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ANKLE

Local anaesthetics use does not suppress muscle activity following an ankle injection

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Abstract
Purpose To determine if peroneus longus (PL), peroneus brevis (PB), medial gastrocnemius (MG) and tibialis anterior (TA) muscle activation patterns during inversion perturbation and running tasks are suppressed following lidocaine injection to the anterior talofibular (ATF) and calcaneofibular (CF) ligament regions.
Methods Fourteen recreationally active male subjects (age, 24.8 ± 2.9 years; height, 177.0 ± 6.0 cm; mass, 77.7 ± 6.7 kg) participated. Testing was performed under five injection conditions to the ATF and CF regions: 1 ml saline, 1 ml lidocaine, 3 ml saline, 3 ml lidocaine or no injection. Following injection condition, traditional ankle taping was applied. Electromyography patterns of the PL, PB, MG and TA were collected while subjects performed continuous lateral jumps on a custom-built device which elicited an ankle inversion perturbation and treadmill running (3.35 m s^{-1}, 0.5 % incline).
Results No significant differences were demonstrated in muscle activation patterns of the PL (n.s.), PB (n.s.), MG (n.s.) or TA (n.s.) for any variable across injected conditions during both tasks. Statistical power was 0.214–0.526 for the PL, 0.087–0.638 for the PB, 0.115–0.560 for the MG and 0.118–0.410 for the TA.
Conclusions Injection of lidocaine up to 3 ml to the ATF and CF regions did not suppress muscle activity of the PL, PB, MG or TA during the inversion perturbation or running tasks. Injection up to 3 ml of 1 % lidocaine to the ATF and CF regions may be used without sacrificing the muscle activation patterns about the ankle. This finding is clinically relevant since the use of the injection does not put the patient at any higher risk of reinjury to the site.
Level of evidence I.

Keywords Ankle sprain · Injection · Anaesthetics · EMG · Perturbation

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Introduction

The use of local anaesthetic injections to return an athlete to play has been part of sports medicine practice for many years [29]. The practice of using these injections is controversial in part because it is poorly studied and, in theory, could increase the risk of subsequent injury. The injections are typically administered at the highest level of sports, limiting the experience of most physicians to perform. The majority of sports organizations do not have official policies regarding the use of local anaesthetics or provide vague usage guidelines [27, 29].

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Orchard [28] demonstrated low-risk complications and high predictability of use.

Previous studies have investigated the effects of anaesthetic injection on balance [3, 13, 19, 33], proprioception [5, 10, 13, 19] and muscle activity [19, 26]. These studies have produced conflicting results in part explained by methodological differences such as injection site and volume of injection utilized [3, 10, 13, 26, 33], use of placebo [5, 26] or use of a static task [13, 23, 26], which may not be applicable to athletic activity.

Ankle sprains have been reported to be the most common injury in sports [1, 14, 24, 36], accounting for approximately 15 % of all injuries and resulting in more time lost from athletic participation than any other injury. Assessing the muscle firing patterns of anesthetized ankle ligaments during a dynamic task as opposed to a static task may help determine if anesthetized injections would increase the risk of recurring ankle sprain. The purpose of this study was to simulate clinical use of local anaesthetics and determine if a lidocaine injection to the anterior talofibular (ATF) and calcaneofibular (CF) ligament regions would alter muscle activation patterns of the peroneus longus (PL), peroneus brevis (PB), medial head of the gastrocnemius (MG) and tibialis anterior (TA) during dynamic tasks. It was hypothesized that the muscle activation patterns about the ankle would be suppressed following lidocaine injection with greater suppression at the higher dosage.

Materials and methods

A total of 14 physically active men volunteered to participate (age, 24.8 ± 2.9 years; height, 177.0 ± 6.0 cm; mass, 77.7 ± 6.7 kg). Physically active was defined as performing recreational exercises or sports for a minimum of 30 min, 3 days per week. All subjects were free of any previous ankle injury history, compromised visual or vestibular–cochlear systems or musculoskeletal injury which would affect the ability to maintain balance, run, or jump. All subjects provided written informed consent in accordance with and as approved by the University Institutional Review Board prior to participation.

Instrumentation

A custom-designed jumping platform was constructed with a gravity-induced tilt plate mechanism, which elicited an inversion perturbation at the ankle while the subjects performed a continuous lateral/medial jump protocol. The tilt plate mechanism was controlled with a stepper-motor and custom program (LabVIEW, National Instruments, Austin, TX) to automatically drive the unlocking/locking mechanism. The tilt plate sequences were selected using a random number generator and were set in the custom program prior to each trial. Release of the locking mechanism was initiated by the subject's contact with the tilt plate, which then resulted in the perturbation. The amplitude of the inversion perturbation was held constant at 20° by a mechanical block affixed to the frame of the device. The running task was performed on a treadmill (Biodex Medical Systems, Shirley, NY).

A 3D motion capture system (Vicon Motion Systems, Englewood, CA), interfaced with eight high-speed infrared cameras sampling at 200 Hz, was utilized to track retro-reflective markers affixed to the tilt plate during the jumping task and affixed to the second metatarsal (toe) and lateral malleolus during the running task. For the jumping task, synchronized tracking of the marker trajectories was utilized to identify the onset of vertical displacement of the tilt plate markers as initial contact. For the running task, a modification of the method described by Schache et al. [34] was used to identify initial contact. Initial contact was identified by the initial downward spike of the toe marker velocity that coincided with the beginning of the valley in the vertical displacement of the malleolus marker. An FM telemetry electromyography (EMG) system (Noraxon USA, Inc., Scottsdale, AZ) sampling at 1,200 Hz was used to record activation of muscles about the ankle joint. The EMG signal was passed through a single-ended amplifier (gain, 500) to an 8-channel transmitter. The signal was wirelessly transmitted to the receiver using FM telemetry where the signal was amplified (gain, 500) and filtered (16–500 Hz band-pass filter, common-mode rejection ratio of 130 dB). The analogue signal was converted to digital using a DT3010/32 (32-channel, 24-bit) A/D board (Data Translation Inc., Marlboro, MA). Previous studies have demonstrated good test–retest reliability for kinematic [9, 17, 22, 31] and EMG measurements [2, 6, 7, 21, 25].

Procedures

A within-subjects, repeated measures crossover design was utilized for this study. The dependent variables for the inversion perturbation task were peak amplitude (initial contact, peak and time to peak within 150 ms prior to initial contact, peak and time to peak within 20–150 ms post-initial contact) and integrated muscle activity (within 150 ms prior to initial contact, within 20–150 ms post-initial contact) for the PL, PB, MG and TA. The dependent variables for the running task were mean amplitude during the following for the same muscles: stance, pre-activation (100 ms prior to initial contact), initial loading (initial contact up to 50 ms post-initial contact) and main loading phase (50–200 ms post-initial contact). The independent variables were solution (lidocaine, saline) and volume (1, 3 ml).

Subjects reported to a University sports medicine research laboratory for four 1.5–2 h sessions, a minimum

of 48 h apart. A total of five testing conditions were performed over four testing sessions. Randomized experimental test conditions were derived from injection condition sequences using a Latin square. An injection sequence was then assigned to each subject using a random number generator. During the first session, subjects were taped, and then they performed the inversion perturbation protocol followed by the running protocol with no injection (control), after which the tape was removed. Subjects then received the first of the four injection conditions as indicated by the injection sequence, were taped, and they repeated the inversion perturbation and running tasks. During each of the subsequent testing sessions, subjects received one injection condition as indicated by the injection sequence followed by taping and the inversion perturbation and running protocols. The muscle activity about the ankle joint of the dominant limb was assessed for all test conditions during both protocols. Limb dominance was determined by asking the subject which foot he would use to kick a ball maximally.

EMG preparation

Prior to each test session, the midpoint of the muscle bellies of the PL, PB, MG and TA were identified through palpation and marked by the same Certified Athletic Trainer (ATC). Any visible hair was shaved, and the skin was lightly abraded and cleaned with isopropyl alcohol at all electrode sites to minimize skin–electrode impedance. Two 20-mm oval self-adhesive, bipolar Ag/Ag–Cl surface electrodes (AMBU Blue Sensor N; AMBU, Glen Burnie, MD) were placed on the skin over the marked sites in series with the muscle line of function with an interelectrode distance of approximately 20 mm. A single ground electrode was placed on the anteromedial tibial flare. The electrodes were secured using strips of hypoallergenic tape, and the leads connecting the electrodes to the FM transmitter were secured to the subject's lower leg using underwrap to minimize motion artefact.

Electromyography activation during a five-second maximal voluntary isometric contraction (MVIC) was recorded for each muscle to ensure proper electrode placement, verify minimal crosstalk between electrodes and normalize EMG data collected during the lateral jump protocol as a percentage of MVIC. Manual resistance was provided by an ATC for all MVIC trials of the PL, PB, MG and TA, which were performed with the subject long sitting and the ankle in neutral position.

Experimental procedure

The following are specific to the four experimental test conditions. After EMG preparation, a sports medicine orthopaedic physician injected the appropriate volume (1 or 3 ml) of either 1 % preservative-free lidocaine HCl (Hospira, Inc., Lake Forest, IL) or a placebo solution (preservative-free 0.9 % sodium chloride, Hospira, Inc., Lake Forest, IL), as determined by injection sequence, with a 25-gauge 5/8-inch needle. Myers et al. [26] reported an impairment of the muscle protective response following injection of 1.5 ml into the ATF and CF ligaments; therefore, a volume of 1 ml was selected for lower volume in the current study in order to remain below the threshold where changes in muscle activation may be induced. The ATF was initially palpated, and when the ATF was not discernable as a thickening, the needle was placed approximately 1 cm anteriorly from the tip of the fibula. The CF was palpated, and the injection was performed distal to the fibular tip. In both injections, the ligament was approached from the side rather than directly injecting into the ligament (Fig. 1). Subjects were blind to both the injected solution and dosage.

Prophylactic ankle taping

In order to simulate the standard of care for ankle sprains, the subject's ankle was taped under all testing conditions. A traditional closed basket weave ankle taping technique

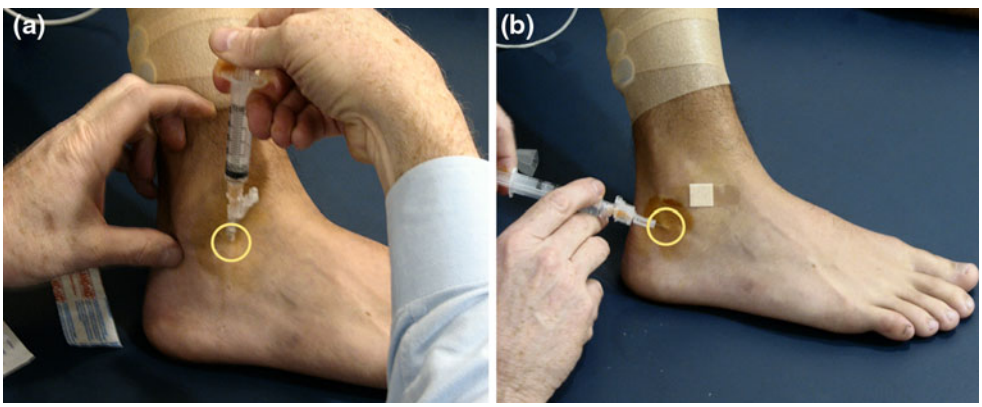

Fig. 1 Injection into the anterior talofibular (**a**) and calcaneofibular (**b**) ligaments

was applied using 3.81-cm adhesive athletic tape (Johnson and Johnson, New Brunswick, NJ) prior to the jumping protocol. A layer of underwrap was used to minimize any irritation related to the tape or tape adhesive. The basket weave taping technique utilized a series of three alternating stirrups and horseshoes, two figure-eight straps and two consecutive medial and lateral heel locks. The procedure was completed by applying circumferential strips to close and anchor the technique, with the proximal anchor ending on the skin. For any given subject, the taping procedures were performed by one ATC. In all, three ATCs were used as a result of personnel changes.

Inversion perturbation procedure

The subjects performed a series of lateral/medial jumps starting from a position such that both lower limbs were on the solid surface of the perturbation device and the tilt plate was to the side of the dominant limb (Fig. 2). The subjects were instructed to jump laterally towards the tilt plate and land with the dominant test limb on the marked location on the tilt plate with the contralateral limb landing in the original position of the test limb. Without stopping, the subjects were to transition back to the starting position and then repeat. Subjects completed a total of 15 continuous lateral/medial jump repetitions with successful foot placement, during which the tilt plate mechanism was randomly released four times to facilitate an inversion perturbation of the test ankle. The cadence of jumping was standardized using a computer display placed approximately 5 m in front of and at eye level of the subject, flashing a 'JUMP' command set at 92 beats/min. Subjects were instructed to maintain visual attention on the computer display, which helped to eliminate visual cue of the tilt plate as well as to help produce continuous lateral/medial jumping throughout each test. To eliminate auditory cues, subjects wore foam ear plugs and headphones playing white noise (static). Subjects were permitted adequate practice trials until they were able to jump laterally on/off the tilt plate at the standardized cadence without visually targeting the location or action of the tilt plate. The tilt plate was locked in an unperturbed position throughout the practice trials.

Running procedure

The subjects performed continuous running on a treadmill at a speed on 3.35 m s^{-1} and with an incline of 0.5 %. Subjects ran for a total of 5 min, with collection of EMG occurring during the final 30 s of the run.

Data reduction

Three-dimensional coordinate data from the tilt plate markers and toe and heel markers during the treadmill running and raw EMG data (MVIC and trial data) were imported and processed in Matlab (The MathWorks, Natick, MA). Raw EMG data were offset to ensure the median of the signal was equal to zero and full-wave rectified. A linear envelope was performed using a fourth-order low-pass Butterworth filter (zero phase shift) with a cut-off frequency of 12 Hz. The filtered EMG data were normalized by the mean of the five-second MVIC to allow between-condition comparisons. For the inversion perturbation protocol, peak EMG amplitude (%MVIC) of each muscle was measured at initial contact, within 150 ms prior to initial contact and 20–150 ms post-initial contact. Time to peak amplitude (ms) was calculated within 150 ms prior to initial contact and 20–150 ms after initial contact. Integrated EMG (%MVIC) was calculated within 150 ms prior to initial contact and 20–150 ms after initial contact. For the running protocol, mean EMG amplitude (%MVIC) was calculated for stance phase as well as 100 ms up to initial contact (pre-activation), initial contact up to 50 ms after initial contact (initial loading phase) and 50–200 ms post-initial contact (main loading phase).

Statistical analysis

Differences between conditions for the dependent variables were assessed with SPSS 17.0 (SPSS, Inc., Chicago, IL).

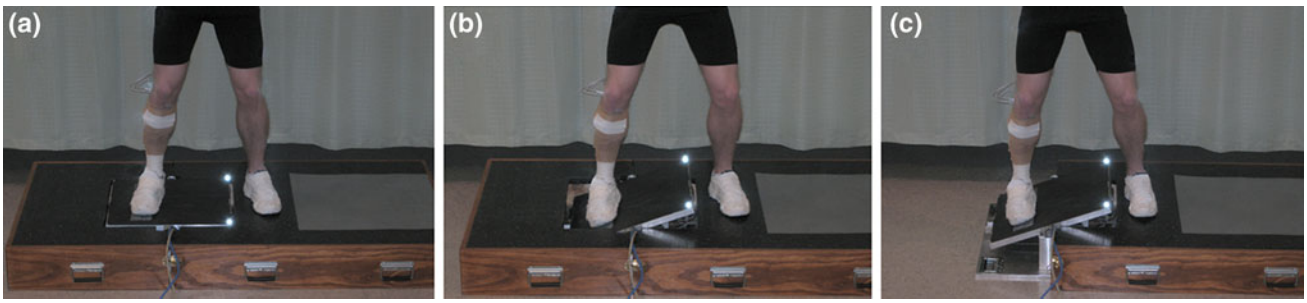

Fig. 2 A subject positioned on the ankle inversion perturbation device non-perturbated (**a**), perturbated (**b**) and perturbated showing tilt plate mechanism (**c**)

Separate one-way repeated measures ANOVA tests were used to analyse the dependent variables between conditions for the PL, PB, MG and TA muscles ($p < 0.05$).

Results

Descriptive data from the inversion perturbation task and running task for the PL are presented in Table 1. No significant differences in EMG activity of the PL were demonstrated for peak amplitude at initial contact (n.s.), peak amplitude (n.s.) and time to peak amplitude (n.s.) within 150 ms prior to initial contact, peak amplitude (n.s.) and time to peak amplitude (n.s.) within 20–150 ms post-initial contact (n.s.), integrated activity within 150 ms prior to initial contact (n.s.) and integrated activity within 20–150 ms post-initial contact (n.s.). Statistical power for the PL was 0.214–0.526. No significant differences in EMG activity of the PL were demonstrated for mean amplitude during stance (n.s.), pre-activation (n.s.), initial loading (n.s.) or main loading (n.s.) phases. Statistical power for the PL was 0.226–0.351.

Descriptive data from the inversion perturbation task and running task for the PB are presented in Table 2. No significant differences in EMG activity of the PB were demonstrated for peak amplitude at initial contact (n.s.), peak amplitude (n.s.) and time to peak amplitude (n.s.) within 150 ms prior to initial contact, peak amplitude (n.s.) and time to peak amplitude (n.s.) within 20–150 ms post-initial contact, integrated activity within 150 ms prior to initial contact (n.s.) and integrated activity within 20–150 ms post-initial contact (n.s.). Statistical power for the PB was 0.087–0.638. No significant differences in EMG activity of the PB were demonstrated for mean amplitude during stance (n.s.), pre-activation (n.s.), initial loading (n.s.) or main loading (n.s.) phases. Statistical power for the PB was 0.346–0.450.

Descriptive data from the inversion perturbation task and running task for the MG are presented in Table 3. No significant differences in EMG activity of the MG were demonstrated for peak amplitude at initial contact (n.s.), peak amplitude (n.s.) and time to peak amplitude (n.s.) within 150 ms prior to initial contact, peak amplitude (n.s.) and time to peak amplitude (n.s.) within 20–150 ms

Table 1 Muscle activation of the peroneus longus (Mean ± SD)

	Solution and volume				
	Control	1 ml saline	1 ml lidocaine	3 ml saline	3 ml lidocaine
Jumping protocol					
PkAmpIC[a]	82.3 ± 69.2	123.9 ± 111.6	112.9 ± 92.3	103.2 ± 62.1	102.5 ± 52.0
PkAmp150-IC[b]	90.1 ± 79.4	135.7 ± 111.9	120.3 ± 92.3	105.7 ± 61.5	115.6 ± 46.6
TimePkAmp150-IC[c]	127.5 ± 29.7	134.7 ± 13.4	125.4 ± 24.3	139.2 ± 11.0	122.9 ± 25.4
PkAmp150-PostIC[d]	173.5 ± 120.5	314.8 ± 359.3	322.3 ± 311.6	210.4 ± 129.7	214.5 ± 112.4
TimePkAmp20-150-PostIC[e]	90.8 ± 19.7	98.0 ± 20.1	84.8 ± 31.7	77 ± 27.3	81.1 ± 26.8
IPre150-IC[f]	12,912.3 ± 12,827.5	17,094.2 ± 9,067.2	15,771.5 ± 11,609.6	13,221.1 ± 6,901.6	16,158.6 ± 7,461.6
IPost20-150-IC[g]	29,230.7 ± 22,455.8	55,120.7 ± 67,171.6	50,283.8 ± 41,969.2	37,752.0 ± 23,596.5	35,065.7 ± 21,657.4
Running protocol					
Stance[h]	92.2 ± 66.5	128.8 ± 65.7	132.3 ± 148.0	104.6 ± 59.8	100.6 ± 52.4
Pre-activation[i]	45.3 ± 33.5	54.6 ± 25.0	48.1 ± 28.0	44.8 ± 20.0	43.7 ± 17.1
Initial loading[j]	101.0 ± 66.0	133.1 ± 73.6	118.4 ± 72.8	123.4 ± 68.5	111.1 ± 56.6
Main loading[k]	74.7 ± 57.2	107.7 ± 69.2	124.0 ± 178.8	80.0 ± 46.8	77.6 ± 39.6

[a] PkAmpIC: Peak amplitude at initial contact

[b] PkAmp150-IC: Peak amplitude within 150 ms to initial contact (%MVIC)

[c] TimePkAmp150-IC: Time to peak amplitude within 150 ms prior to initial contact

[d] PkAmp150-PostIC: Peak amplitude post-initial contact (%MVIC)

[e] TimePkAmp20-150-PostIC: Time to peak amplitude within 20–150 ms post-initial contact

[f] IPre150-IC: Integrated activity within 150 ms prior to initial contact

[g] IPost20-150-IC: Integrated activity within 20–150 ms post-initial contact

[h] Stance: Mean amplitude during stance phase (%MVIC)

[i] Pre-activation: Mean amplitude 100 ms to initial contact (%MVIC)

[j] Initial loading: Mean amplitude initial contact to 50 ms post-initial contact (%MVIC)

[k] Main loading: Mean amplitude 50–200 ms post-initial contact (%MVIC)

Table 2 Muscle activation of the peroneus brevis (Mean ± SD)

	Solution and volume				
	Control	1 ml saline	1 ml lidocaine	3 ml saline	3 ml lidocaine
Jumping protocol					
PkAmpIC[a]	90.2 ± 38.3	103.9 ± 60.8	161.7 ± 168.6	88.3 ± 39.5	98.3 ± 25.2
PkAmp150-IC[b]	101.1 ± 35.2	113.8 ± 60.4	184.6 ± 220.9	94.8 ± 41.8	112.9 ± 32.7
TimePkAmp150-IC[c]	130.2 ± 21.4	132.7 ± 15.8	135.1 ± 12.5	134.3 ± 13.4	133.8 ± 16.4
PkAmp150-PostIC[d]	198.3 ± 42.4	218.1 ± 96.3	312.1 ± 312.5	180.8 ± 91.6	191.4 ± 76.3
TimePkAmp20-150-PostIC[e]	81.6 ± 34.1	84.6 ± 32.3	94.8 ± 32.8	89.8 ± 19.2	81.1 ± 20.8
IPre150-IC[f]	14,867.3 ± 6,952.9	18,509.5 ± 10,573.9	25,975.2 ± 25,252.6	13,957.8 ± 6,375.5	16,488.1 ± 6,126.2
IPost20-150-IC[g]	30,383.4 ± 9,073.5	37,085.9 ± 17,519.6	45,717.4 ± 31,300.3	30,504.4 ± 15,784.9	29,631.3 ± 11,853.6
Running protocol					
Stance[h]	103.1 ± 49.1	92.2 ± 39.2	91.8 ± 49.1	149.1 ± 163.3	88.9 ± 42.1
Pre-activation[i]	50.1 ± 40.0	38.5 ± 21.3	36.3 ± 19.4	61.6 ± 62.0	37.2 ± 15.3
Initial loading[j]	112.7 ± 88.3	82.9 ± 44.4	87.7 ± 50.9	152.8 ± 168.7	86.1 ± 46.8
Main loading[k]	87.1 ± 39.5	84.2 ± 36.3	83.5 ± 56.0	129.8 ± 149.3	77.4 ± 40.3

[a] PkAmpIC: Peak amplitude at initial contact

[b] PkAmp150-IC: Peak amplitude within 150 ms to initial contact (%MVIC)

[c] TimePkAmp150-IC: Time to peak amplitude within 150 ms prior to initial contact

[d] PkAmp150-PostIC: Peak amplitude post-initial contact (%MVIC)

[e] TimePkAmp20-150-PostIC: Time to peak amplitude within 20–150 ms post-initial contact

[f] IPre150-IC: Integrated activity within 150 ms prior to initial contact

[g] IPost20-150-IC: Integrated activity within 20–150 ms post-initial contact

[h] Stance: Mean amplitude during stance phase (%MVIC)

[i] Pre-activation: Mean amplitude 100 ms to initial contact (%MVIC)

[j] Initial loading: Mean amplitude initial contact to 50 ms post-initial contact (%MVIC)

[k] Main loading: Mean amplitude 50–200 ms post-initial contact (%MVIC)

post-initial contact, integrated activity within 150 ms prior to initial contact (n.s.) and integrated activity within 20–150 ms post-initial contact (n.s.). Statistical power for the MG was 0.115–0.560. No significant differences in EMG activity of the MG were demonstrated for mean amplitude during stance (n.s.), pre-activation (n.s.), initial loading (n.s.) or main loading (n.s.) phases. Statistical power for the MG was 0.186–0.260.

Descriptive data from the inversion perturbation task and running task for the TA are presented in Table 4. No significant differences in EMG activity of the TA were demonstrated for peak amplitude at initial contact (n.s.), peak amplitude (n.s.) and time to peak amplitude (n.s.) within 15 ms prior to initial contact, peak amplitude (n.s.) and time to peak amplitude (n.s.) within 20–150 ms post-initial contact, integrated activity within 150 ms prior to initial contact (n.s.) and integrated activity within 20–15 ms post-initial contact (n.s.). Statistical power for the TA was 0.134–0.274. No significant differences in EMG activity of the TA were demonstrated for mean amplitude during stance (n.s.), pre-activation (n.s.), initial loading (n.s.) or main loading (n.s.) phases. Statistical power for the TA was 0.118–0.410.

Discussion

The finding of most importance in this study was that muscle activation patterns did not change when a local anaesthetic was injected. The purpose of the study was to determine if the muscle activation patterns of the PL, PB, MG and TA were compromised following injection of a local anaesthetic to the ATF and CF ligament regions in healthy subjects. No significant differences were identified in any of the variables, regardless of the solution or the volume, as compared to the non-injected condition during the inversion perturbation or running tasks. These findings indicate that muscle activation patterns about the ankle joint may not be compromised by local anaesthetic injections of up to 3 ml to the ATF and CF ligament regions. These findings refute our original hypothesis that muscle activity would be suppressed following injection.

Previous studies have examined the effect of injection of a local anaesthetic at the ankle joint on balance, proprioception and/or muscle activity. Most studies that investigated the effect of anaesthetic injection at the ankle on balance reported no differences in balance between injected and non-injected conditions, regardless of stance

Table 3 Muscle activation of the medial gastrocnemius (Mean ± SD)

	Solution and volume				
	Control	1 ml saline	1 ml lidocaine	3 ml saline	3 ml lidocaine
Jumping protocol					
PkAmpIC[a]	103.9 ± 51.7	110.5 ± 73.8	105.4 ± 57.5	93.5 ± 50.1	96.4 ± 46.0
PkAmp150-IC[b]	152.7 ± 70.2	144.3 ± 66.7	138.8 ± 51.3	137.4 ± 43.7	126.8 ± 48.7
TimePkAmp150-IC[c]	117.4 ± 12.6	119.8 ± 17.4	122.5 ± 16.0	109.6 ± 28.0	115.3 ± 21.2
PkAmp150-PostIC[d]	144.1 ± 63.4	218.8 ± 219.3	194.1 ± 74.1	259.3 ± 276.8	172.1 ± 72.9
TimePkAmp20-150-PostIC[e]	59.7 ± 18.7	75.8 ± 35.4	85.5 ± 33.4	85.6 ± 19.2	69.8 ± 27.2
IPre150-IC[f]	24,760.2 ± 13,414.5	24,120.4 ± 9,924.4	22,316.2 ± 8,332.4	23,736.4 ± 10,495.1	20,819.8 ± 9,180.5
IPost20-150-IC[g]	22,336.2 ± 11,793.2	35,135.2 ± 37,272.3	28,836.1 ± 11,597.5	36,503.1 ± 30,751.0	24,671.1 ± 13,443
Running protocol					
Stance[h]	136.6 ± 58.5	118.5 ± 29.0	134.0 ± 53.5	141.4 ± 43.4	128.8 ± 35.0
Pre-activation[i]	54.2 ± 19.0	48.9 ± 23.1	52.3 ± 21.7	58.8 ± 22.1	51.3 ± 19.7
Initial loading[j]	138.8 ± 44.7	123.2 ± 46.4	138.1 ± 39.4	145.1 ± 35.8	128.8 ± 30.2
Main loading[k]	109.8 ± 51.5	97.4 ± 22.7	110.5 ± 50.3	118.0 ± 52.6	106.4 ± 32.3

[a] PkAmpIC: Peak amplitude at initial contact

[b] PkAmp150-IC: Peak amplitude within 150 ms to initial contact (%MVIC)

[c] TimePkAmp150-IC: Time to peak amplitude within 150 ms prior to initial contact

[d] PkAmp150-PostIC: Peak amplitude post-initial contact (%MVIC)

[e] TimePkAmp20-150-PostIC: Time to peak amplitude within 20–150 ms post-initial contact

[f] IPre150-IC: Integrated activity within 150 ms prior to initial contact

[g] IPost20-150-IC: Integrated activity within 20–150 ms post-initial contact

[h] Stance: Mean amplitude during stance phase (%MVIC)

[i] Pre-activation: Mean amplitude 100 ms to initial contact (%MVIC)

[j] Initial loading: Mean amplitude initial contact to 50 ms post-initial contact (%MVIC)

[k] Main loading: Mean amplitude 50–200 ms post-initial contact (%MVIC)

position (single or bilateral) or visual input (eyes open vs. eyes closed) [3, 19, 33]. Hertel et al. [13] reported alterations in the centre of pressure following injection, with a more lateral displacement during static single leg stance and a more medial displacement during dynamic single leg stance in which the surface was moved into plantarflexion/dorsiflexion or inversion/eversion. However, no differences were seen in postural sway between injected and non-injected conditions. It was suggested that the centre of pressure may have been altered as a compensatory mechanism for the lost sensory input resulting from anaesthesia.

Although not part of the current study, conflicting results have also been reported in the effect of anaesthetic ankle injection on ankle proprioception and balance. Hertel et al. [13] reported no difference in passive joint position sense between injected and non-injected conditions, whereas Konradsen et al. [19] reported a significant decrease in passive joint position sense following injection. Hertel et al. [13] injected the ATF/joint capsule with 8 cc of lidocaine, passively inverted the ankle at 3°/s while Konradsen et al. [19] intravenously injected 20 ml of carbocain, passively inverted the ankle at 2°/s. Several other studies have demonstrated a lack of active joint position sense following ankle ligament injection in both weight-bearing and non-weight-bearing conditions [5, 10, 19, 23].

Previous research studies that evaluated muscle activity via EMG following anaesthetic ankle injection have looked at PL alone [19] or at the TA, PL and PB muscles [26]. Konradsen et al. [19] reported no differences in peroneal reflex reaction time. Similarly, Myers et al. [26] reported no differences in muscle latency, maximum amplitude or time to maximum amplitude in any of the muscles. However, suppression of the protective muscle response was reported, as indicated by a decrease in the mean muscle activity in the 100 ms postmuscle activation onset. Utilizing a running task, Myers et al. [26] also demonstrated a significant decrease in the mean amplitude of the TA during swing phase and the PL and PB muscles during the stance phase of running. These authors compared a 1.5-cc injection of lidocaine to a similar injection of saline and postulated a pressure effect as there was no difference between a saline injection and a lidocaine injection in their study.

The choice of anaesthetic agent should also be considered for its potential impact on the results. Orchard [28]

Table 4 Muscle activation of the tibialis anterior (Mean ± SD)

	Solution and volume				
	Control	1 ml saline	1 ml lidocaine	3 ml saline	3 ml lidocaine
Jumping protocol					
[a]PkAmpIC	57.5 ± 31.8	69.8 ± 67.3	55.4 ± 36.0	61.3 ± 41.3	68.6 ± 50.6
PkAmp150-IC[b]	65.6 ± 34.4	75.1 ± 67.7	60.6 ± 38.0	66.4 ± 42.1	84.1 ± 65.9
TimePkAmp150-IC[c]	120.7 ± 37.7	130.9 ± 21.6	132.5 ± 19.0	129.1 ± 25.1	127.8 ± 16.9
PkAmp150-PostIC[d]	128.6 ± 80.7	146.5 ± 157.5	108.9 ± 55.0	143.9 ± 133.7	119.6 ± 69.7
TimePkAmp20-150-PostIC[e]	82.2 ± 32	75.4 ± 27.5	88.9 ± 18.9	77.2 ± 34.9	86.5 ± 19.4
IPre150-IC[f]	8,587.5 ± 5,323.9	8,448.4 ± 5,836.3	7,500.0 ± 5,488.7	8,203.5 ± 5,519.4	10,118.4 ± 8,001.1
IPost20-150-IC[g]	20,878.0 ± 15,098.7	24,568.3 ± 28,146.4	17,428.7 ± 10,286.0	22,855.3 ± 21,474.2	17,615.9 ± 10,896.5
Running protocol					
Stance[h]	27.4 ± 16.2	29.1 ± 23.7	40.84 ± 47.24	23.8 ± 18.3	29.5 ± 20.1
Pre-activation[i]	71.3 ± 29.5	74.1 ± 30.0	74.53 ± 37.84	66.2 ± 15.1	69.0 ± 19.0
Initial loading[j]	37.5 ± 28.6	38.7 ± 35.7	45.16 ± 59.78	26.1 ± 17.7	34.7 ± 32.9
Main loading[k]	21.6 ± 12.0	23.8 ± 18.0	41.7 ± 54.32	21.4 ± 15.3	23.7 ± 10.7

[a] PkAmpIC: Peak amplitude at initial contact

[b] PkAmp150-IC: Peak amplitude within 150 ms to initial contact (%MVIC)

[c] TimePkAmp150-IC: Time to peak amplitude within 150 ms prior to initial contact

[d] PkAmp150-PostIC: Peak amplitude post-initial contact (%MVIC)

[e] TimePkAmp20-150-PostIC: Time to peak amplitude within 20–150 ms post-initial contact

[f] IPre150-IC: Integrated activity within 150 ms prior to initial contact

[g] IPost20-150-IC: Integrated activity within 20–150 ms post-initial contact

[h] Stance: Mean amplitude during stance phase (%MVIC)

[i] Pre-activation: Mean amplitude 100 ms to initial contact (%MVIC)

[j] Initial loading: Mean amplitude initial contact to 50 ms post-initial contact (%MVIC)

[k] Main loading: Mean amplitude 50–200 ms post-initial contact (%MVIC)

injected predominantly bupivacaine in his clinical study, whereas Myers et al. [26] utilized lidocaine. Both lidocaine and bupivacaine are members of the aminoamide group of anaesthetics and are weak bases. Bupivacaine is more lipophilic and has longer half-life than lidocaine (2.7 vs. 1.7 h). Both medications block motor and sensory function presumably by reversibly blocking voltage-gated sodium channels [12].

To the best of our knowledge, this is the first time a continuous dynamic protocol has been used to investigate the effect of local anaesthetics on ankle function. Previous studies have utilized a tilt plate mechanism, with subjects standing on the tilt plate until the perturbation task took place [19, 26]. Quiet stance time enabled previous authors to study muscle latency, which we were unable to evaluate, as there was no time during the task when muscles were not activated. It is possible that the decreased protective response reported by Myers et al. [26] is not present during a dynamic task, as muscle firing patterns are continuous in anticipation of landing. The difference between the dynamic task utilized in our study and prior studies may make comparisons difficult.

There are several limitations to the study. Subject enrolment was restricted to healthy men with no prior ankle injury, indicating no ankle effusion was present. Injured ankles of male subjects may respond differently to the injections as may injured ankles of female subjects. Ankle effusion has been shown to impair muscle function [4, 8, 11, 15, 16, 18, 35, 37]. Hall et al. [11] reported a decrease in the H-reflex in the flexor digitorum longus but not the PL following acute inversion ankle sprain and that this decrease was correlated to ankle girth. Palmieri et al. [30] induced an ankle joint infusion with 10 ml of saline and found increased H-reflex of the PL, TA and the soleus. Hopkins et al. [16, 37] and Tsang et al. [16, 37] noted suppressed peroneal muscle activity without compromising the TA, gastrocnemius and soleus. It is plausible that injections into other structures (e.g. deltoid ligament, ankle syndesmosis) may influence cutaneous and/or articular sensory receptors differently; therefore, the current findings cannot be extrapolated to the use of local anaesthetics in other acute ankle injuries. Additionally, effectiveness of the injection to induce anaesthesia of the area was not evaluated, which could have been determined using a

two-point discrimination assessment. The use of athletic tape, as would be used clinically, may have affected our results. Taping has been shown to impact cutaneous mechanoreceptors [20, 32] stimulation or excitability of the motor neuron pool [20, 32] and has resulted in conflicting results [20, 32]. Future research should investigate whether individuals with a subacute inversion ankle sprain, chronic ankle instability or functional ankle instability respond similarly to healthy controls following injection. Future studies should incorporate higher injection volumes, approaching doses that induce significant changes in previous studies [13, 19] as well as approaching doses typically utilized by sports medicine physicians on the sidelines in order to clarify if such a volume threshold exists. These higher injection volumes should also be tested with ankle effusion to determine the additive effect. Despite the lack of significant findings (positive in the context of this study) for the tested variables, the results were based on a small sample that demonstrated low power. Additional research is necessary to confirm these findings.

This finding is of clinical importance due to the common use of local anaesthetic injections in athletics. Having the knowledge that muscle activation patterns remain the same with or without a 3-ml dose of anaesthetic can help practitioners make an informed decision regarding the use of local anaesthetic injections following injury. Since there is no change in muscle activation patterns, the athlete will be at no higher risk of injury following an injection, and the injection can assist with pain management of the injured site.

Conclusion

Using a dynamic perturbation task, we were unable to demonstrate a statistically different change in muscle firing patterns in the PL, PB, TA or MG. The results suggest that up to 3 ml injection can be used safely for pain control without reducing dynamic restraints.

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Conflict of interest The authors declare that they have no conflict of interest.

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