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Intra-Articular Anesthesia and Knee Muscle Response

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Abstract

Background—Many receptors located within the intra-articular knee structures contribute to the neuromuscular responses of the knee. The purpose was to compare the automatic postural response induced by a perturbation at the foot before and after an intra-articular injection of a local anesthetic (bupivicaine), after a saline (sham) injection, and after no intra-articular injection (control) in the knee.

Methods—Muscle onset latencies and automatic response magnitudes for the vastus medialis, vastus lateralis, biceps femoris, medial hamstrings, tibialis anterior, and gastrocnemius were measured using EMG when anteriorly directed perturbations were applied to the feet of 30 subjects. All subjects then received a lidocaine skin injection followed by: an intra-articular bupivicaine injection (treatment group); an intra-articular saline injection (sham group); or no injection (control group), depending on their randomized group assignment. The perturbation tests were then repeated.

Findings—Muscle onset latencies and automatic response magnitudes did not change as a result of the intra-articular injections. Latencies were significantly greater for the vastus medialis and vastus lateralis when compared to the medial hamstrings, biceps femoris and tibialis anterior ($p<0.001$). Automatic response magnitudes for the tibialis anterior were significantly greater than those of the hamstrings, which were greater than those of the quadriceps ($p<0.001$).

Interpretation—There were no differences in muscle response when anteriorly directed perturbations were applied to the foot with or without an injection of local anesthetic in the knee. Intra-articular receptors were either unaffected by the anesthetic or the extra-articular receptors or receptors of the other joints were able to compensate for their loss.

Keywords

Muscle; Neuromuscular; Perturbation; Proprioception; Biomechanics

1. Introduction

Capsular, muscular, and ligamentous receptors are responsible for initiating automatic neuromuscular responses that protect the knee (Dhaher et al., 2003, Di Fabio et al., 1992, Friemert et al., 2005, Wojtys and Huston, 1994). The interaction of the receptors and the mechanisms of the response are unclear. The intra-articular receptors could initiate a reflex response to provide input and feedback to the automatic postural response (APR) of the body.

Stimulation of receptors within the ACL and joint capsule has been shown to produce reflexive contractions in the hamstring muscles (Tsuda et al., 2001, Fujita et al., 2000, Friemert et al.,

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2005, Di Fabio et al., 1992, Dhaher et al., 2003). Tsuda et al. (2001) verified the presence of the ACL-hamstring reflex loop by applying electrical stimulation to the ACL before and after a local anesthetic injection in the joint. They found that the anesthetic blocked the afferent impulses from the neural receptors in the ACL.

Barrack et al. (1983) evaluated the changes in knee joint proprioception and gait in response to injections of local anesthetic or saline (simulated effusion) in the knee. Tests were performed on subjects with normal knees, and were repeated after an intra-articular injection of lidocaine or saline. They reported that intra-articular anesthesia and simulated effusion had no effect on knee proprioception or gait patterns. Fluid injections of 20cc to 100cc have also been shown to reduce quadriceps muscle activation when testing the Hoffman reflex, suggesting that effusion from injury may affect muscle reflex activity (Palmieri et al., 2003, McNair et al., 1996). Palmieri et al. (2003) reported an increased ability to maintain single leg stance in healthy subjects after a 60mL saline injection. McNair et al. (1995) found no change in the ability to track limb movement after a 90mL injection of saline. In designing a study to evaluate the effects of intra-articular anesthesia on knee function, it is essential to include a sham group in which an equivalent fluid volume is injected into the joint to isolate anesthesia effects from those of the associated effusion.

The objective of this study was to examine the APR when an anterior-directed perturbation is applied at the foot before and after an intra-articular injection of local anesthetic (bupivicaine), sham (saline) injection, and no injection (control) into the knee. Comparisons of muscle onset latency (time) and automatic muscle response magnitudes were made. Our hypotheses were that intra-articular injection of local anesthesia would increase muscle onset latency and decrease the muscle response magnitude in comparison to sham injection and no intra-articular injection. Additional perturbation tests were performed on the control subjects to establish the reproducibility of muscle onset measurements during repeated testing. These data are necessary to establish if the APR is altered with an intra-articular injection of local anesthesia, or when an effusion is present. If no changes are observed then it will be possible to measure ACL strains when dynamic perturbations are applied to the limb in vivo (Fleming et al., 2001a, Beynnon and Fleming, 1998).

2. Methods

2.1. Subject

Thirty healthy subjects (15 men, 15 women, ages 18–59) were recruited from the University of Rhode Island and Rhode Island Hospital campuses (Table 1). The inclusion criteria were no history of neurological disease, lower extremity injury, low back pain, cardiovascular disorders, alcoholism, balance or vision disorders, or drug allergies. The study received IRB approval from Rhode Island Hospital and the University of Rhode Island. All subjects provided written informed consent prior to their participation.

2.2. Electromyography (EMG)

Surface EMG was used to measure muscle activity before, during and after the perturbation was applied to the feet (Hodges and Bui, 1996, Henry et al., 1998a, Di Fabio et al., 1992, Williams et al., 2003, Wojtys and Huston, 1994). The vastus medialis (VM), vastus lateralis (VL), biceps femoris (BF), medial hamstrings (MH), tibialis anterior (TA) and the gastrocnemius (GA) were measured. Surface electrodes were applied to the skin overlying the respective muscle bellies, as described by Cram and Kasman (1988), using on-site preamplifier modules (gain = 35) hardwired to an eight-channel differential amplifier (Model EMG-67, Therapeutics Unlimited, Iowa City, IA, USA). The bipolar silver/silver chloride electrodes (7 mm diameter) had a fixed intra-electrode distance of 2 cm. The signals were transmitted to the

main differential amplifiers (input impedance > 15 Megaohms at 100 Hz, frequency response = 20 – 4,000 Hz, common mode rejection ratio = 87 db at 60 Hz) where they were amplified (gain = 500 – 10,000) and RMS processed (time constant = 2.5msec). An oscilloscope (Model 2232, Tektronix, Inc., Beaverton, OR, USA) was used to check raw EMG signals for noise and artifact. Data acquisition and reduction were completed using the Enhanced Graphics Acquisition and Analysis system (RC Electronics, Goleta, CA, USA) and a personal computer. The analog-to-digital sampling rate was 1,000 samples/second/channel. Data were continuously sampled to a buffer, but permanently stored 1000ms before to 1000ms after the onset of the perturbation event.

2.3. Perturbation platform

The perturbation platform produced an unexpected anterior translation of the feet (Figure 1). In theory, an anterior perturbation at the ground-foot interface would shift the center of gravity of the body posteriorly, and provide an anteriorly directed shear load at the tibia relative to the femur. The APR would elicit a response from tibialis anterior, quadriceps, and rectus abdominis muscles, generally in that order (Henry et al., 1998b,Horak and Nashner, 1986). Thus, the APR will elicit a quadriceps contraction to maintain equilibrium, which in turn would strain the ACL (Beynnon and Fleming, 1998). The hamstrings will also respond to maintain balance and possibly protect the ACL by reducing anterior tibial translation.

The perturbation platform consisted of a sliding plate measuring 50cm x 50cm. It was supported by four pillow blocks containing ball bearings, which were permitted to slide on a pair of 1.25cm diameter bearing rails. Two high-tension springs loaded the system. A solenoid release was used to initiate plate motion. An accelerometer (Model 2001–3; Genisco Technology Corp, Simi Valley, CA, USA) was mounted beneath the plate to trigger data acquisition relative to plate motion onset and to document the kinematic profile of the platform (Figure 2). When activated, the plate moved 10cm anterior with average (standard deviation) peak anterior accelerations and velocities of 21.4 (4.42)m/s^2 and 1.05 (0.29)m/s, respectively (Figure 2). Perturbations of this magnitude have been shown to elicit an APR without placing subjects at risk to injury (Di Fabio et al., 1992,Henry et al., 1998a).

2.4. Protocol

Subjects' height and weight measurements were recorded. Six surface EMG electrodes were mounted over the bellies of the BF, MH, VL, VM, TA and GA after shaving and cleaning with an isopropanol scrub (Soderberg and Knutson, 2000, Cram and Kasman, 1998). Electrodes were oriented with the approximate fiber direction of the respective muscle group as described by Cram & Kasman (1998). A reference electrode was placed over the tibial tubercle of the contralateral limb. After a brief warm up, isometric tests were performed to establish the maximum voluntary contractions (MVCs) for each muscle group (Soderberg and Knutson, 2000). MVCs for the hamstrings and quadriceps muscles were measured using an isokinetic dynamometer (KinCom 500H; Chattecx, Chatanooga TN, USA), while those of the GA and TA were evaluated by pulling against inelastic resistive bands. The knee and ankle angles were set at 15° and 0°, respectively. Average MVC data for each muscle group were used to normalize the integrated EMG for that muscle produced during perturbation tests.

For the perturbation tests, subjects were placed in a safety harness to protect them from falling. Reference lines on the platform surface were used to standardize foot position (heels aligned, toes pointed anterior, and the feet spaced 40cm apart). Subjects were instructed to place equal weight on both feet with their knees flexed to 15°. The knee angle was checked by a therapist with a goniometer. 15° was chosen because the ACL strains are relatively high when the leg muscles are contracted at this knee angle (Beynnon et al., 1997, Beynnon and Fleming,

1998, Fleming et al., 2001b). Four repeated trials of the anteriorly directed perturbation were applied.

Perturbation timing was under investigator control. Although subjects knew the perturbation direction, they did not know when it would occur. Steps were taken to minimize the subject's ability to anticipate the event. Subjects wore ear plugs and were instructed to focus their eyes on an inanimate object to minimize external cues.

Subjects were then block-randomized to one of three experimental groups; 1) treatment, 2) sham, and 3) control. The randomization protocol was established to minimize selection bias while ensuring that 5 males and 5 females were assigned to each of the three experimental groups. All subjects received a 1cc 1% lidocaine skin injection to reduce skin discomfort using a 1.6cm (5/8") 25-gauge needle in the skin overlying the lateral parapatellar area. The superficial injection was followed by: 1) a 30cc intra-articular injection of 0.25% (75mg) bupivicaine (treatment group); 2) a 30cc intra-articular injection of saline (sham group); or 3) no intra-articular injection (control group), depending on the group assignment. The intra-articular injections were performed with a 3.8cm (1.5") 18-gauge needle through a lateral parapatellar injection site. A blottable effusion was noted and patients tolerated the injections. Subjects ambulated for 15-minutes to distribute the injected fluid and allow the local anesthetic to take effect (McNair et al., 1996).

Subjects were repositioned on the perturbation platform. Those assigned to the treatment and sham groups received four more anterior perturbation trials. Subjects in the control group received three more sets of four trials of the anterior perturbation test. The additional sets of trials for the control group were performed to document the reproducibility of the latency measurements and to evaluate potential learning effects.

2.5. Data Analysis

After the EMG signals were RMS processed, the data were evaluated from -250ms (pre-trigger) to 250ms (post-trigger). The trigger was marked by the accelerometer when board motion was initiated. The four trials within a set were ensemble averaged by taking a point-by-point average relative to the trigger. The pre-trigger data were used to establish baseline EMG activity for each muscle while the post trigger data established the perturbation response (Wojtys and Huston, 1994, Shultz et al., 2001).

Muscle latencies were determined from the EMG ensemble average tracings relative to the start of the perturbation board movement for each muscle. A computer algorithm low pass filtered (Butterworth) the signal at 50 Hz and calculated the RMS EMG threshold of three times the standard deviation of the pre-perturbation EMG signal. When the post-perturbation signal crossed the threshold and remained above it for at least 25 ms for the BF, MH, TA and GA, the onset of the perturbation was identified (Hodges and Bui, 1996) (Figure 2). We adopted the recommendation of Shultz et al. (2000) to use a 10ms window for the VL and VM. Muscle onsets were also determined manually by two investigators. If a difference greater than 15ms was noted between the computer generated value and one of the manually selected points, the EMG versus time plot was reviewed to determine if an anomaly in the output (i.e. muscle twitch, subject anticipation) may have caused the computer to select an erroneous point. If not, the computer selection was accepted. Agreement between the manual reviewers and the computer selected onset values occurred in 96% of the trials.

Automatic response magnitudes were determined using integrated EMG. We followed an approach similar to that of Di Fabio et al. (1992) but tuned the method to each muscle by measuring the area under the EMG versus time curve from muscle onset to volitional onset for each muscle group (Henry et al., 1998a). The onset of volitional contraction was established

by manual review of the trials performed on the control subjects. Volitional contraction was defined as the sustained higher level activity that occurred subsequent to the automatic postural response (Figure 2), and was visually determined for each muscle of each control subject. Onset times were extremely similar for the VL and VM, and for the ST and BF; data for these pairs of muscles were collapsed. When averaged across subjects, the onset of the volitional response was found to be 131ms for the VL and VM, 146ms for the ST and BF, and 166ms for the TA after the involuntary onset. These windows were then used to establish the response magnitudes for the comparison between treatment groups. Automatic response magnitudes were reported as a percentage of EMG relative to the mean peak MVC value previously obtained for each muscle over the same time window.

We attempted to measure EMG output from the gastrocnemius muscle in this study. Unfortunately, the gastrocnemius output was negligible during this perturbation, which is not surprising because the anterior translation would cause subjects to fall posteriorly, engaging the TA and quadriceps muscles (Di Fabio et al., 1992).

2.6. Statistical Analyses

Repeated measures analysis of variance was used to examine differences in muscle onsets due to treatment, time, and muscle. Treatment was an across-subject factor with three levels (anesthesia, saline, control). Time and muscle were within subject factors with two (pre-treatment, post-treatment), and five levels (VL, VM, MH, BF, TA), respectively. When appropriate, pair-wise comparisons were performed using Fishers Least Significant Difference test. A similar analysis was performed to test for differences in automatic response magnitudes. The study was designed a priori to have power of 0.80 to detect a 25ms mean difference in muscle onset.

Repeated measures analysis of variance was used to examine the reproducibility of the muscle onset latencies due to trial set (1, 2, 3, 4) and muscle (VL, VM, MH, BF, TA). The four sets of trials, which were performed on each control subject, were evaluated for each muscle group.

Results

All subjects successfully underwent the injection protocols and were able to endure the perturbation testing protocol without complications or pain. Of the 30 subjects recruited, complete data sets were obtained from 28. In two subjects, either the pre-injection (1 subject) or post-injection data (1 subject) were lost due to technical issues with the data acquisition software. These patients were excluded from the statistical analyses leaving 9 patients in the treatment group, 9 patients in the sham group, and 10 patients in the control group.

3.1. Muscle onset latency

There were no significant differences between muscle onset latencies due to treatment ($p=0.91$) or time ($p=0.11$). There were significant differences in latency due to the muscle ($p<0.001$). Muscle onset latencies for the quadriceps (VM & VL) were significantly longer than those for the MH, BF or TA (Figure 3). The mean differences (standard deviation) in muscle onset latencies (pre-post), pooled across muscles, were -1.1 (12.7)ms, 2.2 (11.7)ms, and 3.4 (16.9) ms for the treatment, sham, and control groups, respectively.

3.2. Automatic response magnitude

There were no significant differences in the automatic response magnitudes due to treatment ($p=0.85$) or time ($p=0.76$). However, there were significant differences in response magnitudes due to muscle group ($p<0.001$). The response magnitude for the TA was significantly greater than those of the hamstrings (MH & BF), which were significantly greater than those of the

quadriceps (VM & VL) (Figure 4). The mean (standard deviation) for the pre-and post-differences, pooled across muscle groups, were -0.25 (10.8)%, –0.5 (19.5)%, and 0.76 (17.3)% MVC for the treatment, sham, and control groups, respectively.

3.3. Reproducibility

There was no significant effect in muscle onset latency in the control subjects due to the trial set ($p=0.28$), and no significant interaction between trial set and muscle group ($p = 0.20$). The mean (standard deviation) onset latencies for sets 1, 2, 3, and 4 were 91 (30.5), 85 (24.2), 89 (24.2), and 87 (26.2)ms respectively, when pooled across muscle groups. These data verify that no learning effects were present for any muscle (Figure 5).

4. Discussion

The results did not support the hypotheses that an intra-articular injection of local anesthetic would increase muscle onset latency and decrease automatic response magnitude in the knee in comparison to sham (saline effusion) injection or no intra-articular injection for an anteriorly directed perturbation applied to the foot. The experimental design included both a sham group, because knee effusions have been linked to the inhibition of the neuromuscular response of the joint (McNair et al., 1996, Palmieri et al., 2003), and a control group to provide a true baseline. Since there were no differences in muscle onset latencies or response magnitudes between groups, we also conclude that the 30cc effusion (followed by 15 minutes of mild exercise) did not affect the APR when anteriorly directed perturbations were applied to the foot. This finding suggests that local anesthetic or the simulated effusion does not affect the intra-articular receptors; or the extra-articular receptors or receptors of other joints were able to compensate for their loss. Mechanotransducers that provide input into the APR are present in all ligament and capsular structures (Kim et al., 1995, Buchanan et al., 1996, Schultz et al., 1984).

The muscle onset latencies for each muscle group were comparable to literature values. Di Fabio et al. (1992) used a perturbation similar to that utilized in our study and found similar muscle latencies in the TA and the quadriceps to those reported in the present study. However, they found that the average response of the hamstrings were highly variable and slower than that of the quadriceps. Wojtys and Huston (1994) initiated an anteriorly directed perturbation directly to the proximal tibia using a pendulum. They reported hamstring onset latencies of ACL-intact subjects that were similar to those measured in the present study. However, the quadriceps responded more quickly than in our study. Shultz et al. (2000; 2001) measured muscle onset latencies when applying an anteriorly directed perturbation in combination with either an internal or external rotatory component. Again, muscle response times were very similar to those documented in the present study, with the hamstrings responding quicker than the quadriceps. Differences in perturbations, subject position, and muscle onset algorithms may explain the minor differences between studies.

Proprioceptive and reflexive responses of the knee have been evaluated with and without anesthesia (Khabie et al., 1998, Barrack et al., 1983, Clark et al., 1979). Tsuda et al. (2001) demonstrated the presence of an ACL-hamstring reflex arc when directly stimulating the ACL with an electrode. The electrode was implanted after a skin injection and EMGs from the MH and BF were recorded. The reflex response disappeared once a local anesthetic (2% lidocaine) was injected into the knee joint capsule to interrupt the intra-articular sensation and mechanoreceptors of the cruciate ligaments. Fahrer et al. (1988) found that an injection of lidocaine following joint aspiration in patients with rheumatoid arthritis resulted in increased isometric muscle strength compared to joint aspiration without local anesthesia. Thus, it is likely that the intra-articular receptors were affected by the local anesthesia in the present study.

In testing the Hoffman reflex, joint effusions have been linked to arthrogenic muscle inhibition at the knee (Hopkins et al., 2002, Hopkins et al., 2001, Palmieri et al., 2004). This could be caused by expansion of the capsule, stretching the capsular receptors, which in turn could inhibit the reflex loop. In our study we did not test the Hoffman reflex, only the global APR. We found that a 30cc saline injection did not alter the APR after 15 minutes of exercise, a finding which is supported by McNair et al. (1996). They concluded that the inhibitory response produced by a simulated effusion was negated following 4 minutes of sub-maximal exercise. The initial inhibition produced by the saline injection may dissipate as the injected fluid is dispersed within the joint, or as the compliance of the joint capsule increases from the initial pressure gradient introduced by the injection. McNair et al. (1995) also found that a 90ml injection of saline did not affect subjects' ability to track limb movement, while Palmeiri et al. (2003) found an increased ability to maintain single leg stance after subjects received a 60ml injection of saline. These findings may differ from those established using the Hoffman reflex. When examining the postural and proprioceptive responses of a joint, the many receptors within the somatosensory system of the body provide feedback to the neuromuscular network, which may alleviate localized reflex loss.

Measurements of muscle latency and automatic response magnitude are complex. Many manual and computer-based techniques have been developed in an effort to filter and threshold the EMG response to differentiate between baseline activity, involuntary onset, and volitional onset (Hodges and Bui, 1996). Difficulties with EMG analyses are due to several factors influencing EMG interpretation including background EMG, low-pass filtration, ensemble averaging of multiple trials, threshold criteria for muscle onset, and the sample width utilized.

Investigators typically assess the earliest detectable rise in the EMG output over baseline (steady state) conditions when manually selecting muscle onset. Manual selection may be susceptible to intra-observer sources of errors, however, Hodges and Bui (1996) have shown that onset is highly repeatable when performed by experienced examiners. More than 27 computer-based algorithms have been described for determining muscle onset (Hodges and Bui, 1996, Brinkworth and Turker, 2003, Moffroid et al., 1992), many of which disagree with each other, or do not match those selected manually. In a comparative study of EMG analysis, Hodges and Buie (1996) determined that the computer-based method most closely matched that which was selected manually when the EMG output was processed using a 50 MHz low pass filter, a threshold level equal to 3 times the standard deviation above baseline, and a 25ms sample width window above that threshold. We utilized these parameters to enable computer selection of muscle onset for the TA, MH and BF after the trials were ensemble averaged, though we used a 10 ms window for the VM and VL.

The methods used to quantify the muscle response magnitude have not been standardized. Investigators typically integrate the EMG signal between defined time points (Di Fabio et al., 1992, Henry et al., 1998a, Henry et al., 1998b). Henry et al. (1998a, 1998b) utilized the epoch between 100 and 200ms after perturbation to compare reflex magnitudes between different loading conditions. Di Fabio et al. (1992) integrated the EMG signal both over a 100 and 200ms time windows from the muscle onset. We followed a similar approach used by Di Fabio et al. (1992) but we tuned the method to each muscle group by measuring the area under the EMG versus time curve from involuntary muscle onset to the onset of volitional contraction for each muscle group. The differences found in the volitional response onsets between muscles suggests that if the same window of analysis was used for all muscles, some of the APR would be missed in some muscles while some of the volitional response would be included in others.

Several study limitations must be considered. First, we assumed the anterior perturbation at the foot would strain the ACL. A perturbation that shifts the ground anterior relative to the foot would cause the subject's center of gravity to shift posterior relative to the feet. In turn, an

anterior shear force would be applied to the tibia relative to the femur. It is well known that an anterior shear force causes the tibia to move anterior relative to the femur (Butler et al., 1980), and we have shown that an anterior shear force applied to the tibia will strain the knee in the weightbearing and non-weight bearing knee (Fleming et al., 2001a). Therefore, we are confident that the perturbation used in this study will elicit a response in the ACL, which may influence the knee musculature.

The perturbations were only applied anteriorly. Although several steps were taken to ensure that the subjects could not predict the timing of perturbation, they always knew the direction. Nonetheless, muscle onset latencies were consistent with literature values (Di Fabio et al., 1992, Shultz et al., 2001, Shultz et al., 2000). The reproducibility data also verified that there was no learning effect.

A secondary perturbation occurs when the plate comes to a stop approximately 140ms after the initial perturbation. Given an involuntary onset time of approximately 80ms, the APR to the secondary perturbation would occur outside the involuntary response window relative to the first perturbation.

Perturbations were applied to the subject standing on two legs with the knee flexed to 15°, while the injection was performed in one knee. It is possible that sensory information from the unaffected leg could compensate for sensory loss of the affected knee. In a subset of patients, we tried to measure the response during single leg standing with the knee flexed at 15°. However, the baseline muscle activity was very high, making it difficult to derive accurate onset latency.

Pain has also been shown to affect the APR (Moseley and Hodges, 2005). We did not formally document subjects' perception of pain during testing. However, no subjects complained of pain. Although it is possible for pain to affect APR, the lack of differences between the sham and anesthetized subjects should dampen this concern.

We assumed the intra-articular injections were consistent between subjects. Imaging was not done to ensure that the needle tip was in the intra-articular space, so it is possible that some of the fluid was not in the intra-articular space. However, all injections were performed by an orthopaedic surgeon following a routine protocol to ensure that all the fluid reached the knee's intra-articular space. The surgeon monitored the resistance of flow out of the needle since a high resistance would indicate that the fluid was not reaching the desired location.

Finally, a power analysis was performed a priori to determine the sample size for this experiment. The study was 80% powered to detect a difference in latency of 25 ms. The mean differences in muscle onsets for the control, sham and anesthetized knees were less than 3.5ms. The mean differences in muscle response magnitudes for the treatment, sham, and control groups were less than 0.77% MVC. These differences are small and unlikely relevant.

Conclusions

Neither the 30cc intra-articular injection of bupivicaine, nor the effusion created by a similar injection of saline in the knee affected the APR to an anterior surface perturbation at the feet. The intra-articular receptors were either unaffected by local anesthesia, or the extra-articular receptors and receptors of other joints were able to compensate for them once the local anesthetic was injected.

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References

Barrack RL, Skinner HB, Brunet ME. Functional performance of the knee after intra-articular anesthesia. Am J Sports Med 1983;11:258–261. [PubMed: 6614298]

Beynnon BD, Fleming BC. Anterior cruciate ligament strain in-vivo: A review of previous work. J Biomech 1998;31:519–525. [PubMed: 9755036]

Beynnon BD, Johnson RJ, Fleming BC, Stankewich CJ, Renstrom PA, Nichols CE. The strain behavior of the anterior cruciate ligament during squatting and active flexion-extension: A comparison of an open- and a closed-kinetic-chain exercise. Am J Sports Med 1997;25:823–829. [PubMed: 9397272]

Brinkworth RS, Turker KS. A method for quantifying reflex responses from intra-muscular and surface electromyogram. J Neurosci Methods 2003;122:179–193. [PubMed: 12573477]

Buchanan TS, Kim AW, Lloyd DG. Selective muscle activation following rapid varus/valgus perturbations at the knee. Med Sci Sports Exerc 1996;28:870–876. [PubMed: 8832541]

Butler DL, Noyes FR, Grood ES. Ligamentous restraints to anterior-posterior drawer in the human knee. J Bone Joint Surg Am 1980;62:259–270. [PubMed: 7358757]

Clark FJ, Horch KW, Back SM, Larson GF. Contributions of cutaneous and joint receptors to static knee position sense in man. J Neurophysiol 1979;42:877–888. [PubMed: 107288]

Cram, JR.; Kasman, GS. Introduction to surface electromyography. Gaithersburg MD: Aspen; 1998.

Dhaher YY, Tsoumanis AD, Rymer WZ. Reflex muscle contractions can be elicited by valgus positional perturbations of the human knee. J Biomech 2003;36:199–209. [PubMed: 12547357]

Di Fabio RP, Graf B, Badke MB, Breunig A, Jensen K. Effect of knee joint laxity on long-loop postural reflexes: evidence for a human capsular-hamstring reflex. Exp Brain Res 1992;90:189–200. [PubMed: 1521607]

Fleming BC, Renstrom PA, Beynnon BD, Engstrom B, Peura GD, Badger GR, Johnson RJ. The effect of weightbearing and external loading on anterior cruciate ligament strain. J Biomech 2001a;34:163–170. [PubMed: 11165279]

Fleming BC, Renstrom PA, Ohlen G, Johnson RJ, Peura GD, Beynnon BD, Badger GJ. The gastrocnemius muscle is an antagonist of the anterior cruciate ligament. J Orthop Res 2001b;19:1178–1184. [PubMed: 11781021]

Friemert B, Bumann-Melnyk M, Faist M, Schwarz W, Gerngross H, Claes L. Differentiation of hamstring short latency versus medium latency responses after tibia translation. Exp Brain Res 2005;160:1–9. [PubMed: 15322783]

Fujita I, Nishikawa T, Kambic HE, Andrish JT, Grabiner MD. Characterization of hamstring reflexes during anterior cruciate ligament disruption: in vivo results from a goat model. J Orthop Res 2000;18:183–189. [PubMed: 10815817]

Henry SM, Fung J, Horak F. EMG response to maintain stance during multidirectional surface translations. J Neurophysiol 1998a;80:1939–1950. [PubMed: 9772251]

Henry SM, Fung J, Horak FB. Control of stance during lateral and anterior/posterior surface translations. IEEE Trans Rehabil Eng 1998b;6:32–42. [PubMed: 9535521]

Hodges PW, Bui BH. A comparison of computer-based methods for the determination of onset of muscle contraction using electromyography. Electroencephalography Clin Neurophysiol 1996;101:511–519.

Hopkins JT, Ingersoll CD, Krause BA, Edwards JE, Cordova ML. Effect of knee joint effusion on quadriceps and soleus motoneuron pool excitability. Med Sci Sports Exerc 2001;33:123–126. [PubMed: 11194097]

Hopkins JT, Ingersoll CD, Krause BA, Edwards JE, Cordova ML. Effect of knee joint effusion on quadriceps and soleus motorneuron excitability Med. Sci Sports Exerc 2002;33:123–126.

Horak FB, Nashner LM. Central programming of postural movements: adaptation to altered support-surface configurations. J Neurophysiol 1986;55:1369–1381. [PubMed: 3734861]

Khabie V, Schwartz MC, Rokito AS, Gallagher MA, Cuomo F, Zuckerman JD. The effect of intraarticular anesthesia and elastic bandage on elbow proprioception. J Shoulder Elbow Surg 1998;7:501–504. [PubMed: 9814930]

Kim AW, Rosen AM, Brander VA, Buchanan TS. Selective muscle activation following electrical stimulation of the collateral ligaments of the human knee. Arch Phys Med Rehabil 1995;76:750–757. [PubMed: 7632131]

Mcnair PJ, Marshall RN, Maguire K. Swelling of the knee joint: Effects of exercise on quadriceps muscle strength. Arch Phys Med Rehabil 1996;77:896–899. [PubMed: 8822681]

Moffroid M, Tranowski J, Ricamato A, Henry S. Computer solutions to identify EMG latency of automatic postural reactions. IEEE Eng Med Biol Mag 1992;12:48–51.

Moseley GL, Hodges PW. Are the changes in postural control associated with low back pain caused by pain interference? Clin J Pain 2005;21:323–329. [PubMed: 15951650]

Palmieri RM, Ingersoll CD, Cordova ML, Kinzey SJ, Stone MB, Krause BA. The effect of a simulated knee joint effusion on postural control in healthy subjects. Arch Phys Med Rehabil 2003;84:1076–1079. [PubMed: 12881838]

Palmieri RM, Tom JA, Edwards JE, Weltman A, Saliba EN, Mistry DJ, Ingersoll CD. Arthrogenic muscle response induced by an experimental knee joint effusion is mediated by pre- and post-synaptic spinal mechanisms. J Electromyogr Kinesiol 2004;14:631–640. [PubMed: 15491837]

Schultz RA, Miller DC, Kerr CS, Micheli L. Mechanoreceptors in human cruciate ligaments. A histological study. J Bone Joint Surg Am 1984;66:1072–1076. [PubMed: 6207177]

Shultz SJ, Perrin DH, Adams JM, Arnold BL, Gansneder BM, Granata KP. Assessment of neuromuscular response characteristics at the knee following a functional perturbation. J Electromyogr Kinesiol 2000;10:159–170. [PubMed: 10818337]

Shultz SJ, Perrin DH, Adams MJ, Arnold BL, Gansneder BM, Granata KP. Neuromuscular response characteristics in men and women after knee perturbation in a single leg, weightbearing stance. J Athletic Train 2001;36:37–43.

Soderberg GL, Knutson LM. A guide for use and interpretation of kinesiologic electromyographic data. J Phys Ther 2000;80:485–498.

Tsuda E, Okamura Y, Otsuka H, Komatsu T, Tokuya S. Direct evidence of the anterior cruciate ligament-hamstring reflex arc in humans. Am J Sports Med 2001;29:83–87. [PubMed: 11206261]

Williams GN, Barrance PJ, Snyder-Mackler L, Axe MJ, Buchanan TS. Specificity of muscle action after anterior cruciate ligament injury. J Orthop Res 2003;21:1131–1137. [PubMed: 14554229]

Wojtys EM, Huston LJ. Neuromuscular performance in normal and anterior cruciate ligament-deficient lower extremities. Am J Sports Med 1994;22:89–104. [PubMed: 8129117]

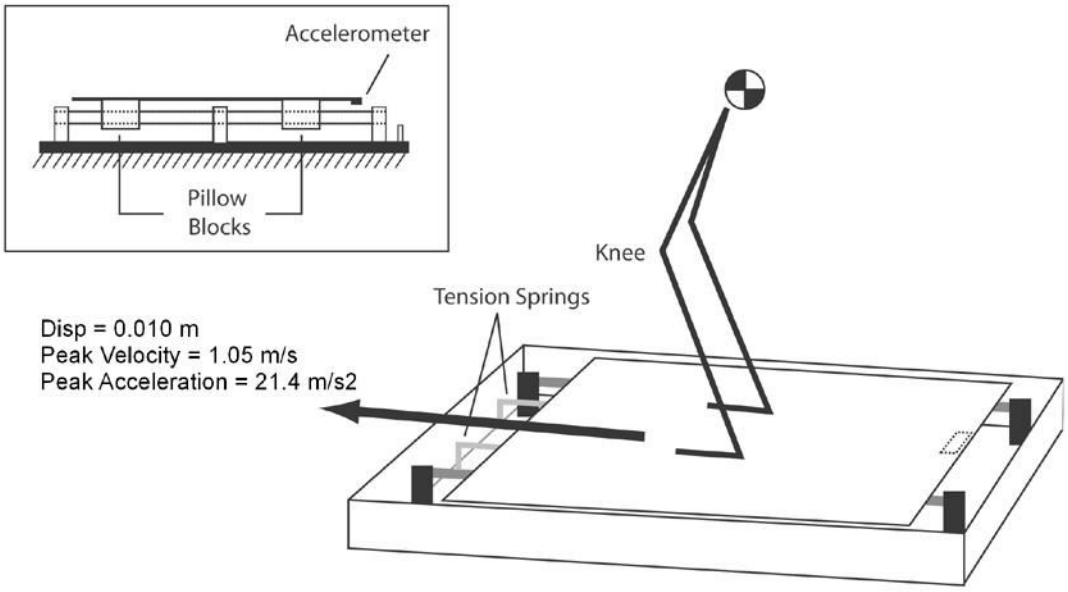

Figure 1.
A custom designed perturbation platform was developed for this study. The platform translated anterior by 10cm with a mean peak acceleration of 21.3m/s^2 and a mean velocity of 1.05 m/s.

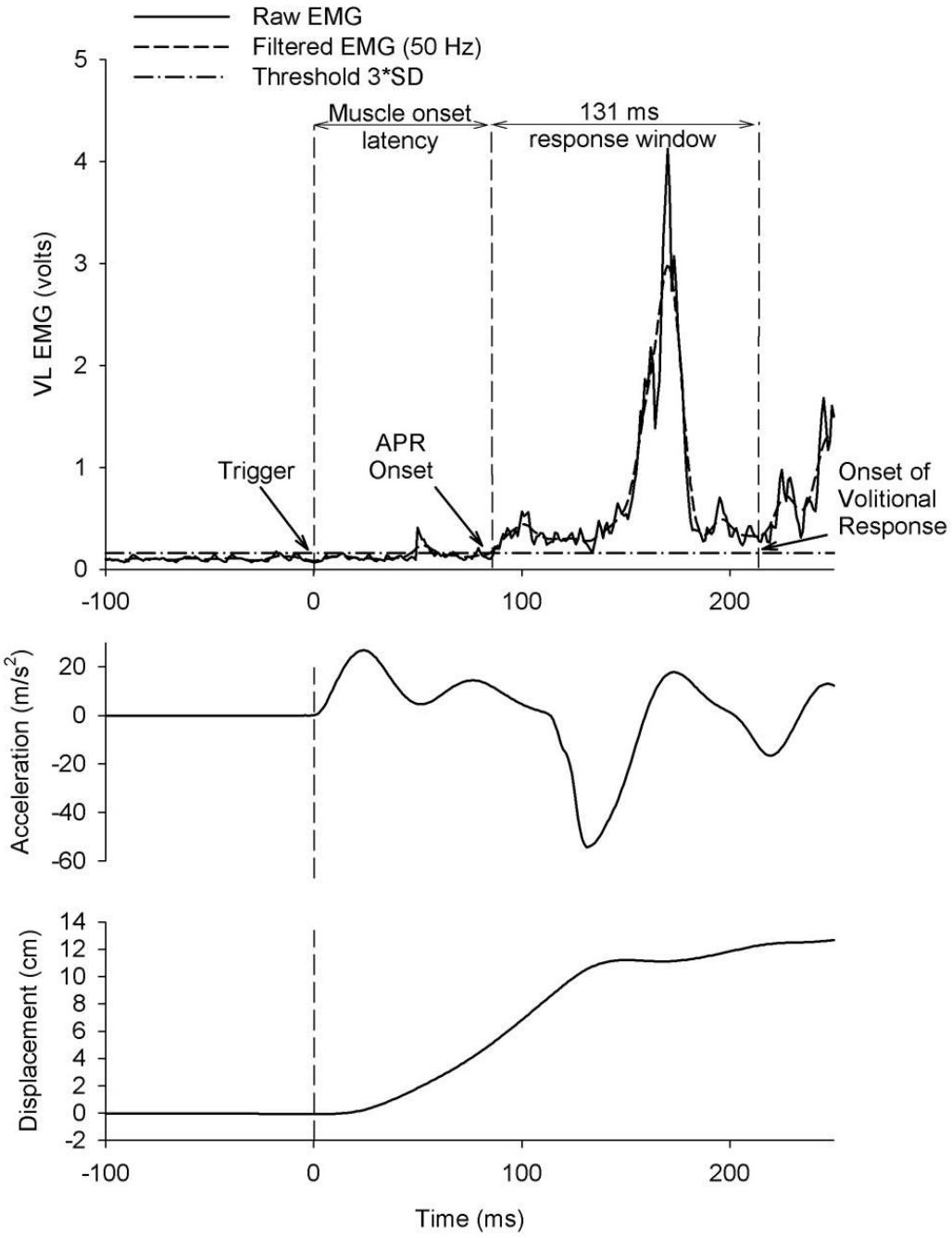

Figure 2.

Muscle onset latency was equal to the time between the trigger and the start of the automatic postural response (APR). Muscle response magnitude was equal to the area under the curve for the response (APR onset time to the onset of volitional response; "response window" minus the baseline area). The duration of the response window was dependent on the muscle group and fixed for each muscle across subjects. The data shown is from the VL of a typical patient.

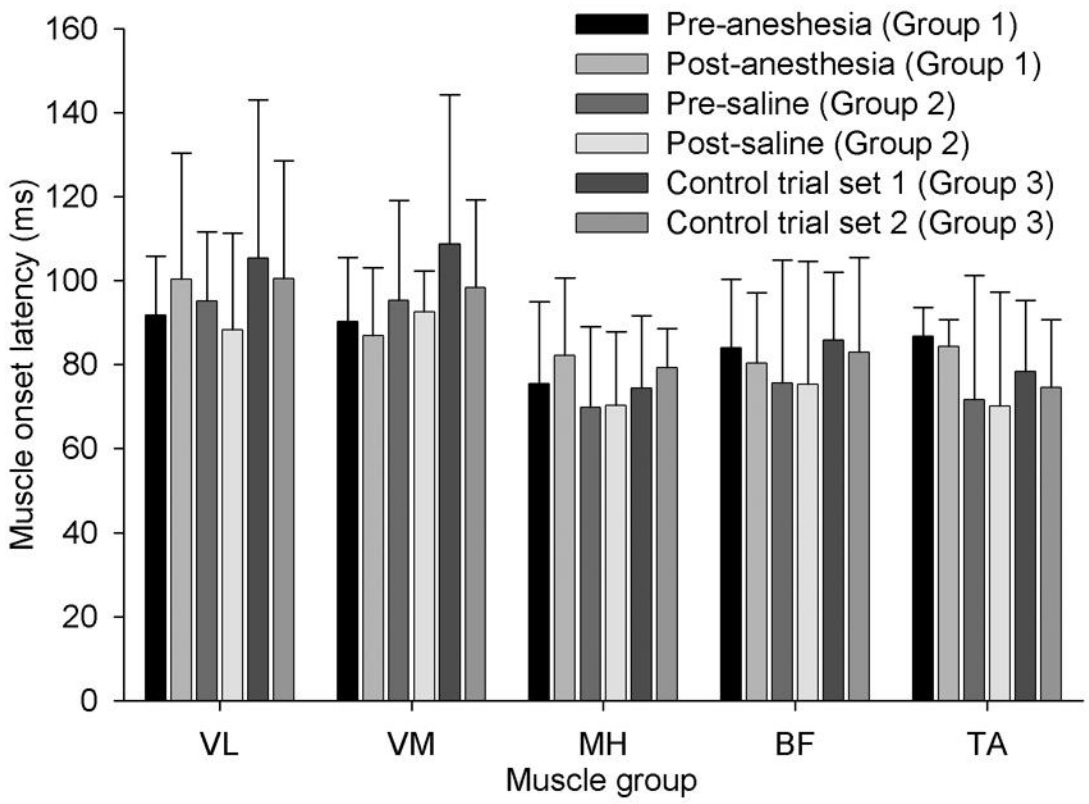

Figure 3.
Mean muscle onset latencies for the five muscle groups (VL = vastus lateralis; VM = vastus medialis; MH = medial hamstrings; BF = Biceps Femoris; and TA = tibialis anterior) for the subjects in each experimental group. Muscle onset latencies of the VL and VM were significantly greater than the MH, BF and TA. No differences were observed pre- and post-treatment within any group.

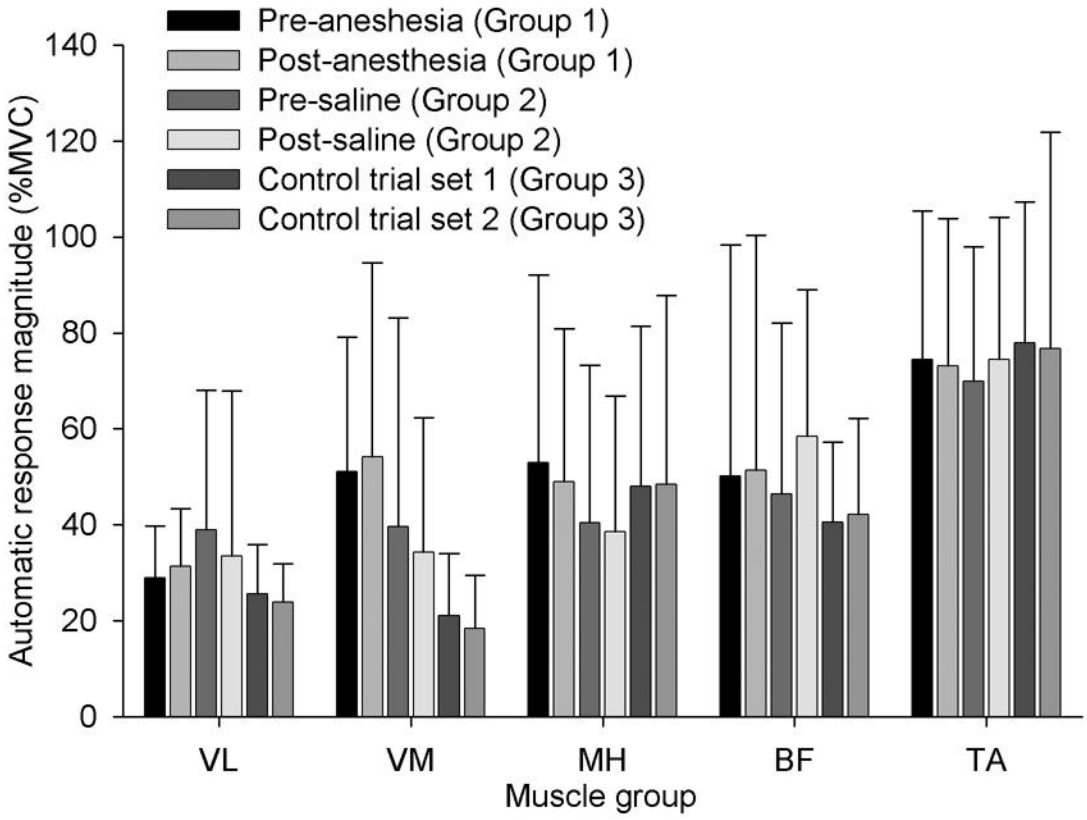

Figure 4.
Mean automatic response magnitudes for the five muscle groups for the subjects in each experimental group. Automatic response magnitudes of the VL and VM were significantly less than the MH and BF which were significantly less than the TA. No differences were observed pre- and post-treatment within any group.

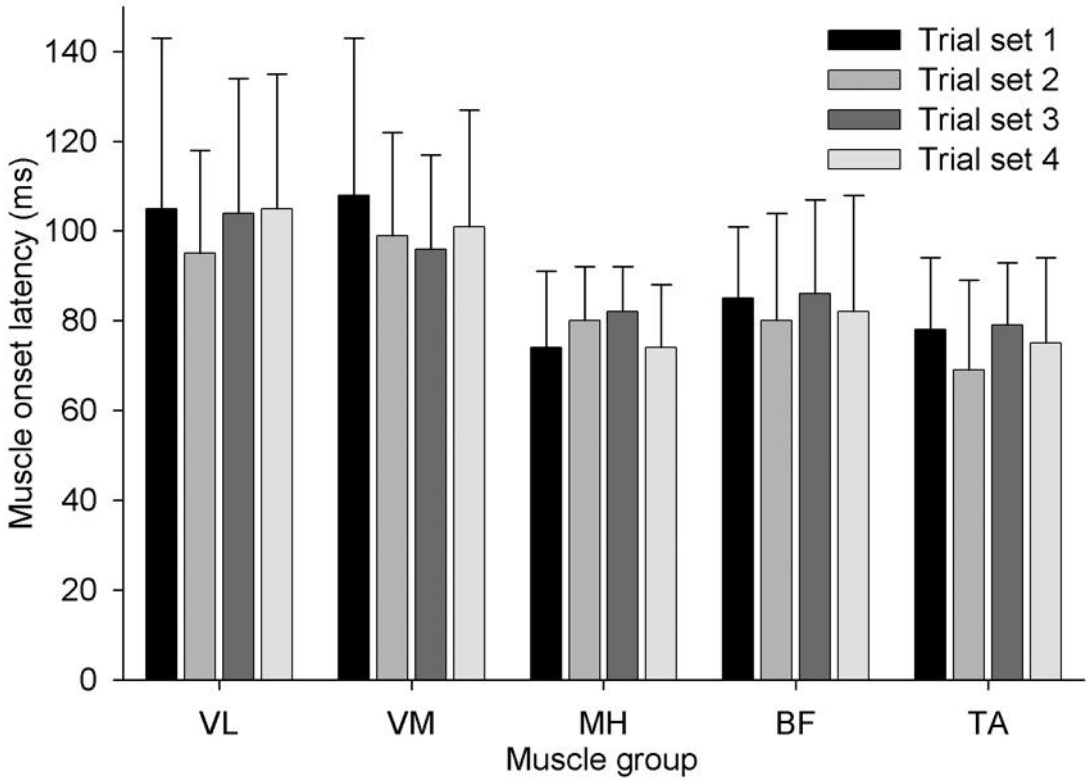

Figure 5.
Muscle onset latency reproducibility evaluation. These data were obtained from repeated testing on the control subjects.

Table 1

Subject data table. It should be noted that the EMG data for 2 of the 30 subjects were incomplete and thus were excluded from the analysis.

Treatment	N	Age	Gender	Height (cm)	Weight (Kg)
Bupivicaine	9	31 (12.3)	5F/4M	172 (6.4)	80 (16.4)
Saline	9	24 (5.0)	4F/5M	173 (9.6)	77 (12.4)
Control	10	27 (5.0)	5F/5M	170 (9.3)	74 (12.2)