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ORIGINAL RESEARCH

Immediate Effects of Therapeutic Ultrasound on Quadriceps Spinal Reflex Excitability in Patients With Knee Injury

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Abstract

Objective: To investigate the effects of nonthermal therapeutic ultrasound on quadriceps spinal reflex excitability in patients with knee joint injury.

Design: Double-blind, randomized controlled laboratory study with a pretest posttest design.

Setting: University laboratory.

Participants: Recreationally active volunteers with a self-reported history of diagnosed intra-articular knee joint injury and documented quadriceps dysfunction (N = 30).

Interventions: A nonthermal ultrasound, or sham, treatment was applied to the anteromedial knee.

Main Outcome Measures: Hoffmann reflex measurements were recorded at baseline, immediately postintervention, and 20 minutes post-intervention. The peak Hoffmann reflex amplitude was normalized by the peak motor response (H/M ratio) measured from the vastus medialis using surface electromyography as an estimate of quadriceps motorneuron pool excitability. A repeated-measures analysis of variance was used for comparisons.

Results: A significant group-by-time interaction was observed for mean ($P = .016$) and change ($P = .044$) in H/M ratio. The ultrasound group demonstrated significantly higher mean ($P = .015$) and change ($P = .028$) in H/M ratio 20 minutes postintervention than did the sham ultrasound group.

Conclusions: Quadriceps motoneuron pool excitability was facilitated 20 minutes after a nonthermal therapeutic ultrasound treatment, and not a sham treatment. These data provide supporting evidence of the contribution of peripheral receptors in modulation of the arthrogenic response in patients with persistent quadriceps dysfunction. Future research in this area should attempt to identify optimal treatment parameters and translate them to clinical outcomes.

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Persistent quadriceps weakness and central activation failure are widely reported after knee joint injury.[1] Posttraumatic muscle dysfunction is linked to a sequelae of impairments detrimental to global health and joint-specific health, including decreased physical activity,[2,3] increased risk of reinjury,[4] and an accelerated onset of knee joint osteoarthritis.[5,6] Central and peripheral neural adaptations accompany these consequences and are established as an underlying contributor to muscle impairment.[7] Quadriceps dysfunction is reported to manifest via altered excitability from spinal and cortical regions.[8,9] The diminished ability to activate otherwise healthy periarticular muscular tissue in the presence of joint injury is termed arthrogenic muscle inhibition (AMI) and is proposed as a neural phenomenon responsible for limiting the progression of rehabilitation.[10] After injury, this arthrogenic response can manifest as an ongoing reflex inhibition due to aberrant sensory information arising from mechanoreceptors located in periarticular structures, which the central nervous system (CNS) interprets as inhibitory.[10,11] Inhibition of surrounding musculature may therefore result from transmission of aberrant

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afferent stimuli and has been examined after artificial joint effusion,[12] pain,[13] and structural damage.[9]

The location of sensory nerves has been suggested to be of particular importance to clinicians when treating AMI.[10] Somatosensory information originating from peripheral receptors is reported to influence motor function via projections to spinal motoneurons and supraspinal structures.[14] Articular mechanoreceptors originating from sensory nerves is theorized to play a primary role in modulating inhibition.[15] It has been established that articular mechanoreceptors play a significant role in regulating afferent signals to the CNS and appear sensitive to change in the presence of joint trauma,[16] making the neurophysiological response of these receptors a clinical interest. Interventions aimed to reduce AMI have been termed "disinhibitory" modalities and have been examined in an effort to combat posttraumatic muscle dysfunction. Mechanical vibration is reported to activate mechanoreceptors in skin and muscle tissue at varying frequencies,[17] whereas deep mechanical stimulation has been used to activate articular mechanoreceptors purported to exert strong effects on motoneuron excitability in animal models.[18] Given the ability of mechanical energy to affect receptors of interest, it is plausible that interventions that transmit energy to deep articular structures may alter the afferent information received by the CNS.

Therapeutic ultrasound is a widely used clinical modality that uses mechanical properties to stimulate tissues. Although often used as a heating agent, the application of superficial heat has not yielded success as a disinhibitory modality,[19] making the nonthermal mechanical properties a particular interest. Nonthermal ultrasound is reported to influence osseous and cutaneous receptors important in fracture[20,21] and wound healing[22] using pulsed energy; however, the influence on articular mechanoreceptors is unknown. In contrast, low-intensity continuous ultrasound is reported to exert stable and prolonged cavitation to structures ≥5cm in depth,[23] which may provide clinical benefit if able to target periarticular tissues via mechanical stimulation. Therefore, the primary purpose of this investigation was to compare quadriceps motoneuron pool excitability (MNPE) in patients with knee joint injury after a single treatment of and sham treatment. Secondarily, this study aimed to explore patient characteristics related to magnitude of change in quadriceps motoneuron pool activity after ultrasound application.

Methods

This was a double-blind, randomized controlled laboratory study with a pretest posttest design. Independent variables included group (ultrasound, sham), limb (involved, uninvolved), and time (baseline 1, baseline 2, immediately postintervention, 20 minutes postintervention). The primary outcome measure was peak Hoffmann reflex normalized by peak motor response (H/M ratio). Group assignment was randomly allocated using a random number generator and concealed in opaque envelopes opened after baseline measurements. To reduce bias, the researcher recording all measurements remained blinded to group assignment throughout the duration of the study, even during data entry, and was not involved in the randomization of group assignment. All participants remained blinded to intervention.

List of abbreviations:

AMI	**arthrogenic muscle inhibition**
CAR	**central activation ratio**
CNS	**central nervous system**
H/M ratio	**peak Hoffmann reflex normalized by peak motor response**
H-reflex	**Hoffmann reflex**
MNPE	**motoneuron pool excitability**
M-response	**motor response**
TENS	**transcutaneous electrical neuromuscular stimulation**
VM	**vastus medialis**

Participants

Forty young recreationally active volunteers with a self-reported history of diagnosed intra-articular knee joint injury were consented and screened. This population was selected to maintain generalizability of findings and to study a unique subset of individuals experiencing persistent posttraumatic muscle dysfunction. To be eligible, participants must have had documented quadriceps dysfunction, defined as having a central activation ratio (CAR) below 90%.[24] Participants were excluded if they reported a lower extremity injury within 6 months, contralateral knee injury, pain rated 4cm or greater on a 10cm visual analog scale, or had a visibly noticeable effusion.[7,25] Nine volunteers with CAR≥90% and 1 without measurable Hoffmann reflex (H-reflex) were excluded, leaving 30 participants (fig 1). Participants were randomized to ultrasound or sham group. Participants were asked to refrain from caffeine use and significant change in activity within 24 hours prior to testing. Measurements were obtained in a university laboratory setting. The study protocol was approved by the Institutional Review Board at the University of Virginia and performed in accordance with the guidelines of the Declaration of Helsinki. All participants gave informed consent before enrollment.

Instrumentation

Quadriceps CAR

Isometric knee extension torque was recorded using the Biodex System 3 dynamometer.[a] Data were exported and digitized at 125Hz using the MP150 system.[b] A square-wave stimulator (S88[c]) and a stimulus isolation unit (SIU8T[c]) were used to produce an electrical stimulus for the superimposed burst technique as previously described.[26]

Hoffmann reflex

Electromyography of the vastus medialis (VM) was performed using pregelled (10-mm-round) Ag/AgCl electrodes in accordance with surface electromyography for noninvasive assessment of muscle guidelines.[27] The signal was amplified (gain = 1000) and digitally converted using a 16-bit data acquisition system.[b] The electromyographic signal was collected at 2kHz for 80ms (common mode rejection, 110dB; input impedance, 1.0MΩ; noise voltage, 0.2μV; signal-to-noise ratio, 70dB). Signals were visualized and processed using AcqKnowledge 3.7.3.[b] A stimulator module and a stimulus isolation unit (STIMISOC)[b] were used to deliver the stimulus using the 4-mm-round electrode.

Ultrasound

The Omnisound 3000E[d] system delivered a nonthermal ultrasound or sham treatment. A transducer with a surface area of 5.0cm, beam nonuniformity ratio of 2.7:1, and effective radiating area of 4.1cm^2 was used for all treatments. This device was chosen because of low