MUSCLE WEAKNESS is a common impairment in patients following joint injury. Arthrogenic muscle inhibition (AMI) is a condition that involves neural inhibition of uninjured musculature surrounding an injured joint. Although AMI can affect the musculature surrounding any joint, it has primarily been studied in the quadriceps muscles following various knee pathologies, including anterior cruciate ligament injury, meniscal injury, anterior knee pain, and osteoarthritis and in the soleus and peroneal muscles of patients with chronic ankle instability.

Following joint injury, neural mechanisms decrease the number of motor neurons that can be voluntarily activated to produce a muscle contraction. This reflexive decrease in motor output may be a protective strategy employed by the central nervous system to diminish loads acting on an injured joint. Unfortunately, this muscle activation deficiency is not easily removed with conventional rehabilitation and has been reported years following acute joint injury. Furthermore, AMI has been identified as a “limiting factor” in joint rehabilitation, suggesting that our ability to treat this “invisible” impairment may significantly affect patient outcomes.

**The Need for Disinhibition**

The inability to activate musculature following a joint injury or surgery is very common and obvious to both clinicians and patients. Currently, standard practice is to engage a weakened muscle in strengthening exercises, without targeting neural inhibition. Although inhibited patients may still be capable of achieving strength gains, failure to address the underlying neurologically-mediated dysfunction may lead to the perpetuation of the inhibited state of the muscle, leaving the previously injured extremity weaker, more susceptible to fatigue, and incapable of properly attenuating loads imposed upon an injured joint during gait or landing. Evidence is mounting that AMI may be a factor contributing to the development of post-traumatic osteoarthritis.

Performing therapeutic exercise in an inhibited state may force patients to utilize suboptimal motor recruitment patterns to perform a task (i.e., decreased motor neuron responsiveness requires recruitment of different motor units to perform a task in a manner that closely resembles the preinjury
movement pattern). When progressing an inhibited muscle through a conventional strength training regimen without addressing the decreased neural activation, clinicians may be directing patients to utilize new motor patterns that are based on a diminished pool of available motor neurons. Without reengagement of the inhibited motor neurons, suboptimal motor recruitment patterns could lead to decreased level of performance, increased rate of fatigue, increase risk of subsequent injury, and risk of chronic dysfunction.

A more effective rehabilitative strategy may be to remove the neural inhibition prior to engaging in exercise, thereby allowing the patient to optimize motor recruitment patterns, which may facilitate greater strength gains and improve both functional movements and joint force attenuation. Recently, cryotherapy and transcutaneous electrical nerve stimulation (TENS) have been reported to have disinhibitory effects.13

Cryotherapy as a Disinhibitory Modality

Cryotherapy is often administered acutely and subacutely to manage pain and minimize swelling following injury. This modality has also been used in conjunction with exercise, a practice termed cryokinetics, which has been reported to allow for earlier restoration of range of motion and return to normal activity.14 The goal of cryokinetics is to provide an analgesic effect, which allows a patient to perform therapeutic exercises that would otherwise be painful. Improved function following cryokinetics has been attributed to a reduction in the volume of pain impulses that are transmitted from the dorsal horn of the spinal cord to the brain.14 Recently, focal joint cooling has been reported to increase the neural excitability of the musculature surrounding the knee and the ankle.15,16 The beneficial effects of cryokinetics may be at least partially due to disinhibition of the inhibited musculature surrounding the injured joint.

Increased torque production for knee extension and ankle plantar flexion have been reported following 20-minute cryotherapy treatments to the respective joints.15,16 Because these studies involved participants who were not experiencing pain, the researchers attributed increased neural excitability to a decrease in reflex inhibition of the motor system.15 We hypothesize that cooling the skin around an injured joint increases sensory signals traveling to the central nervous system from superficial receptors (i.e., mechanoreceptors, thermoreceptors).9 This increase in excitatory signals may diminish the influence of afferent signals from the injured joint, which would otherwise produce a greater inhibitory effect on the motor system. The effects on the neural system following a 20-minute cryotherapy treatment have been reported to last up to 40 minutes, which may provide an adequate period to perform therapeutic exercise.13

TENS as a Disinhibitory Modality

Sensory TENS applied to the anterior knee has been reported to increase quadriceps neural excitability in healthy subjects with knee effusion.13 There have also been reports that TENS administration in conjunction with exercise has improved strength gains and normalized gait in patients with osteoarthritis.17 The proposed mechanism for the disinhibitory effect provided by TENS is similar to that associated with cryotherapy. The TENS sensory stimulus provides the neural system with an increase in excitatory signals traveling to the spinal cord, thereby counteracting signals arising from the injured joint that would otherwise produce an inhibitory effect on the motor system. In contrast to cryotherapy, the disinhibitory effect of TENS appears to decrease immediately following discontinuation of the stimulation.13 The difference between the effects of TENS and cryotherapy may be attributable to a difference in central nervous system processing of the neural signals associated with the respective forms of sensory stimulation. The immediate increase in neural excitability following TENS administration is not as great as that reported following cryotherapy.13 To date, there is no evidence that either of these modalities poses any type of inherent risk or benefit when combined with therapeutic exercise. It should also be noted that previous disinhibitory modality data13,15,16,18-20 were collected in the absence of pain, and pain is a likely contributor to muscle inhibition. TENS and cryotherapy may have additional effects on pain-associated muscle inhibition, which may produce more robust disinhibitory benefits in a pathological state.

Incorporating Disinhibitory Modalities Into Clinical Practice

Although we have presented evidence supporting the use of cryotherapy and TENS as disinhibitory modalities, clinical trials have not yet provided
Evidence for the effectiveness of these treatments for this specific indication. Both of these modalities are inexpensive, easy to use, accessible to most clinicians, and present minimal risk for adverse effects. Cryotherapy and TENS are also disinhibitory modalities that can be incorporated into a home-based exercise program with simple instructions from a trained clinician.

We have included suggested parameters for administration of TENS (Table 1) and cryotherapy (Table 2) that have been derived from the relevant literature. Although these modalities are widely used in sports medicine, the indication for muscle disinhibition may be new to many clinicians. The timing of disinhibition treatments must immediately precede therapeutic exercise to take advantage of the enhanced excitability of the motor system that they promote.

Previous research indicates that a 20-minute cryotherapy treatment to the knee joint is effective in increasing neural activity in the vastus medialis muscle for 40 minutes following treatment. This suggests that muscle strengthening following joint injury may be enhanced by 20 minutes of joint cooling prior to performance of the strengthening exercises. We advocate cooling of the joint, rather than cooling of the musculature (see Figure 1).

![Figure 1](image1.png)

**Figure 1** Suggested application of TENS electrodes at the knee. Electrodes are positioned around the patella with as little contact with the muscle as possible. Two separate currents were crossed (1 & 2) to increase the surface area being stimulated.

Although the magnitude of neural excitation provided by TENS is less than that provided by cryotherapy, TENS can be administered while the patient is exercising. A strong sensory stimulus that does not elicit muscle contraction is advocated. Electrodes should be positioned around the periphery of the patella (Figure 1). Previous authors have advocated use of two sets of electrodes to increase the amount of surface area that is stimulated. TENS can be administered outside the clinic as patients progress to participation in sport-specific activities.

**Conclusion**

Although direct evidence of a disinhibitory effect in injured subjects is lacking, laboratory observations support the use of disinhibitory modalities. Disinhibition is a new indication for cryotherapy and TENS, which may revolutionize the manner in which joint rehabilitation is performed in the future.

**References**


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