Targeting Quadriceps Inhibition with Electromyographic Biofeedback: A Neuroplastic Approach

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ABSTRACT: Weakness of the quadriceps is a common occurrence in patients after knee injury or surgery; this weakness is due to a natural mechanism known as arthrogenic muscle inhibition. If inhibition of the quadriceps persists, it can become detrimental to a patient’s function and lead to additional pathologies. A number of therapeutic interventions have been used in the rehabilitation of these patients, but few have proven to be successful. Electromyographic biofeedback is one modality that has demonstrated positive outcomes in patients by restoring quadriceps function. However, the reason for the effectiveness of this modality has yet to be fully explained in the area of rehabilitation. Neuroplasticity is a phenomenon that has gained much attention in rehabilitation, and its potential continues to grow. After an injury, the brain has the ability to enhance recovery by strengthening its neural circuitry. Through rehabilitation, clinicians can use attentional strategies to foster neuroplasticity and promote the recovery of their patients. In this article we provide reasoning for the effectiveness of electromyographic biofeedback using the evidence of neuroplasticity. With this information, we hope to provide clinicians a rationale for using this tool in the rehabilitation of patients with persistent quadriceps inhibition.

KEY WORDS: arthrogenic muscle inhibition, knee injury, knee surgery, rehabilitation, attention

I. INTRODUCTION

Noticeable weakness and atrophy of the quadriceps are conditions commonly observed in patients following knee injury or surgical intervention,1–4 and these conditions often linger for an extended period of time.5,6 This becomes a major challenge for clinicians during rehabilitation because the ability to restore quadriceps function largely dictates whether these patients will recover successfully. Persistent quadriceps weakness can be partially attributed to a neuromuscular mechanism known as arthrogenic muscle inhibition (AMI). AMI is a reflexive response that initially serves as a protective mechanism to prevent further damage to a joint. Because it is slow to return to normal it often becomes a hurdle toward regaining quadriceps strength during rehabilitation.7 AMI results in an inability to completely activate a muscle despite no structural damage to the muscle or innervating nerve.7 Having normal quadriceps strength is essential for lower extremity function. Prolonged quadriceps dysfunction has been found to affect dynamic knee stability,8,9 physical function,9–12 and quality of life,11 as well as increase the risk of knee re-injury and the development of osteoarthritis.13–16

There are several interventions used to overcome AMI and restore quadriceps strength after knee injury. Electromyographic biofeedback (EMGBF) is one modality that has been used in conjunction with exercise to increase quadriceps activation and strength. EMGBF uses surface electrodes to detect and measure electric activity of the muscle while providing the patient with visual or auditory feedback about the magnitude of their muscular activation.17,18 This modality allows muscular activation to be brought to a conscious level without a visible contraction of the muscle. By providing patients with a method to quantify their muscular activation, they have a better understanding of their muscular deficit, thus motivating them to increase their motor output and reach a desired
goal of activation. The use of EMGBF has been shown to increase quadriceps strength and activation.\textsuperscript{18–23} However, the underlying neuromuscular mechanism behind EMGBF is not completely understood. It has been hypothesized that EMGBF can potentially increase muscular strength and activation by improving motor unit recruitment\textsuperscript{18,19,24} and optimizing firing rates through cortically generated mechanisms.\textsuperscript{22,24}

These theories suggest that neuroplasticity may provide a partial explanation for the positive effects elicited by EMGBF in rehabilitation. Neuroplasticity is a phenomenon that refers to changes in neural pathways and synapses due to changes in behavior, the body, or the environment.\textsuperscript{25,26} The concept of brain plasticity was first introduced in 1890 by William James,\textsuperscript{27} who proposed that the brain and its functions are not fixed throughout a person’s life. This idea was neglected during most of the 20th century because neuroscientists believed that after a critical period in early childhood, the structure and function of the nervous system was permanent or hardwired throughout adulthood. However, since the 1960s significant evidence of neuroplasticity has been produced through animal and human studies that demonstrated modifications in the structure and function of the nervous system in response to an experience.\textsuperscript{28–35} Therefore, the purpose of this article is to explain the effectiveness of EMGBF from a neuroplasticity perspective.

II. CONSEQUENCES OF ARTHROGENIC MUSCLE INHIBITION

AMI is a reflex inhibition of uninjured musculature surrounding a joint after swelling or structural damage occurs to that joint.\textsuperscript{7} After joint injury or swelling, an increased amount of afferent information is transmitted from the sensory receptors within that joint to the spinal cord. This afferent discharge of sensory information from the affected joint activates inhibitory interneurons within the spinal cord to decrease the excitability of the motor neuron pool in the joint musculature.\textsuperscript{6,36,37} The result is a decreased ability to recruit the motor neuron pool of the muscle and therefore produce a sufficient muscle contraction.\textsuperscript{6} As a muscle loses its ability to activate, the cross-sectional area of a muscle begins to decrease, causing it to weaken.

The rationale for AMI is to initially serve as a protective mechanism by “turning off” the adjacent muscles to immobilize the joint and prevent further joint damage. However, persistent AMI of the quadriceps muscle can become detrimental to patients. The quadriceps comprise 4 muscles (rectus femoris, vastus medialis oblique, vastus lateralis oblique, and vastus intermedius) that extend the knee and function together to propel and absorb forces during tasks such as walking, running, and jumping. They also play an important role in providing dynamic stability of the knee joint during activity. After knee injury, if a patient’s knee loses mobility because of the continued inability to contract the quadriceps at a sufficient level, the muscle begins to atrophy and weaken, which can alter knee function and make them susceptible to future injury.\textsuperscript{38} AMI essentially becomes the driving factor of a vicious injury cycle (Fig. 1).

Becker and colleagues\textsuperscript{39} have shown that residual levels of quadriceps activation deficits (approximately 8% inhibition compared to healthy, age-matched controls) may remain up to 4 years in patients after arthroscopic meniscectomy. Likewise, patients with anterior cruciate ligament (ACL) rupture along with additional joint damage (e.g., ligamentous, capsular, meniscal) have been found to demonstrate quadriceps activation deficits of 15–41% several months to years after the initial injury.\textsuperscript{40,41} Persistent AMI of the quadriceps has been shown to contribute to persistent quadriceps weakness as well.\textsuperscript{42–48} Quadriceps strength deficits between 5% and 18% of the reconstructed limb (compared to the healthy limb) have been reported between 5 and 15 years after ACL reconstruction and extensive rehabilitation.\textsuperscript{49–52} Weakness of these muscles has been related to poor functional outcomes\textsuperscript{8–12} and the onset of both patellofemoral and tibiofemoral osteoarthritis.\textsuperscript{13–16,53–56}

The combined functional outcomes and osteoarthritis may be explained by the altered biomechanics that result from quadriceps inhibition and weakness.\textsuperscript{57–60} A decrease in quadriceps function may be caused by the ability of the quadriceps to eccentrically absorb forces and provide joint stability during activities of daily living. As a result, the knee would
encounter excessive joint loading and be subject to articular joint damage. After ACL reconstruction, Lewek et al.\textsuperscript{58} discovered that patients who had <80\% of quadriceps strength compared to the nonsurgical limb displayed a gait that avoided using the quadriceps (decreased knee flexion) while walking and jogging. In addition, artificial knee joint effusion has been used to elicit temporary quadriceps inhibition and measure changes in lower extremity biomechanics.\textsuperscript{57,59,60} While inhibited, subjects have been found to exhibit decreased knee flexion and increased ground reaction forces upon landing.\textsuperscript{59}

It is interesting that AMI of the quadriceps has been reported to be present bilaterally following unilateral injury, and quadriceps activation deficits in the contralateral limb have been reported to be nearly equivalent to that of the injured limb.\textsuperscript{40,41,61} It is suggested that a crossover effect from the central nervous system is the cause of bilateral quadriceps inhibition.\textsuperscript{42,48} When compared to healthy, matched controls, there is also evidence of bilateral quadriceps weakness following unilateral knee injury, placing the uninjured limb at risk for injury.\textsuperscript{51,47,48,62} These findings suggest that decisions for athletes to return to play should not be based solely on bilateral comparisons and that rehabilitation efforts should focus attention on both the injured and uninjured limbs.

III. EMGBF IN REHABILITATION

Various therapeutic modalities have been used in combination with exercise for rehabilitation after knee injury or surgery to control and decrease the influence of AMI. One such modality that aims to overcome AMI by improving voluntary neuromuscular control of the quadriceps is EMGBF, a therapeutic modality that is used in rehabilitation to help patients develop greater awareness and voluntary control of their motor unit recruitment, which is otherwise involuntary and unfelt because of AMI.\textsuperscript{63}
It has been hypothesized that EMGBF facilitates initial strength gains by improving motor unit recruitment\textsuperscript{[18,19,24]} and optimizing firing rates through cortically generated mechanisms.\textsuperscript{[22,24]} EMGBF uses surfaces electrodes that detect and measure muscular activation beneath the skin. The electromyographic signals are converted to either auditory or visual cues that provide the patient with feedback regarding the amount of muscular activity that is produced. This feedback enhances the patient’s awareness of their muscular activity by bringing it to a conscious level. Although a patient may not be able to visibly see a muscular contraction being produced by their quadriceps, the use of EMGBF provides them with an alternative method to observe and quantify their quadriceps activation level. This information allows the patient to modulate their quadriceps activation by consciously adjusting their motor output. A greater conscious control of descending motor pathways can theoretically override the neural inhibition at the spinal cord and restore quadriceps activation levels.\textsuperscript{[64]} As a result, restored quadriceps activation would eventually lead to muscle hypertrophy and quadriceps strength would then increase.

The use of EMGBF in conjunction with exercise has been reported to positively affect both quadriceps activation\textsuperscript{[18,21,65,66]} and strength.\textsuperscript{[18–20,22,23,66–68]} In a study that assessed the effectiveness of EMGBF in postoperative patients who underwent meniscectomy, their quadriceps activation was increased 10-fold compared to patients who performed standard rehabilitation. Likewise, Maitland et al.\textsuperscript{66} conducted a case study of a patient who was 8 months after ACL reconstruction and reported progressively declining knee stability. They found that by incorporating EMGBF into a closed kinetic chain rehabilitation program, the patient’s quadriceps activation was improved by 52% and their peak torque during knee extension was increased by 203%. The patient’s neuromuscular improvements also were found to result in better gait and knee stability.

**IV. EMGBF AND NEUROPLASTICITY**

The combination of exercise with EMGBF is thought to target cortical mechanisms associated with generating muscular force.\textsuperscript{[18,69,70]} It has been discovered that when muscular force is produced, there is increased neuronal activity in the motor cortex of the brain.\textsuperscript{69} Furthermore, when visual feedback is provided during a movement task, neuronal activity in the motor cortex and production of muscular force are symmetrically enhanced.\textsuperscript{70} These findings imply that neuroplasticity may provide an additional explanation for the effectiveness of EMGBF in restoring quadriceps activation and strength after knee injury or surgery. Neuroplasticity is a phenomenon that refers to the ability of the brain to modify its neural pathways and synapses in response to changes in behavior, the body, or the environment.\textsuperscript{[25,26]} In other words, neuroplasticity is the brain’s ability to reorganize itself and form new connections as a result of experiences throughout one’s life.

Before discussing the role neuroplasticity plays in regaining quadriceps function through the use of EMGBF, it is important to first discuss the potential cortical implications that result from persistent AMI of the quadriceps. As AMI continues to restrict activation of the quadriceps, the cortical topographic representation of the quadriceps may also begin to fade. Similar to the sensory cortex of the brain, the motor cortex has a topographical map that corresponds to regions of the body. However, unlike the sensory cortex, the topography of the motor cortex is more abstract. Through the use of positron emission tomography and functional magnetic resonance imaging techniques, studies have been able to noninvasively discover the topographical mapping of the human motor cortex. Rather than the parts of the body being mapped separate from each other, like in the sensory cortex, these studies have found the mapping of the motor cortex to be overlapped and intermingled.\textsuperscript{71–74} This evidence suggests that the motor cortex is functionally organized by muscle synergies to promote coordination during movement.

The inability to properly extend the knee because of quadriceps inhibition results in a topographical representation in the motor cortex that is diminished and invaded by other muscle groups. Experiments on adult rats have shown that the topography of the motor cortex can change if motor pathways to a muscle are disrupted.\textsuperscript{31,75,76} Sanes et al.\textsuperscript{75} examined the dynamic reorganization of the motor cortex in
rats after forelimb amputation or facial motor nerve transection. Those rats who underwent forelimb amputation demonstrated expanded representation of the shoulder at the motor cortex, which invaded the motor area originally represented by the forelimb. For those rats that underwent facial motor nerve transection, the area of the motor cortex that was originally occupied by the vibrissa musculature was now invaded by enlargements of forelimb and eye/eyelid motor areas. Furthermore, it was shown not only that cortical changes occur after motor nerve lesion but also that these changes can take place as early as 1 week.

Although AMI does not cause denervation, several human studies of lower limb immobilization and ACL deficiency also have demonstrated cortical plasticity through the use of transcranial magnetic stimulation (TMS), a noninvasive technique that transmits an electromagnetic pulse through the motor cortex to measure the cortical output of certain motor areas. Roberts and colleagues used TMS to evaluate corticomotor plasticity before and after lower limb immobilization. Eight healthy subjects had their left leg placed in a full-length cast for 10 days, and TMS was performed before casting, immediately after removing the cast, and again 24 and 48 hours after the cast was removed. A control group that did not wear casts underwent the same TMS sessions to serve as a comparison. Roberts et al. found that corticomotor excitability in the experimental group peaked at 24 hours after cast removal, whereas no changes were observed in the control group. Changes in corticomotor excitability also have been discovered in subjects who have an ACL deficiency. In comparison to healthy controls, Heroux and Tremblay found that subjects with chronic ACL-deficient knees elicited lower resting motor thresholds using TMS, corresponding to enhanced corticomotor excitability.

Increased corticomotor excitability is suggested to be indicative of cortical reorganization that occurs during motor learning. Both lower limb immobilization and ACL deficiency can affect the function of the lower extremity and force individuals to learn compensatory strategies to remain functional. Any biomechanical changes that are made are reflected in the motor cortex by the reorganization of maps. In a similar way, AMI of the quadriceps can constrain knee function and alter biomechanics in the lower extremity. By not being able to adequately extend the knee, compensatory strategies may be adopted by the hip and ankle to maintain function. As a result, the area of the motor cortex that originally represented the quadriceps may be partially invaded by representations of the hip and ankle musculature. These compensatory strategies can ultimately lead to a “learned nonusage” of the quadriceps, making it increasingly difficult to regain quadriceps function. This compensatory behavior is referred to as maladaptive plasticity.

Therapeutic exercises can be used during rehabilitation to help reverse maladaptive plasticity and recover quadriceps function. Recovered function of the quadriceps through therapeutic exercise can partially be attributed to the concept of activity-dependent plasticity. The idea of activity-dependent plasticity (associative learning) was first conceptualized by Donald Hebb in 1949. Hebb proposed that the formation and strengthening of neuronal synapses are directly correlated with learning an activity ("neurons that fire together wire together"). As the practice of an activity increases, synaptogenesis occurs, and the strength of the synaptic connections increase accordingly. A persistent increase in synaptic strength eventually leads to neural stability/memory, a process referred to as long-term potentiation. Conversely, as the practice of an activity declines, synapses begin to deteriorate and their connections become weakened (long-term depression).

Activity-dependent plasticity of the motor cortex has been demonstrated in multiple animal studies through motor learning. Nudo et al. trained squirrel monkeys to perform highly skilled hand and forearm tasks. The monkeys were taught to retrieve food from a Kluver board using the fine motor skill of their digits. As a result, the monkeys' digit representation in their motor cortex expanded and their wrist/forearm representations contracted. Studies of rats have demonstrated that functional and structural plasticity co-occurs within the same region of the motor cortex. Kleim et al. trained one group of rats to perform a skilled reaching task and another group of rats to perform a simple bar-
pressing task. They discovered that the rats that performed the skilled task increased their wrist and digit cortical representations, but their elbow/shoulder representations decreased. On the other hand, in the rats that performed the simple task the opposite cortical representations were observed. The results from these animal studies show that topographical changes in the motor cortex are dependent on the strategies used during a practiced activity.

Several human studies also have supported activity-dependent plasticity within the motor cortex following skill training and resistance training. Pascual-Leone et al. used TMS to map the cortical motor areas of the finger and hand in subjects learning a one-handed, 5-finger exercise on the piano. Compared to the control subjects, trained subjects enlarged their cortical motor areas over the course of 5 days. These plastic changes also have been observed in lower extremity skill training. Furthermore, Weier et al. reported increased cortical excitability with TMS in subjects who underwent a 4-week resistance training program consisting of a squatting exercise with a heavy load.

This evidence on activity-dependent plasticity suggests that using therapeutic exercise after knee injury or surgery may help to override AMI and regain quadriceps function. However, there is one critical factor that drives neuroplasticity, which therapeutic exercise sometimes lacks: the presence of attention. Attention has been found to enhance activity-dependent plasticity by increasing the release of modulatory neurotransmitters. In other words, for an individual to accelerate and maximize their neuroplasticity, the exercise must be meaningful to them and they must be attentively engaged when performing the exercise.

Attention may be especially important when attempting to correct the cortical and neuromuscular ramifications from persistent AMI. Repetitive exercise alone is not enough to maximize activity-dependent plasticity. EMGBF is one such modality that can be used in conjunction with therapeutic exercise to provide the attention aspect for enhancing activity-dependent neuroplasticity. The visual and/or auditory cues from EMGBF force the patient to focus on their quadriceps activation level and make the necessary adjustments through volitional effort. Instead of the patient viewing an exercise as a routine task, EMGBF provides a motivational aspect to the exercise and challenges the patient to consciously increase their motor output. The use of EMGBF potentially becomes more important when the patient is unable to produce a visible muscular contraction of their quadriceps. Providing a patient with a method to visibly observe their quadriceps activation when they are unable to see a muscular contraction not only focuses their attention, it also provides them with encouragement and motivation as well.

In theory, the enhanced activity-dependent neuroplasticity from the attention aspect of EMGBF would reestablish the topographical representation of the quadriceps in the motor cortex that was altered by AMI. As the patient continues to use EMGBF in conjunction with therapeutic exercise, the cortical synapses continually strengthen, and long-term potentiation would eventually be established. The solidified quadriceps representation in the motor cortex would allow the patient to efficiently bypass the inhibitory mechanism at the spinal cord and facilitate motor unit recruitment at the quadriceps through conscious control. Quadriceps activation level would be restored, hypertrophy would occur, and their strength would eventually be regained. These improvements would then allow proper knee extension and lower extremity kinematics/kinetics, thus improving quality of life and reducing the development of subsequent knee pathologies such as osteoarthritis.

This being said, there is a remaining theoretical gap that must be explained. How do patients transfer the control of their quadriceps activation from a conscious to an unconscious state? Once a patient develops the ability to obtain conscious control of their quadriceps, they must not only be able to retain this control but also do so unconsciously to be functional. Otherwise, if an individual had to consciously activate their quadriceps during every task, they would not be able to perform that task very well and their awareness of the environment would be diminished. This supports the importance of incorporating activities of daily living into rehabilitation practice to help successfully transfer learned skills.

Although the transition from conscious to unconscious control is real and necessary, there is no defini-
tive explanation as to how it occurs. Cortical motor planning areas such as the supplementary motor area and premotor area are involved in unconscious planning of movements, but there are other areas of the central nervous system that must be responsible for the execution and retention of unconscious movement. The most likely prospects can be narrowed down to the spinal cord and the cerebellum.

When you walk or run, your lower limbs seem to automatically fall into a repetitive and rhythmic pattern by alternating flexion and extension between each lower limb. This reciprocal movement can be attributed to neural circuits within the lumbar spinal cord called stepping pattern generators (SPGs), which control locomotion of the lower limbs. SPGs function to trigger lower motor neurons within the lumbar spinal cord to activate muscles and produce alternative flexion and extension of the hips and knees. These neural networks also receive proprioceptive information from the lower limbs with regard to the task and environment to allow for adaptation. For example, if the terrain that you were walking upon changed from grass to sand, your SPGs would alter their output to your lower limbs and adapt your walking pattern to the changed terrain. In addition, both skill training and strength training have been found to increase motor neuron excitability in the spinal cord. To be more specific, skill training is shown to heighten the spinal stretch reflex in muscles, whereas strength training elicits synaptogenesis within the ventral horn of the spinal cord. Therefore, both the presence of SPGs and increased spinal excitability would allow an individual to unconsciously adjust their quadriceps activation to the task and environment and recruit motor units with greater ease.

It is well known that the cerebellum plays a major role in motor control, but its potential has yet to be fully understood in the realm of neurophysiological research. The primary role of the cerebellum is to compare the intended movement to the executed movement. Once a movement is performed, sensory information from the spinal cord is sent to the cerebellum regarding the quality of that movement. If an error in movement is detected, information is sent from the cerebellum to the motor areas so that the movement can be corrected. However, several researchers have proposed that the cerebellum also plays an important role in the automaticity of learned movements. Comparisons of subjects with cerebellar damage to healthy controls have shown that their performance of a learned movement deteriorates when their attention is focused away from the movement task; they are thus unable to effectively perform dual tasks. This evidence suggests that the cerebellum plays a role in maintaining quadriceps control during activities of daily living without conscious involvement.

V. CONCLUSION

After knee injury or surgical intervention, AMI of the quadriceps is commonly observed and can often have a lingering effect. If not appropriately treated, the ramifications of persistent quadriceps inhibition can be detrimental and place the individual at higher risk for subsequent knee pathologies. Therefore, rehabilitation plays a vital role in overcoming AMI and regaining adequate quadriceps function. Persistent quadriceps inhibition results in not only neuromuscular deficits; it may also lead to cortical effects explained through the phenomenon of neuroplasticity. For this reason, the use of EMGBF in conjunction with therapeutic exercise can enhance activity-dependent plasticity to resolve the cortical and neuromuscular effects of AMI and restore quality of life. However, further research is needed to examine the potential cortical effects of persistent quadriceps inhibition. Because of the limited amount of literature on EMGBF, future investigations should examine the full effects of this modality and its applicability in rehabilitation.

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