ACRM
AMERICAN CONGRESS OF REHABILITATION MEDICINE

Archives of Physical Medicine and Rehabilitation

journal homepage: www.archives-pmr.org

Archives of Physical Medicine and Rehabilitation 2014;95:2021-8

ORIGINAL ARTICLE

Whole Body and Local Muscle Vibration Reduce Artificially Induced Quadriceps Arthrogenic Inhibition

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Abstract

Objective: To evaluate the effects of whole body vibration (WBV) and local muscle vibration (LMV) on quadriceps function after experimental knee effusion (ie, simulated pathology).

Design: Randomized controlled trial.

Setting: Research laboratory.

Participants: Healthy volunteers (N=43) were randomized to WBV (n=14), LMV (n=16), or control (n=13) groups.

Interventions: Saline was injected into the knee to induce quadriceps arthrogenic muscle inhibition (AMI). All groups then performed isometric squats while being exposed to WBV, LMV, or no vibration (control).

Main Outcome Measures: Quadriceps function was assessed at baseline, immediately after effusion, and immediately and 5 minutes after each intervention (WBV, LMV, control) via voluntary peak torque (VPT) and the central activation ratio (CAR) during maximal isometric knee extension on a multifunction dynamometer.

Results: The CAR improved in the WBV (11.4%, $P=.021$) and LMV (7.3%, $P<.001$) groups immediately postintervention, but they did not improve in the control group. Similarly, VPT increased by 16.5% ($P=.021$) in the WBV group and 23% ($P=.078$) in the LMV group immediately postintervention, but it did not increase in the control group. The magnitudes of improvements in the CAR and VPT did not differ between the WBV and LMV groups.

Conclusions: Quadriceps AMI is a common complication following knee pathology that produces quadriceps dysfunction and increases the risk of posttraumatic osteoarthritis. Quadriceps strengthening after knee pathology is often ineffective because of AMI. WBV and LMV improve quadriceps function equivocally after simulated knee pathology, effectively minimizing quadriceps AMI. Therefore, these stimuli may be used to enhance quadriceps strengthening, therefore improving the efficacy of rehabilitation and reducing the risk of osteoarthritis.

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Knee osteoarthritis (OA) is a leading cause of disability[1,2] affecting 9 million Americans at an annual cost of $51 billion.[2,3] Although gradual idiopathic degeneration of articular cartilage appears to be the primary cause of OA, traumatic orthopedic injuries (eg, anterior cruciate ligament injury and reconstruction) commonly result in posttraumatic OA, accounting for 10% of knee OA cases.[1] Quadriceps arthrogenic muscle inhibition (AMI) is a common chronic complication of knee pathologies that results in quadriceps dysfunction.[4-6] Because the quadriceps functions as a shock absorber during the early stance phase of gait, this dysfunction results in impulsive loading,[7-10] which is linked to onset and progression of OA.[11,12] Both retrospective[13,14] and prospective[15,16] evidence implicates quadriceps dysfunction as a contributor to OA development and progression. Therefore, minimizing AMI may delay OA development and progression.

Presented in part to the National Athletic Trainers' Association, June 24-26, 2013, Las Vegas, NV.

Supported by the University of North Carolina at Chapel Hill/North Carolina State University Rehabilitation Engineering Center Seed Grant Program.

Disclosures: none.

0003-9993/14/$36 - see front matter © 2014 by the American Congress of Rehabilitation Medicine
http://dx.doi.org/10.1016/j.apmr.2014.07.393

AMI is attributable to factors that alter sensory information derived from the joint and signal pathology, including pain, swelling, inflammation, and damage to mechanoreceptors.[17] Quadriceps AMI can also be induced experimentally via injection of saline into the knee joint, which mimics the effects of swelling/effusion.[18] The resulting inhibition can be quantified via measures of muscle function, such as voluntary peak torque (VPT) and central activation ratio (CAR), during maximal isometric contraction.[5,19]

Traditional rehabilitation and strengthening exercises are often ineffective in individuals with knee pathologies, likely because AMI prevents adequate activity to stimulate strength gains.[20,21] However, whole body vibration (WBV) facilitates quadriceps function in individuals with nonpathologic knees[22,23] and may therefore minimize AMI and quadriceps dysfunction in individuals with knee pathologies. Clinical application of WBV, however, may be limited because these devices require a fixed location, permit limited types of rehabilitation exercises because of the constrained area of the device ($\sim$0.5m^2), and may be cost prohibitive. Local muscle vibration (LMV) also facilitates quadriceps activity[24-26] and may be integrated into a portable, cost-effective device for use in a variety of rehabilitation tasks and settings. However, the effects of these vibratory stimuli on quadriceps AMI and the underlying mechanisms of knee OA development and progression have yet to be investigated.

The purpose of this study was to evaluate the effects of WBV and local muscle vibration (LMV) on quadriceps AMI after experimental knee effusion. The experimental effusion model was chosen rather than pathologic knees to avoid the potential confounding effects of pain, inflammation, and sensory deficits. We hypothesized that both WBV and LMV would improve quadriceps function and that these improvements would be similar in magnitude.

Methods

Participants

Forty-five healthy, physically active volunteers (17 men, 28 women) were randomly assigned to 3 groups: WBV (n=15), LMV (n=16), and control (n=14) (fig 1). Subjects were required to be between 18 and 35 years of age, be physically active (at least 20min of physical activity 3 times per week), and have no history of anterior cruciate ligament or meniscal injury, neurologic disorder, knee OA or rheumatoid arthritis, acute knee injury within 6 months prior to participation, or lower-extremity surgery. The study was approved by the university's biomedical institutional review board, and all subjects provided written informed consent prior to participation.

Subjects reported to the laboratory on 2 occasions separated by 1 to 3 days. The first occasion served to familiarize subjects with the testing procedures. During the second occasion, quadriceps function was assessed as subjects performed maximal voluntary isometric quadriceps contractions during which VPT and the CAR were measured. Subjects were seated on a multifunction dynamometer[a] with the knee in 60° of flexion, and stimulating electrodes were secured over the quadriceps (fig 2). Subjects were instructed to extend the knee maximally as quickly as possible in response to a visual stimulus. Isometric torque data were sampled from the dynamometer at 1000Hz and low-pass filtered at 50Hz, and VPT and the CAR were derived from the resulting torque versus time curve (fig 3). VPT was calculated as the maximal voluntary torque value normalized to body mass. The CAR was measured via the superimposed burst technique during which an electrical stimulus (2-pulse train, 600μs duration, 100Hz, 150V), delivered by an isolated stimulator,[b] was applied to the quadriceps after plateau of the voluntary torque signal. This stimulus excites the remaining motor units, which are not recruited voluntarily, therefore activating all available motor units. VPT is referenced to the peak torque resulting from the superimposed burst, and the resulting ratio (CAR) indicates the percentage of the total motor unit pool that can be recruited voluntarily (see fig 3).

Following baseline measures of quadriceps function, subjects were anesthetized with a periarticular injection of 3mL of 1% lidocaine followed by an ultrasound-guided[c] supralateral intra-articular injection of 60mL of saline[d] into the knee joint space using a 25-gauge, 3.8-cm (1.5-in) hypodermic needle to induce AMI.[18] Immediately after the saline injection, subjects returned to the dynamometer, and quadriceps function was assessed again to ensure that AMI had been induced (posteffusion).

The WBV and LMV groups were then exposed to vibratory stimuli previously demonstrated to facilitate quadriceps function,[23,24] and the control group performed these same procedures without vibratory stimuli. Immediately after the posteffusion assessment of quadriceps function, subjects moved to a WBV device[e] where they stood in $\sim$40° of knee flexion (fig 4). The WBV device was activated only for the WBV group, who received a standardized WBV stimulus (30Hz, 2*g*). Pilot testing was conducted to characterize the vibration stimulus that the WBV device introduced to the quadriceps. A triaxial accelerometer[f] was secured over the quadriceps tendon while subjects (n=5) were exposed to the aforementioned WBV stimulus, and acceleration was sampled at 1000Hz for 10 seconds. The mean 3-dimensional resultant root mean square acceleration at the quadriceps tendon was 2.1$\pm$0.2*g* and was provided primarily in the anterior-posterior direction (ie, into the quadriceps). This stimulus is similar to the mechanics of a reflex hammer when evaluating the tendon-tap reflex in that it creates rapid changes in quadriceps length, therefore exciting the muscle via the muscle spindle system. The vibratory parameters for the LMV intervention (30Hz, 2*g*) were established based on these pilot acceleration data, and the stimulus was delivered via a custom-built stimulator secured over the quadriceps tendon (see fig 4). As such, the characteristics of the WBV and LMV stimuli at the level of the quadriceps were identical. Subjects remained in the squatted position while vibratory stimuli (WBV, LMV) were delivered for 1 minute, 6 times, with 2 minutes of rest between exposures based on the protocol developed by Tihanyi et al.[23] The control group performed the same isometric squat for identical periods of activity and rest, but no vibratory stimulus was provided. All interventions were conducted by the same coinvestigator, and the principal investigator was blinded to group assignment. After the interventions, subjects immediately returned to the dynamometer for postintervention assessments (immediately, 5min postintervention).

List of abbreviations:

AMI	**arthrogenic muscle inhibition**
CAR	**central activation ratio**
LMV	**local muscle vibration**
OA	**osteoarthritis**
VPT	**voluntary peak torque**
WBV	**whole body vibration**