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Research article

EFFECTS OF ANKLE JOINT COOLING ON PERONEAL SHORT LATENCY RESPONSE

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ABSTRACT

While cryotherapy has direct physiological effects on contractile tissues, the extent to which joint cooling affects the neuromuscular system is not well understood. The purpose of the study was to detect changes in ankle dynamic restraint (peroneal short latency response and muscle activity amplitude) during inversion perturbation following ankle joint cryotherapy. A 2x3 factorial design was used to compare reaction time and EMG amplitude data of treatment conditions (cryotherapy and control) across time (pre-treatment, post-treatment, and 30 min post-treatment). Thirteen healthy volunteers (age 23 ± 4 yrs, ht 1.76 ± 0.09 m, mass 78.8 ± 16.6 kg), with no history of lower extremity joint injury participated in this study. Surface EMG was collected from the peroneus longus (PL) of the dominant leg during an ankle inversion perturbation triggered while walking. Subjects walked the length of a 6.1 m runway 30 times. A trap door mechanism, inducing inversion perturbation, was released at heel contact during six randomly selected trials for each leg. Following baseline measurements, a 1.5 L bag of crushed ice was applied to the lateral ankle of subjects in the treatment group with an elastic wrap. A bag similar in weight and consistency was applied to the lateral ankle of subjects in the control group. A repeated measures ANOVA was used to compare treatment conditions across time ($p < 0.05$). Maximum inversion range of motion was 28.4±1.8° for all subjects. No overall condition by time difference was detected ($p > 0.05$) for PL reaction time. Average RMS EMG, normalized to an isometric reference position, increased in the cryotherapy group at the 30 min post-treatment interval relative to the control group ($p < 0.05$). Joint cooling does not result in deficiencies in reaction time or immediate muscle activation following inversion perturbation compared to a control.

KEY WORDS: Dynamic stability, reaction time, cryotherapy.

INTRODUCTION

Cryotherapy continues to be a popular intervention in the management of acute and chronic musculoskeletal conditions. It has been promoted for its beneficial effects on pain, mediation of the inflammatory process, and reduction of secondary injury (Knight, 1995). Used in combination with active exercise, cryotherapy is included as part of the rehabilitative process of cryokinetics (Knight, 1995). It is also used clinically as a treatment prior to performance or activity, although some clinicians may consider use of cryotherapy prior to activity to be an inappropriate treatment.

The effects of cryotherapy on muscle function remain controversial. Cooling muscle tissue appears to have distinct physiological effects on muscle contraction, including depressed muscle spindle activity (Oksa et al., 2000), decreased ATP-hydrolysis, and impaired calcium release and uptake

in the muscle (Ferretti, 1992). Investigators disagree as to whether muscle cooling has an effect on force production (Burke et al., 2000; Cornwall, 1994, Hatzel and Kaminski, 2000; Kimura et al., 1997; Mattacola, 1993; Verducci, 2000), joint position sense (Thieme et al., 1996; Uchio et al., 2003), or performance (Cross et al., 1996; Evans et al., 1995). However, cooling the joint, independent of the muscles, appears to be beneficial to isolated soleus motor recruitment (Hopkins and Stencil, 2002; Krause et al., 2000) and have no effect on lower extremity kinetics and kinematics in the closed chain during a semirecumbant stepping motion (Hopkins and Adolph, 2003). The disparity observed in the literature appears to be the result of the type of measurement used, the location of cryotherapy treatment (muscle or joint), and the time at which the motor output measurement was taken (during or following ice application) (Hopkins and Stencil, 2002).

Figure 1. Runway with built in trap door segment used for ankle inversion perturbation.

Few studies have examined the effects of cryotherapy on the ability of joint musculature and the sensorimotor system to stabilize the joint. Miniello et al. (2005) concluded that cold water immersion of the entire lower leg resulted in no impairment of ankle stabilization following landing from a jump. However, Kinzey et al. (2000) found a decrease in vertical impulse during a single leg vertical jump following cold water immersion of the ankle. The authors suggested that since the average vertical ground reaction force was not changed, the time component was primarily responsible for deficits in vertical impulse. In other words, either the time necessary to produce force following cooling is greater, or the muscle contraction time is slower. These authors suggested that a decrease in nerve conduction velocity was a primary contributor to the decrease in vertical impulse (Kinzey et al., 2000). In both of these studies the ankle was cooled along with musculature of the lower leg. While some (Hopkins and Adolph, 2003; Hopkins and Stencil, 2002) suggest that joint cooling produces a different motor response than muscle cooling, it remains to be seen whether joint cooling (independent of muscle cooling) may have an effect on dynamic stabilization of the joint.

The purpose of this study was to determine if ankle joint cooling has an effect on peroneal short latency response and activation following ankle inversion perturbation during walking. These data will provide insight into whether joint cooling adversely affects the timing and quality of the peroneal contraction following inversion perturbation.

METHODS

Subjects

Thirteen (7 male, 6 female; age 23±4 yrs, ht 1.76 ± 0.09 m, mass 78.8 ± 16.6 kg) healthy, physically active volunteers participated in this study. Physically active was defined as participating in at least 20 min of exercise 3 days per week or more. Volunteers had no history or symptoms of any disorder of the neuromuscular system or acute lower extremity injury. Subjects provided informed consent in accordance with the University Institutional Review Board.

Instrumentation

Muscle activity was recorded using a Biopac MP150 system (BIOPAC Systems Inc., Santa Barbara, CA). Signals were amplified (TEL100M, BIOPAC Systems Inc., Santa Barbara, CA) from disposable, pre-gelled Ag-AgCl electrodes. The input impedance of the amplifier was 1.0 megaohm, with a common mode rejection ratio of 110 dB, high and low pass filters of 10 and 500 Hz, a signal to noise ratio of 70 dB, and a gain of 1000. EMG data were collected at 1000 Hz using the Acknowledge 3.73 software package (BIOPAC Systems Inc., Santa Barbara, CA). Raw EMG signals were processed using a root mean square (RMS) algorithm with a 10 msec moving window.

A trap door mechanism built into a runway (6.1 m long x 0.76 m wide x 0.25 m high) was used to model an ankle inversion injury mechanism (Figure 1). The runway consists of five 1.22 m

interchangeable segments, with the trap door mechanism incorporated into one segment. Within this segment a vertical support on each side can be removed by a mechanical lever allowing for a walking surface rotation (inversion) of 30° upon foot contact. When the mechanical lever removes the vertical support, the door rests on spring ball plungers for support until 0.45 kg of pressure is applied to the trap door. Two adhesive, non-slip strips (Figure 1) mark the foot path to ensure appropriate foot placement and to prevent the foot from slipping when the trap door falls. Electromagnetic switches on the platform trap doors output a signal sampled with the EMG data to mark the trapdoor release for subsequent analysis.

Procedures

All subjects reported to the lab on 3 separate occasions: an orientation session, testing session 1, and testing session 2. All sessions were separated by 1 week. One week prior to the initial testing subjects reported for an orientation session. Subjects were oriented to EMG electrode placement and the function of the trap door mechanism. Each subject walked several lengths of the 6.10 m runway to the cadence of a metronome (100 beats·min^{-1}) to practice the step rate and to establish the subject's step length. The step length was used to determine a starting point from which the subject would consistently step on the runway segment containing the trapdoor mechanism.

On the initial test day, surface EMG electrodes were applied on the skin over the peroneus longus (PL) of the dominant leg. Leg dominance was defined as the stance leg from which the subject preferred to jump. The proximal electrode was centered on the PL 3 cm distal to the fibular head. Electrodes were placed 2 cm center to center in line with the longitudinal axis of the muscle. The skin was prepared by lightly abrading and cleaning with isopropyl alcohol. A ground electrode was placed over the tibial tuberosity. Electrodes were outlined with a permanent marker (Sharpie) for the 2nd testing session. Appropriate electrode placement was visually verified by observing tracings from isolated isometric eversion contractions. To monitor ankle inversion, an electrogoniometer (SS20, BIOPAC Systems Inc., Santa Barbara, CA) was applied laterally over the ankle joint, in line with the fibula during stance. One support was secured to the foot distal and in line with the lateral malleolus, and one support secured to the leg just proximal to the lateral malleolus.

Prior to data collection, each subject warmed up on an exercise bike for 5 min at a moderate intensity. All subsequent data were collected from the subject's preferred leg for kicking. Subjects wore their own low-top athletic court shoes during testing, and subjects wore blinders that obstructed the field of vision below eye level.

Prior to testing, subjects performed a 5 sec isometric contraction for subsequent scaling of EMG data. The isometric reference contraction (IRC) was performed while the subject was sidelying with the dominant (superior) ankle hanging off the end of the treatment table. A 4.5 Kg weight was hung from the midfoot, and subjects were asked to maintain the foot in a neutral position. IRCs were repeated following completion of each of the two conditions. Average processed activity from a 100 msec window starting at 2.5 seconds was used in computing the mean value of the three isometric contractions (IRC). This value was used for normalizing the EMG data.

The subject began at the predetermined point on the runway, with instructions to walk to a sign at the end of the runway at the cadence of 100 steps/min audibly maintained by a metronome. The sign guided the subject to walk straight, and also indicated the end of the runway. Data were collected for 5 seconds which permitted time to walk the length of the runway. To ensure the subject did not lose balance and/or fall during perturbation, hand rails were available (Figure 1) and a research assistant walked behind the subject. Data were collected for 30 walking trials for each measurement interval (pre-treatment, post-treatment, 30 min post-treatment), with the trap door randomly triggered on foot contact for a total of 6 trials per leg. In other words, the subject walked the length of the runway 18 times with no perturbation, 6 times with right leg perturbation, and 6 times with left leg perturbation. The order was randomized. The subject's foot was visually monitored to be certain each stride was in line with a 10.2 cm friction strip applied to the runway (Figure 1). The subject was instructed to continue to walk forward along the runway following perturbation. Each perturbed trial was inspected to ensure that muscle activation was not premature in anticipation of trap door release. If premature muscle activation was detected, or the foot was not completely on the strip during perturbation, then the trial was not saved for analysis and the next random trial was performed. Each measurement interval (30 walking trials) took 5-8 min with 5-10 sec rest between each trial.

Immediately following pre-treatment measurements a 1.5 L bag filled with crushed ice (cryotherapy) or crushed, dry clay (sham control) was applied to the lateral ankle with an elastic wrap. The treatment was left in place for 30 min while the subject remained seated. Following treatment, electrodes were reattached and post-treatment

Table 1. Means (±SD) for peroneus longus reaction time (msec) following inversion perturbation during walking.

Treatment	Pre-treatment	Post-treatment	30 min Post-Tx
Cryotherapy	59.07 (6.22)	59.29 (5.54)	60.51 (5.22)
Control	59.45 (2.57)	59.54 (2.27)	60.09 (2.76)

measurements were collected. Ankle inversion testing began within 2 min following removal of the treatment. Thirty min following the initiation of post-treatment measurements, testing was initiated for the 30 min post-treatment measurements. Subjects returned 1 week later for the 2nd testing session, at which time they received either cryotherapy or sham treatment, whichever was not received in the previous testing session. Electrodes were reapplied at the locations previously outlined with the permanent marker. Treatment order was counterbalanced between subjects and all procedures were carefully administered to maintain consistency between testing sessions.

Filtered EMG and goniometer data were processed (RMS over 10 msec moving window) exported in a text file format for processing with custom software. Trap door release was identified from the appropriate channel data, and muscle activity onset was defined as an EMG level 4 standard deviations above the mean value of the EMG from the midpoint of the swing phase prior to trap door release. A custom-made automated program (Microsoft Visual Basic.NET) performed the necessary calculations. A graphical representation of each trial was viewed to confirm the timing of the trap door release and onset of muscle activity. Muscle reaction time was calculated as the time interval between the onset of the trap door release and the onset of muscle activity. The average EMG values (in %IRP) were calculated from the 100 ms interval following muscle onset.

Statistical analysis

Dependent variables included the peroneal short latency response time (reaction time) and peroneal mean EMG normalized to an isometric reference position. Electrogoniometer measurements were used to ensure all subjects inverted to approximately 30°. Reaction time and average EMG means were computed from the 6 trials collected from the preferred leg. A repeated measures 2 X 3 factorial ANOVA was used to detect differences in treatments (cryotherapy and sham control) over time (pre-treatment, post-treatment, and 30 min post-treatment). Tukey's honestly significant difference test was used to detect any post hoc differences. The *a priori* alpha level was set at $p \leq 0.05$.

RESULTS

Means and standard deviations are reported in Tables 1 and 2. Figures 2 and 3 present the data as a percent change from baseline. Maximum inversion range of motion was 28.4 ± 1.8° for all subjects. No treatment by time differences were detected for PL reaction time measurements ($F_{2,24} = 0.388$, $p = 0.682$, partial E^2=0.031, observed power = 0.105). A treatment by time interaction was detected for average PL EMG ($F_{2,24} = 9.146$, $p = 0.001$, partial $E^2 = 0.433$, observed power = 0.957). Follow up testing revealed a decrease in average PL EMG at the post-treatment interval (relative to baseline) for both groups ($p < 0.05$). The control group maintained depressed average EMG at the 30 min post-treatment interval ($p < 0.05$).

DISCUSSION

The PL short latency response was unchanged by cryotherapy treatment. Further, no PL activation deficiencies were observed in the 100 msec following onset compared to the control group. These observations suggest that cooling the ankle joint (independent of the muscle) has no negative effects on the timing and magnitude of the PL short latency response to inversion perturbation during walking.

During a jump landing, Miniello et al. (2005) reported that PL activation decreased in the 100 msec following the landing. However, it should be noted that the entire lower leg was cooled (up to the knee), including the PL. In the current study, cooling was limited to the lateral ankle joint only. Despite

Table 2. Means (± SD) for peroneus longus average EMG (% isometric reference position) following onset of muscle activation after inversion perturbation during walking.

Treatment	Pre-treatment	Post-treatment	30 min Post-Tx
Cryotherapy	300.4±165.6	273.7±162.3*	305.1±162.0
Control	336.9±155.0	297.2±114.4*	292.1±127.9*

*less than pre-treatment measures ($p < 0.05$)

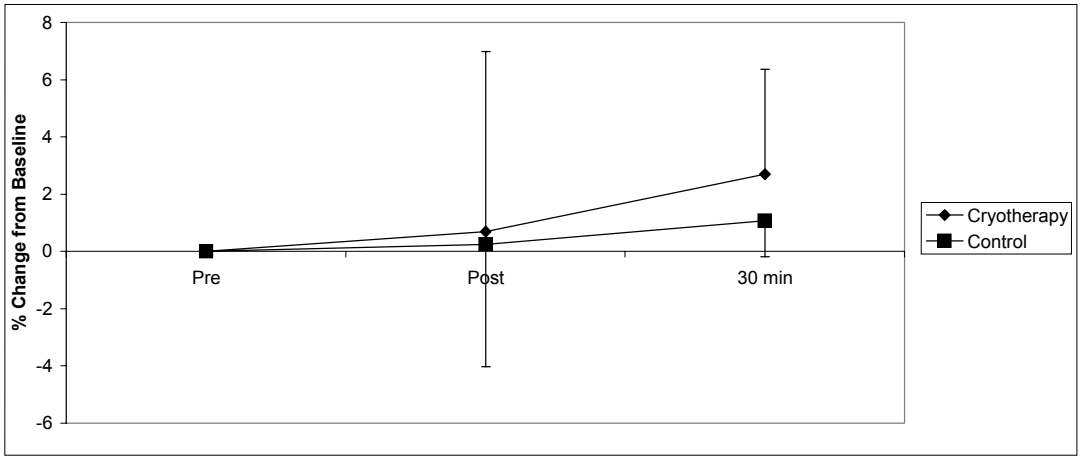

Figure 2. Peroneus longus reaction times presented as percent change from baseline.

the temporary decrease in PL activation and given the fact that change was observed in time for stabilization following the jump landing, Miniello et al. (2005) concluded that cooling the lower leg did not impair dynamic stability. Our data appear to be consistent with this conclusion. We speculate that the sensitivity or threshold of the muscle spindles within the PL were not cooled and therefore were unaffected by the cryotherapy treatment at the ankle. Therefore PL latency was not affected by the treatment in the current study.

Average EMG amplitude of the PL short latency response decreased in both groups following cooling (post-treatment interval). We speculate that this is primarily due to neural adaptation to the perturbation. Over several trials subjects became more comfortable with the perturbation mechanism, and fewer motor units were activated in response. Another factor to consider is that pressure was applied to the ankle in both groups as a bag was compressed to the ankle during one treatment and a sham bag was compressed to the ankle in the other treatment. The afferent feedback from the compression could have played a role in the decrease observed in both treatment groups at the post-treatment interval. It is also possible that fatigue played a role in the decrease observed at the post-treatment interval. However, given the time between measurement intervals (pre, post, and 30) we believe that fatigue played a minor role if any. Further, we have previously observed strong reliability [ICC(2,1) = 0.918] over the 6 repetitions in a single measurement session. For future work using this type of inversion perturbation model, we recommend that each subject practice several trials to help alleviate the accommodation observed between the pre-treatment and post-treatment intervals.

At 30 min post-treatment we observed an increase in PL activation relative to the control group. While this finding is difficult to explain, it is in part supported in the literature. Following knee joint cooling, voluntary quadriceps activation increased relative to a control during rewarming of the tissue (30 min post treatment) (Hopkins et al., 2004). Involuntary activation during the rewarming phase following joint cooling is also well documented (Hopkins et al., 2001; 2002, Krause et al., 2000). Following ankle joint cryotherapy, soleus activation remained facilitated above baseline levels

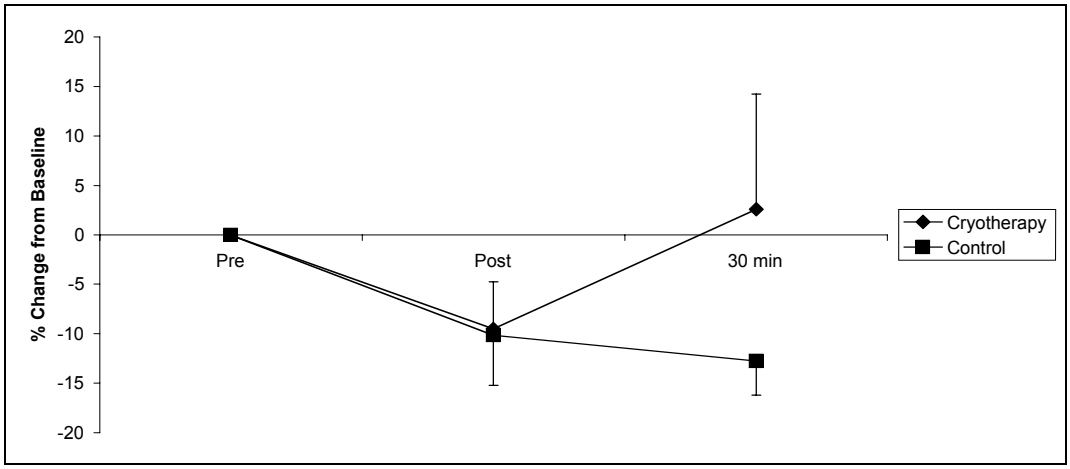

Figure 3. Average normalized peroneus longus EMG presented as percent change from baseline.

at the 60 min post treatment interval (Hopkins and Stencil, 2002). Increases in activation during rewarming are likely due to alterations in afferent input from skin and joint receptors and/or altered supraspinal drive. Oksa et al. (2000) argued that muscle activation changes due to cooling and rewarming are likely centrally regulated due to muscle agonist/antagonist pattern changes following cooling. While more data are needed to determine the contribution of increased PL activation to movement and dynamic stability, these data are consistent with the idea that ankle joint cryotherapy may be used prior to activity without a reduction in PL activation. Further, joint cryotherapy may be an effective adjunct intervention to assist in active exercise where an increase muscle activation may be indicated.

The model used in this experiment to examine dynamic muscle response to an inversion perturbation during walking is a novel approach to study dynamic stabilization characteristics of the ankle. Previous researchers (Benesch et al., 2000; Konradsen and Ravn, 1991; Konradsen and Ravn, 1990; Isakov et al., 1986) have not sufficiently tested response time of the peroneal musculature using an ankle inversion mechanism that examines dynamic restraint characteristics while the subject maintains a static postural stance. In order to more closely mimic the dynamic mechanism of an ankle sprain injury and the motor patterns active during gait, a runway with built in trapdoors was used in this study. This permits measurement and inspection of the timing and quality of the muscular response to perturbation while walking, taking into consideration sensorimotor factors only present during ambulation.

Clinically, these data suggest that joint cooling is a safe intervention prior to activity in terms of short latency response of the peroneals. Joint cooling has also been shown to have no effect on lower chain kinetic variables (peak and average torque and power) during activity (Hopkins and Adolph, 2003) nor time to stabilization (Miniello et al., 2005). While the use of cryotherapy prior to physical activity has been questioned (Ferretti, 1992), when the muscle is not cooled, the motor activity around the joint appears to be unaffected in most cases. However, more data are needed to examine other aspects of dynamic stability, postural control, and muscular function before a clinical conclusion is made.

A few limitations should be mentioned in regards to this study. Our use of healthy subjects was intended to provide an indication of how cryotherapy affects the dynamic response to ankle inversion perturbation. However, we recognize that subjects with acute or chronic ankle injury might respond differently to cryotherapy. It should also be noted however, that joint cooling was previously found to resolve deficits in motor recruitment due to joint effusion (Hopkins et al., 2002; 2004). Another limitation is our analysis of only the short latency response. Certainly we acknowledge that differences could exist in polysynaptic and centrally mediated responses. However, we chose to examine the short latency response as the first line of defense against an ankle injury mechanism. We also should note that the short latency response could change with varying gait speeds. We felt that the controlled, moderate gait speed used in this study would be indicative of functional movement.

CONCLUSION

Joint cryotherapy, independent of muscle cooling, produces no deficit in the timing of PL short latency response. Thus, it is safe to use cryotherapy as an intervention in terms of latency response. Further, joint cryotherapy may provide increased PL activity during the rewarming period following joint cooling. Further work should consider whether joint cooling may affect other areas of sensorimotor control.

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KEY POINTS

- Joint cooling is used as a treatment intervention prior to activity. Whether ankle cooling will affect dynamic restraint during functional movement is unknown.
- Short latency response should be measured during functional movement instead of during stance to take into consideration alterations in motor drive.
- Joint cooling has no effect on peroneal short latency response, and joint cooling may result in increased short term peroneal activation.
- Joint cooling has no effect on the peroneus longus as a dynamic stabilizer during walking.

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