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Original article

A modified straight leg raise test to differentiate between sural nerve pathology and Achilles tendinopathy. A cross-sectional cadaver study

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ABSTRACT

Background: A modified straight leg raise test for the sural nerve (SLR_{SURAL}) has been proposed to assist in the differential diagnosis of sural nerve pathology in people with posterior calf or ankle pain, or lateral foot pain. The biomechanical rationale is that strain in the dorsolateral ankle and foot structures following dorsiflexion-inversion can be selectively increased in the sural nerve with hip flexion. There are however no studies which have investigated whether hip flexion can increase strain in the sural nerve at the ankle.
Objectives: To measure strain and longitudinal excursion of the sural nerve and Achilles tendon during a modified SLR.
Design: Cross-sectional cadaver study, with a repeated-measures design.
Method: Strain and excursion were measured unilaterally in seven embalmed cadavers using differential transducers and a digital calliper. Data were analysed with repeated-measures ANOVAs ($p < 0.05$).
Results: With hip flexion (mean (SD): 54.6 (10.6) degrees), strain increased in the sural nerve (0.9 (0.5)%; $p = 0.008$), but not in the Achilles tendon (0.3 (0.3)%; $p = 0.16$). The sural nerve moved 1.0 (0.5) mm proximally with hip flexion ($p = 0.02$).
Conclusions: The load placed on the sciatic nerve following hip flexion is transmitted distally to the sural nerve. These findings provide biomechanical support for the SLR_{SURAL}. The relatively small changes in strain and excursion were most likely due to limited available ankle mobility in the tested cadavers. Further research is required to establish the diagnostic accuracy of SLR_{SURAL} in a clinical setting.

1. Introduction

Calf pain, ankle pain and foot pain can be due to a large variety of causes (Hawke and Burns, 2009; Choudhary and McNally, 2011). Sural nerve pathology is an uncommon diagnosis (Stickler et al., 2006), but is a condition that may be overlooked as differential diagnosis. For example, a retrospective analysis of 18 elite athletes with suspected chronic Achilles tendinopathy who failed conservative tendon management revealed that sural nerve entrapment was the cause of the symptoms (Fabre et al., 2000). Surgical release of the nerve resulted in positive outcomes. Also, standard textbooks in physiotherapy, and orthopaedic and sports medicine do not discuss sural nerve pathology extensively (Kolt and Snyder-Mackler, 2007; Magee, 2008; Brukner and Khan, 2012).

The sural nerve is a sensory nerve that innervates the posterolateral side of the distal third of the leg and lateral aspect of the foot (Paraskevas et al., 2014). Anatomically, the formation and course of the proximal sural nerve show great variability (Riedl and Frey, 2013; Paraskevas et al., 2014). In the majority of people (73%), the sural nerve branches off the tibial and fibular nerve. In 23% of people it originates from the tibial nerve only, and in a small minority (3%), the sural nerve branches off the fibular nerve only

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(Riedl and Frey, 2013). The sural nerve courses distally between the two heads of gastrocnemius, pierces through the deep fascia from inside the mid-calf and travels down over the lateral part of the Achilles tendon to terminate in the lateral foot (Apaydin et al., 2009).

Various pathological mechanisms have been described in the literature that may result in sural nerve pathology. Entrapment of the nerve can be caused by compression due to fascial thickening, increased calf muscle mass, local scar tissue or mass-lesions, such as a benign or malignant tumour, haemorrhage or abscess (Fabre et al., 2000; Nicklas et al., 2006; Paraskevas et al., 2014). Ski boots, casts and tight footwear may increase pressure from externally (Brukner and Khan, 2012). Injury to the sural nerve can also result from trauma (e.g., an ankle inversion sprain (Jotwani et al., 2008), or a fracture or direct blow during sports (Babwah, 2012)). Percutaneous repair of the Achilles tendon also places the sural nerve at risk for injury (Apaydin et al., 2009; Porter et al., 2014). Furthermore, distal peripheral neuropathy is one of the most frequent complications of diabetes and diabetes also affects the sural nerve (Kundalic et al., 2014). Clinical manifestations are burning pain, electrical or stabbing sensations, paraesthesia, hyperesthesia, deep pain, and allodynia. Diabetes results in morphological changes in the nerve fibres and in the connective tissue sheaths of the sural nerve, such as perineurial thickening and endoneurial fibrosis (Severo Do Nascimento et al., 2013; Kundalic et al., 2014).

Sural nerve pathology can mimic Achilles tendon pain and, due to the close proximity of the two structures, differentiation between the two conditions remains difficult (Fabre et al., 2000; Cook et al., 2002). A modified SLR test (SLR_{SURAL}) has been proposed to differentiate between Achilles tendinopathy and sural nerve pathology (Butler, 2000). In this test, dorsiflexion-inversion, which mechanically challenges both the sural nerve and Achilles tendon (Lyman et al., 2004; Wilmes and von Piekartz, 2010), is followed by hip flexion. As mechanical forces are transmitted and distributed along long sections of the nervous system (Coppieters et al., 2006; Ridehalgh et al., 2014), it is argued that hip flexion can increase the load on the sural nerve without loading the Achilles tendon (Butler, 2000). However, to date, there are no studies that evaluate whether hip flexion can increase the mechanical provocation of the sural nerve.

The primary aim of this study was therefore to investigate whether mechanical loading of the sural nerve could be increased with hip flexion in a modified SLR procedure. Strain and excursion of the sural nerve were evaluated. The secondary aim was to evaluate whether the strain in the Achilles tendon altered with hip flexion. Besides the nervous system, a continuous fascia network has been described from the hip, via the knee, to the ankle (Gerlach and Lierse, 1990; Webborn et al., 2014). It has been argued that forces can be transmitted over fascia networks and distributed across multiple segments (Vleeming et al., 1995; Barker and Briggs, 1999). There is currently no strong evidence to support or refute this assumption.

2. Materials and methods

2.1. Cadavers

Strain and excursion of the sural nerve and Achilles tendon were evaluated in seven cadavers (6 females; 1 male) with a mean (SD) age at time of death of 85.4 (10.4) years. The number (Coppieters and Alshami, 2007; Gilbert et al., 2007) and age (Coppieters et al., 2006; Alshami et al., 2007b) of the cadavers was comparable to previous studies. All procedures were tested in a pilot trial in one additional female cadaver before the actual experiment commenced. All cadavers were embalmed using genelyn 2% phenol. The lower quadrant, trunk and spine were intact in all cadavers. There were no visual anatomical abnormalities or signs of trauma on any of the cadavers to the skeletal, articular, muscular or neurovascular systems. Ethical approval for this study was obtained from the institutional ethics committee prior to the commencement of the study.

2.2. Dissection

To expose the sural nerve and Achilles tendon at the distal dorso-lateral part of the lower leg, a skin flap (size: ~12 × ~6 cm) was created just proximal to the lateral malleolus. Subcutaneous tissues were removed if it was anticipated they would interfere with the measurements. Removal of subcutaneous tissues was kept to a minimum in order not to compromise normal biomechanics of the sural nerve or Achilles tendon. The dissections were performed on the right side in four cadavers, and on the left side in three cadavers.

2.3. Straight leg raise test procedure

In the starting position, the hip was positioned in neutral and the ankle in plantar flexion. Maximal plantar flexion was avoided in the starting position to prevent buckling of the neurovascular bundle (Coppieters and Alshami, 2007). In the first stage of the procedure, the ankle was maximally dorsiflexed, while the hip was maintained in a neutral position. In the second stage, the hip was flexed to the available ROM while maintaining the ankle position obtained in the first stage. Ankle inversion could not be included in the test due to the restricted ROM of the ankle joint. Prior to the dissection, the ankle and hip were mobilised to increase ROM, but this procedure was only effective for the hip. Because the primary aim of this study was to evaluate the effect of hip flexion on sural nerve biomechanics at the ankle (Stage 2 of the procedure), obtaining a physiological hip ROM was important.

A universal stainless steel goniometer was used to measure ankle and hip angles according to standard goniometry guidelines (Norkin and White, 1995). Anatomical landmarks were marked on the skin with a surgical pen to reduce measurement error. In each stage of the test, ankle and hip angles were verified. Hip and ankle ROM were kept constant between repetitions within one cadaver, but could vary between cadavers depending on the available ROM.

The modified SLR was performed with the cadaver positioned in side-lying. This position was chosen to prevent the potentially varying gravitational effect on the strain gauge components when the hip would be flexed with the cadaver in supine. Three investigators stabilised the pelvis, trunk and opposite lower limb to maintain the cadaver in the side-lying position.

2.4. Strain measurements

Strain in the sural nerve and Achilles tendon were measured with strain gauges (Differential variable reluctance transducer (DVRT), Microstrain, Burlington, VT, USA) with a stroke length of 6 mm and a resolution of 1.5μ m. Strain gauges were inserted into the nerve and tendon with two barbed pins (Fig. 1). Calibration equations provided by the manufacturer were used to convert voltage output into length measurements. Output from the strain gauges was displayed on two digital voltmeters.

2.5. Excursion measurements

Longitudinal excursion of the sural nerve was measured relative to a reference marker (Coppieters et al., 2006). The reference