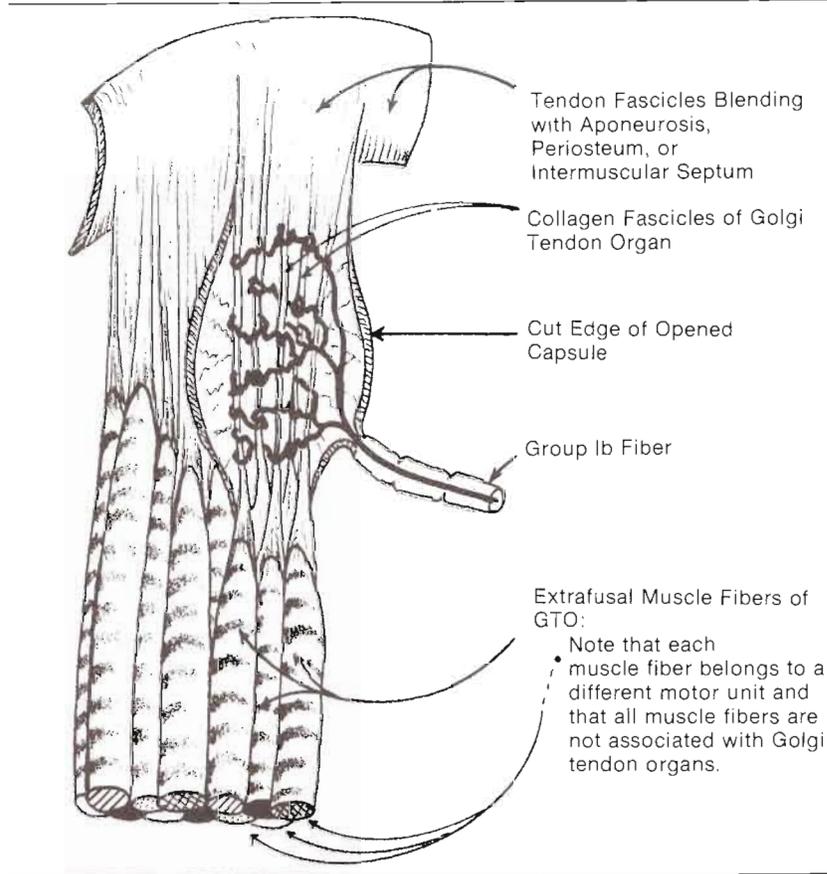
## Structural Relationships
The Golgi Tendon Organ (GTO) is perhaps the easiest receptor to understand structurally, and perhaps functionally, even though all of its functions are still unknown. As the name implies, this receptor organ is associated with tendons, and/or tendinous-like structures called aponeuroses. Almost all extrafusal (skeletal or voluntary) muscle fibers attach to bone, aponeuroses, or intramuscular septa via tendon fascicles located at the origins and insertions of muscle fibers. In nonmammalian species, the GTOs are *un*encapsulated receptors located along the length of the tendon fascicles. In mammals, GTOs are encapsulated and are located at the myotendinous (musculotendon) or musculoaponeurotic junctions, that is, where the extrafusal muscle fibers attach to or blend with the tendon fascicles (7-10) (see Figure 2). This change in location and structure from nonmammals to mammals is believed to be important for several reasons: (a) Research on the structural and functional aspects of the GTO in lower animals (nonmammalian) is not directly applicable to the way in which these receptors function in mammals, including man; (b) Encapsulated organs are believed to be more sensitive to a given amount of stimuli and are more precise in localizing and relaying information to the CNS

than are nonencapsulated receptors; and (c) The close association between the extrafusal muscle fibers and the collagen bundles of the encapsulated tendon organ enables these receptors to be extremely sensitive to any degree of change in tension in the individual muscle fibers to which they are attached (2, 8, 9, 11-14) (see Figure 2). In comparison, in nonmammalian tendon organs, any change in muscle fiber tension initially has to be translated along tendon fascicles before reaching the receptor organ. This could result in a less sensitive receptor because some of the tension-forces generated from the muscle fibers would be dampened along the length of the tendon fascicles interposed between the muscle fibers and the GTO receptor.

In mammals there is an interesting relationship between GTOs, extrafusal muscle fibers, muscle fiber types, and motor units. (A motor unit is defined as a group of extrafusal muscle fibers innervated by a single motoneuron and/ or an alpha (α) motoneuron (mn).) In large postural muscles, one α mn may supply several hundred muscle fibers. In muscles used for fine manipulation or control, only a few muscle fibers may be supplied by a single α mn. In either case the muscle fibers that belong to a motor unit are never grouped together, that is, they do not make up a discrete area within a muscle (2, 8, 12, 15). Rather, they are interdigitated among muscle fibers belonging to other motor units. In like manner, individual muscle fibers that belong to *different* motor units are associated with a single GTO (8, 9, 11, 12, 14) (see Figure 2). For example, one GTO may have five muscle fibers associated with it, with each muscle fiber be-

Tendon Fascicles Blending with Aponeurosis, Periosteum, or Intermuscular Septum

Collagen Fascicles of Golgi Tendon Organ

Cut Edge of Opened Capsule

Group Ib Fiber

Extrafusal Muscle Fibers of GTO:
Note that each muscle fiber belongs to a different motor unit and that all muscle fibers are not associated with Golgi tendon organs.

longing to a different motor unit, or there may be as many as one or two dozen muscle fibers per GTO, each belonging to a different motor unit (8, 11, 12). In this regard one needs to realize that different motor units are made up of different kinds of muscle fibers. In mammals four types of extrafusal muscle fibers are recognized: (a) slow oxidative (SO), fatigue resistant (red) fibers; (b) fast oxidative glycolytic (FOG) or fatigue resistant (pink) fibers; (c) fast, intermediate F(int), which are a "blend" between types FOG and FG; and (d) fast glycolytic (FG), fatigable (white) fibers (2, 4, 12, 15). Each GTO is known to have dif-

ferent combinations of muscle fiber types associated with it (8, 12, 14). Therefore each receptor is able to respond individualistically or somewhat differently from every other GTO due to (a) its fiber composition; (b) the muscle in which it is located and the size of the motor units associated with it; (c) the number of muscle fibers per GTO; and (d) the circumstances of the moment regarding position and movement in relation to the activity in which the animal is engaged (8, 9, 16).

The important fact about the GTO is not so much the complexity of the relationships between muscle fibers, types of fi-

bers, motor units, and GTOs, but the fact that this receptor appears to be extremely sensitive to all degrees of change in muscle tension (2, 9-12, 17). GTO receptors are just as capable of responding to the contraction of one or two muscle fibers belonging to a single small motor unit as to the forces generated by fibers belonging to a much longer unit (9, 11, 12, 14). However, some evidence indicates that the most sensitive GTOs are those that are associated with the smallest motor units, which are primarily made up of SO or slow oxidative muscle fibers. All of which means that the GTO is now known to be an extremely sensitive monitor of all degrees of muscle tension, in contrast to the older theory that stated that the GTO was only sensitive to the upper or extreme levels of muscle tension (11, 18).

The GTO is part of a complex feedback system that continually sends signals to the CNS concerning all gradations or changes in muscle tone or tension, both in relation to recruitment of additional motor units and/or decreases in tension (8, 12, 19). However, the GTO appears to be much more sensitive to changes in muscle tension produced by active muscle contraction (concentric) than to tensions produced by passive stretching of a muscle or active muscular elongation, that is, eccentric contraction (8, 9, 11, 12). In some muscles that have been studied extensively, there appears to be a close numerical relationship between the number of muscle spindles per muscle and the number of GTOs. This would indicate that the GTO and certain components of the muscle spindle are comonitoring muscle tension and sending parallel feedback sig-

nals, each of a slightly different kind, to the CNS (3, 12).

GTOs are reported to be much more numerous in antigravity musculature than in prograrvity muscles (2). In this regard, one has to equate the terms "anti- and progravity musculature" to the normal stance of an animal when it assumes an upright adult posture. For example, in quadrupeds, the extensors and synergists of all four limbs, the neck, and posterior trunk muscles, are classified as anti-gravity musculature. In bipeds the extensors and synergists of the lower limbs, posterior neck, and trunk muscles are classified as anti-gravity muscles, but the upper limb extensors and synergists are not. Instead the flexors and synergists of the upper extremities are antigravity, while the extensor group is prograrvity musculature. It is important to keep these differences in mind because most of the research data and terminology (in relation to reflexes, movements, and pro- and antigravity musculature) refer to quadrupedal animals, and therefore are not always directly applicable to humans.

If future research confirms that GTOs are much more numerous in antigravity muscles than in progravity (2), then the GTO may play an important role in monitoring muscle tension in relation to the forces of gravity. In this respect the GTO and the gravity receptors of the vesticular system (the maculae of the utricle and sacculus) undoubtedly are involved in parallel processing of feed-forward and feedback input to interneurons and lower motoneurons (LMNs) concerning gravity and muscle tension in relation to the position of the body in space. However, in the case of paralleling

processing, different kinds of inputs would be influencing the interneurons and lower motoneurons; that is, the vestibular input (especially from the lateral vestibular nucleus) would be facilitating antigravity muscle tone, while the GTO might be inhibiting or facilitating this tone. This would enable each system to have either a "counterbalancing" or a parallel effect upon the interneurons and LMNs and would allow for delicate adjustment of muscle tension and movement in relation to gravitational forces.

## Functional Relationships

The afferent fibers from GTO receptors to the CNS are classified as slowly adapting mechanoreceptors or Group Ib (Gp.Ib) fibers. These are heavily myelinated fibers having a conduction velocity (in humans) of about 50 to 85$^{+}$ M/sec (10, 20). Group Ib fibers do not synapse directly upon $\alpha$ mns; rather, they act disynaptically or polysynaptically via Ib inhibitory and facility interneurons (2, 8, 12, 19) (see Figures 3-7). The Gp.Ib input, via the Ib inhibitory and excitatory interneurons, was once believed to be limited to autogenic inhibition, that is, inhibition of homonymous muscles (agonists) and synergists, and facilitation of heteronymous (antagonistic) muscles. Sufficient evidence has accumulated today to indicate that GTOs and Gp.Ib fibers are also involved in cocontraction and in autogenic excitation; that is, they facilitate agonists and synergists, and inhibit antagonists (8, 11, 13) (see Figures 3-5). This, of course, is different from what was once believed to be the "real" function of GTOs, autogenic inhibition.

In order to understand how

**Figure 3**
Simplified circuitry illustrating how Golgi tendon organ–Group Ib fibers, in conjunction with central nervous system regulation of Ib interneurons, can cause autogenic inhibition (See text for details.)



Supraspinal and
Propriospinal Influences

GTO–Gp.Ib
Re-afferents
from
Agonists & Synergists

Lower motoneuron pool:

Ib inhib. interneurons ●—<
Ib excit. interneurons ○—<
α motoneurons ⊙—→

Agonists & synergists
inhibited.
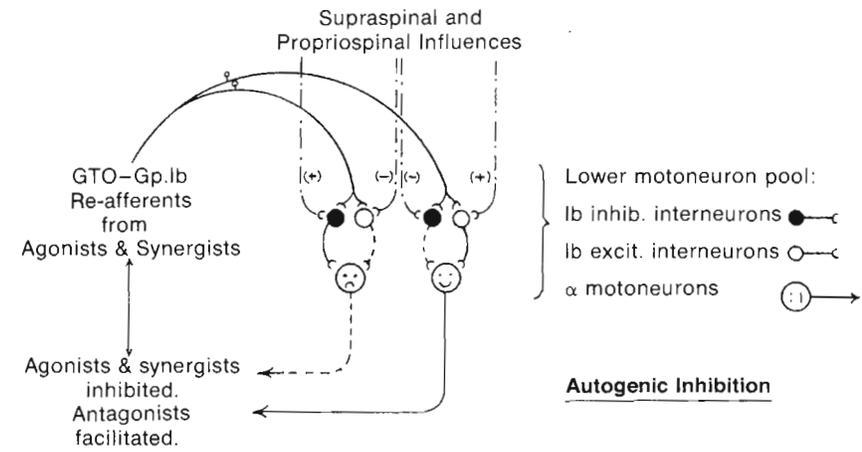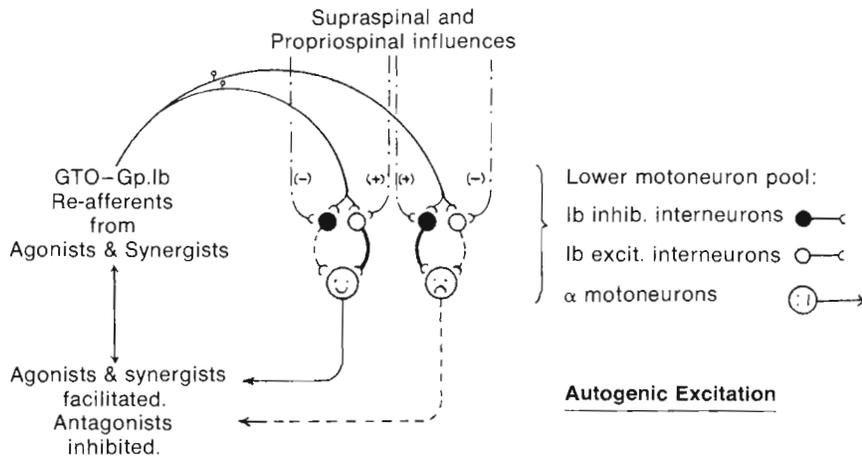Antagonists
facilitated.

**Autogenic Inhibition**

**Figure 4**
Simplified circuitry illustrating how Golgi tendon organ–Group Ib fibers, in conjunction with central nervous system regulation of Ib interneurons, can cause autogenic excitation (See text for details.)



Supraspinal and
Propriospinal influences

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Lower motoneuron pool:

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α motoneurons ⊙—→

Agonists & synergists
facilitated.
Antagonists
inhibited.

**Autogenic Excitation**

GTOs function in light of today's theories, one has to look at the older research. When the GTO-Gp.Ib fibers were originally investigated, the animals that were used were not only anesthetized, but they were spinalized, that is, only an isolated segment of the spinal cord was investigated. (Unfortunately, this technique is still being used today.) Also the animal was held in a rigid contraption while various measurements were made. Later experiments used either decerebrate cats, who were under lighter anesthesia and were less confined, or cats and monkeys without gross CNS lesions (3, 8, 11-13, 21). During this same period various operative techniques, recording equipment, and microprobes were being refined, and in-

vestigators who were working on the cells of the CNS began to unravel more of the functions and intricate connections between interneurons, supraspinal and propriospinal pathways, and the lower motoneurons. This combined knowledge led to new theories on the structure and function of the nervous system. Even though it will be many decades before everything is understood at just the spinal cord level of the nervous system, most investigators today agree that the final determinant of how receptors function depends on the thresholds of the interneurons of the CNS, that is, the delicate balance between excitatory and inhibitory synapses impinging upon these interneurons in relation to the moment-to-moment needs of the organism. In other words, if the animal suddenly detects something threatening, its immediate reaction may be to "stiffen up" or tense almost all of its muscles (agonists, synergists, and antagonists) in order to present itself as a formidable foe prepared to spring to the attack. If GTOs functioned only in autogenic inhibition, this posturing would be impossible, for in theory, as the animal tenses up, the GTO-Gp.Ib fibers would function to "un-tense" all of the tensed muscles.

For ages it has been known that animals, including humans, can optimally tense agonists, synergists, and antagonists concurrently and not experience autogenic inhibition. (The muscle spindle, along with other receptors, were implicated as being primarily responsible for this kind of activity even though this was not a fair explanation of this physiological event.) However, this example illustrates that the CNS has the final

word; that is, it is the central generator or regulator that determines the way in which receptors will influence function, and not the other way around (that the receptors and their afferents are the ultimate determinants of function) (3, 10, 11, 17).

Next, one has to consider a common "human factor" that often interferes with advances in understanding how receptors and/or the CNS functions. For example, once investigators "locked in" to an idea that GTOs-Gp.Ib fibers only functioned in autogenic inhibition, they failed to look for other explanations. Instead, they continued to refine their theories to prove that they were correct in their assumptions. It was not until the mid 1970s that new theories began to appear in the literature. This was due in part to the realization that the GTO was a much more complex receptor than was originally surmised, especially in regard to its structural relationships with different kinds of muscle fibers belonging to different motor units. Also, investigators using newer techniques found that the GTO was extremely sensitive to all degrees of contraction tension-forces (and especially to sudden small motor unit recruitment), and it was much less sensitive to stretch, especially passive stretch.

New experimental evidence demonstrated that there were alternate ways in which the GTO-Gp.Ib fibers could react in relation to central programming by the CNS. These "alternate ways" concerned (a) the supraspinal pathways (a minimum of 10 to 12 descending excitatory and inhibitory tracts are recognized as being important in regulating the thresholds of the interneurons through

**Figure 5**

Simplified circuitry illustrating how Golgi tendon organ–Group Ib fibers, in conjunction with central nervous system regulation of Ib interneurons, can cause co-contraction (See text for details.)
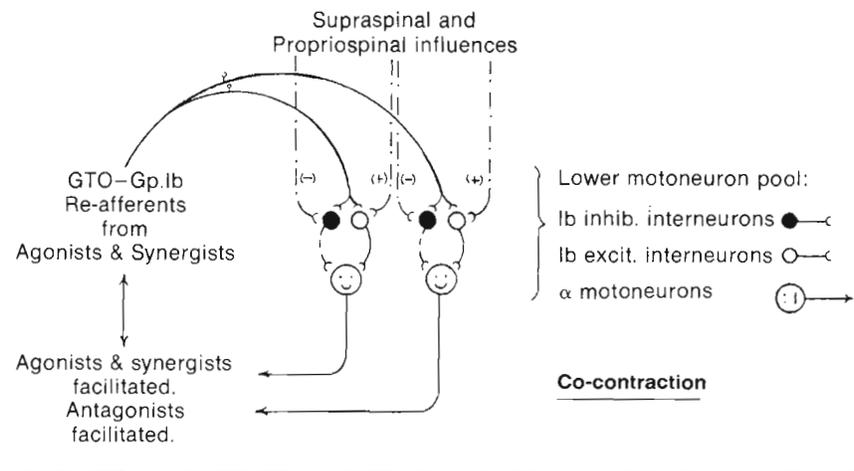


which the GTO-Gp.Ib fibers function) (3, 10, 11, 17); (b) the ipsilateral and contralateral propriospinal tracts that influence the thresholds of interneurons and the lower motoneuron pool (LMN); and (c) the afferents and feedback-afferents from the peripheral nervous system, which have long been known to influence the thresholds of the LMN pools either directly or indirectly via spinal interneurons (see Figure 6). In earlier investigations on spinalized or decerebrate cats and monkeys, the regulation by the supraspinal and propriospinal systems on cell thresholds of interneurons and LMNs was quite abnormal. In animals without lesions and with all systems intact (though the animals were lightly anesthetized), the GTO-Gp.Ib fibers actually were tension-force transducers or recorders, that is, they only informed the CNS about the amount of force (tension) being generated in extrafusal muscle fibers. The GTO-Gp.Ib fibers did not determine if the feedback re-
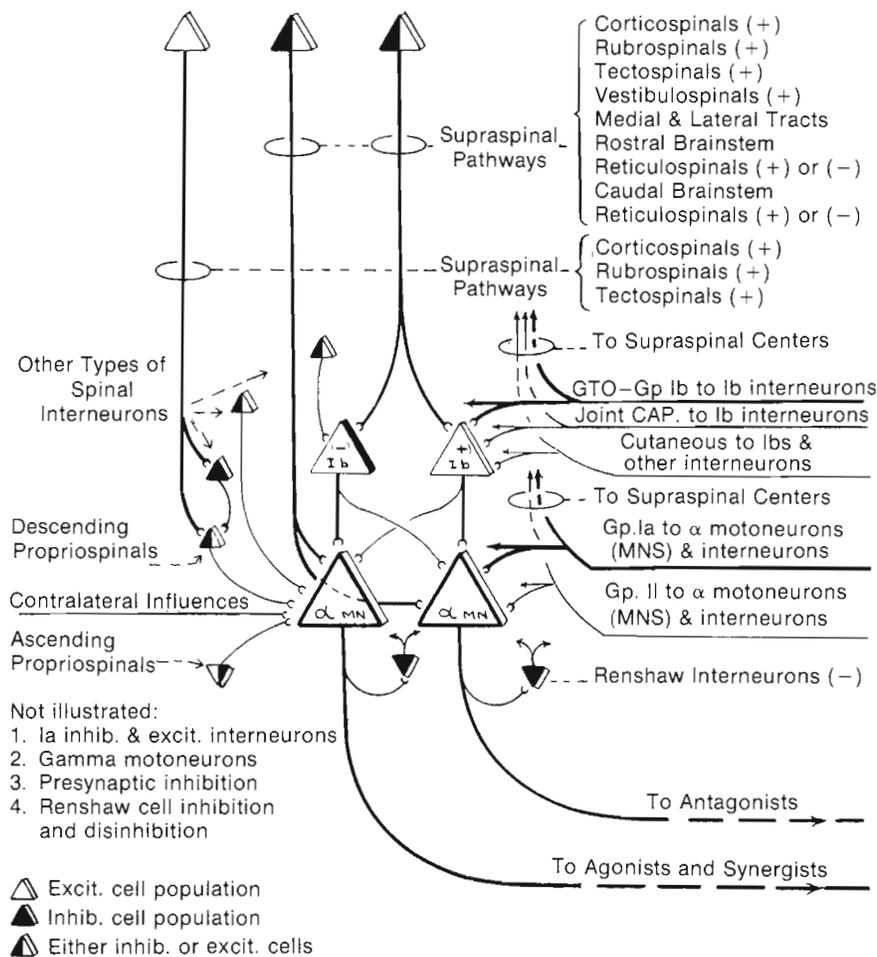
sulted in reducing tension (autogenic inhibition) or increasing tension (autogenic excitation). Rather, the central control mechanisms, monitoring all of the signals both from within and outside of the CNS, regulated the amount of force by changing (i.e., exciting or inhibiting) the thresholds of the Ib interneurons and LMNs. In this way the CNS determined the amount of force necessary to perform a given task, and the GTO-Gp.Ib fiber system merely continued to monitor the results of these changes and send new signals to the CNS so that the most appropriate force-tensions would be generated for a given task.

One might equate the GTO-Gp.Ib system to the brake pedal on a car. The brake by itself cannot decrease or increase the speed of the vehicle, but when controlled by the driver (the CNS), it can either decrease the speed, stop the vehicle, or, by letting up on the brake, enable the vehicle to resume speed.

In some cases the GTO and at

Supraspinal Pathways
- Corticospinals (+)
- Rubrospinals (+)
- Tectospinals (+)
- Vestibulospinals (+)
- Medial & Lateral Tracts
- Rostral Brainstem Reticulospinals (+) or (−)
- Caudal Brainstem Reticulospinals (+) or (−)

Supraspinal Pathways
- Corticospinals (+)
- Rubrospinals (+)
- Tectospinals (+)

To Supraspinal Centers

GTO–Gp Ib to Ib interneurons

Joint CAP. to Ib interneurons

Cutaneous to Ibs & other interneurons

To Supraspinal Centers

Gp.Ia to α motoneurons (MNS) & interneurons

Gp. II to α motoneurons (MNS) & interneurons

Renshaw Interneurons (−)

To Antagonists

To Agonists and Synergists

Other Types of Spinal Interneurons

Descending Propriospinals

Contralateral Influences

Ascending Propriospinals

Not illustrated:
1. Ia inhib. & excit. interneurons
2. Gamma motoneurons
3. Presynaptic inhibition
4. Renshaw cell inhibition and disinhibition

△ Excit. cell population
▲ Inhib. cell population
◮ Either inhib. or excit. cells

least two other known receptors (low threshold joint capsule receptors and low threshold cutaneous receptors) are involved in parallel processing of information, which is limited to or results in autogenic inhibition. These are related to "protective functions" (22, 23). If a muscle develops excessive tension or meets sudden resistance to an immovable object, or if a body part reaches its physiological limit in relation to range of motion, these different receptors send parallel excitatory signals to the CNS, which can override other influences so that autogenic inhibition results. The excitatory input from these receptors synapses on the Ib inhibitory interneurons, which inhibits the α mns of the agonist and synergists and concurrently signals the Ib excitatory interneurons to facilitate the α mns of antagonists (see Figure 7). This is a rather powerful reflex, and it helps prevent injury in the associated muscles, tendons, ligaments, and joints.

Low threshold pain receptors located throughout the body in muscles, tendons, joint capsules, and skin probably act in the same way, reflexively inhibiting further movement and facilitating movement in the opposite direction (the withdrawal reflex), or causing cessation of movement (2, 14, 23). In many cases the pain may not be liminal, that is, it need not reach consciousness in order to cause autogenic inhibition. Subliminal pain sensations acting at a spinal or brainstem reflex level are sufficient to cause diminished strength and/or the inability on the part of the individual to recruit additional motor units that would be needed to perform an activity. This is commonly seen in arthritic conditions, subluxations, postoperative states, trauma cases, and in elderly persons.

The GTO appears to function in reducing muscle fatigue (2, 3, 17). As tension builds up in a group of muscle fibers, the GTO and Gp.Ib afferents send an increasing number of signals to the CNS. This results in autogenic inhibition to the muscle fibers under tension. This momentarily reduces the tension in these fibers and concurrently reduces the amount of feedback being received by the CNS from these muscles. This momentary reduction in feedback enables more tension to develop in these muscles (via the α-γ co-activation system) resulting in less fatigue and/or a renewed strength in these muscles.

Another function that was once believed to be one of the primary actions of the GTO has now been disproven. This is the "clasp-knife" reflex. It was theorized that the sudden "giving away" response of a passively stretched group of muscles was the result of activating the "high threshold" GTO recep-

tors and Gp-Ib afferents, which, in turn caused autogenic inhibition and/or the sudden relaxation of the stretched muscle. This theory correlated with the belief that GTOs functioned only as high threshold stretch or tension receptors. Both ideas have since been disproven (2, 11, 18). The GTO is now known to be a tension receptor capable of monitoring all thresholds of muscle tension. Likewise, the clasp-knife reflex (an abnormal response found only in cases of upper motoneuron lesions) is now believed to be the result of afferent input from Gp.II fibers coming from the muscle spindles and perhaps thinly myelinated (C fibers) subserving pain (1, 11, 14, 18).

Undoubtedly the final word today on the functions of the GTO, especially in relation to how it interacts with other receptors in parallel processing and with the CNS, is not known. However, investigators are realizing this receptor is more complex than was originally thought (1-4, 6, 8, 11, 12, 15, 22). This should come as no surprise. It is well known that the circumstances of the moment (in regard to position, muscle tone, degree of alertness, innate as well as stored memories, and/or various kinds of nervous system lesions) are critical factors in determining how different receptors function in relation to the immediate demands of the central control mechanisms of the nervous system (11-13, 16). Does this new information place some of our therapeutic techniques into question? Fortunately the answer is no. Therapists do not use one kind of receptor to gain a desired response. Nor do they base their theories on the supposed function of a single type of receptor. Rather,

**Figure 7**
Simplified circuitry showing Golgi tendon organs, joint capsule receptors, and low threshold cutaneous receptors functioning reflexively in autogenic inhibition in order to prevent injury in muscles, tendons, ligaments, and joints (See text for details.)
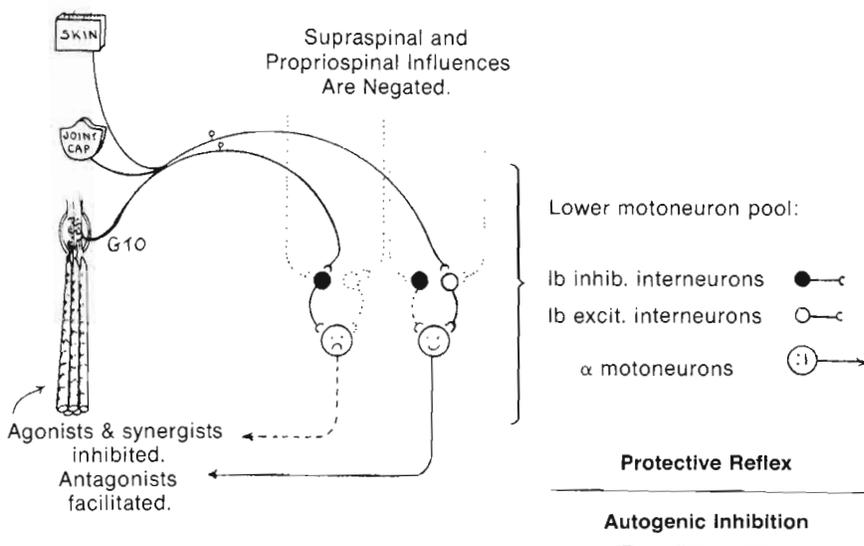


therapists work with the entire body and nervous system using appropriate multisensory stimuli, active movement patterns, and positions for minimizing deficits and maximizing more normal responses. The only precaution of note is to realize that the GTO and Gp.Ib fiber system is not limited to autogenic inhibition as was once postulated in the research literature. Perhaps it is wiser to think of the GTO as being part of a group of mechanoreceptors or proprioceptors involved in parallel processing that are important for monitoring all thresholds of muscle tension, length, and velocity of movement in relation to spatiotemporal orientation and the immediate demands of the organism within its environment.

In conclusion, GTOs are encapsulated receptors located (in mammals) at the myotendinous or musculoaponeurotic junction of extrafusal muscle fibers. The function

of these receptors was once considered to be limited to autogenic inhibition, that is, inhibition of agonists and synergists and facilitation of antagonists. Also the GTO was thought to be a high threshold tension receptor and was said to be responsible for the clasp-knife reflex in individuals with upper motoneuron lesions. Today these theories have been disproven. New research has demonstrated that GTOs monitor all degrees of muscle tension (being most sensitive to tension forces generated by muscle contraction), while the Gp.II fiber from the muscle spindle is now implicated in the clasp-knife reflex. In some situations, such as pain, muscle fatigue, excessive muscle tension, sudden resistance to movement, or reaching the limit of range of motion of a joint, autogenic inhibition does occur on a reflex level. The GTO-Gp.Ib fibers, along with other receptors and their afferents, are be-

lieved to be involved in this reflex. However, under normal conditions, the GTO-Gp.Ib fiber system, in concert with the CNS, can be involved in either autogenic excitation, autogenic inhibition, or co-contraction depending on the CNS's regulation of the excitatory or inhibitory balance of the Ib interneurons, and the immediate needs of the organism.

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