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Tibialis posterior muscle pain effects on hip, knee and ankle gait mechanics

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ABSTRACT

Background: Tibialis posterior (TP) dysfunction is a common painful complication in patients with rheumatoid arthritis (RA), which can lead to the collapse of the medial longitudinal arch. Different theories have been developed to explain the causality of tibialis posterior dysfunction. In all these theories, pain is a central factor, and yet, it is uncertain to what extent pain causes the observed biomechanical alterations in the patients. The aim of this study was to investigate the effect of experimental tibialis posterior muscle pain on gait mechanics in healthy subjects.

Methods: Twelve healthy subjects were recruited for this randomized crossover study. Experimental pain was induced by ultrasound-guided injection of 1 mL hypertonic saline into the upper part of the right tibialis posterior muscle with the use of isotonic saline as non-pain-inducing control. Subsequently, kinematic data during three self-paced over ground walking for each condition were collected. Ground reaction forces and external moments were measured from force plates installed in the floor. Painful areas were evaluated using body charts and pain intensity scoring via a verbal numerical rating scale.

Findings: Decreased hip internal rotation was observed during the pain condition at the end of the stance phase. There were no changes in gait velocity and duration of stand phase between the pain and no pain conditions. Reduced external joint moment was found for external knee rotation and for external hip rotation.

Interpretation: The study has demonstrated that induced pain in the TP muscle evokes kinematic alteration in the hip and the knee joints, but not in the ankle, which suggest an underlying early stage joint compensatory mechanism. These findings suggest the need to include those joints in current physical evaluations of tibialis posterior dysfunction.

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1. Introduction

Tibialis posterior (TP) dysfunction is associated with gait dysfunction and a progressive flat foot deformity and pain development (Michelson, Easley, Wigley, & Hellmann, 1995) affecting multiple patient groups (Holmes, 1992; Keenan, Peabody, Gronley, & Perry, 1991; Myerson, Solomon, & Shereff, 1989). Prevalence data of TP dysfunction are limited (Semple, Murley, Woodburn, & Turner, 2009), but has previously been reported with a prevalence as high as 64% among patients with rheumatoid arthritis (RA) (Michelson et al., 1995). The pain associated to TP dysfunction is initially localized to the medial side of the ankle, and in later stages the pain can also be present in the lateral side (Johnson & De, 1989). Different theories exist on a possible association between TP dysfunction and adult-acquired flat foot deformity (Yeap, Singh, & Birch, 2001). It has been suggested that mechanical alterations caused by soft tissue instability (Spiegel & Spiegel, 1982; Woodburn, Cornwall, Soames, & Helliwell, 2005) or increased pronation forces (Keenan et al., 1991) lead to flat foot deformity. Subtalar and midfoot abnormalities have also been suggested to be the cause (Spiegel & Spiegel, 1982). Further theories include chronic tenosynovitis (Masterson, Mulcahy, Mcelwain, & Mcinerney, 1995). In all these scenarios, pain is a common factor, and yet, it is uncertain to what extent pain itself is causing the observed biomechanical alterations.

In a previous study comparing foot kinematics between patients with TP dysfunction and healthy controls, presence of increased rearfoot eversion has been observed in the patient group (Tome, Nawoczenski, Flemister, & Houck, 2006). In accordance to this observation, runners with early stage PT tendon dysfunction have been found with prolonged and increased rear-foot eversion compared to controls (Rabbito, Pohl, Humble, & Ferber, 2011). Further, a previous study have found that subjects with pronated feet have higher TP muscle activity compared to those with neutral foot structure (Murley, Buldt, Trump, & Wickham, 2009). As a result, it has been hypothesized that pronation of the rear-foot puts a greater load on the TP tendon and may be the reason behind the progressive nature of TP dysfunction (Murley et al., 2009; Rabbito et al., 2011).

Both acute and chronic pain have demonstrated capability for causing alteration in movement patterns (Henriksen, Graven-Nielsen, Aaboe, Andriacchi, & Bliddal, 2010). It is challenging to examine isolated effects of pain in chronic pain patient populations because of the different contemporary confounding factors that are present in patients with chronic pain (e.g. muscle atrophy and structure changes) (Son, Kim, Seeley, & Hopkins, 2017). Experimental chemical pain models have therefore been developed for healthy subjects (e.g. joint pain and muscle pain (Arendt-Nielsen, Graven-Nielsen, Svarrer, & Svensson, 1996)) to control for the effect of these confounding factors in patients with chronic pain. These models allow for the investigation of movement modifications due to pain in an otherwise healthy system, and have shown good replication of patients with musculoskeletal pain (Arendt-Nielsen et al., 1996; Henriksen et al., 2010).

Experimental pain models have also been used as a tool to investigate referred pain patterns, where pain are perceived at an adjacent site to that of the pain (Arendt-Nielsen & Graven-Nielsen, 2008). A previous study injecting hypertonic saline into the tibialis anterior muscle resulted in referred pain to a distant area from the injection site, primarily in the area of the ankle joint (Graven-Nielsen et al., 2000).

The current trend within footwear science is to model the foot as a multi-segmental structure. Even though these models have improved the knowledge and the in-vivo function during locomotion, it is often at the expense of excluding segments above the ankle joint (Barn et al., 2014; Pataky, Daou, Pataky, De Clercq, & Aerts, 2009; Tome et al., 2006). However, these excluded joints above the ankle joint may play an important role in the toe-out-gait observed among patients with TP dysfunction and potentially lead to consequential damages (Rabbito et al., 2011). Previous literature have reported reduced knee and hip moments in similar populations (Ringleb et al., 2007; Svoboda, Janura, Kutilek, & Janurova, 2016). However, existing literature within TP dysfunction has primarily focused on the kinetics and kinematics of the foot and ankle (Barn, Turner, Rafferty, Sturrock, & Woodburn, 2013a; Ferber & Pohl, 2011; Pohl, Rabbito, & Ferber, 2010; Ringleb et al., 2007) and not the causes of the motion or its effect on the other joints of the lower extremity. The foot is the foundation of all lower extremity locomotion, and inhibited foot motion may cause abnormalities in other joints, which potentially may develop into referred pain in distant anatomical sites (Hill, Gill, Menz, & Taylor, 2008).

The aim of this study was, therefore, to examine kinematic and kinetic variables in the joints of the ankle, knee and hip in healthy subjects with experimentally induced pain in the TP muscle. It was hypothesized (I) that experimental pain in the TP muscle would trigger referred pain in the location of the TP tendon and at the ankle joint. It was also hypothesized (II) that pain would cause greater ankle joint eversion.

2. Methods

2.1. Subjects

Twelve healthy subjects participated in this study. None of these had muscular, neurological, ischemic or other impairments that could affect their gait. Since this is the first study to induce pain in the TP muscle experimentally by injection technique, no power analysis was performed prior the experiment. However, in other similar experimental pain studies from 9 to12 subjects have been enrolled (Hirata, Arendt-Nielsen, & Graven-Nielsen, 2010; Shiozawa, Hirata, & Graven-Nielsen, 2013, 2015).

In this randomized, placebo controlled, crossover study experimental pain was induced by injection of hypertonic saline via a single injection into the TP muscle and isotonic saline was used as control (Hirata et al., 2010). Subjects were not informed that they would receive a control injection; they were informed that two injections with different sodium chloride concentrations were used. Injection order of the two types of injections was performed in a randomized manner using an ultrasound-guided technique by. Three trials were performed before each of the two injection types (baseline) and likewise another three immediately afterwards. Hence, a total of twelve gait trials was performed for each study subject. All gait trials were performed with a standardized neutral running

shoe (Nike, Beaverton, Oregon, USA).

Mean age was 28.3 (SD 1.8) years, body mass 83.7 (SD 12.0) kg, and height 180.3 (SD 9.8) cm. Written informed consent was obtained from all participants. This study was conducted in accordance with the Declaration of Helsinki. The local committee on health research ethics granted ethical approval for this study (application reference: N20170066).

2.2. Experimental muscle pain

Ultrasound examination of the right TP muscle was performed with the subject placed in a supine position with the leg slightly internally rotated. The safety window was identified to avoid artery, vein and neurovascular bundles (Rha, Im, Lee, & Kim, 2010). After prepping the skin with an alcohol wipe and iodine, 1 mL of hypertonic saline (5.0% NaCL) or isotonic saline (0.9% NaCl) was injected near the upper third point of the tibia with the anterior approach using an ultrasound-guided injection technique (Rha et al., 2010). Injections were performed inside the motion capture laboratory, thereby allowing starting data collection immediately after the injection. Joint angles of the ankle, knee and hip joint were measured. Additionally, external joint movements of the ankle, knee and hip, gait speed, pain scores and body charts were also collected.

2.3. Numerical rating scale and pain mapping

The experimental pain intensity was assessed on a 0–10 numerical rating scale (NRS) where “0” indicated ‘no pain’ and “10” ‘maximum pain’. Subjects were asked to indicate the severity of their pain after each gait trial and to register the perceived pain on anatomical body charts of the lower leg. Pain area was then measured using ImageJ v.1.8.0_112 (National Institutes of Health, Madison, Wisconsin, USA). Body charts were scanned, layered, and averaged through MATLAB, version 2017B (The MathWorks, Inc., Natick, Massachusetts, USA).

2.4. Gait analysis

Kinematic data during self-paced gait were collected at 100 Hz by an eight-camera infrared Qualisys system (Oqus 300 series, Qualisys, Sweden). A protocol of 32 passive, reflective markers was used to track the lower extremity: 12 markers were placed on palpable anatomical landmarks on the left and right side (medial malleus, lateral malleolus, femur medial epicondyle, femur medial epicondyle, anterior superior iliac spine and posterior superior iliac spine). Four markers on each shoe (Heel, on top of the 3rd toe and approximately above the 1st and 5th metatarsal head), four clusters with three markers in each placed on the shank and thigh using double-sided tape (Fig. 1). The same investigator placed all the markers. Kinematic and kinetic data were processed in Qualisys Track Manager Version 2.16, (Qualisys, Gothenburg, Sweden) and exported in a C3D format. Ground reaction forces and moments were collected at 1000 Hz from force plates installed in the floor (AMTI, USA).

2.5. Estimation of joint angles, joint moments and gait velocity

A musculoskeletal model was applied using the AnyBody Modelling System (AMS) version 7.0.1 (AnyBody Technology A/S, Aalborg, Denmark) to estimate joint angles, joint moments and muscle forces. However, only joint angles and moments estimates were applied in the study due to the uncertainty of the muscle redundancy problem caused by the experimental pain (Simonsen et al., 2018).

An anatomical landmark scaled musculoskeletal model was used (Lund, Andersen, de Zee, & Rasmussen, 2015). The model was scaled based on a standing reference without any additional input. Briefly, a nine-segment stick-figure with 15 degrees of freedom was generated from a standing reference trial. The standing reference included four additional markers, compared to the gait trials, representing the medial femoral epicondyles and medial malleolus used to compute knee and ankle axis (Lund et al., 2015). The standing reference included four additional markers, compared to the gait trials, representing the medial femoral epicondyles and medial malleolus used to compute knee and ankle axis (Lund et al., 2015), the markers was not removed during the experiment. These markers were used to construct the stick figure model but not included in the analysis of the dynamic trials. The hip joint was defined as a spherical joint and its position was calculated from the reference trial via a regression formula with leg length, pelvic depth and width as predictors (Harrington, Zavatsky, Lawson, Yuan, & Theologis, 2007). The knee and talocrural joints were defined as revolute joints. The subtalar joint, which is not identifiable from the marker positions, was placed 10 mm inferior to the ankle joint and oriented with a joint inclination of 42° and a deviation of 23° with respect to the midfoot line joining the heel and front toe marker (Inman, 1976). The total body mass was distributed to the individual segments using the anthropometric model by Dempster (Dempster, 1955). Joint angles were computed over the entire gait trial in the kinematic analysis (Lund et al., 2015), which is accomplished by minimizing the least-square difference between modeled and experimental markers (Andersen, Damsgaard, MacWilliams, & Rasmussen, 2010; Andersen, Damsgaard, & Rasmussen, 2009). The joint angles and the scaled musculoskeletal model were thereafter used as input in the inverse dynamic analysis to estimate the joint moments. Both joint angles and joint moments were represented in joint coordinate systems defined in accordance with the International Society of Biomechanics’ recommendations (Wu et al., 2002).

The horizontal gait velocity was calculated by averaging the horizontal velocity of the four pelvis markers during the stance phase. The stance phase started when the heel struck the force plate and ended when the toes left the plate at the beginning of the swing phase. The stance phase was time normalized to 100%. Joint moments were normalized to percentage of body weight and body

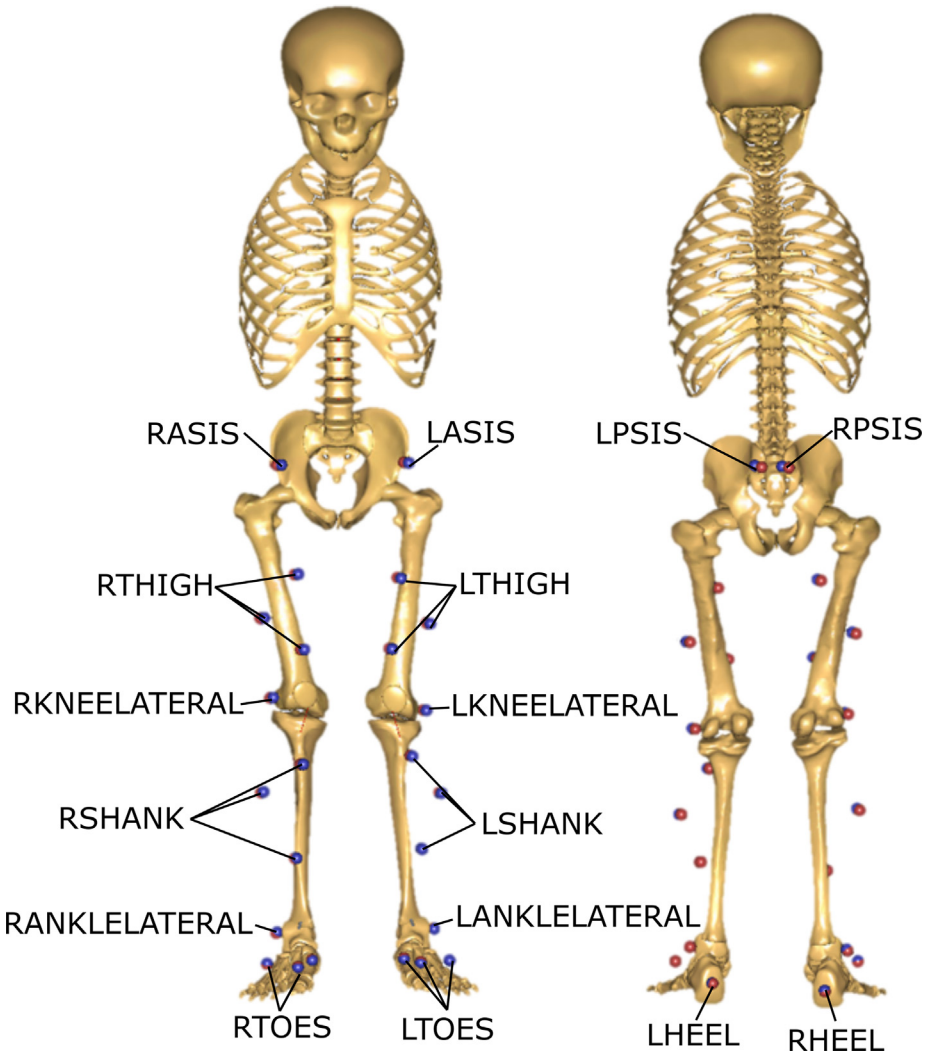

Fig. 1. Placement of the markers for the motion capture. Locations of the clusters were calculated by using an optimization method.

height.

2.6. Statistical analysis

All data were tested for normality using a Shapirio Wilk test. A paired *t*-test was used to compare the NRS scores after isotonic and hypertonic injections SPSS v25 (SPSS Inc., Chicago, IL, USA). Statistical-non parametric mapping (SnPM) (Friston, 2007; Pataky, 2012) was used to assess differences in joint moments, joint angles and ground reaction forces (GRF) between all conditions. Statistical parametric mapping (SPM) and SnPM are techniques originally developed within the field of functional brain MRI, but it have also been applied within the field of biomechanics (Pataky, 2012; Pataky et al., 2008). SPM and SnPM analysis allows for statistically analysis of entire time series of a cyclic motion, such as gait (Pataky et al., 2008). Both approaches describe the behavior of random data, but SnPM does not assume a normal Gaussian distribution. The SnPM version of the one-way ANOVA with repeated measures of was performed. Further, post-hoc analysis was performed with Bonferoni correction. The resulting critical alpha value was 0.012. No differences between baselines and the isotonic conditions was found. Subsequently, only post-hoc results between the isotonic and hypertonic injection is presented. All SPM analyses were applied using the open source spm1d code (Pataky, 2012) (v.0.4, spm1d.org) in MATLAB (R2017B), (The MathWorks, Inc., Natick, Massachusetts, USA).

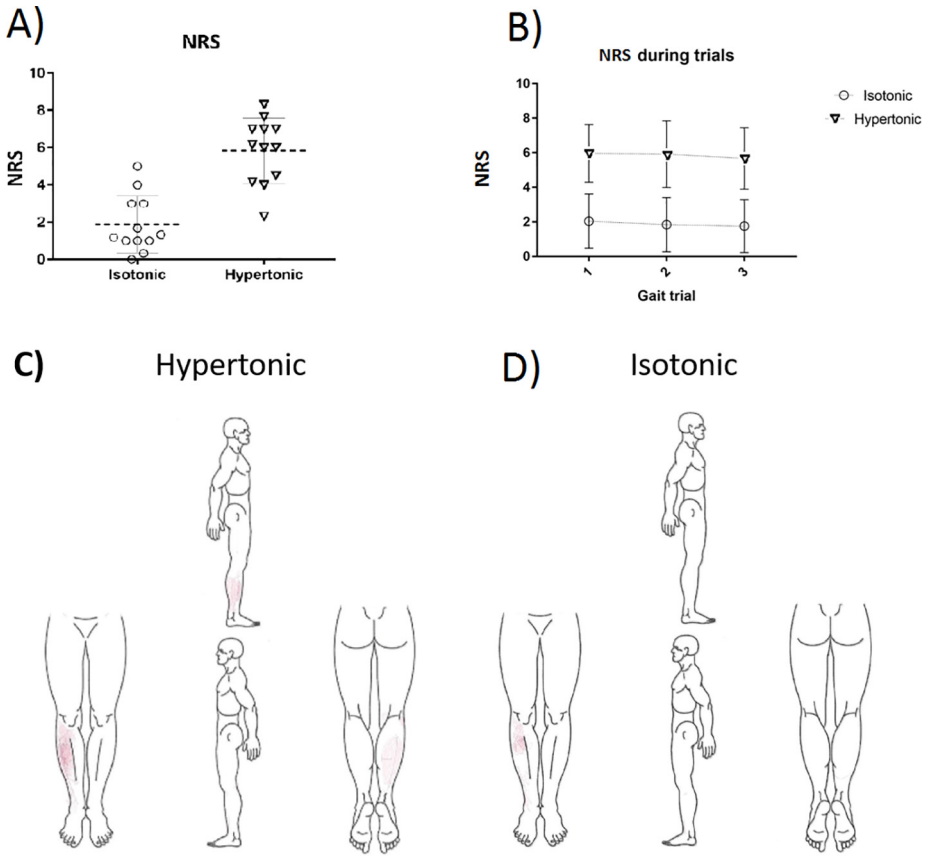

Fig. 2. A) NRS scores for the hypertonic and isotonic condition, dashed line is the mean value and whiskers is the SD. B) Average NRS scores for each gait trial with SD. C) Layered body chart for the pain condition induced by hypertonic saline injection. D) Layered body chart for the control condition by use of isotonic saline.

3. Results

3.1. Pain perception

Pain intensity (NRS scores) following the hypertonic saline injection (mean 5.8 (SD 1.7) arbitrary unit (a.u.)) was higher than in the controlled setting by injection of isotonic saline (mean 1.8 (SD 1.4) a.u.), ($t(11) = -3.97$, $p < 0.001$). Fig. 2A shows NRS scores for the hypertonic and isotonic condition. The experimental pain disappeared after approximately 2 min, average NRS scores directly after each gait trial are shown in Fig. 2B. The area on the body chart was larger upon injection of hypertonic saline ($t(11) = -3.859$, $p = 0.003$). Fig. 2B and C show layered body charts for both control and pain conditions.

3.2. Joint angles, gait kinematics and ground reaction force

There was no difference in angles for the ankle joint (Fig. 3). However, Difference between conditions was found for the internal hip rotation angle (($F(3,44) > 5.337$, $p = 0.030$)) post-hoc analysis revealed increased internal hip rotation angle for the hypertonic condition compared to isotonic condition at the end of the stance (time point 91%–100% ($t(11) > 3.167$, $p = 0.010$, Fig. 3F)).

No statistical difference was found in horizontal gait velocity (Pre-Isotonic = 1.06 m/s, Pre-Hypertonic = 1.07 m/s, Isotonic = 1.07 m/s and Hypertonic = 1.05 m/s ($F(2.215, 24.362) = 1.215$, $p = 0.318$)) or for the duration of the stance phase (Pre-Isotonic = 0.74 s, Pre-Hypertonic = 0.75 s, Isotonic = 0.74 s and Hypertonic = 0.74 s). $F(1.3(14.431 = 0.287$, $p = 0.655$)). Additionally, no difference was found in ground reaction force between the two conditions (Fig. 4).

3.3. Hip, knee and ankle joint moments

No differences were found for the ankle joint moments (Fig. 5A–C). However, difference was found between conditions for the knee external rotation ($F(3,44) > 5.092$, $p = 0.010$), post-hoc test found difference between time points, 59%–84% ($t(11) > 4.776$, $P < 0.001$, Fig. 5F), during hypertonic saline condition compared with isotonic condition. Finally, the hip external rotation moment

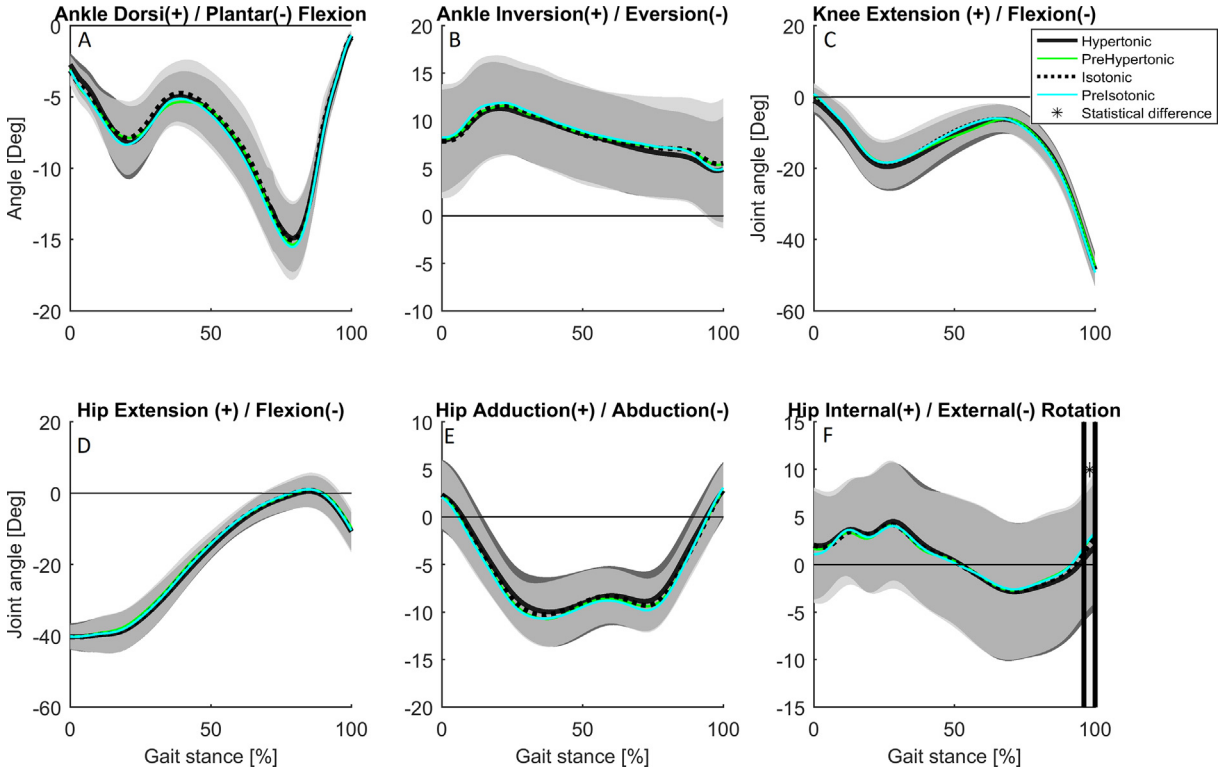

Fig. 3. Mean joint angles for the ankle, knee and hip joint of all subjects. The dotted line is the isotonic condition, the solid line is the hypertonic condition and the light gray shaded area with a star signals a significant difference between the conditions.

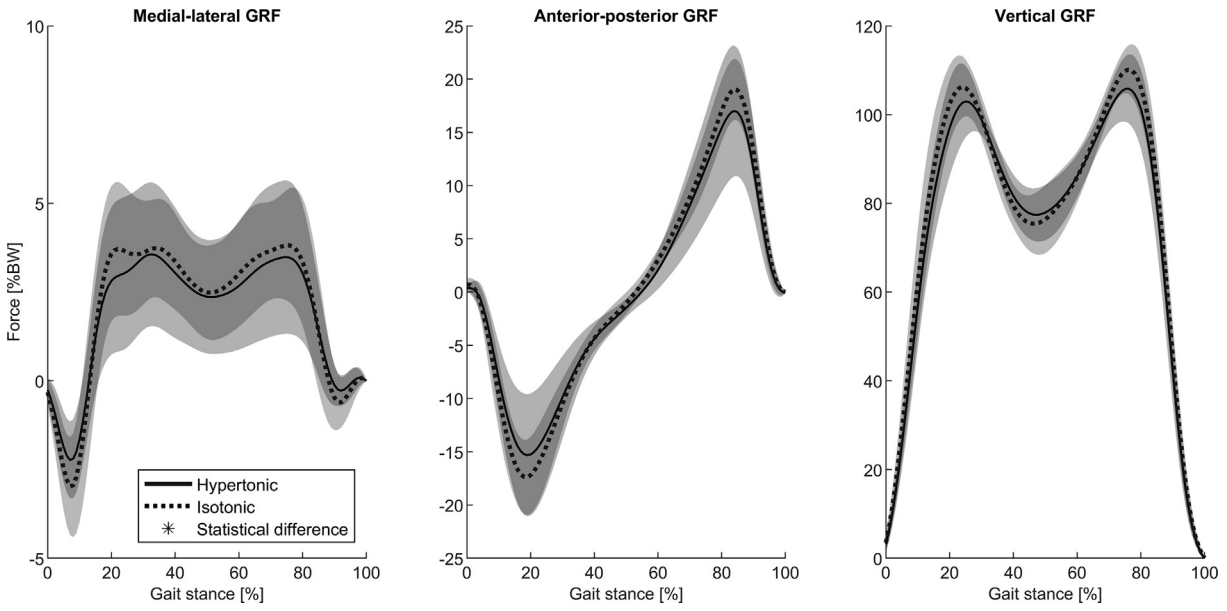

Fig. 4. Mean medial-lateral, anterior-posterior and vertical ground reaction forces (GRF). The dotted line is the isotonic condition; the solid line is the hypertonic condition.

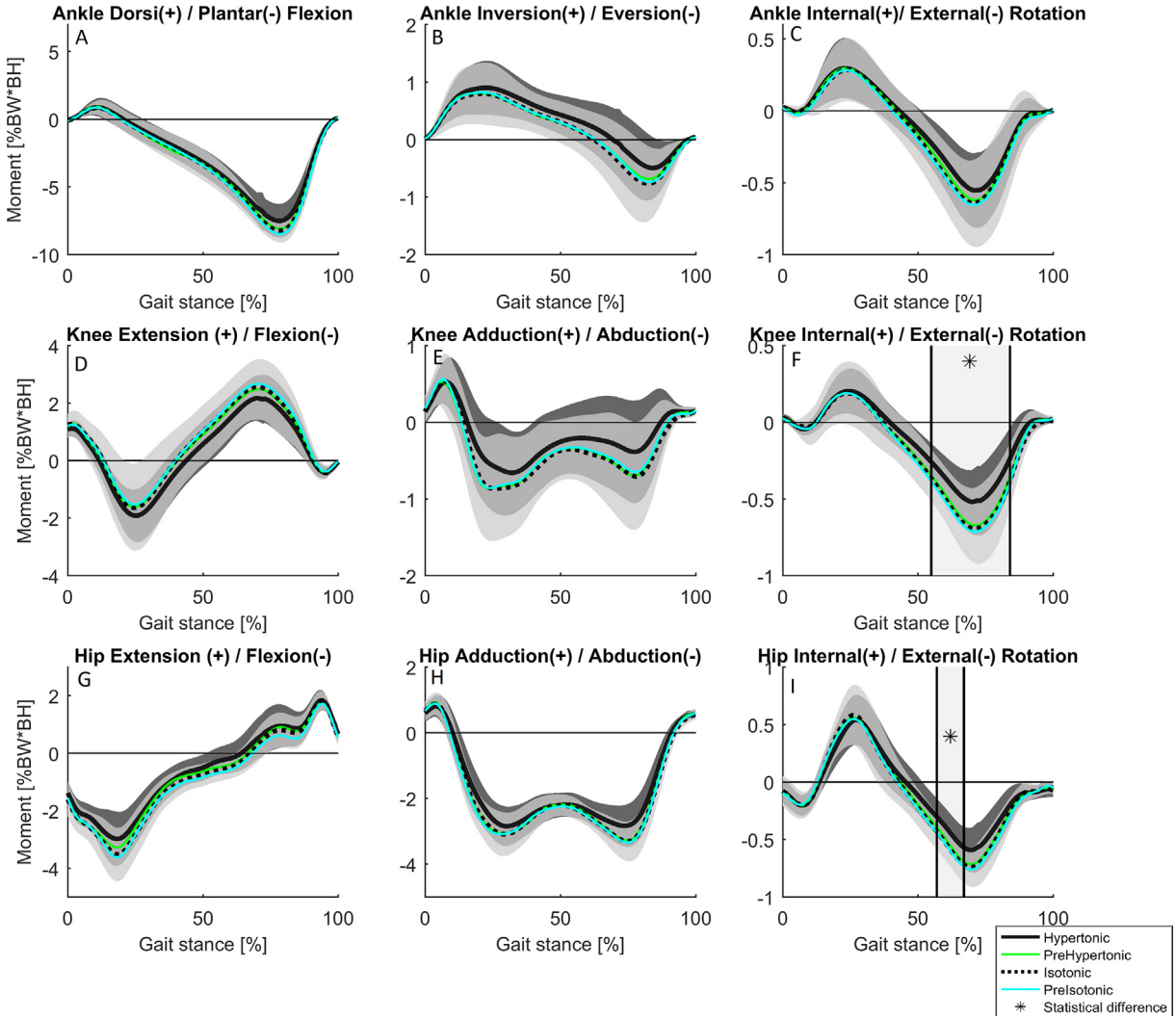

Fig. 5. Mean moment values for all degrees of moment of the ankle, knee and hip joint. The dotted line is the isotonic condition, the solid line is the hypertonic condition and the gray shaded area with a star signals a significant difference between the conditions. The dark gray and gray shaded areas show one SD for the hypertonic and isotonic condition, respectively.

($F(3,44) > 5.844$, $p = 0.022$). The hip external rotation moment was reduced during the hypertonic condition compared to the isotonic between time points, 57%–67% ($t(11) > 4.762$, $p < 0.001$, Fig. 5I).

4. Discussion

This study explored the effect of experimentally induced pain in TP muscle on pain perception itself, lower extremity joint angles and moments in healthy subjects.

4.1. Pain perception

To the best of our knowledge, this is the first study to assess induced pain in the TP muscle. NRS scores for both the hypertonic and isotonic saline injection test were higher than shown in previous studies in which pain has been induced in the superficial part of the tibialis anterior muscle (Graven-Nielsen, Arendt-Nielsen, Svensson, & Jensen, 1997). This difference might be due to the deep injection technique used in the present study, where a needle was inserted through the tibialis anterior muscle and the interosseous membrane (Rha et al., 2010). Another possible contributing factor for the increased pain could be the nociceptor density of the TP muscle. Spindle density of the small plantar flexor muscles has been observed to be up to 5.5 times larger compared to the larger plantar flexor muscles (Peck, Buxton, & Nitz, 1984), which potentially makes the TP muscle more sensitive to pain stimuli.

A previous study by Graven-Nielsen et al. (2000) found that experimental pain in the tibialis anterior muscle gave rise to referred

pain in the ankle (Graven-Nielsen et al., 2000; Graven-Nielsen, 2006). Pain mapping from the present study revealed that the subjects reported pain around the area of the injection site in the isotonic (control) test as well as in the hypertonic (painful) test. However, in the hypertonic condition, subjects also reported pain on the back (n = 5) and lateral part (n = 6) of the lower leg. Additionally, four subjects reported pain around the ankle. Together, those observations suggest that the applied pain experimental model to some extent had induced referred pain in a subgroup of the study subjects. Subjects in the present study did not receive specific instructions to be aware of referred pain. A possible mechanism behind the observed referred pain could be the close innervation at the L5 and S1 roots in the spinal cord. These innervations carry signals from different parts of the lower body. The close overlapping innervation might explain why some subjects felt pain at the ankle joint, despite the nociceptors at the ankle level were not simulated directly by the injections (Palsson & Graven-Nielsen, 2012). The finding of referred pain in the present study and the findings of Graven-Nielsen et al. (2000) supports that pain in the tibialis anterior and TP trigger referred pain near the ankle joint site. Future studies could introduce pain by injection in other structures than muscles (e.g. tendons) and investigate if experimental pain proximal to the ankle joint would result in referred pain at the upper part of the lower leg. In addition, investigations on patients with early stage TP dysfunction could be performed to see if similar changes are found near the knee.

4.2. Joint angles, gait kinematics and ground reaction forces

Both acute and chronic pain cause alterations in movement patterns in both healthy and patients (Carroll, Parmar, Dalbeth, Boocock, & Rome, 2015; Graven-Nielsen, Svensson, & Arendt-Nielsen, 1997). Previous studies have reported reduced gait velocity in patients with TP dysfunction compared to healthy subjects (Ness, Long, Marks, & Harris, 2008). However, in the present study, neither the horizontal gait velocity, duration of the stance phase nor GRF were different between the two conditions. This finding suggests that isolated TP muscle pain is not the direct cause for decreased gait velocity in patients with TP dysfunction (Ness et al., 2008).

In contrast with one of our hypotheses, no difference was found for the ankle joint angles. A previous muscle fatigue study by Pohl et al. (2010) showed that a 30% reduction of maximal voluntary contraction of the TP muscle did not change foot kinematics during gait (Pohl et al., 2010). It should be noted that a limitation of using fatigue-inducing models is the uncertainty to what extent the targeted muscle has been fatigued, since synergistic muscles might be involved (Pohl et al., 2010). In contrast, injection-based models allow for controlled investigations (Shiozawa, Hirata, & Graven-Nielsen, 2013). All together, the results of Pohl et al. (2010) and the present study suggest that an acute pain or fatigue of the TP muscle does not cause ankle kinematic alterations. This is contrary to what has been reported in studies investigating patients with TP dysfunction (Barn et al., 2013a; Rabbito et al., 2011; Semple et al., 2009; Tome et al., 2006). A study by Rabbito et al. (2011), investigating gait kinematics in runners with diagnosed stage I TP tendon dysfunction, found increased and prolonged peak rear-foot eversion, speculating that increased foot pronation puts greater load on the TP tendon, which may be an explanation of the progressive nature of TP dysfunction (Rabbito et al., 2011). The later antalgic gait pattern found in these patients suffering from TP dysfunction and rheumatoid arthritis might be the result of an adaptation strategy over time aiming to decrease pain in the affected areas (Hirata et al., 2010). The foot model used in the present study only consist of one segment; therefore, future investigations could try to use multi-segmental foot models combined with the rest of the lower extremity. This could potentially improve the insights into the interplay between the foot with TP dysfunction and the rest of the lower extremity.

In the present study, the external rotation of the hip increased during the last period of the stance phase for the hypertonic pain condition compared to the isotonic condition. This finding suggests that the foot is more externally rotated during toe-off and this could potentially lead to increased changed loading and tension in the toe joints due to the increased rotation (Resende, Pinheiro, & Ocarino, 2019). However, this strategy could also lead to an unloading of the foot segments or the muscles controlling the foot segments (Lynn, Kajaks, & Costigan, 2008). Previous studies have found an relation between external rotation of the hip joint and transverse rotation of the foot (Gaston, Rutz, Dreher, & Brunner, 2011; McMulkin, Baird, Caskey, & Ferguson, 2006; Van Kuijk, Kosters, Vugts, & Geurts, 2014). However, a regression analysis between the transverse foot angle and internal/external hip angle revealed a weak relation ($r^2 = 0.105$) Though, it is questionable whether this has any clinical relevance isolated for the hip joint, since the hip internal rotation moment is close to zero during the end of the stance phase.

4.3. Hip, knee and ankle moments

Muscles located in the lower leg primarily control movement of the foot during walking (Martini & Nath, 2009). Pain-evoked dysfunction of one muscle can lead to compensation from other anatomic structures and potentially lead to an antalgic gait (Lalli et al., 2013). The limited existing literature about patients with TP dysfunction has primarily focused on foot kinematics and electromyography measurements of the muscles of the lower leg (e.g. TP, tibialis anterior, soleus, gastrocnemius) (Barn et al., 2014; Barn, Turner, Rafferty, Sturrock, & Woodburn, 2013b). This is the first study to investigate internal moments at the hip and knee joints.

The experimental TP muscle pain reduced external knee rotation (55%–85% of stance phase) moment compared with the isotonic condition. Furthermore, the external hip rotation moment was reduced (57%–67% of stance phase) compared with the isotonic condition. These results suggest a protective strategy to decrease joint mechanical load by reducing the joint moments during phases where the TP muscle is normally active during gait (Murley et al., 2009). Protection strategies for reducing pain have been proposed by multiple studies, for instance, Hirata et al. (2010) found in an experimental pain study that the non-painful leg compensated for the painful leg during a quiet standing task (Hirata et al., 2010). Another example is Friel, Mclean, Myers, and Caceres (2006), who found reduced ankle dorsiflexion and plantar flexion in patients recovering from ankle sprains, assessed by goniometric

measurements (Friel et al., 2006). Friel et al. (2006) suggested that it was a protective strategy to reduce mechanical loading of the injured ankle during motion. Interestingly, Friel et al. (2006) also found reduced hip abduction strength indicating that compensation strategies due to foot injury can influence muscles of the hip. Altogether, these results suggest that pain leads to motor adaptations aiming to reduce mechanical load in the injured area (Friel et al., 2006; Hirata et al., 2010; Hodges, 2011). These motor adaptations seem to involve joints distant from the painful area (Friel et al., 2006; Hirata et al., 2010). Indeed, our results showed no changes in ankle joint moments during gait even though it was expected that TP pain would have caused altered ankle joint kinematics and kinetics and, potentially, decreased gait velocity, which would potentially reduce ankle joint mechanical loads. Experimental TP pain did not cause similar antalgic gait patterns seen in patients with chronic rheumatoid arthritis and TP dysfunction (Barn et al., 2013a; Tome et al., 2006). However, the experimental TP pain induced changes in the mechanical load of the adjacent joint (hip and knee). These changes might be reflecting the impairments found in patients at very early stage of the disease, and should be investigated in greater detail in future studies.

4.4. Limitations

The first limitation is the limited number of subjects enrolled in this study. The small number of subjects and the variance of the gait data might be the explanation of why no statistical difference were found at the ankle joint. Even though this is the case, statistical differences was found between the conditions for the hip and knee joint. It is also possible that kinematic data would have been different if the subjects had more time to adjust to the pain. Pain from the hypertonic saline disappeared quicker (after two minutes) than has been reported in other muscles and ligaments, where it is not unusual for the pain to be present for up to ten minutes (Hirata et al., 2010). However, many of these studies have used a static experimental setup (Tsao, Tucker, Coppieters, & Hodges, 2010). Previous studies using experimental hypertonic saline test models during dynamic tasks have found that that pain can change and disappear quicker due to dynamic movement such as stretching and movement of the painful muscle, thereby increasing the blood flow, which removes the saline quicker (Tsao et al., 2010). Consequently, this makes it difficult to maintain pain intensity with single injections. There were also limitations with the musculoskeletal model applied in the current study (e.g. the foot of the model was modelled as a single segment), although the model used in the present study has previously shown good accuracy compared to in-vivo measured knee forces (Marra et al., 2015).

4.5. Conclusion

In summary, acute pain in the TP muscle does not cause similar gait alterations as observed in patients with TP dysfunction and rheumatoid arthritis. This might indicate that TP pain is not the immediate cause for the antalgic gait observed among early stage patients suffering from TP dysfunction (Ness et al., 2008). In the present experimental study, the antalgic gait from the TP pain was not observed locally at the ankle joint, although reductions at the internal rotation of the hip joint were found, suggesting a compensation motor strategy in a joint distant from the pain site. Further, reduced moments in the knee and hip joints were observed around the push-off phase. The changes found in the healthy patients in this study might reflect the early adaptations when the patients first have pain (Rabbito et al., 2011). The early compensatory motor strategy observed at the knee and hip joints might be interpreted as the initial signs of impaired TP function in patients. Future studies should investigate if the compensatory mechanisms found in healthy subjects under experimental acute pain are indeed present in patients suffering from RA and TP dysfunction in earlier stages.

Disclosure

We, the authors, have no real or perceived financial and personal relationships with other individuals or organizations that could inappropriately influence (bias) our work.

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