Arthrogenic Muscle Inhibition: A Limiting Factor in Joint Rehabilitation

J. Ty Hopkins and Christopher D. Ingersoll

Objectives: To define the concept of arthrogenic muscle inhibition (AMI), to discuss its implications in the rehabilitation of joint injury, to discuss the neurophysiologic events that lead to AMI, to evaluate the methods available to measure AMI and the models that might be implemented to examine AMI, and to review therapeutic interventions that might reduce AMI.

Data Sources: The databases MEDLINE, SPORTDiscus, and CIHNAL were searched with the terms *reflex inhibition, joint mechanoreceptor, Ib interneuron, Hoffmann reflex, effusion,* and *joint injury.* The remaining citations were collected from references of similar papers.

Conclusions: AMI is a limiting factor in the rehabilitation of joint injury. It results in atrophy and deficiencies in strength and increases the susceptibility to further injury. A therapeutic intervention that results in decreased inhibition, allowing for active exercise, would lead to faster and more complete recovery.

Key Words: reflex inhibition, Hoffmann reflex, effusion model, cryotherapy, transcutaneous electrical nerve stimulation

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Current joint rehabilitation depends on active exercise. Early active exercise in the rehabilitative process is essential for decreased healing time, increased vascular ingrowth, quicker regeneration of scar tissue, and stronger ligament and tendon healing.[1] The process of early active exercise in joint rehabilitation is significantly hindered by the patient's inability to contract surrounding musculature, as is common after joint injury. This diminished ability to contract, or inhibition, is termed arthrogenic muscle inhibition (AMI). The muscle shuts down even though it is not damaged.

The purposes of this review are to (1) discuss the clinical implications of AMI, (2) explore the neurophysiological factors associated with AMI, (3) discuss the methods to measure AMI in a research setting, (4) discuss models used to measure AMI, and (5) explore several interventions that might slow or block AMI.

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The Nature of the Problem

AMI is a presynaptic, ongoing reflex inhibition of musculature surrounding a joint after distension or damage to structures of that joint. It is a natural response designed to protect the joint from further damage.

Pain and disuse are often blamed for the inhibition and muscle atrophy after joint injury. However, AMI results from activity from many different joint receptors, which act on inhibitory interneurons synapsing on the motoneuron (MN) pool of joint musculature.[2,3] The information from inhibitory interneurons decreases the ability of recruitment within the MN pool and therefore decreases the force of any contraction stemming from that MN pool. Free nerve endings and specialized nociceptors might play a role in inhibition, but the primary effect seems to stem from mechanoreceptor activity.[4-7] With this in mind, the limiting factor in joint rehabilitation is not necessarily only pain but also the neurophysiological response of joint mechanoreceptors.

The role of active exercise in joint rehabilitation is well understood, and clinicians use techniques that can strengthen joint musculature while maintaining joint stability. We can help athletes regain range of motion and general fitness, but a factor that often eludes clinicians is functional, bilateral strength. These athletes often return to competition deficient in strength and neuromuscular control, and this results in an increased susceptibility to injury.[6] This is caused by inhibition of joint musculature, even though the musculature is not itself damaged.

AMI takes a central role in the injury cycle (Figure 1). After joint injury, an athlete experiences deficits in range of motion and movement (immobilization). Immobilization could result from swelling, pain, muscle spasm, and/or the surrounding joint musculature's inability to contract at a normal level. This leads to muscle wasting and weakness. Finally, these factors result in an increased susceptibility to joint injury. AMI plays a central role in this cycle,

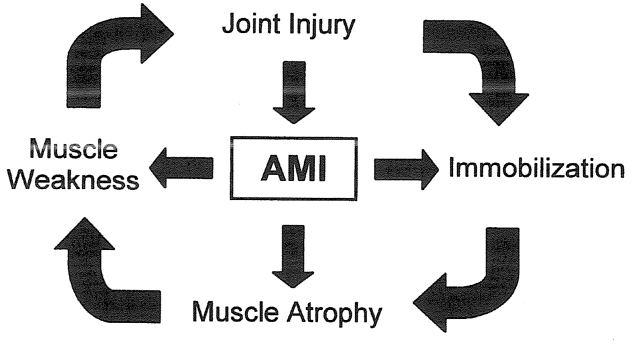

Figure 1 The injury paradigm. Adapted with permission from Stokes and Young, 1985, *Clinical Science,* **67,** 7-14.[4] © the Biochemical Society and the Medical Research Society.

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directly affecting each of these factors: immobilization, muscle wasting, muscle weakness, and increased susceptibility to further injury.[6]

This injury cycle can continue if it is not blocked or slowed (AMI) in some way. AMI is important; it is a built-in mechanism that forces an injured patient to rest and not aggravate the injury. However, in a controlled environment for rehabilitation, active exercise is necessary for clinicians to intervene in the injury cycle. A therapeutic intervention that could block or slow AMI would allow clinicians to return an athlete to participation with no strength or kinesthetic limitations when healing has occurred, instead of laboring to return the athlete in a deficient state.

Neurophysiological Factors Associated With AMI

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The spinal cord consists of a complex system of channels relaying information in electronic form from several parts of the body. The central and peripheral nervous systems work together to gather, transmit, and process information from many different neurophysiological systems in order to coordinate movement. From a neurophysiological perspective, joint movement provides supraspinal centers with constant information about environment, position, and movement.[8] The joint transmits information regarding each of these factors. Change in afferent input to the spinal cord from the joint appears to be the most influential factor associated with AMI.[2,3] Increased afferent activity created by joint effusion has resulted in quadriceps inhibition.[9-13] Additionally, rupture of the ACL has been argued to result in quadriceps inhibition.[6,14] A review of the processes that result in changes in afferent activity and many factors associated with these processes follows.

Joint Receptors

Receptors are specialized cells or subcellular structures that change their properties in response to specific stimuli of various types.[2] Receptors that respond to physical or mechanical stimuli are termed mechanoreceptors. Mechanoreceptors act to transduce energy from one form, for example tension, into a specific nerve signal.[15] Receptors that transduce information about the relationship between body segments are proprioceptors.[2] Joint receptors are mechanoreceptors. They can also act as proprioceptors. Therefore, joint receptors have 2 major functions: to provide position sense or information about the relative configuration of body segments and to initiate protective reflex mechanisms that protect and help stabilize the joint.[16]

The knee joint contains 3 types of mechanoreceptors: Ruffini endings, Golgilike endings, and Pacinian corpuscles.[11,15,17-19] Ruffini endings are slowly adapting receptors that have been identified in the joint capsule,[15] the anterior cruciate ligament (ACL),[17-19] and in the perimeniscal tissue and outer third of the meniscus.[20] These receptors have a very low threshold, and they respond to very slight changes in ligament tension and capsular

pressure. Ruffini endings adapt very slowly to a stimulus and are therefore capable of a prolonged period of discharge.[18] It is suggested that these receptors play a role in signaling proximity of the joint to its range-of-motion limitations.[18,19] It is also apparent that these receptors are active during capsular pressure from joint effusion.[12]

Golgilike receptors are morphologically distinct from Ruffini endings, resembling tendon organs. These receptors are found primarily in the ligaments of the knee.[8] They fire rapidly on first movement of the joint and then slow to a steady discharge. These receptors help provide information about joint position.[8]

The Pacinian corpuscle, like the Ruffini ending, has been found in the joint capsule,[15] the ACL,[17-19] and the perimeniscal tissue and outer third of the meniscus.[20] This receptor, unlike the others, adapts quickly to a stimulus. Any movement of the joint, regardless of position, activates it.[18] Its brief, high-velocity discharges indicate joint acceleration and deceleration.[15]

Free nerve endings are nonspecialized, nonencapsulated, unmyelinated (or finely myelinated) receptors. They function as pain receptors[8,15] and probably provide a crude awareness of initial joint movement.[21,22] Although these nerve endings are found throughout the joint tissue and are sure to be active with any joint damage, it is not known whether they play a significant role in AMI.

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Afferent Pathway to the Spinal Cord

Most receptors are specialized endings to sensory nerve fibers. When the receptor is stimulated it allows for a change in membrane potential, depolarizing the membrane and creating an action potential. The action potential travels along the dendrite until it reaches the cell body of the nerve fiber. Sensory nerve cells contain a cell body located in a dorsal root ganglion very close to the spinal cord. The cell body then projects through the dorsal horn of the spinal cord, where it can make connections with several different types of neurons.[2,3]

The sensory distribution of specific nerves of the knee joint has been documented.[11,23] Two consistent groups of nerve fibers have been identified: a posterior group and an anterior group.[11,23] The posterior group consists of the largest nerve supplying the knee, the posterior articular, and a branch from the obturator. The anterior group of nerve fibers includes the articular branches of the femoral nerve, common peroneal, and saphenous nerves.

The posterior articular nerve is a branch of the tibial nerve. It separates in the popliteal fossa, diving lateral to the popliteal vein and perforating the popliteal ligament. Branches supply the posterior capsule, the outer portions of the menisci, the cruciate ligaments, and even the infrapatellar fat pad. The terminal portion of the obturator nerve follows the femoral artery into the popliteal fossa, receiving information from the posterior portion of the joint capsule.

The anterior group of afferent fibers supplies the anteromedial and anterolateral joint capsule with sensory fibers. The terminal portions of the femoral nerve supplying the quadriceps muscles make up the articular branches. The branch to the vastus medialis supplies a wide area of the anteromedial capsule. The branch to the vastus lateralis then supplies the superolateral capsule. Deep in the suprapatellar pouch is the terminal portion of the branch to the vastus intermedius. A branch from the common peroneal nerve, the lateral articular nerve, arises from the posterolateral joint line to supply the inferolateral capsule and the collateral ligament. The recurrent peroneal nerve enters the joint on the anterolateral joint line. The infrapatellar branch of the saphenous nerve (a branch of the femoral nerve) enters the area between the sartorius and gracilis tendons supplying the inferomedial capsule, the patellar tendon, and the skin over the anterior portion of the knee.

The specific location of sensory nerves could be of particular importance when devising treatment techniques that could affect the magnitude of AMI. If the aim of slowing or modifying AMI is to target the afferent fibers involved, then information regarding the specific distribution and pathway is important.

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The Interneuron

Once the sensory fiber enters the dorsal horn of the spinal cord, it usually branches to synapse on several interneurons.[3] An interneuron can be defined as a neuron receiving information from a neuron and transmitting it to other neurons.[2] A single neuron can receive information from many other neurons and project to many different neurons. Most interneurons have axons that branch widely, ascending or descending in the white matter over distances of 2–3 segments before reentering the gray matter.[24] Organization might be something that one would expect from the central nervous system, but the network of interneurons and the incredible amount of information from sensory fibers and supraspinal centers traveling through these interneurons make this network nearly impossible to completely comprehend. With this in mind, a general explanation of interneurons in the spinal cord follows.

Interneurons are the intermediates of pathways to α- and γ-MNs and autonomic efferent neurons and to ascending pathways. They receive projections from sensory afferent fibers, descending fibers, and other interneurons. It would seem that interneurons are merely relay stations, but the existing information indicates that they have an important integrative function.[24] The net effect of all information arriving at the interneuron is expressed in the inhibitory or excitatory response of the MN pool.

Several interneuronal systems are well understood. One of these, the Ia inhibitory interneuron, is active during reciprocal inhibition. Muscle-spindle afferents synapse directly on the MN pool of the affected muscle, causing

it to be excited. They also synapse on inhibitory Ia interneurons. These interneurons have projections to the antagonist MN pool, resulting in inhibition of that MN pool.[3] The interneuron also receives inputs from the corticospinal, rubrospinal, and vestibulospinal tracts.[24] It also receives input from Renshaw cells, which act to inhibit the inhibitory interneuron. This is termed disinhibition.[2] The net effect of the Ia inhibitory interneuron depends on the spatial facilitation between these convergent systems. This means that the postsynaptic potential induced by an input fiber can only cause a change relative to the summation of all other inputs. The intricate involvement of all involved systems makes this simple interneuron somewhat difficult to understand.

Ib excitatory and inhibitory interneurons receive information from Golgi tendon organs, joint and cutaneous afferents, and from some Ia fibers. They also receive information from corticospinal and rubrospinal tracts and from at least 2 descending systems originating in the brain stem.[24] When the tendon organ fires, this results in inhibition of the agonist and excitation of the antagonist. Joint receptors appear to stimulate the Ib inhibitory interneuron.[24]

Traditionally, we picture descending activity as being modulated by afferent activity.[24] It is the interneuron in which this integration takes place, which in turn affects MN excitability. The net effect resulting from integration of information at the interneuron is considered state dependency. Because the integration is taking place in an interneuron instead of all factors coming together at the MN, the net effect of the interneuron could be considered a change of state.

Ascending and Descending Information

When sensory information enters the spinal cord through the dorsal roots, the afferent fibers branch and ascend via the dorsal column pathway. The dorsal column pathway also receives fibers from other interneurons. The ascending fibers terminate in the medulla, continuing via the medial lemniscus to the ventroposterior-lateral nucleus of the thalamus and on to the cerebral cortex.[2]

Descending pathways are more involved. They are arranged into spinal tracts that carry specific information from a supraspinal center. This is not intended to be a comprehensive review of all descending pathways, but rather a simple review of some of the pathways involved in modification of AMI.

The corticospinal tract contains approximately 1 million axons, half from the motor cortex and half from the supplementary motor area.[2] Cortical neurons synapse on α-MNs, γ-MNs, and interneurons.[8] Cortical neurons carry motor information to the MN. Most facilitate, but some cortical neurons are inhibitory.[25] The inhibitory action of cortical neurons is discussed later in the article.

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Valeriani et al[26] suggested that central somatosensory pathways are functionally modified by lesions to peripheral mechanoreceptors. Because knee proprioception is very important in standing and gait, they suggested that the cortex is involved in complex spatial integration of articular proprioceptive inputs. However, others[27] have suggested that joint afferents do not change cortex activity. They have shown that primary cortex activity correlates directly with the EMG of the muscle after stimulation of afferents. From this, they concluded that joint position or torque is not linearly encoded by motocortical neurons. The answer to the question of the comprehensive role the corticospinal system has in AMI is not completely understood.

The vestibulospinal tract regulates postural reflexes through projections to MNs and interneurons.[3,24] These tract neurons play a major role in regulating postural reflexes. The vestibulospinal tract remains tonically active to help maintain upright posture.[3] Some authors[28-30] have noted that prior to voluntary movement, postural reflexes change. These changes are mediated at the interneuron by the vestibular system and the cerebral cortex.[2]

The rubrospinal tract is a smaller group of neurons that, like corticospinal tract neurons, innervate neurons controlling distal musculature.[3] The rubrospinal tract has also been implicated in inhibitory actions affecting interneurons.[31]

Iles[32] and Iles and Pisini[33] concluded that corticospinal and vestibulospinal neurons converge on inhibitory interneurons to inhibit the inhibitory mechanism. This is further supported by the work of Cervero et al,[34] who reported a constant tonic inhibition from supraspinal centers that inhibits normal afferent activity from causing a motor response. This descending tonic spinal inhibition is essentially inhibition of the inhibition that could result from stimulation of cutaneous receptors. Cervero et al[34] reported that during joint injury, descending tonic spinal inhibition is reduced, allowing for an increase in AMI.

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Types of Inhibition

Inhibition is a very common regulatory occurrence in the neuromuscular system.[3] AMI is one of many inhibitory mechanisms that help regulate musculoskeletal movement. In this section we discuss the basic types of inhibition involved in the inhibitory processes, namely postsynaptic and presynaptic inhibition, and inhibitory processes that could affect AMI. Finally, we discuss evidence supporting the concept of AMI.

Inhibition in the nervous system is either postsynaptic or presynaptic. Synapses between neurons or between neuron and membrane are either excitatory or inhibitory. Both excitatory and inhibitory processes result in the release of a neurotransmitter at the terminal endplate. The neurotransmitter then traverses the synaptic cleft to bind to a specific receptor on the postsynaptic membrane, causing an excitatory or inhibitory potential at

the postsynaptic membrane. If the neurotransmitter is an inhibitory neurotransmitter, the binding to the specific site causes ion channels to open that hyperpolarize the membrane, making it more difficult for the combined action of all synapses to generate an action potential.[2,3] This is postsynaptic inhibition.

The neurotransmitter that is believed to be most involved in postsynaptic inhibition involved with AMI is γ-aminobutyrate (GABA).[35] This neurotransmitter can bind to GABAa or GABAb receptors. Binding to GABAa receptors increases the permeability to chloride ions, whereas binding to GABAb receptors increases conductance of potassium channels and decreases calcium currents.[35]

Presynaptic inhibition is generally caused by a decrease in neurotransmitter release from the presynaptic terminal.[2,3] The purpose of presynaptic inhibition is to decrease the effectiveness of just 1 type of neuron synapsing on the membrane.[2] Presynaptic inhibition can be more specific, whereas during postsynaptic inhibition the entire membrane is affected. The specific factor involved with decreasing the release of the neurotransmitter at the terminal presynaptic membrane is thought to involve interference with calcium influx at the terminal synapse.[3] Calcium is important in helping the vesicles that contain the neurotransmitter on the inside of the terminal bind to the membrane and allow for exocytosis.[36] Exocytosis is a method of transporting a substance across a membrane by engulfing it, fusing with the membrane, and releasing it outside the membrane.[36]

AMI is likely a combination of presynaptic and postsynaptic inhibition. All afferent and supraspinal fibers synapsing on the interneuron conduct their excitatory or inhibitory information. Other fibers synapse on the presynaptic membrane, resulting in presynaptic inhibition or excitation. The result of presynaptic factors on the neuron is then transmitted to the interneuron in an excitatory or inhibitory form. The net effect of the neurons synapsing on the interneuron is then mediated by postsynaptic neurotransmitters. The summation of all involved factors results in excitation or inhibition. The net result of the interneuron traveling to the MN is thought to be presynaptically mediated.[25,32,37]

Other inhibitory processes might also play a role in AMI. Recurrent inhibition is inhibition mediated by Renshaw cells found on the efferent loop near the α-MN. The Renshaw cell is excited by α-MN activity. It then inhibits the Ia interneuron, projecting to its synergists. The net result is an inhibition of the affected MN pool and its synergists and disinhibition of the antagonists. Renshaw cells are under central control, receiving information through descending fibers from the brain stem and cortical pathways, helping to modify the effect of these cells.[3]

Reciprocal inhibition is caused by Ia inhibitory interneuron activity. As discussed previously, muscle-spindle primary fibers respond to stretch, resulting in afferent activity that synapses on interneurons. One of these interneurons is the Ia inhibitory interneuron, stimulation of which results

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in inhibition of the antagonist muscle and its synergists.[3] Also involved in this loop is the γ-MN system, which is important in functional regulation of Ia receptors in the muscle during contraction. Johansson and colleagues[38-40] suggested that joint afferents have a greater effect on γ-MNs than α-MNs do and that stimulation of γ-MNs by joint afferents helps contribute to continuous adjustment of muscle stiffness around the joint, increasing the stiffness and stability of the joint. This mechanism is most prominent when the joint ligament afferents are stimulated.[40]

Knee-joint injury seems to inhibit the knee extensors and facilitate the flexors and its synergists. In sheep, stimulation of ACL mechanoreceptors caused increased hamstring activity and decreased quadriceps activity.[41] Johansson and colleagues[38,39] reported that a tonic force applied to the ACL produced increased hamstring and triceps surae activity. Others[9,11,12,42-46] have reported that increased pressure in the knee capsule decreases quadriceps MN-pool recruitment. We have also demonstrated that there is an increase in soleus MN-pool recruitment after knee-joint effusion.[47] Kariya et al[48] reported a 12% decrease in quadriceps cross-sectional area with no reduction in hamstring or adductor cross-sectional area in patients with chronic ACL pathology. It has also been shown that the quadriceps-to-hamstring ratio in osteoarthritic patients was decreased with no correlation to age, pain, or gravity. These authors concluded that reflex inhibition was the cause.[49]

Clinical observation suggests that the vastus medialis is the quadriceps muscle most affected by AMI. The results of Wise et al,[50] in a study using EMG biofeedback, support this observation. Others[11,12] have observed that the vastus medialis was inhibited with less fluid than any of the other quadriceps muscles during artificial effusion. Voight and Wieder[51] illustrated that the recruitment patterns changed in the quadriceps with patellofemoral dysfunction. They reported that the vastus lateralis fired before the medialis in the pathological condition. Others have also questioned selective muscle-fiber-type inhibition.[4,52] There is no evidence that any specific muscle fiber type is inhibited more than another.

AMI can be caused by increased afferent activity, evidenced by effusion, and an apparent lack of afferent activity, such as is the case with ACL rupture. Given the information presented, the former cause seems to be mediated by quadriceps Ib inhibitory interneurons. However, the AMI caused by ligamentous rupture is not so straightforward. Some authors[53-55] have suggested that mechanoreceptor damage disrupts a central postural control mechanism. Hoffman et al[54] showed this to be true in patients lacking afferent activity from the ACL. This same mechanism could be 1 explanation for AMI after ligamentous tearing.

Measuring AMI

AMI is simply a reduction in MN-pool recruitment. This can be measured at least 2 different ways: the voluntary force output of that MN pool or the

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product of neuromuscular recruitment of the MN pool. Each method has advantages and disadvantages.

Voluntary Force Measurement

Measuring voluntary force output of an MN pool is a simple measure that can be performed with little equipment. Decreased voluntary contraction is 1 of the final outcomes of AMI. The difference in a baseline maximum voluntary contraction (MVC) and an MVC after injury is essentially inhibition. Many investigators[4,13,54,56-59] have demonstrated a decrease in joint extensor torque and quadriceps force output after joint injury. DeAndrade et al[42] reported that voluntary ability to lift the heel off the table was impossible after injection of 10–200 ml of plasma into the knee joint. AMI is evident using this measurement method.

Several investigators[43,56,57] have compared force measurements of the injured leg to the contralateral, uninjured leg. Although this method of comparison has been used to show gross differences, it might not be totally accepted as a valid comparison. Crossed spinal pathways transmit information to the contralateral leg,[60] which might inhibit the joint musculature of the contralateral leg. Others[61,62] have confirmed this, showing that the unaffected leg is inhibited along with the affected leg.

Muscle-force comparisons are very simple to perform and have been used to measure inhibition, but these measurements also have some drawbacks. In order to effectively measure differences in voluntary force production, the subject must be willing and able to perform an MVC. The MVC measurement must also be accurate and reproducible. Although this issue has been debated in the past,[63,64] the injury factor makes this measurement even more difficult to accept. If a subject is asked to perform a voluntary contraction postinjury, there are psychological factors such as perceived pain and lack of confidence that could hinder his or her ability to perform an MVC. This measurement method also uses an entire group of muscles with aid from synergists that might or might not be inhibited. It is impossible to measure independent muscles separately. One last issue is that of obtaining a baseline measurement. If the contralateral leg comparison is not valid, then what do we have to compare the pathological measurement against? The interpolated-twitch technique is a possible solution.

The interpolated-twitch technique combines an MVC and an additional supramaximal external stimulus to make up for the inhibited portion of the MN pool. The technique allows for measuring AMI without a baseline torque measurement; however, its results are mixed.[65-70]

In theory, direct stimulation of the nerve innervating the involved muscle group during MVC recruits any fibers not in use, in essence maximizing use of the MN pool.[65,71] Many authors[65,70,72] have used a train of stimuli ranging in number from 4 at 20 Hz to 10 at 100 Hz. Herzog and Suter[66] reported inhibition in normal subjects and increased inhibition after injury with an eventual

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decrease after surgery, rehabilitation, and rest. Some authors[65,69] have found no inhibition in healthy subjects, and others[67,68,70] have reported no inhibition even after injury or surgery. The validity of this measurement is questionable.

When considering the enormous amount of torque generated by the quadriceps, and the amount of torque that can be generated even with a supramaximal stimulus, it is difficult to understand how changes in the MN pool can be detected with this method. Hales and Gandevia[72] reported that the magnitude of the force generated during a twitch is very small compared with the background force, and it can easily go undetected. In addition to using a train of stimuli, they suggested that force responses to single stimuli that show no superimposed twitch be averaged, in an attempt to help make the superimposed twitch more evident. Another problem with this measurement is that it still relies on a voluntary contraction, which might be difficult with an acute injury. Although this measurement method has problems, the benefit of obtaining a measure of AMI, without a preinjury measurement for comparison, makes it worthy of consideration.

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The Hoffmann Reflex

The Hoffmann reflex (H reflex) is a measure of MN-pool recruitment. Electrical stimulation of a mixed nerve evokes 2 distinct EMG responses from the affected muscle.[73] One response (Figure 2) to the stimulus is an action potential with a latency between 19 and 40 milliseconds, depending on the muscle. This is a result of primary afferent (Ia) stimulation, which in turn excites α-MNs in the anterior horn of the spinal cord. This response is the H reflex. As the intensity of the stimulus increases, more afferent fibers are stimulated, causing more MNs to be recruited within the MN pool. This is represented as an increased amplitude of the twitch of the affected muscle as measured by surface EMG. As the external stimulus intensity is increased even more, a second response (Figure 2) appears between 5 and 15 milliseconds. This response is a direct stimulation of efferent α-MN fibers, and it is termed the M response.

When the threshold for the efferent fiber is reached and an M response appears, the H-reflex amplitude decreases and eventually disappears (Figure 3). This does not mean that the stimulus is not causing a monosynaptic reflex; it means that it is not measurable. The reason for this is the antidromic effect.[2] The antidromic effect is essentially a depolarization of the MN from backward efferent traffic, which discontinues continuation of the reflex because of the refractory period. When the efferent or motor fiber reaches threshold, an action potential travels not only to the muscle but also back to the cell bodies in the anterior horn of the spinal cord. It results in the depolarization of the α-MN and occurs just prior to afferent volleys arriving at the MN. As a result of the depolarization, a refractory period prohibits the afferent activity from depolarizing the α-MN.[2,74] As the stimulus intensity increases, more fibers reach threshold, and eventually all efferent or

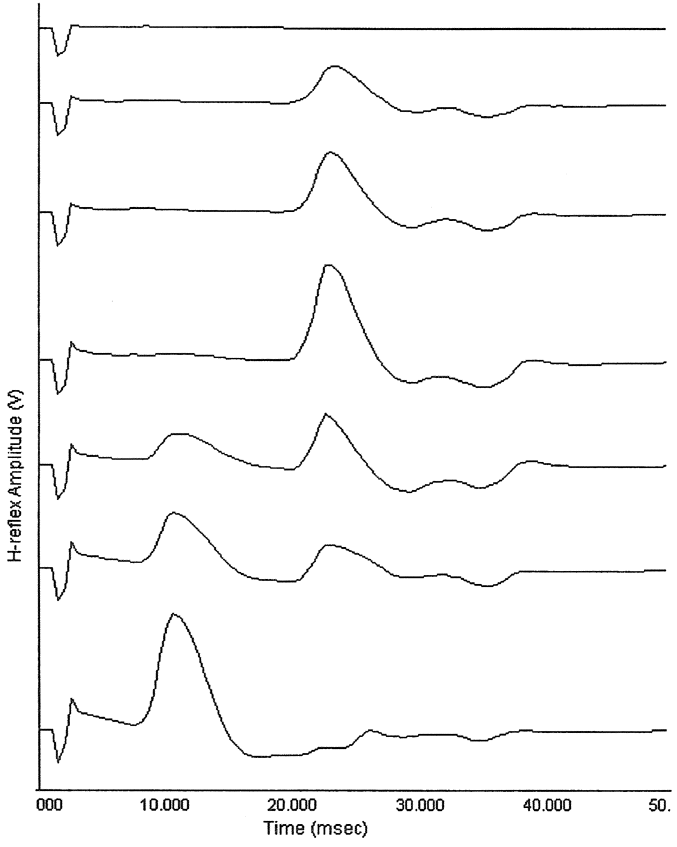

Figure 2 Seven quadriceps H-reflex measurements, each with an increase in stimulus intensity. H-reflex waves occur at approximately 22 milliseconds. M-response waves appear at 11 milliseconds.

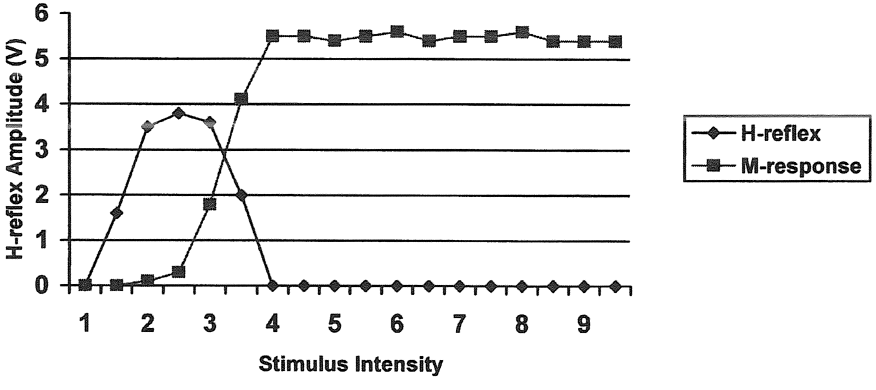

Figure 3 Example recruitment curve of H reflex and M response.

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motor fibers are depolarized. At this point the M response has leveled out and the H reflex is not apparent (Figure 3).

The amplitude of the H reflex represents the portion of the MN pool that was stimulated from afferent activity. Inhibition results in a decrease in MN-pool excitability. In other words, the threshold at which a stimulus can create an action potential changes, requiring a greater stimulus to initiate an action potential. Therefore, a decrease in the H-reflex amplitude after injury represents inhibition. This is the rationale behind this measurement technique. However, it can be very difficult to obtain reliable H-reflex measurements given the number of factors that affect H reflex. Following is a discussion of these factors and ways to normalize the H-reflex measurement.

H-reflex response can vary with such factors as head and body posture, foot position, eye movement, and remote muscle contractions.[75,76] The H-reflex measurement is also extremely variable among individuals.[77,78] Hugon[79] reported that maximum soleus H-reflex-to-M-response (H:M) ratios ranged from 35% to 75% in healthy subjects, and Mongia[80] reported quadriceps H:M ratios ranging from 6.4% to 62%. H-reflex differences have also been noted with differing age.[81] However, under controlled conditions and with the same subjects, the H-reflex measurement is very reliable between measurements[77,78,82] and between days.[83]

Very specific guidelines control for the external factors that influence H reflex. Hugon[79] promoted the use of a special reclining chair that supports the head and arms and maintains knee flexion at 120°. The subject lies supine with the hands at the sides, head resting on a pillow, and eyes open and staring at picture on the ceiling. The knee is supported at approximately 15° of flexion, and the heel rests in a supportive foam block to maintain foot position.[83]

The stimulating electrode is placed directly over the mixed nerve in order to elicit an H reflex in the absence of an M response.[79] Hugon[79] supports the use of an active pad over the nerve and a dispersive pad located on the opposite side of the body part. For example, the soleus H reflex is measured by applying an active electrode over the tibial nerve in the popliteal fossa and a dispersive electrode over the distal portion of the quadriceps. He suggested that this arrangement of electrodes is better than a longitudinal arrangement because (1) the stimulus artifact is less, (2) an anodal block is less likely to develop, and (3) selective stimulation of the nerve trunk is easier. Hugon[79] also suggested that a stimulus 1 millisecond in duration be used, because it is more selective for afferent axons with long utilization times. A stimulus duration with a shorter time favors activation of the α-MN fibers. Ten to twenty measurements were advocated as a standard to be used to find a mean measurement.[79] However, we have shown that 5 measurements are sufficiently reliable ($ICC_{3,1}$ = .932).[83]

The reliability of this measurement depends on placement of stimulating and recording electrodes. If the electrodes are not moved, and all other previously mentioned factors are controlled for, the same stimulus intensity will

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allow for detection of any changes in MN-pool recruitment. It is rarely the case, however, that the electrodes will not be moved. For this reason it is often necessary to normalize the H-reflex measurement. The most commonly used method of normalization is to express the maximum H reflex as a percentage of maximum M response.[10,79,80,84] The M response reaches its peak amplitude and levels off as all the efferent fibers are stimulated. This maximum M response represents the entire MN pool. Because the number of MNs in the spinal cord will not likely change, the M response is a stable measure to use for normalization.

Another method used to normalize H-reflex measures is the threshold of the H reflex to the threshold of the M response.[74,85] This is simply the ratio of the stimulus intensity required to elicit an H reflex to the stimulus intensity required to elicit an M response. It has been advocated as the appropriate measurement technique to evaluate subjects with spastic reflexes.[85]

Funase et al[74] suggested yet a third way to normalize the H-reflex measurement. They proposed that the best method is to measure the developmental slope of the H reflex. In other words, measure the changing rate in MN recruitment as a function of increased Ia input to the MN pool.

The most commonly studied muscle in H-reflex research is the soleus. The soleus H reflex is easily measured and interpreted compared with other muscles. Stimulation of the tibial nerve in the popliteal fossa allows for a latency between the M response and H reflex. This provides a clear separation between the 2 waves, ensuring that the M wave will not affect the amplitude of the H reflex. The quadriceps H reflex is also reported in the literature.[11,12,80] The M-response and H-reflex waves are much closer together in the quadriceps H-reflex measurement, possibly affecting the amplitude of the H reflex. Also observed during quadriceps H-reflex measurements is an F wave, which is an efferent antidromic depolarization of the MN.[2] The F wave appears at approximately the same latency as an H reflex, confusing the actual measurement. Use of the peroneals and ankle invertors has also appeared recently in the literature.[84,86] The peroneal H reflex seems to be confounded by the M response, making it very difficult to assess whether any changes are caused by MN-pool recruitment or merely a change in the antidromic affect of the stimulated motor nerve.

As has been discussed, the H reflex measurement requires great control, but it can be performed while the subject is resting. No voluntary effort is required, which could be extremely useful when examining a pathological population. It is also a very sensitive measure that can be used to detect small changes in MN-pool recruitment. A single muscle can also be investigated as opposed to a large muscle group. This can allow for investigation of selective inhibition of certain muscles in a muscle group.

The Soleus–Quadriceps Relationship

The soleus H reflex has been established as a reliable measure of MN-pool recruitment.[77,78,82] There is clear separation between the soleus M-response

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and H-reflex waves. The quadriceps H reflex is not as easily measured or interpreted. Because the quadriceps H reflex presents some difficulty in measurement and interpretation, there is some benefit in the relationship between these 2 muscles during AMI.[47] If there is a relationship between the quadriceps and soleus H reflexes, the soleus reflex could be used to detect changes in both of the muscle groups.

Meunier and colleagues[87,88] noted that heteronymous projections, or projections from 1 MN pool to another in the spinal cord, from the quadriceps to the triceps surae allowed the soleus to project 39% of the quadriceps excitatory postsynaptic potential after stimulation of the femoral nerve. That is, the triceps surae was excited by stimulation of the femoral nerve. They observed that stimulation of the femoral nerve produced Ia excitation followed by an inhibition of the soleus. This inhibition was suggested to be recurrent inhibition.[87] They further explained that the soleus activity was the result of the cocontraction of the soleus and quadriceps during the stance phase of locomotion. Other authors[89-92] have observed the same response when quadriceps Ia afferents are stimulated. This relationship seems to build the case that the soleus would be inhibited during AMI. The quadriceps have been shown to be inhibited,[10-12] and it seems likely that the soleus would also be somewhat inhibited.

Our work[47] has indicated that the soleus MN pool is not inhibited by knee effusion—in fact, it is facilitated. This might be explained by the interneuron that mediates AMI. The Ib interneuron is inhibitory to the quadriceps and excitatory to the hamstrings and its synergists.[24] The Ia interneuron that was stimulated in the work by Meunier and colleagues apparently does not affect the Ib inhibitory interneuron. Other evidence suggests that a pattern exists with AMI: inhibition of the quadriceps and excitation of the hamstrings and triceps surae.[38,39,41] Our work supports the excitation of the soleus during AMI.

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Models to Study AMI

It is difficult to investigate AMI and any interventions that might affect it in an injured subject, because AMI is confounded by the perception of pain, lack of a baseline measurement, and variability among subjects. Therefore, 3 models have been developed to study this phenomenon.

The Effusion Model

The effusion model was first used by DeAndrade et al,[42] who injected plasma into the knee-joint capsule of healthy subjects and observed a linear decrease in strength with increased volume. Several others[9,11-13,44,45,47] have adapted this model by using sterile saline rather than plasma. Spencer et al[12] found that the vastus medialis was inhibited with 20–30 ml of sterile saline, and the vastus lateralis with 50–60 ml. Iles et al[45] injected up to 100 ml of sterile saline and saw a decrease in MN-pool recruitment at rest and

during MVC. They suggested that the effusion should be controlled by regulating joint pressure. Other authors[43,68,93] have used a clinically effused knee and measured the change in MN-pool recruitment after aspiration.

A major advantage of the effusion model is that mechanoreceptors are active, whereas perceived pain and other injury factors are not.[47] The effusion model does not provide all the answers. Stokes and Young[4] claimed that when the clinically effused knee was aspirated, maximum voluntary activation increased, but not completely up to baseline measures. This demonstrates that there could be other factors that play a role in AMI.

A Pain Model

Pain is a component of injury that is very difficult to measure. Although some authors[94,95] have attributed AMI to pain, it is fairly apparent that pain is not the only factor involved. Other authors[4,6,52,96] advocate that pain has no association with AMI. Stokes and Young[4,96] reported that after meniscectomy, patients were inhibited well after pain had subsided, and that subjects were still inhibited after a 5-ml injection of 0.5% bupivacaine. Furthermore, artificial knee effusion results in significant inhibition of the quadriceps[10-13] in the absence of pain.[4,42,96] Clinically we see evidence to support both sides. There are athletes who appear to be inhibited by pain, and there are those who are functionally unaffected by pain. Neither of these views completely answers the question of the role of pain in AMI. More data are needed.

A pain model should be developed in order to determine the contribution of pain to AMI. Several such models exist, but most concentrate on cutaneous or muscle receptors. There is no model to date that can isolate joint receptors. The cold-pressor model uses ice to elicit pain.[97] The submaximal-effort tourniquet technique is an ischemic pain model.[98] A variation of the tourniquet technique is intramuscular injection of acid phosphate buffer to induce pain.[98] Delayed-onset muscle soreness is often used to induce muscular pain.[99,100] Further work needs to be done to develop this model.

An Atrophy Model

Atrophy is one of the negative effects of AMI, and it also directly affects muscle strength.[6] Although disuse undoubtedly causes morphologic muscle wasting, atrophy is also a direct product of AMI. Sectioning the dorsal root in cats and rabbits after induced arthritis prevented atrophy.[101] This demonstrates that afferent activity from the joint has a direct influence on muscle atrophy. Any intervention that will slow or modify AMI should have a direct effect on reducing muscle atrophy, but this will need to be tested for efficacy.

Perhaps it is possible to create atrophy independent of injury and determine its effects. One idea is to immobilize a joint and determine whether the immobilization itself has an inhibitory effect. Sale et al[102] examined the

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effect of 5 weeks of casting on the thumb. They observed no change in contraction times, half-relaxation times, or twitch tensions of the thenar muscles. Perhaps immobilization from casting or bracing does not induce inhibition, but it is important that a model be developed to study atrophy.

Therapeutic Interventions

The goal of our work with AMI is to find a way to eliminate or reduce its consequences. No studies to date have concentrated on trying to reduce AMI. Much information exists, however, on the effects of several modalities on muscle force, joint swelling, atrophy, and pain. The goal of this section is not to review all of this literature but to examine some of the effects of certain interventions that could be the key in turning off, or at least turning down, AMI.

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Pharmacological Agents

Spencer et al[12] reported that injection of lidocaine into the effused joint capsule negated the inhibitory process. Although this might seem to be the answer, it is a difficult ethical issue. AMI might be turned off, but all perceived pain is also turned off, preventing essential feedback to both the athlete and the clinician. Without pain further damage is inevitable. The gross nerve block created by this medication would also have an effect on kinesthesia, increasing the susceptibility to injury. Arvidsson et al[103] used a lidocaine epidural the day after ACL reconstruction and reported increased quadriceps MVC. Again, this is a gross reduction in AMI, perceived pain, and movement. This intervention would not allow for effective rehabilitation in a clinical setting. Other medications could have similar effects with the same consequences. It is necessary to find an intervention that can work on a local level, reducing AMI while not completely blocking pain and kinesthesia.

Topical anesthetic agents could have an affect on AMI. However, in one study a topical anesthetic spray and a placebo spray each increased soleus MN-pool recruitment when sprayed on the muscle, suggesting that the cutaneous innervation had a greater effect than the anesthetizing agent did.[104]

Cryotherapy

For a complete review of cryotherapy, refer to Knight.[105] Clinically, it can be observed that ice increases our ability to perform gradual, graded active exercise. Knight[105] attributes this to a decrease in residual pain. Residual pain is purported to be pain from damaged tissue and pressure from swelling on nerves, as opposed to sensory pain. Ice might, however, have a direct effect on AMI, allowing for increased active exercise.

Cryotherapy not only decreases general nerve conduction velocity, synaptic transmission, muscle spasm, and pain but it also has a definite slowing and eventual blocking effect on sensory nerve fibers. The relationship appears to be linear; the cooler the nerve becomes, the more slowly the impulse is carried. The temperatures that are feasible in clinical cooling will not block afferent activity.[105] An increase in action potential time results in a decreased peak-to-peak amplitude of depolarization at the interneuron, which could possibly result in decreased firing of the Ib inhibitory interneuron, resulting in increased voluntary activation of the MN pool.

It seems that any cooling of a mixed nerve would have the same effect on both motor and sensory fibers, but the results of studies exploring the effects of cryotherapy on strength and torque output are varied.[105-110] Furthermore, ice has no effect on proprioception[105] or agility.[111] These factors suggest that cooling could affect sensory fibers without affecting MN-pool recruitment.

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TENS

Transcutaneous electrical nerve stimulation (TENS) is another appealing intervention that could reduce AMI. TENS is advocated mostly as a pain intervention.[97,112] It stimulates cutaneous type I nerve endings and could compete for the same type I afferent fibers that carry information from joint receptors to the spinal cord. This makes it a viable candidate for treating AMI. Arvidsson and Eriksson[112] reported a small increase in voluntary activation of the quadriceps after TENS treatment in ACL-reconstruction and meniscectomy patients. This could be a result of decreased pain, competition for type I afferent pathways, or some other explanation not yet understood. Iles[32] reported that stimulation of cutaneous nerve branches and the sural nerve reduced presynaptic inhibition of the soleus. Brushing the distal dorsal and plantar surface of the foot also decreased presynaptic inhibition of the soleus.

This evidence lends support to the fact that TENS, applied to the proper cutaneous region, could decrease AMI. TENS might be beneficial applied over the dermatomal level of the femoral nerve roots instead of the injury area. This treatment area would be over the lower back instead of over the injured knee. Much work is yet to be done in this area.

Conclusions

AMI is a limiting factor in rehabilitation of joint injury. It results in strength deficits, often long after healing has occurred. It causes atrophy, adding to the injured athlete's deficits. AMI can lead to premature return to competition and increased susceptibility to reinjury. AMI prevents the injured athlete from performing the early active exercise necessary to help increase healing. A reduction in AMI would allow the injured athlete to perform active exercise in a controlled environment, facilitating healing and preventing decreases in

strength and muscle mass. It is likely that interventions capable of modifying AMI are already in use. With the proper measurement method and models, these interventions can be examined for their efficacy in reducing or even blocking AMI. From this a greater understanding of the process will be gained. More important, athletes will be able to return to competition after joint injury stronger and less susceptible to further injury.

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