Lack of Effect of Superficial Heat to the Knee on Quadriceps Function in Individuals With Quadriceps Inhibition

Brandon Warner, Kyung-Min Kim, Joseph M. Hart, and Susan Saliba

Context: Quadriceps function improves after application of focal joint cooling or transcutaneous electrical nerve stimulation to the knee in patients with arthrogenic muscle inhibition (AMI), yet it is not known whether superficial heat is able to produce a similar effect. Objective: To determine quadriceps function after superficial heat to the knee joint in individuals with AMI. Design: Single blinded randomized crossover. Setting: Laboratory. Patients: 12 subjects (4 female, 8 males; 25.6 ± 7.7 y, 177.2 ± 12.7 cm, 78.4 ± 18.2 kg) with a history of knee-joint pathology and AMI, determined with a quadriceps central activation ratio (CAR) of <90%. Intervention: 3 treatment conditions for 15 min on separate days: superficial heat using a cervical moist-heat pack (77°C), sham using a cervical moist pack (room temperature at about 24°C), and control (no treatment). All subjects received all treatment conditions in a randomized order. Main Outcome Measures: Central activation ratio and knee-extension torque during maximal voluntary isometric contraction with the knee flexed to 60° were collected at pre, immediately post, 30 min post, and 45 min posttreatment. Skin temperature of the quadriceps and knee and room temperature were also recorded at the same time points. Results: Three (treatment conditions) by 4 (time) repeated ANOVAs found that there were no significant interactions or main effects in either CAR or knee-extension torque (all \( P > .05 \)). Skin-temperature 1-way ANOVAs revealed that the skin temperature in the knee during superficial heat was significantly higher than other treatment conditions at all time points (\( P < .05 \)). Conclusions: Superficial heat to the knee joint using a cervical moist-heat pack did not influence quadriceps function in individuals with AMI in the quadriceps.

Keywords: thermal modality, central activation ratio, knee-extension torque, knee injuries
potential to diminish AMI in the quadriceps, as it could be speculated that therapeutic heat would stimulate sensory receptors in a manner similar to that of joint cryotherapy and transcutaneous electrical nerve stimulation (TENS) in terms of modulating pain.9–11 Regardless of whether significant pain is present, it could be speculated that superficial heat would also stimulate the cutaneous afferents that may mediate quadriceps inhibition. However, this potential therapeutic benefit associated with superficial heat has not been investigated. Therefore, the purpose of this study was to determine whether superficial knee-joint heating with a moist-heat pack increases the quadriceps’ volitional activation and maximal strength in individuals with AMI. We hypothesized that quadriceps activation and peak knee-extension torque would immediately increase after the heating of the knee joint.

Methods

Design

This study was a single-blinded randomized controlled laboratory study with a crossover design. The independent variables were treatment condition and time. Subjects were randomly allocated into 1 of 3 treatment conditions on different days with concealed envelopes: moist-heat pack (heat treatment), a room-temperature moist pack (sham treatment), and no intervention (control). The treatment conditions were applied on 3 separate days at least 48 hours apart. The main outcome variable was quadriceps volitional activation, as expressed with central activation ratio (CAR) and the peak knee-extension torque during a maximal voluntary isometric contraction (MVIC). The outcome variables were recorded pretreatment, immediately posttreatment, 30 minutes posttreatment, and 45 minutes posttreatment. The same examiner assessed all outcome variables and was blinded to treatment conditions throughout the whole study. The examiner was asked to leave the testing room after taking baseline measurements. Another investigator serving as a therapist entered the room and applied the allocated treatment dictated in the concealed envelope.

The examiner returned to the room, where there was a screen placed by the therapist in front of the subject that allowed the examiner to see only the subject’s face, not the knee. Subjects were also instructed to refrain from talking about their intervention to the examiner.

Patients

Our sample size was determined with a study done by Petrosimone et al12 that investigated effects of knee-joint cooling on quadriceps CAR in patients with tibiofemoral osteoarthritis. From that study, we took a baseline variability of 0.13 in CAR and the expected mean change of 0.15 in CAR after knee-joint cooling and put them into our calculation to estimate a sample size, along with an alpha level of .05 and a 1-beta level of .80. The sample size of 12 was found to provide an adequate power for the current study.

Twelve subjects (4 female, 8 males) with a history of knee injury or surgery participated in this study (Table 1). The International Knee Documentation Committee’s (IKDC) subjective knee form was administered to all subjects to quantify their current knee function, with 100% indicating full knee function without presence of symptoms in activities of daily living and sports.13 Our results of the mean score of 76.3% ± 16.3% showed that our subjects had limited knee function that may have resulted from their previous knee injury or surgery.

Subjects were recruited from a university community and were included in this study if they self-reported a previous history of knee-joint injury or surgery, were age 18 to 65 years, and had AMI in the quadriceps as measured with a CAR less than 90% during screening. Subjects were excluded if they reported a history of neurological disorders or lower extremity injury other than knee injury, had skin breakdown on electrode sites, or reported acute pain that would preclude muscle contraction. Subjects were also excluded if they had areas of impaired circulation, a fear of electricity, or current lower extremity injury within past 6 weeks. The university’s institutional review board approved the study. All patients were read and signed an informed-consent form and underwent screening before participation in the study.

<table>
<thead>
<tr>
<th>Injury</th>
<th>Timea</th>
<th>Age (yb)</th>
<th>Height (cm)b</th>
<th>Mass (kgb)</th>
<th>IKDC scoresb</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACL reconstruction (n = 2)</td>
<td>N/A</td>
<td>25.0 ± 5.7</td>
<td>182.9 ± 18.0</td>
<td>78.9 ± 28.2</td>
<td>90.2% ± 13.8%</td>
</tr>
<tr>
<td>Meniscus repair (n = 2)</td>
<td>N/A</td>
<td>22.5 ± 0.7</td>
<td>184.2 ± 9.0</td>
<td>74.6 ± 9.9</td>
<td>87.4% ± 13.0%</td>
</tr>
<tr>
<td>Patellofemoral injury (n = 3)</td>
<td>N/A</td>
<td>24.7 ± 7.2</td>
<td>181.2 ± 5.3</td>
<td>84.4 ± 9.6</td>
<td>73.6% ± 27.0%</td>
</tr>
<tr>
<td>Chronic knee pain (n = 5)</td>
<td>59 (7, 302)</td>
<td>27.2 ± 9.8</td>
<td>168.5 ± 13.1</td>
<td>73.3 ± 23.0</td>
<td>67.2% ± 5.0%</td>
</tr>
<tr>
<td>Total (N = 12)</td>
<td>55 (5, 302)</td>
<td>25.6 ± 7.7</td>
<td>177.2 ± 12.7</td>
<td>78.4 ± 18.2</td>
<td>76.3% ± 16.3%</td>
</tr>
</tbody>
</table>

Abbreviations: ACL indicates anterior cruciate ligament; IKDC, International Knee Documentation Committee.

a Median (minimum, maximum) times, in months, spent since injury. b Means ± SD. c One patient with patellofemoral injury failed to recall the injury date.
Instruments
A Biodex isokinetic dynamometer (Biodex System 3 Pro, Biodex Medical Systems, Shirley, NY) was used to assess force production during MVIC. A custom-made coaxial cable was used to output the corresponding analog signal from the Biodex to a 16-bit data-acquisition system (MP150, BIOPAC Systems, Inc, Goleta, CA) for digital conversion and measurement of force output. A square-wave stimulator (S88, GRASS Telefactor, West Warwick, RI) in conjunction with a stimulation isolation unit (SIU8T) produced a 100-millisecond train of 10 stimuli, at 100 pulses/s, with a pulse duration of 0.6 milliseconds and a 0.01-millisecond pulse delay for the superimposed burst. Two 8 × 14-cm carbon-impregnated electrodes (Bloomex International Inc, Elmwood Park, NJ) were used to deliver a 125-V stimulation, and a highly conductive multipurpose gel (Parker Laboratories Inc, Fairfield, NJ) was used as the coupling medium for the electrodes. In addition, a moist-heat pack commercial unit (model SS, Chattanooga Group, Austin, TX) was used to keep the moist-heat packs at 77°C. The heat pack was a traditional cervical pack that measured 24 in. long (Chattanooga Group) and was covered with a standard cover before application. A room-temperature moist-heat pack with a standard cover was used for the sham. To record skin temperatures, the Physitemp Thermes USB model (Physitemp Instruments, Inc, Clifton, NJ) was used with InstaCal software (Measure Computing, Norton, MA).

Procedure

CAR Assessment. During screening, all the subjective demographic data and the IKDC scores were collected, and the subjects were prepared for CAR testing. The procedure for CAR testing is consistent with previously reported methods.12,14 Two 8 × 14-cm carbon electrodes were placed over the proximal vastus lateralis and the distal vastus medialis muscles of the quadriceps.15 The electrodes were secured on the skin via prewrap to ensure their placement over the course of the trial. The subjects were then placed in the dynamometer at 60° of knee flexion, hips flexed to 85°, and the trunk at 85° of flexion.16 The lower leg was then secured to the dynamometer just proximal to the ankle joint. Three restraint belts were used to secure the subject during testing to reduce accessory muscle activity.17 Once subjects were properly positioned in the dynamometer, 2 surface thermocouples were placed over the patellar tendon and midthigh to record skin temperature, and an additional thermocouple was placed in the air to measure room temperature. The 3 measurements of skin temperature and ambient temperature at each site were recorded at each measurement time point to ensure that the moist-heat pack provided superficial heat solely to the knee joint, not the quadriceps.

The CAR testing began with a graded warm-up that consisted of a series of submaximal contractions at 25%, 50%, 75%, and 100% of the subject’s perceived MVIC during knee extension. Once the subject was familiarized with the MVIC, the volitional contraction was paired with submaximal stimuli of 25%, 50%, and 75%, 100% of the maximum testing voltage of 125 V. The subject was then asked to perform 3 to 5 MVIC trials until the investigator was confident that the subject could complete the task. During testing, the subject performed 3 MVICs separated by 60-second rest periods with the addition of the superimposed electrical bursts. The investigator manually applied the stimulation when there was an observable plateau in the peak quadriceps torque in the output graph. If the average of 3 CARs was less than 90%, the subject was included in the study. After successful screening, participants were scheduled for 3 more visits to examine the effects of the intervention. During those visits, the subjects’ CARs were recorded in the same manner of screening at 4 different time points: preintervention, immediately postintervention (after removal of the intervention), 30 minutes postintervention, and 45 minutes postintervention. Three trials of CAR were performed at each time point.

Superficial Heat. A traditional 61-cm-long cervical moist-heat pack (Chattanooga Group, Austin, TX) was used to deliver superficial heat to the knee joint. The cervical pack was chosen to surround only the knee joint. The heat pack was covered with a standard cervical cover and secured to the knee joint using an elastic bandage for 15 minutes (Figure 1). The sham treatment condition consisted of the same size cervical pack without heat (room temperature) that was applied to the knee joint in the same manner as the superficial heat. The control condition included no treatment; subjects sat in the dynamometer for 15 minutes. The investigator assessing outcomes was blinded to intervention assignments, and another investigator applied all interventions.

Data Processing. CAR was calculated by dividing the torque of the MVIC (TMVIC) by the torque produced by the TMVIC plus the superimposed burst (TSIB) in the following equation as previously described12,14:

\[ \text{CAR} = \frac{\text{TMVIC}}{\text{TMVIC} + \text{TSIB}} \]

The mean knee-extension-torque value during MVIC was calculated by using the force value at 100 milliseconds on the plateau in the force curve just before the application of the exogenous electrical stimulation. All torque data were normalized to subject body mass. The mean values of 3 separate trials of CAR and knee-extension torque were used for statistical analysis.

Statistical Analysis
Separate 4 × 3 repeated-measures ANOVAs were performed to compare the CAR and the knee-extension torque over 4 time points (pretreatment and immediately, 30 min, and 45 min posttreatment) between 3 treatment conditions (superficial heat, sham, and control). Simple contrasts were performed post hoc in the event of a significant main effect or interaction. For temperature measured at each site, separate 1-way ANOVAs were used.
to compare temperature between treatment conditions at each time interval. Tukey post hoc tests were used to locate specific differences. Significance levels were set a priori at .05. All statistical analyses were performed with the Statistical Package for the Social Sciences (version 19, SPSS Inc, Chicago, IL).

Results

There were no treatment condition × time interactions for knee-extension torque ($F_{6,99} = 1.1, P = .40$) or quadriceps CAR ($F_{6,99} = 1.0, P = .43$). Neither knee-extension torque nor quadriceps CAR was different over time (torque $F_{3,99} = 1.7, P = .17$; CAR $F_{3,99} = 1.9, P = .14$) or between sessions (torque $F_{1,33} = 0.3, P = .76$; CAR $F_{1,33} = 0.1, P = .92$). Descriptive data are shown in Table 2.

Neither ambient room temperature nor thigh skin temperatures were different between sessions at any time point ($P > .05$ for all measures), indicating a focal heating of the knee joint only. However, cutaneous temperature at the knee was significantly different between sessions at all time points except baseline. Post hoc tests indicate that temperatures during the knee-heating sessions were significantly higher than in the sham ($P < .001$) and control sessions ($P < .001$) immediately after heating. The surface temperatures at the knee remained elevated at 30 and 45 minutes postheating compared with sham ($P = .002$, $P = .003$, respectively) and control sessions ($P = .001$, $P = .006$, respectively), which are shown in Figure 2.

Discussion

Our hypotheses that superficial knee-joint heating would immediately increase quadriceps volitional activation and strength were not supported by our results. We found that the superficial heat to the knee joint, in the form of moist-heat pack, did not change either the quadriceps voluntary activation, as expressed with CAR, or strength, as measured with knee-extension torque during MVIC. These findings are not consistent with other modality interventions such as focal knee-joint cooling or TENS. Various factors may have affected our results, such as the depth of penetration of the energy, the type of receptor activated with superficial heat, or the reflexive mechanisms of AMI.

There is little evidence on the effects of superficial heat on neuromuscular function. Two studies investigated superficial heat and involuntary motoneuron recruitment of the soleus, as measured with Hoffmann reflex. They found that the motoneuron excitability was not changed after application of superficial heat either over an entire lower leg using an electrically heated blanket or applied to the ankle with a standard moist-heat pack. However, those studies investigated healthy participants, while we examined individuals with volitional activation deficits in the quadriceps. Despite using a pathological population, our findings of no changes in quadriceps CAR or knee-extension torque after superficial heat to the knee joint may be due to heat’s lack of influence on quadriceps motoneuron activity. Other modalities such as joint cryotherapy and TENS have shown disinhibition of the quadriceps when applied to the knee joint. Knee-joint cryotherapy has been hypothesized to affect the inhibitory reflex mechanisms that will in turn result in an excitatory response of the previously inhibited α-motoneurons. Minimizing the inhibition has been found to improve quadriceps function including the knee-extension torque, motor fiber-conduction velocity, and functional performance in the
lower extremity. Similarly, TENS has been hypothesized to cause an increased interpretation of afferent signals as excitatory stimuli that may override the afferent inhibitory signals that cause inhibition. We speculated that heat might be capable of producing similar effects by overloading the sensory system and overriding the afferent inhibitory signals. This mechanism would potentially increase motoneuron-pool excitability and facilitate force output in the previously inhibited quadriceps. Our results, however, did not support this speculation.

A proposed method of sensing heat is through the thermal receptors, as well as nociceptors in the skin. These receptors are responsible for sensing heat or heat-like stimuli (i.e., capsaicin). The cumulative effect of sensory information and the specific type of receptor that is excited provide either a painful or a potentially analgesic effect. The transient receptor potential cation channel subfamily V member-1 (TRPV-1) nociceptors become active above 40°C, and prolonged stimulation can cause a desensitizing effect, which may be responsible for analgesia. Our mean surface temperatures at the knee were 41.1°C and should have been able to activate the TRPV-1 receptors. However, the participants in our study were not in pain at the time of testing, and the effect of the heat may have been minimized as a result.

Superficial heat has been proposed to provide a noxious response, which has been shown to influence the α-motor neuron in animals but could be excitatory or inhibitory. Valeriani et al. found that noxious heat stimulation resulted in an inhibition of the primary motor cortex. Similarly, Falla et al. observed a decrease in net activation and a change in spatial activation of the upper trapezius muscle after noxious stimulation. Conversely, Sargeant demonstrated a significant increase in both

### Table 2 Central Activation Ratio and Knee-Extension Torque, Mean ± SD

<table>
<thead>
<tr>
<th>Time point</th>
<th>Treatment Condition</th>
<th>Control</th>
<th>Sham</th>
<th>Heat</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Pretreatment</td>
<td>Immediately</td>
<td>Posttreatment</td>
</tr>
<tr>
<td>Central activation ratio</td>
<td>0.66 ± 0.18</td>
<td>0.63 ± 0.18</td>
<td>0.67 ± 0.23</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.63 ± 0.19</td>
<td>0.62 ± 0.19</td>
<td>0.65 ± 0.22</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.61 ± 0.19</td>
<td>0.63 ± 0.21</td>
<td>0.66 ± 0.21</td>
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</tr>
<tr>
<td></td>
<td>0.61 ± 0.18</td>
<td>0.63 ± 0.19</td>
<td>0.65 ± 0.21</td>
<td></td>
</tr>
<tr>
<td>Knee-extension torque (Nm/kg) Pretreatment</td>
<td>2.32 ± 1.09</td>
<td>2.02 ± 0.80</td>
<td>2.38 ± 1.31</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2.35 ± 1.14</td>
<td>2.14 ± 0.99</td>
<td>2.57 ± 1.49</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2.30 ± 1.15</td>
<td>2.18 ± 1.07</td>
<td>2.52 ± 1.40</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2.22 ± 1.04</td>
<td>2.19 ± 1.17</td>
<td>2.48 ± 1.46</td>
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</tr>
</tbody>
</table>

**Figure 2** — Skin-surface temperatures on the knee before and after interventions. *Temperature after superficial heat to the knee significantly increased from baseline. †Temperature after superficial heat to the knee significantly higher than other interventions.
force output and power when the legs were heated to 44°C. The effects of superficial heat may be paradoxical, potentially depending on whether the heat is placed on the muscle or joint and the extent of overall heating or whether pathology is present. Our results were similar to those of Huffman et al., who investigated the role of a menthol counterirritant on motoneuron-pool excitability and also found no effect.

We did find that our superficial heat treatment with a cervical moist-heat pack successfully increased skin temperature superficial to the knee joint. This method of focal knee heating may be an effective clinical option for clinicians dealing with patients with knee-joint pain or stiffness. The increased temperature may permit a greater response to interventions directed to increase range of motion, such as joint mobilization or stretching. However, it is not clear that superficial heat applied to the knee joint would affect the musculature of the quadriceps in either a facilitatory or an inhibitory manner.

In conclusion, a 15-minute superficial moist-heat treatment to the knee joint did not change quadriceps central activation or maximal isometric knee-extension torque in asymptomatic individuals with a history of knee injury.

References