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Effect of Extra-osseous Talotarsal Stabilization on Posterior Tibial Nerve Strain in Hyperpronating Feet: A Cadaveric Evaluation

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ABSTRACT

Excessive abnormal strain or tension on the posterior tibial nerve in feet exhibiting talotarsal instability has been considered one of the possible etiologic factors of tarsal tunnel syndrome. The suggested treatment options in such cases include stabilization of the talotarsal joint complex in a corrected position, which might help minimize the abnormal forces placed on the posterior tibial nerve due to over stretching. The primary goal of this study was to quantify strain on the posterior tibial nerve in feet exhibiting hyperpronation caused by talotarsal instability, before and after an extra-osseous talotarsal stabilization (EOTTS) procedure. We hypothesized that the excessive strain placed on the posterior tibial nerve in hyperpronating cadaveric feet would be reduced significantly after intervention using the HyProCure® EOTTS device. Posterior tibial nerve strain was quantified in 9 fresh-frozen cadaver specimens. A miniature differential variable reluctance transducer was used to measure nerve elongation as the foot was moved from its neutral to a maximally pronated position, before and after intervention. The mean elongation of the posterior tibial nerve (with respect to a fixed reference point) decreased by 43% after the EOTTS procedure (i.e., from 5.91 ± 0.91 mm to 3.38 ± 1.20 mm; N = 27). The reduction was statistically significant at $p < .001$. HyProCure® was effective in stabilizing the talotarsal joint complex, thus reducing the excessive amount of strain placed on the posterior tibial nerve. Clinical implications of this study suggest the use of EOTTS devices in the treatment of tarsal tunnel syndrome.

Tarsal tunnel syndrome is a painful foot condition that has recently received a great deal of attention by the medical community. It is generally considered an entrapment neuropathy of the posterior tibial nerve in the tarsal tunnel (1–3). However, a multitude of etiologies have been associated with the occurrence of tarsal tunnel syndrome. These are broadly classified under the categories of trauma, space occupying lesions, structural deformities of the foot, and idiopathic (4–6). Recently, research has focused on understanding the effect of structural deformities of the foot as a causative factor of tarsal tunnel syndrome, more specifically, studying the effects of an unstable hindfoot/midfoot joint complex on posterior tibial nerve tension and strain (7,8). The pathologic and physiologic consequences resulting from stretching of a nerve have been investigated in great detail. Research studies have shown that excessive strain on a nerve results in reduced intraneural blood flow and abnormal action potential patterns, which could be a cause of tarsal tunnel syndrome in patients exhibiting instability of the talotarsal joint complex (7,9–12).

Hyperpronation is a characteristic of talotarsal instability (dislocation of the talus on the tarsal mechanism) and flexible flat feet. Francis et al (13) proposed that benign joint hypermobility syndrome leading to flexible flat feet places the posterior tibial nerve and its branches “on the stretch” with each weight-bearing step. They also consider this as the most common mechanism causing tarsal tunnel syndrome (13). Subsequently, Daniels et al (7) and Lau and Daniels (8) showed that tension on the posterior tibial nerve increases in feet exhibiting instability of the hindfoot and midfoot joint complex. These investigators suggest eliminating the instability of the foot (i.e., the talotarsal joint complex) as a treatment option for tarsal tunnel syndrome. This indicates the elimination of pathologic talotarsal joint motion while allowing the normal range of pronation to occur.

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Conflict of Interest: Michael E. Graham is the inventor of HyProCure®. He is the Founder and President of GraMedica, LLC., the company that manufactures and distributes HyProCure®. He is also the Founder of Graham International Implant Institute.

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HyProCure® (GraMedica, Macomb, MI) is an internal, extra-articular, and extra-osseous talotarsal stabilization (EOTTS) device designed to eliminate excessive abnormal pronation and restore the normal triplanar motion at the talotarsal joint complex (14). In a recent study, our research group showed that the pressures in the tarsal tunnel and porta pedis in hyperpronating feet were reduced significantly after intervention with this EOTTS device (14). We also speculated that along with reducing pressure in these compartments, this EOTTS device would be effective in reducing the excessive abnormal elongation of the posterior tibial nerve in hyperpronating feet. The theory is that hyperpronation leads to both an increased pressure in the tarsal tunnel and porta pedis, as well as excessive abnormal elongation of the posterior tibial nerve. An EOTTS device stabilizes the talotarsal joint complex and therefore reduces excessive pronatory forces. It would therefore decrease the pressure in the tarsal tunnel and porta pedis (which it did) and minimize the excessive abnormal elongation of the posterior tibial nerve. On the basis of that argument, the goal of this study was to quantify the elongation (and hence strain) of the posterior tibial nerve in human adult cadaver specimens exhibiting hyperpronation (partial dislocation of the talus on the tarsal mechanism (i.e., instability of the talotarsal joint complex) before and after intervention using the HyProCure® EOTTS device. To the best of our knowledge, we found no published reports that measured the elongation of the posterior tibial nerve after extra-osseous stabilization of the talotarsal joint complex. Although research has been done to quantify posterior tibial nerve tension after stabilization of a surgically created flat foot with an arthrodesis procedure (8). In the present study, we hypothesized that elongation of the posterior tibial nerve in hyperpronating feet would reduce significantly after placement of the EOTTS device.

Materials and Methods

To measure the posterior tibial nerve elongation, we used the same 9 cadaver specimens in which we had previously measured the tarsal tunnel compartment pressures. The details regarding specimen preparation and test setup have been previously published (14). In brief, each of these specimens exhibited hyperpronation (i.e., instability of the talotarsal joint complex). After the pressure measurements, the flexor retinaculum was excised to expose the posterior tibial nerve. The intact, unbranched portion of the nerve was identified for placement of the elongation measuring device (Fig. 1).

A differential variable reluctance transducer (DVRT®, Microstrain, Williston, VT) with a 9-mm linear stroke range and a resolution of 1.5 μm was used to measure elongation. The output voltage of the DVRT® was amplified using a DEMOD-DVRT signal processor (Microstrain) and recorded using the MB-SMT 4 motherboard with data acquisition software (Microstrain). Using the calibration equation provided by the manufacturer, the output voltage (in volts) was converted to displacement (in millimeters). With the foot in the neutral position, the body of the DVRT® was held in a fixed/immovable position at a level distal to the medial malleolus using a needle pin (clamped to the body). Similarly, a barbed pin clamped to the core of the DVRT® was inserted into the posterior tibial nerve proximal to its terminal branches (i.e., medial plantar, lateral plantar, and calcaneal nerves). This was the movable end of the DVRT® (i.e., as the nerve elongated, the core would move relative to the fixed DVRT® body) (Fig. 1). With the foot held in a neutral position, the DVRT® output voltage was recorded to calculate the length between the needle and the barbed pin; this was denoted as the reference or initial length (L_o). Next, the investigator pronated the foot maximally (hyperpronated) by applying a vertical and abductory force under the fourth and fifth metatarsal head, as previously described (14). The foot was held in this position, and the change in DVRT® voltage was recorded after allowing it to equilibrate for approximately 15 seconds. This voltage change was converted to the change in length (ΔL, i.e., elongation of the nerve with respect to the fixed position of the DVRT® body) to yield the final length between the 2 pins ($L = L_o + \Delta L$). The percentage of strain was calculated as ($\Delta L/L_o \times 100$). The foot was then unloaded back to its neutral position, and the procedure was repeated 3 times (i.e., $n = 3$ for each foot without intervention). Note, the strain or elongation measured is a relative value (i.e., relative with respect to the foot in a neutral position, neither pronated nor supinated).

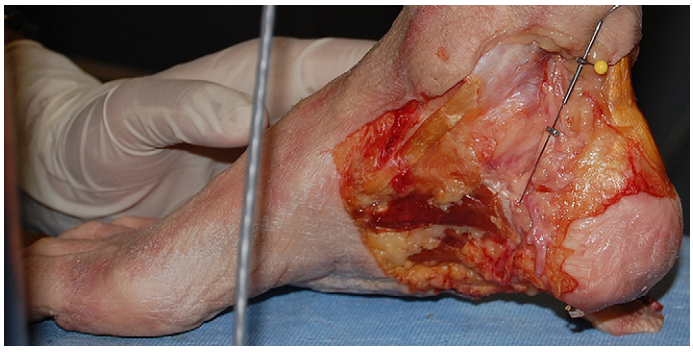

Fig. 1. Posterior tibial nerve with differential variable reluctance transducer. Image of right-footed specimen mounted on the materials testing system (not shown) with the foot in the neutral position and the differential variable reluctance transducer mounted on posterior tibial nerve as described in the "Materials and Methods" section. The output of the differential variable reluctance transducer was recorded in this position to give the initial or reference length, after which the foot was maximally pronated, and the differential variable reluctance transducer output was recorded to calculate the change in the length or elongation of the posterior tibial nerve.

After these measurements, the appropriate size EOTTS device was placed into the sinus tarsi to stabilize the talus on the tarsal mechanism (14). Next, the elongation of the posterior tibial nerve was measured after maximally pronating the foot, as described above (i.e., $n = 3$ for each foot with intervention). Throughout the experiment, the investigator was unaware of the output of the DVRT®.

The data are reported as the mean ± 1 standard deviation, range and 95% confidence interval of the mean of elongation (in millimeters) and strain (in percentages) for each experimental condition (i.e., with and without intervention) and for each of the 9 cadaveric foot specimens. The hypothesis tested was that in feet exhibiting talotarsal instability, the elongation (and hence strain) of the posterior tibial nerve would decrease after an EOTTS procedure (H_a: $\mu_{EOTTS} < \mu_{No\_Treatment}$). A one-tailed paired Student's t test for two-sample means was computed to test for significance at the 95% confidence level. The null hypothesis of no difference was rejected for $p \leq .05$.

Results

A consistent load was applied by the investigator under the fourth and fifth metatarsal head while maximally pronating the foot (14). For each of the 9 foot specimens, the relative elongation measured in the posterior tibial nerve as the foot was moved from its neutral to its maximally pronated position, with and without the EOTTS device, is listed in Table 1. The mean elongation of the posterior tibial nerve was 5.91 ± 0.91 mm (strain 27%) and 3.38 ± 1.20 mm (strain 15%) without and with the EOTTS device, respectively ($N = 27$). The difference between the 2 groups was statistically significant ($p < .001$, with no overlap between the upper and lower limits of the respective 95% confidence intervals; Fig. 2). The extra-osseous talotarsal stabilization procedure was effective in stabilizing the talotarsal joint complex, thus restoring the normal range of pronatory motion and reducing the excessive strain placed on the posterior tibial nerve.

Discussion

In the past, investigators have studied the efficacy of internal, extra-articular, and extra-osseous talotarsal stabilization devices for the treatment of hyperpronation related pathologies in the adult human population by analyzing patient satisfaction scores before and after surgical treatment (15–20). However, there is a dearth of studies focusing on the biomechanical aspects of the cause-and-effect relationship of these devices in the treatment of pathologic features associated with talotarsal instability. Biomechanical changes resulting from talotarsal instability can lead to conditions such as flatfeet, posterior tibial tendon dysfunction, plantar fasciitis, Achilles tendinitis, tarsal tunnel syndrome, and hallux abductovalgus, among others (21–24). The main goal of this study was to understand the biomechanical aspect of talotarsal instability as it relates to tarsal tunnel syndrome and to evaluate the efficacy of an EOTTS device (i.e., HyProCure®) in the treatment of the underlying faulty biomechanics. To the best of our knowledge, this is the first study to report the strain measurements in the posterior tibial nerve in hyperpronating feet in